Bengal Journal of Otolaryngology and Head Neck Surgery

Official Publication of The Association of Otolaryngologists of India, West Bengal

Volume 28 No. 2 - August, 2020

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p-ISSN: 2395-2393
e-ISSN: 2395-2407
RNI No.: 62551/95

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Contd.

Bengal Journal of Otolaryngology and Head Neck Surgery
(Incorporating and directly descended from State Journal of Otolaryngology and Otolaryngology, Calcutta)

Published by
The Association of Otolaryngologists of India, West Bengal
CMC House, 91B Chittaranjan Avenue,
Kolkata - 700073,
West Bengal,
India.

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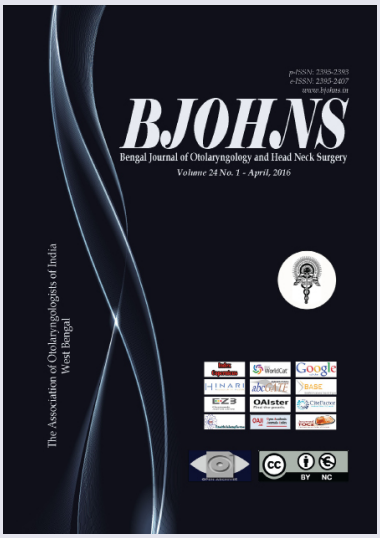

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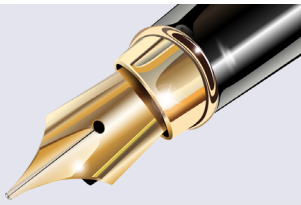

The Board of Governors in supersession of the Medical Council of India has, in its recent amendments in the 'Minimum Qualifications for Teachers in Medical Institutions Regulations, 1998', tried to rectify some of the aberrations that had crept in to the previous notification. These amendments have made the basic courses on medical education and biomedical research mandatory for the medical teachers in addition to prescribing some major changes in the criteria for publications for appointment and promotions of teachers in medical institutions in India.

The number of publications required for promotions has been reduced to 1 for Associate Professor and 3 for a Professor. This would reduce the rush for publication and may provide an impetus for quality. Research projects will also be considered for promotion to the post of Associate professor, in lieu of publication in journals, provided the teacher is either a Principal or Co-Principal (P1/Co P1) Investigator in that research project. Such research projects has to be funded by a national research body such as Indian Council for Medical Research (ICMR), Department of Science & Technology (DST) or Department of Bio Technology (DBT).

The revised basic course in medical education aims to train the teachers with a wide range of topics encompassing all the aspects of medical education. A teacher needs to participate in a course conducted under supervision of either the nodal/ regional centre that caters to the medical college, the teacher is employed with.

The basic course in biomedical research is conducted by the Ministry of Education through the web portal 'SWAYAM', which is also available on Android and iOS platforms. The candidate has to score at least 50% marks in the examination after completing 23 modules of research methodology.

A transitory period of two years has been offered from the date of notification (12 February, 2020) for smooth transition to the new system. The college can appoint or promote to the posts of Associate Professor or Professor, based on the previous criteria up to February, 2022.

The types of articles to be considered, now includes original papers and meta-analysis, systematic reviews and case series. The first three authors or the corresponding author will get recognition for the publication. The new directive has sought to check mushrooming of predatory journals by reducing the quantity of publications required and raising the bar for the quality of journals to choose from. Publications in the journals indexed with Medline, Pubmed Central, Citation index (not Indian Citation Index), Sciences Citation index, Embase, Scopus, Directory of Open access journals (DoAJ) only will be considered to be appropriate and I take the opportunity to advise teachers to ignore lure of the false claims to avoid the trap of the predatory journals and engage themselves in meaningful and quality research.

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Paediatric Type I Cartilage Tympanoplasty with or without Concomitant Adeno-tonsillectomy – An Institutional Experience

https://doi.org/10.47210/bjohns.2020.v28i2.322

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ABSTRACT

Introduction

In the management of COM in paediatric age group, eradication of the septic foci in the tonsil and the adenoids while addressing the dysfunction of Eustachian tube play a key role This study aims to assess the success rate of type I tympanoplasty with cartilage graft in mucosal type of chronic otitis media in paediatric age group and finally, whether concomitant adeno-tonsillectomy plays any role in outcome of surgery or curing the disease process.

Materials and Methods

A prospective clinical study was done over a period of one and half years amongst 59 patients, aged between 5 and 12 years, presenting with chronic otitis media with dry central perforation of ear-drum and pure conductive hearing loss. Patients were divided into two groups. Group 1 (n1= 22) underwent type I cartilage tympanoplasty with adenoidectomy/ tonsillectomy/ both in the same sitting and Group 2 (n2= 37) underwent type I cartilage tympanoplasty without any adeno-tonsillectomy.

Results

Graft uptake was 86.4% in Group 1 at 6 months post-operative follow up while that in Group 2 was found to be 86.5%. The mean hearing gain in two groups were found to be 15.91±6.54 dB and 17.02±8.96 dB respectively. No significant difference was found between graft uptakes and hearing gains in two groups at the end of the study.

Conclusion

Type I cartilage shield tympanoplasty, may be considered as an effective management strategy of paediatric chronic otitis media, mucosal disease. Type I tympanoplasty with concomitant adeno-tosillectomy did not prove to be better than type I tympanoplasty alone in terms of anatomic closure of tympanic perforation and hearing gain.

Keywords

Child; Adenoidectomy, Concomitant; Tympanoplasty

Tympanoplasty in children is a long-debated topic amongst Otolaryngologists and a controversial theme, though it is going through different modifications and alterations from time to time. Children usually suffer from recurrent upper respiratory tract infections. The disease usually begins in childhood as a spontaneous tympanic perforation due to acute otitis media, which occurs during the first 6 years of a child's life, with peak around 2 years.[1] The highest rate of perforation is seen in the 2 to 4 years old age group, at which stage, the rate of perforation is roughly 3 times the rate seen in adulthood.

There is always a possibility of failure of the conventional type I tympanoplasty operation in children

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due to recurrent attacks of otitis media. Cartilage has become a good alternative to temporalis fascia, as grafting material, in cases of type I tympanoplasty in both uncomplicated and revision cases, in adults; but more results are needed to establish its efficacy in paediatric population. In childhood, the spectrum of otitis media (AOM, OME and COM) is most commonly associated with Eustachian tube dysfunction secondary to various factors.[3,4,5] Hypertrophied adenoid and chronically inflamed tonsils act as a septic focus in upper respiratory tract in children; which not only cause mechanical obstruction to Eustachian tube, but also act as reservoir of micro-organisms to play a crucial role in pathogenesis of chronic otitis media in children. So, in the management of COM in paediatric age group, eradication of these septic foci and addressing the dysfunction of Eustachian tube play a key role.

In this study, an attempt is made to find out the effectiveness of cartilage in type I tympanoplasty, in terms of graft take rate and hearing results in inactive type of chronic otitis media in paediatric age group with or without concomittant adeno-tonsillectomy. At the same time, our aim is to assess whether concomitant adeno-tonsillectomy proves beneficial for management or alters the final outcome of paediatric type I tympanoplasty.

Materials and Methods

This study was carried out at Department of Otorhinolarryngology of a tertiary care hospital after getting proper approval from the ethical committee. 59 patients were selected for this study, all between 5 and 12 years of age (both the limits included), attended ENT OPD for chronic otitis media, mucosal disease, presenting with dry central perforation of ear drum and pure conductive deafness, over a period of one year. Patients with squamosal disease/cholesteatoma, patients with per-operative findings of ossicular fixity or discontinuity, h/o previous tympanoplasty or mastoid surgery, with sensori-neural or mixed hearing loss, with congenital anomalies of ear-nose-throat and medically unfit patients were excluded from this study.

These patients were placed into two groups, viz. Group 1: patients undergoing type I cartilage tympanoplasty with concomitant adeno-tonsillectomy (n1=22) and Group 2: patients undergoing type I cartilage tympanoplasty only (n2=37). Intactness of the graft, dryness of the ear, pre- and post-operative air-bone gap (in pure tone audiometry) were the parameters studied. Patients with Grade 3 & 4 tonsillar or adenoid enlargement underwent the additive operative procedures as needed along with tympanoplasty.

All the patients were subjected to thorough pre-operative assessment including detailed history and clinical examination (to rule out possible infective foci, if any), along with otoscopy and tuning fork tests. Examination under microscope, pure tone audiometry and Eustachian tube function tests including impedance audiometry were done in every case to select the children with dry central perforation and pure conductive hearing loss.

After proper counseling of patients and parents, written consent was signed regarding the procedures and after getting the fitness regarding general anaesthesia from the anaesthesiologists, patients were admitted for the surgery. Patients posted for surgery received a dose of intravenous antibiotic (preferably Amoxycillin+Clavulanic acid, dose adjusted as per body weight) before surgery.

All the surgical procedures were done under general anaesthesia with endotracheal intubation done by anaesthesiologists. Patients in Group 1 underwent adenoidectomy/ tonsillectomy/ both (by conventional technique) followed by type I tympanoplasty in the same sitting and those in Group 2 underwent type I tympanoplasty alone. No post-operative complication seen in any of adeno-tonsillectomy cases.

Tympanoplasty was done by post aural route (through Wilde's incision). Ossicular integrity and mobility, condition of middle ear mucosa and the opening of Eustachian tube were checked in all cases. Autologus tragal cartilage (without perichondrium on both sides) was harvested by giving a separate incision over the medial surface of the tragus and a piece of cartilage taken out leaving a rim of tragal cartilage on the lateral most part to maintain the contour of tragus. The harvested cartilage was sliced to 0.4 mm thickness using

cartilage slicer. The sliced cartilage was then cut in the shape of tympanic membrane and a 'V' shaped notch was made to incorporate the handle and lateral process of malleus. Graft was placed as underlay; shield and sometimes additional small pieces of cartilage were needed as architrave to support the graft or for proper fitting in the bony annulus. Middle ear was filled with antibiotic soaked gelfoam pieces before placing the graft to support the graft from within and after repositioning of the tympano-meatal flap, external canal also filled with antibiotic soaked pieces of gelfoam. Finally skin and soft tissues were closed in layers. Thereafter all the patients were followed up at 1, 3 and 6 months post-operative to note the anatomic and functional outcome of the surgery and complication, if any.

Pure tone audiometry was done in every case at 3 and 6 months post-operative follow up and hearing gain (Pre-op A-B gap minus Post-op A-B gap) was assessed by comparing it with pre-operative Air-Bone gap.

Results

The patients were selected between the age of 5 and 12 years, both the limits included and statistical analysis (Kolmogorov-Smirnov test with Lillifors significance correction) showed that the observations were not normally distributed. Pearson Chi-square test revealed no statistically significant difference in proportion of males and females in two groups. (Table I)

The graft uptake was compared between the two groups. (Table II) Statistical analysis (Fisher's Exact

Table I: Distribution of patients by their age and sex

GROUPS	AVERAGE AGE (IN YEARS)		SEX		TOTAL
	MEAN ± SD	STATISTICS, DF, P	MALE	FEMALE	
1. (n1=22) AT + CT	9.09± 2.11	0.16,22,0.115	14 (63.6%)	8 (36.4%)	22 (100%)
2. (n2=37) CT	9.30± 2.13	0.16,37,0.011	20 (54.1%)	17 (45.9%)	37 (100%)

[AT+CT=cartilage tympanoplasty with adeno-tonsillectomy, CT=cartilage tympanoplasty, df=degree of freedom, p=significance]

Table II: Comparison of graft uptake rate in cartilage tympanoplasty with or without adeno-tonsillectomy

GROUPS	FOLLOW UPS						TOTAL
	AT 1 MONTH		AT 3 MONTHS		AT 6 MONTHS		
	F	NF	F	NF	F	NF	
1. AT+CT	19 86.4%	3 13.6%	19 86.4%	3 13.6%	19 86.4%	3 13.6%	22 (100%)
2. CT	35 94.6%	2 5.4%	31 83.8%	6 16.2%	32 86.5%	5 13.5%	37 (100%)
TOTAL	54 91.5%	5 8.5%	50 84.7%	9 15.3%	51 86.4%	8 13.6%	59 (100%)

[AT+CT=cartilage tympanoplasty with adeno-tonsillectomy, CT=cartilage tympanoplasty; F=Favorable outcomes (Dry ear, Graft taken up/ Well epithelialized graft) NF=Not favorable/ Unfavorable outcomes (Discharging ear, Residual perforation/ Antero-inferior dehiscence, Graft failure)]

Table III: Hearing gain or improvements (in dB) obtained at 6 months follow up

GROUPS	MEAN±SD	ST. ERROR OF MEAN	MINIMUM, MAXIMUM, RANGE	TEST OF NORMALITY (SHAPIRO-WILK) STATISTIC/ DF/SIG
1. AT+CT	15.91±6.54	1.39	3 /35 /32	0.936/22/0.163
2. CT	17.02±8.96	1.47	3 /46 /43	0.898/37/0.003

[AT+CT= cartilage tympanoplasty with adeno-tonsillectomy, CT=cartilage tympanoplasty; all values in dB]

Test): At 1 month p=0.351 (>0.05), At 3 months p=1.000 (>0.05), At 6 months p=1.000 (>0.05). So, there was no significant differences between graft uptakes in two groups- cartilage tympanoplasty with or without adeno-tonsillectomy at 1, 3, 6 months follow ups.

We recorded the pre-operative A-B gap and post-operative A-B gap at 3 and 6 months follow ups in each of 59 patients irrespective of intervention underwent and calculated the hearing gain (Pre-op A-B gap minus Post-op A-B gap) in each case. Hearing gain at 3 months and 6 months showed negligible differences and we took the results at 6 months for comparison. (Table III).

Analysis showed that hearing gain data in Group 1 were normally distributed (p=0.163>0.05) and those in other group were not normally distributed (p=0.003<0.05). After log transformation of the data in two groups, the test of normality was again performed and the results are shown in Table IV.

As the hearing gain in these two groups are not normally distributed, so non-parametric Mann-Whitney Test was applied to get statistical analysis and the results are shown in Table V.

Table VI shows that there was no significant difference in Hearing gain among these two groups, i.e. Cartilage tympanoplasty with and without Adeno-tonsillectomy (p=0.919>0.05).

Discussion

Otits media is predominantly a disease of infancy and early childhood with peak age specific attack rate occurring between 6 and 18 months of age.[6] A functionally and structurally immature Eustachian tube system[3,4] and an immature immune system[7] are probably the most important factors related to the increased incidence of otitis media in infants and young children. Repeated episodes of acute upper respiratory tract infections (mostly viral, may be bacterial) lead to acute otitis media and otitis media with effusion; which with or without pre-existing pathophysiology of Eustachian tube,[8] result in chronic otitis media in children. Continued E. tube obstruction retards spontaneous closure of the

Table IV: Corrected analysis of data on hearing gain

GROUPS (LOG)	KOLMOGOROV-SMIRNOVA			SHAPIRO-WILK		
	STATISTIC	DF	SIG.	STAISTIC	DF	SIG.
1. AT+CT	0.15	22	0.18	0.88	22	0.01
2. CT	0.15	37	0.02	0.95	37	0.17

Lilliefors significance Correction [AT+CT=cartilage tympanoplasty with adeno-tonsillectomy, CT=cartilage tympanoplasty]

Table V: Results of Mann-Whitney test

	GROUPS	N	MEAN RANK	SUM OF RANKS
HEARING GAIN	1. AT+CT	22	29.7	653.5
	2. CT	37	30.18	1116.5
	TOTAL	59		

[AT+CT=cartilage tympanoplasty with adeno-tonsillectomy, CT=cartilage tympanoplasty]

perforation of eardrum. Chronic otitis media in children is multifactorial[2] which includes genetic, infection, immunologic, allergic, environmental and social factors.

Waldeyer's ring plays an important role in the pathophysiology of upper respiratory tract infection and allergy[7] in paediatric population. Adenoid hypertrophy can cause recurrent acute otitis media (RAOM), otitis media with effusion (OME), Obstructive sleep apnoea syndrome (OSAS) in children.[9] The size of the adenoid causing mechanical obstruction to E. tube, is not the main determinant factor in OME pathogenesis but the degree of bacterial colonization[10] is much more important in COM pathogenesis and a deciding factor whether adenoidectomy should be done in cases of COM or not. Saafan et al10 from Egypt in 2013 and Szalmas et al[9] from Hungary in 2013, have studied extent of surface biofilm of adenoid and evaluated its role in the pathogenesis of COM in children. Large tonsils, per se, have not been found to cause an ear infection to keep coming back; but chronic tonsillitis always acts as a reservoir of infection.

Though tymapnoplasty is considered as gold standard in the management of chronic otits media; when a child presents with a persistent perforation of ear drum, question arises whether early surgery to be attempted to correct the anatomical defect and thereby improving hearing; or the elective surgery is better to be deferred until the peak incidence of AOM has passed.[11,12] Jeffrey et al in 1999 have shown that otologic surgery in children is less successful and argued for tympanoplasty in older age groups.[13] Boronat Echeverria et al in 2012 favored paediatric tympanoplasty on the merit that children present greater risk of retraction, SOM, re-perforation with episodes of AOM and at the same time they coined that, it is technically difficult in doing surgery because of narrowness of external auditory canal and smaller size of ear drum.[14] They have presented arguments in favor of surgery at an earlier age (<5 yrs); though opinion differs in this regard in various studies like not before 7 years (MacDonald et al[15]), 8 years (Koch et al[16]), 10 years (Shih et al[17]) and 12 years (Raine and Singh[18]). On the other hand, long standing or permanent perforation of tympanic membrane can result in long-term irreversible damage to the inner ear in children and early intervention is always needed.[19] So, tympanoplasty in children not only cures the disease but also lessens the hearing handicap and helps in better school performance.

Though temporalis fascia is a time tested grafting material in tympanoplasty, various authors have used cartilage myringoplasty in different situations like E. tube dysfunction, retraction pockets, subtotal to total perforations, revision tympanoplasty, ear discharging at the time of surgery, myringoplasty in children etc.[20,21,22] Because of its thickness, rigidity and mechanical stability, cartilage can resist resorption and retraction; it is more resistant to infection and able to withstand adverse states of the graft bed as its vascular demands are less compared to other materials. Since tragal cartilage is yellow fibroelastic cartilage, formed mainly by type II collagen,[23] which is also the main type in lamina propria of tympanic membrane and it is easily available at operative field, a thin tragal cartilage graft would be a better option as grafting material. The rigidity of the cartilage that prevents re-perforation however has been questioned to interfere with sound conduction.[24] The cartilage slices <0.5 mm thick are similar to the tympanic

Table VI: Statistical analysis of the data on the hearing gain

	HEARING GAIN
Mann-Whitney U	**400.5**
Wilcoxon W	**653.5**
Z	**-0.102**
Asymp. Sig (2-tailed)	**0.919**

membrane in their acoustic properties.[25,26]

In general, studies support no single conclusion about the usefulness of previous adenoidectomy/tonsillectomy for major ear surgery. While Gianoli et al[27] and Charlett et al[28] favored adenoidectomy and showed that success rate of tympanoplasty depends on it; Ophir et al[29] concluded that adenoidectomy is not related to the success of paediatric myringoplasty. Vartiainen et al found that all failure cases of paediatric tympanoplasties occurred in those who underwent previous adeno-tonsillectomy.[30]

Many authors consider that a 4 to 6 weeks interval is needed between adeno-tonsillectomy and tympanoplasty to resolve post-operative mucosal oedema that may block E. tube function.[31] Although large studies regarding paediatric cartilage tympanoplasty (seven such in PubMed listed below) and its relation with previous adeno-tonsillectomy (two such in PubMed listed below) are available in contemporary literature, paediatric tympanoplasty with or without concomitant adeno-tonsillectomy study has not been done in recent times (no study found in pub med). (Table VII)

In the present study, type I tympanoplasty and adeno-tonsillectomy were done in the same sitting in one of the study groups in view of the facts that- i) it prevents the child from repeated exposure to general anaesthesia and related hazards, ii) parents counseling is also easier, iii) more easy to follow up in our set up, iv) if adenoidectomy or tonsillectomy or both be done in expert hands and in precise way with modern instruments, it does not cause any E.tube injury or local oedema to hamper E.tube function so as to be the reason behind tympanoplasty failure.

So, the anatomic and functional results of this study are comparable with the available series in the literature

Conclusion

From this study, we can see that tympanoplasty is still the gold-standard in the management of inactive mucosal type of chronic otitis media in paediatric age group and can be well advocated in the patients of age 5 years and above. Type I cartilage shield tympanoplasty, using sliced (<0.5 mm thickness) tragal cartilage in post-aural route is a good solution to paediatric COM, inactive type, where it can effectively tackle the issues like E.tube dysfunction, recurrent URTI, retraction of ear drum, re-perforation and different anatomic and physiologic variations of middle ear cleft in paediatric age group. Graft uptake and hearing results of Type I cartilage shield tympanoplasty were satisfactory (>86% and >15 dB hearing gain respectively) in this study and hence cartilage may be used more liberally in paediatric type I tympanoplasty as grafting material. And Removal of septic focus (adenoidectomy/ tonsillectomy/ both) plays a vital role in the management of mucosal disease, however, type I tympanoplasty with adeno-tonsillectomy in the same sitting did not prove to be better or beneficial than type I tympanoplasty alone in terms of anatomic closure and hearing gain.

Table VII: Comparison of Graft Uptake and Hearing Results among Different Studies

STUDY/ AUTHOR	ANATOMIC SUCCESS RATE	HEARING RESULTS
Paediatric Cartilage Tympanoplasty		
Diaa M El-Hennawi, 2001, Egypt.[32]	86.60%	Excellent post-operative hearing gain, though delayed up to 6 months
Couloigner Vincent et al, 2005, France.[33]	71%, may be increased up to 81%	Hearing levels were not different from those obtained with underlay temporalis fascia tympanoplasty
Gaslin M et al, 2007, USA.[34]	85.70%	93.8% patients achieved post-op A-B gap< 20 dB; Mean improvement in A-B gap 10.7 dB
Albirmawy OA, 2010, Egypt.[35]	95%	Trends towards better post-operative results in cartilage 'ring' graft compared to temporalis fascia
Nevoux J et al, 2011, France.[36]	87.30%	Closure of the average A-B gap within 20 dB achieved in 62.2% at 1 year; Mean pre-op ABG 25±11.8 dB, post-op ABG 18.9±10.3 dB
Adva B Friedman MD et al, 2012, USA.[37]	95%	Improvement in PTA post-operatively – 10.23(<7yr), 12.5(7-10yr), 3.95(10-13yr) dB
Yilmaz MS et al, 2013, Turkey.[38]	41 out of 45 i.e. 91.1%	Mean pre-op PTA 30.6±7.7 dB and post-op PTA 17.8±7.8 dB
Paediatric Cartilage Tympanoplasty + Adenoidectomy/ Tonsillectomy		
Gianoli G et al, 199527	75%	
Charlett SD et al, 200928	79.10%	
Present study		
Cartilage tympanoplasty + Adenotonsillectomy	86.40%	Mean hearing gain/improvement 15.91±6.54 dB at 6 months
Cartilage tympanoplasty	86.50%	Mean hearing gain/improvement 17.02±8.96 dB at 6 months

References

1. Charles D. Bluestone; Epidemiology; Eustachian Tube: structure, function, role in otitis media. Vol 2, BC Decker 2005; 11-12
2. Hamilton J. Chronic otitis media in childhood; Scott Brown's Otorhinolaryngology, Head and Neck Surgery, 7th ed, 2008; volume 1; Hodder Arnold; 933
3. Bluestone CD. Anatomy; Eustachian Tube: structure, function, role in otitis media; Vol 2, Hamilton BC Decker, 2005; 25-32
4. Bluestone CD. Physiology; Eustachian Tube: structure, function, role in otitis media; Vol.2, Hamilton BC Decker, 2005; 51-66
5. Bluestone CD, Eustachian Tube: Structure, Function, Role in Otitis Media; Vol. 2; Ch. 5, Ed. Bluestone MB; 2005, BC Decker 2005; 67-91
6. Darrow D H, Derkay CS. Otitis Media; Complications in Pediatric Otolaryngology; Ed. Josephson GD, Wohl DL. Broken Sound Parkway NW, Taylor & Francis 2005; 445
7. Bluestone CD. Eustachian tube function: physiology, pathophysiology and role of allergy in pathogenesis of otitis media. J Allergy Clin Immunol. 1983; 72(3): 242-51
8. Bluestone CD. Conquering Otitis Media, An illustrated guide to understanding, treating and preventing ear infections; Ed. Bluestone MB; Ch.5, BC Decker 1999; 16-24
9. Szalmás A, Papp Z, Csomor P, et al. Microbiological profile of adenoid hypertrophy correlates to clinical diagnosis in children. Biomed Res Int. 2013;2013:629607. doi:10.1155/2013/629607
10. Saafan ME, Ibrahim WS, Tomoum MO. Role of adenoid biofilm in chronic otitis media with effusion in children. Eur Arch Otorhinolaryngol. 2013 Sep; 270(9): 2417- 25
11. Dornhoffer JL. Cartilage tympanoplasty. Otolaryngol Clin N Am. 2006; 39: 1161-76
12. Buchwach KA, Birck HG. Serous otitis media and type 1 tympanoplasties in children. A retrospective study. Ann Otol Rhinol Laryngol Suppl. 1980; 89(3 Pt 2):324-5
13. Vrabec JT, Deskin RW, Grady JJ; Metaanalysis of Pediatric Tympanoplasty; Arch Otolaryngol Head Neck Surg.1999; 125(5):530-4
14. Boronat-Echeverría NE, Reyes-García E, Sevilla-Delgado Y et al. Prognostic factors of successful tympanoplasty in pediatric patients: a cohort study. BMC Pediatrics 2012, 12:67. https://doi.org/10.1186/1471-2431-12-67
15. MacDonald RR 3rd, Lusk RP, Muntz HR. Fasciaform myringoplasty in children. Arch Otolaryngol Head Neck Surg. 1994; 120(2):138-43. doi:10.1001/archotol.1994.01880260010003
16. Koch WM, Friedman EM, McGill TJ, Healy GB. Tympanoplasty in children. The Boston Children's Hospital experience. Arch Otolaryngol Head Neck Surg. 1990;116(1):35-40. doi:10.1001/archotol.1990.01870010039013
17. Shih L, de Tar T, Crabtree JA. Myringoplasty in children. Otolaryngol Head Neck Surg. 1991; 105: 74-7
18. Raine CH, Singh SD. Tympanoplasty in children: A review of 114 cases. J Laryngol Otol. 1983; 97(3):217-21
19. Knapik M, Saliba I. Pediatric Myringoplasty: a study of factors affecting outcome. Int J Pediatr Otorhinolaryngol. 2011; 75(6): 818-23
20. Milewski C. Composite graft tympanoplasty in the treatment of ears with advanced middle ear pathology. Laryngoscope 1993; 103:1352-1356
21. Amedee RG, Mann WJ, Riechelmann H. Cartilage palisade tympanoplasty. Am J Otol. 1989; 10(6):447-50
22. Duckert LG, Muller J, Makielski KH, et al. Composite autograft "shield" reconstruction of remnant tympanic membranes. Am J Otol. 1995; 16(1):21-6
23. Ross MH, Romrell LJ. Connective tissue. In: Histology: a text and atlas, vol. 89, 2nd ed. Baltimore, Williams and Wilkins 1989.
24. Singh I. editor. Textbook of human histology. New Delhi Jaypee Brothers Medical Publishers 2004; 89-93
25. Zahnert T, Huttenbrink KB, Murbe D, Bornitz M. Experimental investigations of the use of cartilage in tympanic membrane reconstruction. Am J Otol. 2000; 21:322-8 26. Mürbe D, Zahnert T, Bornitz M, Hüttenbrink KB. Acoustic properties of different cartilage reconstruction techniques of the tympanic membrane. Laryngoscope 2002; 112(10):1769-76. doi:10.1097/00005537-200210000-00012
26. Gianoli GJ, Worley NK, Guarisco JL; Pediatric Tympanoplasty: the role of adenoidectomy; Otolaryngol Head neck Surg. 1995;113(4): 380-6
27. Charlett SD, Knight LC. Pediatric myringoplasty: does previous adenoidectomy improve the likelihood of perforation closure? Otol Neurotol. 2009; 30(7):939-42
28. Ophir D, Porat M, Marshak G. Myringoplasty in the pediatric population; Arch Otolaryngol Head Neck Surg. 1987; 113(12): 1288-90
29. Vartiainen E, Vartiainen J. Tympanoplasty in young patients: the role of adenoidectomy; Otolaryngol Head neck Surg. 1997; 117(6): 583-5
30. Wohl DL, Belenkay WM. Otologic Surgery; Complications in Pediatric Otolaryngology. Eds. Josephson GD, Wohl DL. Broken Sound Parkway NW, Taylor & Francis 2005; 480
31. El-Hennawi DM. Cartilage perichondrium composite graft (CPCG) in pediatric tympanoplasty. Int J Ped Otorhinolaryngol.

2001; 59(1):1-5

32. Couloigner V, Baculard F, El Bakkouri W, Viala P, et al. Inlay Butterfly Cartilage Tympanoplasty in Children. Pediatric Otology and Neurology 2005; 26(2):247-5
33. Gaslin M, O'Reilly RC, Morlet T, McCormick M. Pediatric cartilage
34. interleave tympanoplasty. Otolaryngol Head Neck Surg. 2007; 137(2):284-8
35. Albirmawy OA. Comparison between cartilage-perichondrium composite "ring" graft and temporalis fascia in type one tympanoplasty in children. J Laryngol Otol. 2010; 124(9); 967-74
36. Nevoux J, Roger G, Chauvin P, Denoyelle F, Garabedian EN. Cartilage shield tympanoplasty in children: review of 268 consecutive cases; Arch Otolaryngol Head Neck Surg. 2011; 137 (1): 24-9
37. Friedman AB, Dornhoffer JL. Outcomes of cartilage tympanoplasty in pediatric patients. Otolaryngol Head Neck Surg. 2012; 147(2 suppl):102
38. Yilmaz MS, Guven M, Kayabasoglu G, Varli AF. Comparison of the anatomic and hearing outcomes of cartilage type 1 tympanoplasty in pediatric and adult patients. Eur Arch Otorhinolaryngol. 2015; 272(3):557-62. doi:10.1007/s00405-013-2869-2.

Main Article

Hospital Based Study of Traumatic Neck Injuries: A Decision Making Approach

https://doi.org/10.47210/bjohns.2020.v28i2.320

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ABSTRACT

Introduction

Traumatic neck injuries can cause serious morbidity and mortality. They have very serious outcomes and require emergent surgical interventions. This study illustrated 31 traumatic neck injury cases and their management over a period of 3 years.

Materials and Methods

Thirty nine (39) patients were studied retrospectively and analyzed. Patients admitted through emergency room with a history suggestive of vascular trauma in the neck, between February 2014 and March 2017 were included. Thirty one (31) patients which were treated surgically were selected for analysis. All the patients were taken to the operating room immediately and made hemodynamically stable, other parameters were quickly corrected and urgent measurements were undertaken to maintain vascular integrity.

Results

Out of 31 patients suspected to have vascular neck injuries, only 29 (74%) were established to have vascular neck injuries. Forty seven percent 47% were stabilized hemodynamically and underwent operations under elective conditions. 16 (55%) patients were found to be hemodynamically unstable with fair signs of vascular injuries and were taken to the operating room immediately. 41% had arterial injuries and 59 % had venous injury. No patients had any post-operative vascular complication. One patient expired on the 11th postoperative day as a result of multiple organ injury and brain injuries.

Conclusion

Patients with severe neck injuries leading to acute bleeding and hematoma must be taken to the surgery for best outcome. Prompt decision making plays very important role to minimize disastrous consequences leading to further vascular and neurological derangements.

Keywords

Neck Injuries; Vascular System Injuries; Decision Making

Neck injuries often involve airway compromise, potential spinal cord injuries and large amounts of blood loss from external wounds.[1,2] All the patients with neck injuries should be taken seriously, and rapid transport to the closest appropriate facility should be ensured. The care provided in the field is extremely important for the patient and it increases survival.[1,2]

Blunt trauma has varied severity. It is always difficult to identify blunt trauma, but the injuries to the neck and spine can have significant consequences. These injuries require multimodal assessment. Spinal cord injury entails high level of concern as do fractures of the larynx and trachea, along with blunt injury to carotid arteries.[1]

Other injuries are penetrating neck injuries (PNIs) caused by gunshot wounds, stab wounds, or penetrating debris such as shrapnel. All PNIs are treated expectantly and mortality rates were high before World War II.[3] Aiming to reduce the high death rates associated with neck injuries, a mandatory surgical approach was adopted, even for stable patients to reduce mortality.[4]

Surgical exploration remained widely accepted as necessary well into the 1990s, when it became obvious that while the mortality rate was low, the rate of negative surgical explorations was unacceptably high (58%) and

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the concept of selective surgical management emerged with the advent of a spectrum of diagnostic modalities.[5]

PNIs, defined by platysma violation, comprise a significant number of traumatic injuries in adults and their treatment is trick.[3] Approximately 15 to 20 percent of PNI cases required treatment. The management of a hemodynamically stable patient is still debatable[3,6,7]

Penetrating neck trauma may present with unpredictable signs and symptoms in the emergency. Around 5% to 10% of all neck injuries comprises of penetrating neck trauma and 30% of patients present with multiple injuries. Thorough knowledge of the anatomy of the neck, physical assessment, and current recommendations for diagnostic and therapeutic interventions are necessary for appropriate management. Express decision making is often required to prevent catastrophic airway, vascular, or neurologic sequelae.

Overall mortality attributed to neck trauma is 11%. Injuries to the carotid or subclavian vessels bear the lion's share of such mortality and are generally associated with poorer outcome.

Injury to certain anatomic structures (e.g., the carotid or subclavian vessels) may be fatal in two thirds of cases. Surgeons show lack of consensus regarding which injuries warrant surgical intervention, especially in cases where conservative approach was taken primarily.8

Materials and Methods

Thirty one (31) patients who were admitted through casualty room with a history of neck injury between Feb 2015 and March 2018 were analyzed by a team of otolaryngology surgeons. Twenty nine (29) of them were found to have vascular lesion. The necks of the other 2 patients were also explored, but no major vascular lesion was found.

Patients who presented with wounds in the neck and were admitted to our side but investigated and kept under observation for 24 hours without surgical interference were not included in our study.

All patients who were unstable were resuscitated according to Advanced Trauma Life Support protocol. Patients with heavy uncontrolled bleeding, expanding or pulsatile hematoma or no response to resuscitation were given prioritized for immediate surgery.

In patients with evidence of a vascular injury, the Glasgow Coma Score (GCS), systolic blood pressure (SBP) and positive neurological signs were recorded prior to surgical intervention. Neurological assessment was done pre and post operatively. Computerized axial tomography (CAT) scan of the brain was done in all the patients who were in coma for more than four hours duration with focal neurological signs.

Patients with injury to internal carotid artery and common carotid artery were taken to operation theatre immediately for repair even in coma or neurological insufficiency.

Seven patients whose conditions were stable underwent carotid artery Doppler ultrasonography.

Some patients posed difficult intubation through oro-tracheal route due to a big hematoma in the neck or difficulty in opening the mouth; trans-nasal intubation was performed in such patients using fibre-optic bronchoscope.

Arterial lesions were either repaired primarily or with venous graft interposition or ligated. Venous injuries were mostly ligated as all venous injuries were unilateral. Two patients with venous injuries were repaired primarily.

Patients were followed up at the outpatient clinic and duplex scan was done for those with arterial repairs.

Results

Twenty-nine patients, 21 males (72%) and 8 females (28%), found to have a vascular injury in the neck, were operated upon. Their ages ranged from 12-61 years with a mean of 41 years.

Sixteen patients (55%), who were unstable at presentation to the ER with definite signs of vascular injury, were taken immediately to OR. Rest of the patients (45%) were stabilized and evaluated by further investigation especially, duplex scan and or CTA.

Arterial injuries were found in 12 patients (41%) and venous injuries in 17 patients (59%).

Exploration was done through the anterior border of the sternocleidomastoid muscle. One patient also

needed a left anterolateral thoracotomy for subclavian artery injury.

The common carotid artery was injured in 5 (17%) patients. The internal carotid artery with the common carotid artery was injured in 3 (10%) patients. The internal carotid artery was injured in 1 (3%) patient. The external carotid artery was injured in 1 patient. The common, internal and external carotid arteries were found injured in 1 (4%) patient. The subclavian was injured in 1 (4%). Vertebral arteries were injured in 2 (7%) patients.

Venous injury was mostly in the internal jugular vein (in 10 patients, i.e. 40%). Four patients presented with injury to the external jugular vein.

Most of the vascular neck injuries were on the right side (19 patients, i.e. 66%). The cause of the vascular injury was penetrating injury by gunshot, knife, sharp rod or iatrogenic during insertion of a permcath for hemodialysis. Other injuries (6 patients, i.e. 21%) were associated with road traffic accidents, fall from a height or falling of heavy objects on the neck and shoulder.

Patients' morbidity was mainly due to bullet injury to carotid artery. There was no postoperative vascular bleeding, hematoma, thrombosis or infection in any patient treated for their vascular injuries.

Mortality was seen in one patient. He was a patient with multiple trauma, fall from a height with head fracture, left transverse cervical spine fracture, left vertebral artery injury, fracture ribs with lung contusion. He succumbed on the 12th post-operative day. The cause of death was suspected to be ARDS.

Routine follow up of the patients was done in the outpatient clinics. Duplex scan was done after 3 months for all the patients on whom arterial repair and revascularization. It showed patency of the arterial flow.

Discussion

Expeditious decision making is often required to prevent catastrophic airway, vascular, or neurologic sequelae.[1,2,8]

A large number of studies since the 1970's have attempted to determine the reliability of the physical exam in screening patients who should undergo further evaluation and treatment for penetrating neck injuries. Mandatory neck exploration for penetrating neck wounds often led to a negative exploration rate (in excess of 50%). A review of the literature published in 1994 by McConnell et al. compares several studies that use physical examination to screen patients. The review notes that in those studies in which asymptomatic patients were placed in observation groups (serial q6 hour exams by a physician), the rate of negative neck exploration was low, while the rate of false negatives was negligible.[4]

Serious morbidity and mortality caused by carotid artery injuries must be intervened promptly and monitored closely after surgery. Arterial Doppler ultrasonography provides useful information in these injuries, conventional method being angiography of aorta visualizing its branches.[9] However, patients with unstable condition, hematoma and uncontrolled bleeding were posted for immediate surgery.[10] Usually young patients with neck injury were taken up for surgery, and arterial integrity was promptly restored leading to satisfactory outcome. Time elapsed between injury and surgery is a very important factor and it is evident that lesser time elapsed ensures better outcome.

Injury due to sharp objects show with cuts. In blunt injury, the surface may not reveal anything, making examination and management difficult. Neck and spinal injuries should be considered in such types of blunt trauma.[11]

It will be useful in case of cervical injuries to examine and investigate the neck in three parts as described by Manson et al. in "discrimination zone criteria" of cervical injury.[12]

A routine surgical exploration should be performed in injuries around neck. Some surgeons and medical centres prefer to go for angiography with elective surgery.

In case of active bleeding, surgical exploration should be done electively.

In patients of penetrating trauma especially in zone I or II who have sufficient clinical evidence of a vascular injury and are hemodynamically unstable, an immediate oblique incision along with anterior border of sternocleidomastoid muscle should be considered for

exploration.[13]

In patients with a gunshot wound in zone II with a hematoma , an unidentified level of a carotid-jugular arteriovenous damage, lost carotid pulse without a neurologic deficit, or proximity to the vertebral artery, diagnostic tests are indicated.[14] Current diagnostic options include duplex ultrasound, computed tomography (CT) angiography, digital subtraction arteriography, and magnetic resonance angiography. In most of the hospitals, arteriography remains the gold standard to explore carotid artery injuries.[14] Duplex scanning has limitations for cervical wounds with large, soft tissue insults. CT angiography could be a better choice as a noninvasive investigation vis-a-vis conventional arteriography. A potential disadvantage is the degradation of image quality due to artifacts produced by metallic fragments embedded in the soft tissue.[15]

For penetrating injuries, the absence of hard signs rules out surgically significant vascular injury.[16]

Studies that examine penetrating trauma to all zones of the neck show consistently that zone 3 injuries are the least likely to occur and the most difficult to treat surgically.[17-19]

The availability and accuracy of angiography in most institutions since the 1970s have brought about the concept of "selective management," which has become standard in some trauma centers for hemodynamically stable patients.[15,20]

This approach results in many negative angiograms, as well as positive studies that are not surgically significant.[21,22]

Liekweg and Greenfield recommended a reconstruction of the injured vessel in a review of 170 patients with isolated injury to the internal carotid artery or common carotid artery with normal neurologic examinations before surgery. After revascularization, 5% of the patients had neurologic deficit versus 33% in patients where the injured carotid artery was ligated ($p < 0.002$).[23] Ramadan et al. reported an even more pronounced difference in neurologic deficit after vascular repair compared with ligation in neurologic asymptomatic patients (8% vs 50%, $p < 0.05$).[24] Other reports concluded that restoration should always be performed in the case of neurologically asymptomatic patients.[25]

The most important complication of a ligated internal carotid artery or an untreated internal carotid artery occlusion is that it may develop embolic sequelae.[13] We believe ligation should be reserved for (1) those patients who had prolonged coma for more than 4 to 6 hours, (2) technical reasons where surgical revascularization or endovascular repair is not possible, and (3) injuries to the external carotid artery and its branches.[26]

As a non-surgical treatment, watchful waiting is the only option. Some authors suggest systemic heparinization for proven occlusion of the carotid artery due to gunshot injury to neck without involving brain or other body parts.[2]

These recommendations are based on available data from patients with blunt carotid injuries with identical injuries in which heparinization has been observed to be linked to improvements in neurologic condition and survival. However, the role of anticoagulants in relation to safety and efficacy in the management of stroke following carotid injury has not been established.[28]

Conclusion

Excellent knowledge of anatomy of the neck, proper systematic physical evaluation and current and updated recommendation-based utilization of appropriate modalities of intervention are necessary for management. Rapid decision-making capability and skill of a surgeon are required to prevent further deterioration of vascular, airway and neurologic status.

Patient with serious bleeding and in unstable condition with hematoma should be taken to surgery immediately and managed by multidisciplinary approaches. Stable patients with suspected vascular injury can be planned for elective surgery after performing in-depth examination, Doppler ultrasonography and CTA. Reconstruction of the arterial and venous injury shows the best outcome in most of the patients.

References

1. Burda TM, Cotton BA. Straight for the jugular: managing blunt

& penetrating neck trauma in the field. JEMS. 2007;32(8):40-9. doi:10.1016/S0197-2510(07)72293-3

2. American College of Surgeons Committee on Trauma. Advanced Trauma Life Support for Doctors: Student Course Manual, 2004 7th ed. American College of Surgeons: Chicago
3. Thal ER, Meyer DM. Penetrating neck trauma. Curr Probl Surg.1992; 29: l
4. McConnell DB, Trunkey DD. Management of penetrating trauma to the neck. Adv Surg. 1994;27:97-127
5. Apffelstaedt JP, Muller, R. Results of mandatory exploration for penetrating neck trauma. World J Surg. 1994; 18:917
6. Asensio JA, Valenziano CP, Falcone RE, Grosh JD. Management of penetrating neck injuries. The controversy surrounding zone II injuries. Surg Clin North Am. 1991; 71(2):267-296. doi:10.1016/s0039-6109(16)45379-x
7. Bryant AS, Cerfolio RJ. Esophageal trauma. Thorac Surg Clin. 2007;17(1):63-72. doi:10.1016/j.thorsurg.2007.02.003
8. Rezende-Neto J, Marques AC, Guedes LJ, Teixeira LC. Damage control principles applied to penetrating neck and mandibular injury. J Trauma. 2008;64(4):1142-1143. doi:10.1097/TA.0b013e318166d2d29.
9. Montalvo BM, LeBlang SD, Nunez DB Jr, Ginzburg E, Klose KJ, Becerra JL, Kochan JP. Color Doppler sonography in penetrating injuries of the neck. AJNR Am J Neuroradiol.1996; 17(5):943-51
10. Sekharan J, Dennis JW, Veldenz HC, Miranda F, Frykberg ER. Continued experience with physical examination alone for evaluation and management of penetrating zone 2 neck injuries: results of 145 cases. J Vasc Surg 2000; 32
11. Martin MJ, Mullenix PS, Steele SR, Asensios A, Anderson CA, Demetriudes D, Salim A. Functional outcome after blunt and penetrating carotid artery injuries :analysis of the national trauma data bank. J trauma 2005; 59(4):860-4; 5/22/2012
12. Manson DO, Saletta JD, Freeark RJ. Carotid vertebral trauma. J Trauma 1969; 9: 987-99
13. Feliciano DV. Management of penetrating injuries to carotid arterv. World J Surg. 2001; 25(8):1028-35
14. Roon AJ, Christenscn N. Evaluation and treatment of penetrating cervical injuries. J Trauma 1979; 19: 391-7
15. van As AB, van Deurzen DF, Verleisdonk EJ. Gunshots to the neck: selective angiography as part of conservative management. Injury 2002; 33(5):453-6. doi:10.1016/s0020-1383(02)00056-6
16. Dennis JW, Frykberg EF, Veldenz HC, et al. Validation of nonoperative treatment of occult vascular injuries and accuracy of physical examination alone in penetrating extremity trauma: 5 to 10 year follow-up. J Trauma 1998; 44:243-53
17. Gerst PH, Sharma SK, Sharma PK. Selective management of penetrating neck trauma. Am .Surg. 1990; 56:553-5
18. Cohen ES, Breaux CW, Johnson PN, et al. Penetrating neck injuries: experience with selective management. South Med J. 1987 ; 80:26-8
19. Sclafani SJ, Panetta T, Goldstein AS, Phillips TF, et al. The management of arterial injuries caused by penetration of zone Ill of the neck. J Trauma 1985; 25:871-9
20. Bumpous JM, Whitt PI, Ganzel TM, McClane SD. Penetrating injuries of the visceral compartment of the neck. Am J Otolaryngol. 2000; 21:190-4
21. Azuaje RE, Jacobson LE, Glover J, et al. Reliability of physical examination as a predictor of vascular injury after penetrating neck trauma. Am Surg. 2003; 69(9):804-7
22. Nason RW, Assuras GN, Gray PR, et al. Penetrating neck injuries: analysis of experience from a Canadian trauma centre. Can J Surg. 2001:44: 122-6
23. Liekweg WG, Greenfield LJ. Management of penetrating carotid arterial injury. Ann Surg.1978; 188:587-92
24. Ramadan F. Rutledge R, Oiler D. Howell P, Baker C, Keagy R. Carotid artery trauma: a review of contemporary' trauma center experiences. Vasc Surg. 1995; 21:46-56
25. Nunez DB, Ibrres-Leon M, Munera F. (2004): Vascular injuries of the neck and thoracic inlet: helical CTangiographic correlation. Hadiog Taphles 2004; 24:1087-1100
26. Rogier H, J. Kropman, Jean-Paul P. M. de Vries, and Michiel J. M. Segers. Surgical Repair of a Gunshot Injury to the Left Carotid Artery: Case Reportand Review of Literature. Vascular and Endovascular Surgery 2008; 42 (2): 180-3
27. Biffl WL, Moore EE, Ryu RK, et al. The unrecognized epidemic of blunt carotid arterial injuries: early diagnosis improves neurologic outcome. Ann Surg. 1998; 228(4):462-70. doi:10.1097/00000658-199810000-00003
28. Hacke W, Stingele R, Steiner T, Schuchardt V, Schwab S. Critical care of acute ischemic stroke. Intensive Care Med. 1995; 21(10):856-62. doi:10.1007/BF01700973.

A Comparative Study of the Effects of Anterior Nasal Packing versus Trans-Septal Suturing in Post-Septoplasty Patients

https://doi.org/10.47210/bjohns.2020.v28i2.323

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ABSTRACT

Introduction

Following septoplasty, the usual norm is to pack the nose using polyvinyl alcohol or white petroleum jelly impregnated gauze. But due to side effects like pain, respiratory problems, headache or pain during pack removal, trans-septal suturing is recommended as it provides stability and prevents septal hematoma formation without causing the above mentioned complaints. This study aims to conduct an in-depth comparison of the outcomes of the two aforementioned methods in an Indian population.

Materials and Methods

A prospective randomized comparative study was done for 30 patients between 18-50 years undergoing septoplasty, while excluding those requiring additional surgical interventions. Trans-septal suturing was done for 15 patients and polyvinyl alcohol nasal packing for the rest. Visual Analogue Scale was used to record discomfort levels 1 day post-operatively. A vast multitude of symptoms were assessed during regular visits for 3 months after surgery.

Results

Post-operatively, nasal pain, headache, dyspnoea, sleep disturbance, post-nasal drip, crusting, and epiphora were found to be reduced in patients who underwent trans-septal suturing when compared with packing, deeming it to be a superior choice of method. However, nasal bleeding was comparable in both groups.

Conclusion

Trans-septal suturing reduces pack related problems and post-operative complications, without significantly increasing the duration of surgery. Hence, we recommend the practice of placing sutures to positively improve patients' quality of life post-surgery.

Keywords

Septoplasty; Nasal Packing; Sutures, Trans-septal

Septoplasty is a common surgery in otorhinolaryngology, performed primarily on patients with deviated nasal septum causing nasal obstruction, mouth breathing, snoring and recurrent episodes of sinusitis and otitis media. It is also indicated in patients having recurrent epistaxis due to a septal spur, and as a part of septorhinoplasty procedures. Nasal packing has been considered to be an essential step of the surgery, performed via different techniques.[1] However, there is lack of definitive standards entailing the preferred materials which should be used for nasal packing, how long the pack should be left in place, or the specific indications for nasal packing.[2]

Some of the common scenarios warranting packing in nasal septal surgeries include control of

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haemostasis, avoidance of septal haematoma, and prevention of displacement of replaced cartilage.[3] But nasal packing comes with its own disadvantages, mainly patient discomfort and the need for hospital stay.[3] Nasal packing has been reported as one of the leading causes of early postoperative pain,[4] and pack removal in the postoperative period is accompanied by pain.[5] Additional complications include the worsening of sleep-disordered breathing and risk of postoperative infections, predominantly toxic shock syndrome resulting from post-septoplasty packing.[6]

Trans-septal suturing alleviates all the afore-mentioned problems without causing any complications. In 1984, Sessions et al studied continuous suture quilting, where he used 4-0 plain catgut suture mounted on a small cutting needle in order to approximate the mucosal flaps.[7] A similar technique was discussed by Lee et al , the difference being the usage of a curved needle instead of the cutting type.[8] These techniques aid in closure of mucosal tears and provide support to the remaining cartilage.[9]

Although there is no paucity of studies comparing selective outcomes of nasal packing with trans-septal suturing following septoplasty, there appears to be a dire need of a broad all-inclusive study, which delves into the entire spectrum of post-procedural complications in the same subset of patients, so as to avoid any confounders or bias caused by differences in patient demographics or criteria of selection for study. Our study has hence been conducted to achieve the same and overcome the limitations of research articles on this topic conducted in the past, while simultaneously evaluating the post-operative quality of life.

Materials and Methods

After obtaining institutional ethical clearance, a single blinded prospective randomized comparative study was conducted in a district general hospital from September 2018 to February 2019. Thirty patients of either sex between the age groups of 18 to 50 years having symptomatic deviated nasal septum, who were willing to undergo the procedure of septoplasty, were selected for the study. Those requiring additional interventions such as turbinectomy, conchoplasty or endoscopic sinus surgery were excluded from the study. Patients were divided into two groups of 15 patients each using envelope method. polyvinyl alcohol (Merocel®) nasal packing was proposed for patients of Group A, while Group B patients were planned to undergo trans-septal suturing after septoplasty.

Informed written consent was taken from all patients, following which all cases were taken up

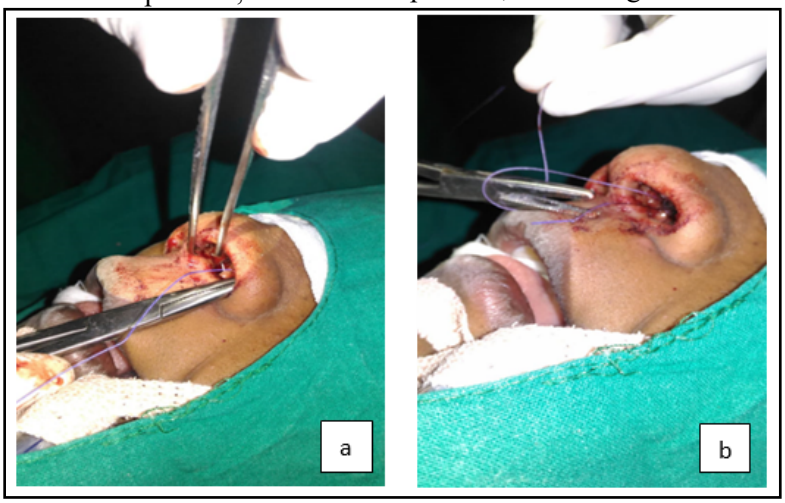

Fig. 1. a & b : Fracture of the body of mandible; c: fracture at the condyle of mandible; d : fracture at the symphysis of the mandible

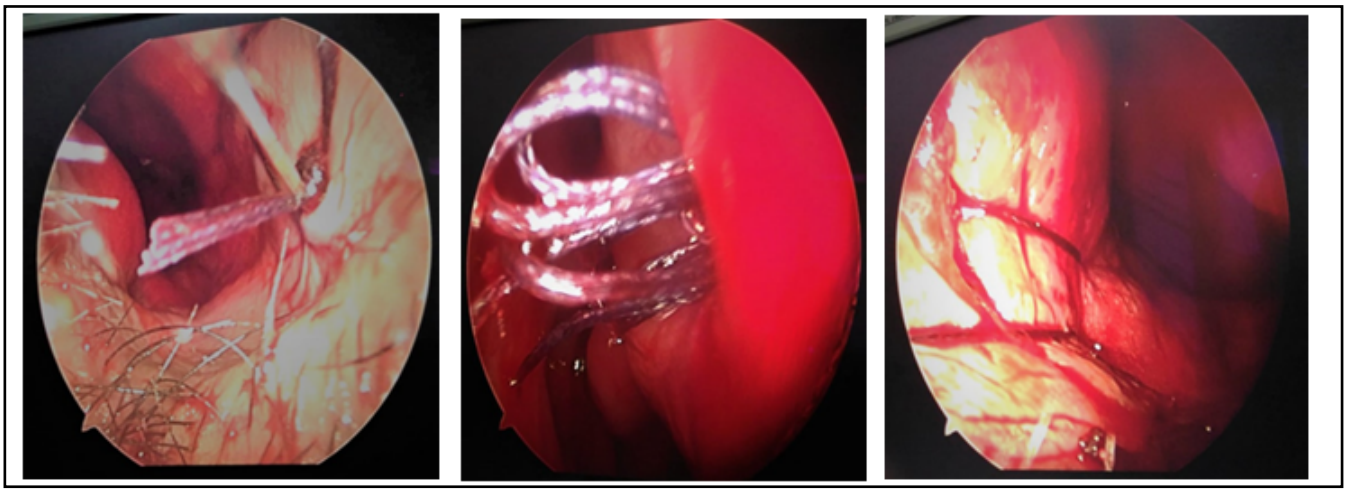

Fig. 2. Endoscopic view of trans-septal suturing

under general anaesthesia. Surgery for all cases was done by a single surgeon to maintain uniformity. Infiltration using 2% lignocaine with 1 in 1,00,000 adrenaline was given over the septal mucosa on both sides. Killian's incision was given 2–3 mm behind the caudal end of septal cartilage on the concave side. Mucoperichondrial and mucoperiosteal flap was elevated on one side, following which bony-cartilaginous junction was identified and dislocated. Deviated part of bony septum was removed using Luc's forceps. Inferior strip of cartilaginous septum was removed. Maxillary crest was removed using gouge and hammer. Hemostasis achieved and flaps were repositioned.

During this stage, it was revealed to the surgeon if patient belonged to Group A or Group B. Patients of Group A underwent nasal packing using polyvinyl alcohol nasal packing. Trans-septal quilting sutures were placed (Fig. 1- a, 1-b) to Group B patients using Vicryl® 3-0 absorbable suture material. (Fig. 2) After surgery, patients received parenteral antibiotics, and adequate analgesia for 3 days. Nasal packing was removed for patients of Group A on second post-operative day. Patients were advised to use saline nasal wash for three months post-surgery.

Patients were asked to record post-operative symptoms using a Visual Analogue Scale (VAS) on first post-operative day. (Fig. 3) The intensity of post-operative symptoms was graded from 1 to 10, with 10 being the score for maximal discomfort and 1 being the least.

Various symptoms, namely nasal pain, nasal bleeding, headache, difficulty in breathing, sleep disturbance, increased lacrimation, post nasal dribbling, and excessive crusting were assessed using VAS during immediate post-operative period and during subsequent visits. Complications like septal hematoma, septal adhesions, and synechiae were also documented. Three follow-up visits were scheduled at first week, second week and third month following surgery.

Results were presented as Mean ± SD and range values of VAS and categorical data as numbers and percentages. Since the observations were made in scores (VAS), which is a non-parametric method, Mann-Whitney test was used to compare between two groups. Chi-square test was used for analyzing categorical data. p value of 0.05 or less was considered for statistical significance. SPSS software (version 17) was used for analysis.

Results

The mean age of the sample (30 cases) , who were ranging from 18 to 50 years, was 23.2 ± 5 years in Group A and 24.2 ± 6.0 years in Group B. (Table I)

The first assessment was carried out on the first

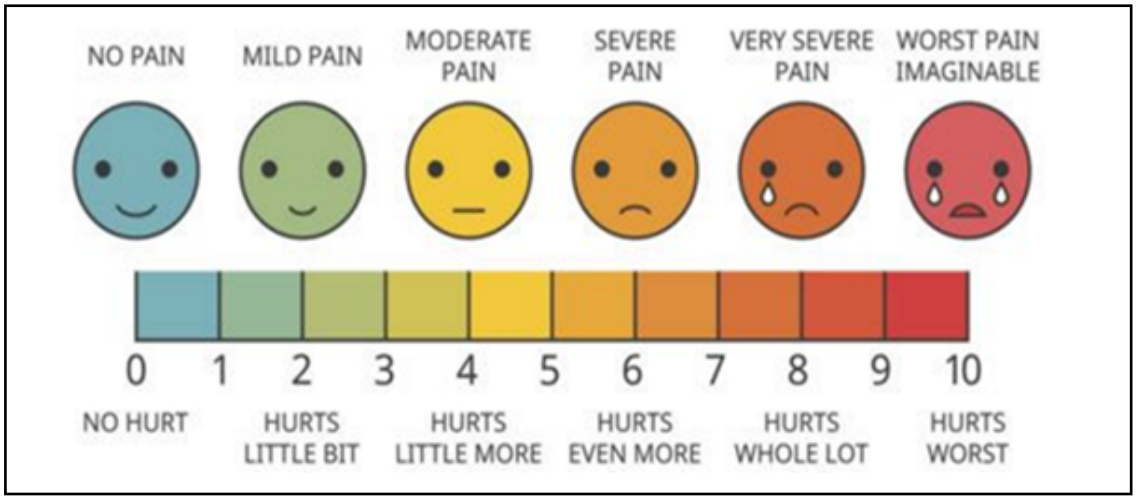

Fig. 3. Visual analogue score to document responses from patients

postoperative day, and the severity graded between 0 to 10 based on the Visual Analogue Scale. The results obtained were tabulated and compared for each symptom separately, which are discussed in the subsequent paragraphs.

Post-operative nasal pain was found to be significantly higher in Group A, (mean - 4.5±1.8) as compared to Group B (mean - 2±1.6) . On comparison, p was found to be less than 0.001, which was highly significant. (Fig. 4)

Post - operative nasal bleeding was found to be equal in both the groups, with mean values being 3.3±2.3 in group A and 3.2±2.5 in group B. (Fig. 5) It was more in cases of correction of gross deviation of septum and prominent spurs, irrespective of the method used.

Patients in Group A complained of headache more than the patients in Group B. Mean was 4.3 ± 2.2 in Group A, whereas it was 1.5 ± 1.4 in Group B. Hence, headache was found to be significantly reduced on trans-septal suturing ($p < 0.001$).

Post-operative difficulty in breathing was significantly lower in Group B (1.1±1.3) as compared to Group A (4.7± 1.5). p value was found to be less than 0.001 which was statistically highly significant, proving trans-septal suturing to be a better method in this parameter.

Post-nasal drip and sleep disturbance were found to be significantly lower in Group B when compared with Group A , with mean values of 0.8± 1.1 and 1.2±1.4 respectively in group B as opposed to 3.3±2.3 and 3.8±2.5 respectively in Group A.

Increased lacrimation and excessive crusting were assessed, but results were not found to be statistically significant.

Overall, patients who underwent trans-septal suturing had a better quality of life as compared to patients who underwent nasal packing due to

Table I: Mean age of patients in both the groups

AGE DISTRIBUTION		GROUP A	GROUP B	GROUP A V/S GROUP B	
				T	P
Age (years)	Mean±SD	23.2±5.1	24.2±6.0	0.5	0.62, NS

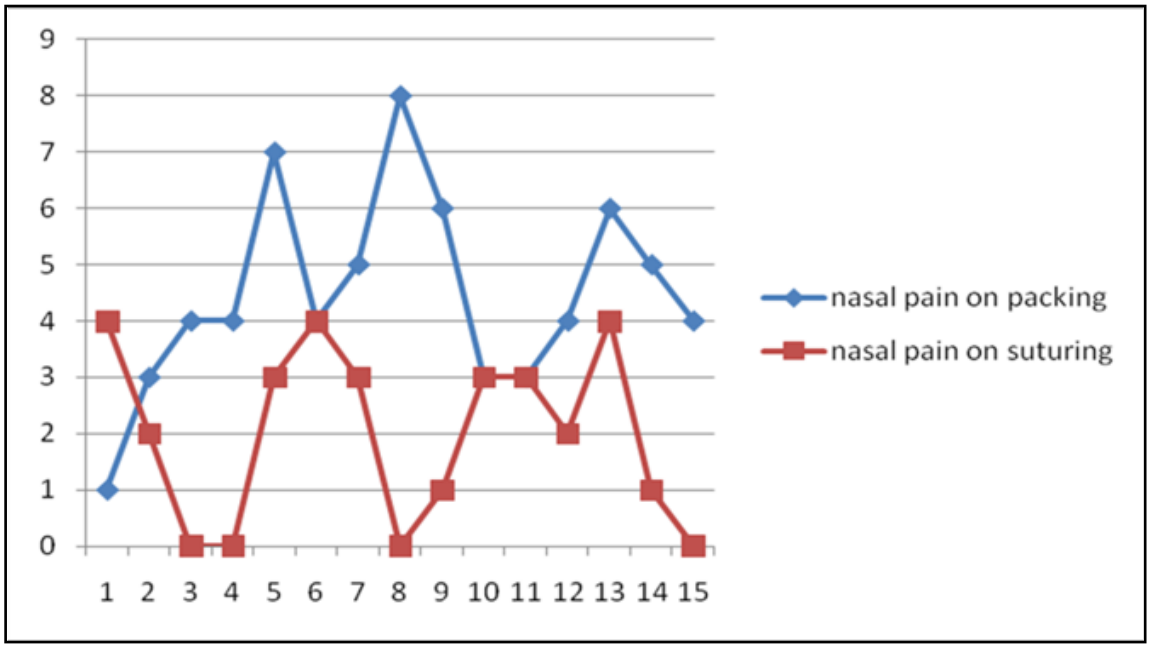

Fig. 4. Comparison of nasal pain between nasal packing and trans-septal suturing

a marked reduction in symptoms of nasal pain, headache, decreased quality of sleep and post nasal drip (Fig. 6). There was no incidence of complications such as septal hematoma or synechiae in either group. Hence, it is a safer and easier alternative to nasal packing post-septoplasty, as proved by our

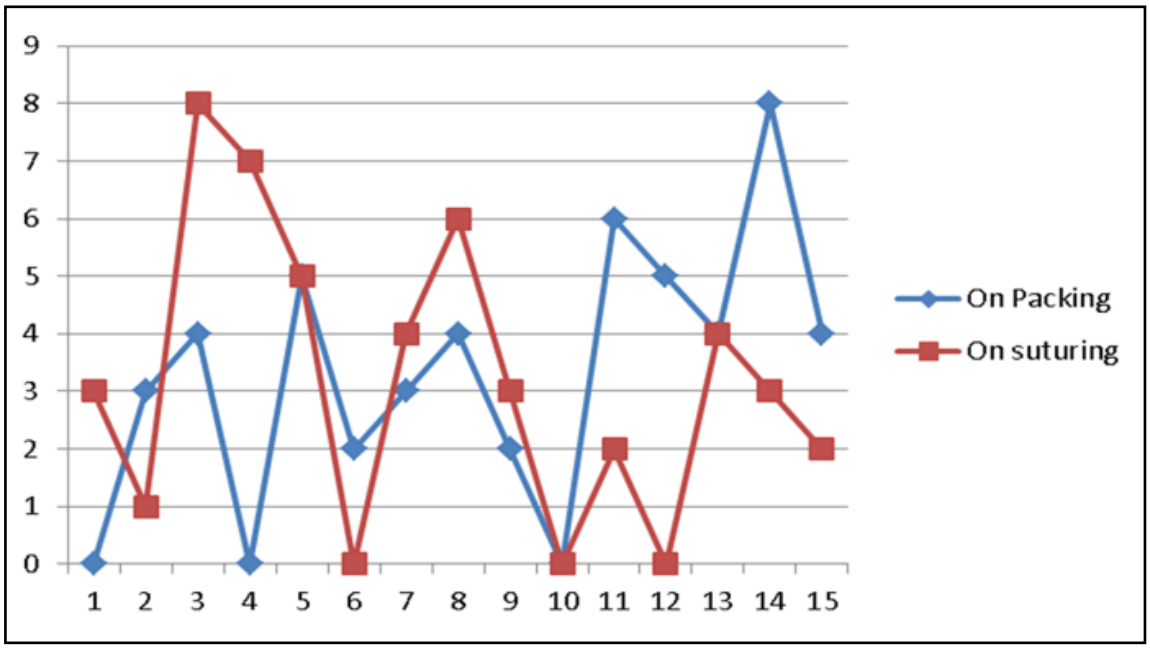

Fig. 5. Comparison of nasal bleeding between nasal packing and trans-septal suturing

TABLE II: Comparison of symptom scores between both the groups on post-operative day

SYMPTOMS ASSESSED POST-SEPTOPLASTY	GROUP A	GROUP B	COMPARISON
	MEAN ± SD	MEAN ± SD	P VALUE
1.Nasal pain	4.5±1.8	2.0±1.6	< 0.001, HS
2.Nasal bleeding	3.3±2.3	3.2±2.5	0.78, NS
3.Headache	4.3±2.2	1.5±1.4	0.001, S
4.Difficulty in breathing	4.7±1.5	1.1±1.3	< 0.001,HS
5.Post nasal drip	3.3±2.3	0.8±1.1	0.002, S
6. Sleep disturbance	3.8±2.5	1.2±1.4	0.004, S
7.Increased lacrimation	1.7±1.9	1.1±1.4	0.51, NS
8. Excessive crusting	1.3±1.6	0.9±1.4	0.51, NS

Mann-Whitney test

p < 0.05, Significant (S) **Group A - nasal packing**

p < 0.001, Highly Significant (HS) **Group B - trans-septal suturing**

p < 0.05, Not Significant(NS)

study. (Table II)

The follow up visits were conducted at the end of first week, second week and third month after surgery. But symptom score for all patients in both groups were less than or equal to 1 according to VAS in all parameters at the end of first week, and 0 in subsequent visits. Hence, these were treated as clinically insignificant.

Discussion

Worldwide, nasal packing is commonly practised after septoplasty to augment mucoperichondrial flap apposition and to facilitate closure of dead space.[3] The commonly used nasal packing materials are paraffin gauze, gauze impregnated with antibiotics, Vaseline gauze, bismuth iodoform paraffin paste, fibrin glue, gelfoam, merocel, silastic sheets, and rarely glove fingers.[10]

We have used Merocel as a packing material in our study as it is comparatively easier to remove after surgery and does not cause foreign body reaction. However, nasal packing itself causes complications. As enlisted by Huang et al, the complications of packing include mucosal injury leading to septal perforation, displacement of packing material or its aspiration, allergic reaction, toxic shock syndrome, and occasionally eustachian tube dysfunction.[11]

In order to overcome these complications, Cukurova et al conducted a retrospective analysis of 697 septoplasty cases, and concluded that trans-septal suturing techniques used in septoplasty cause negligible pain and post-procedural complications, allowing patients to resume their routine day-to-day

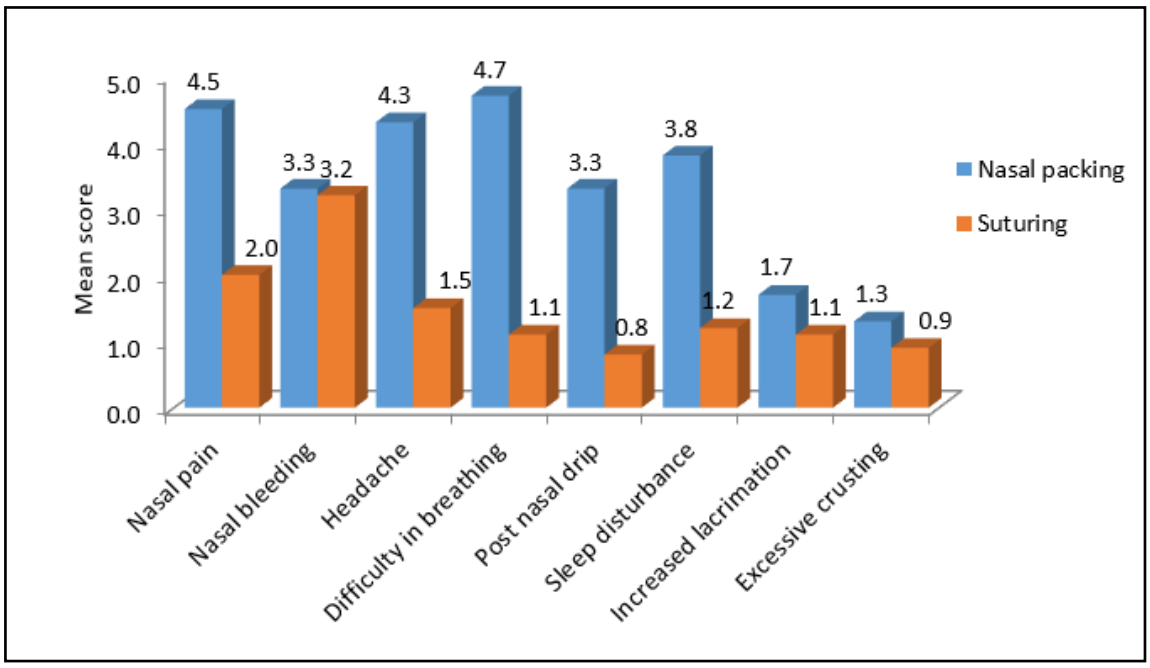

Fig. 6. Comparison of mean of visual analogue scores between the two groups

functions within a relatively short duration.[12] Their study affirmed that nasal packing is a risk-associated procedure having no justified reason for its continued use in routine practice, while concurrently emphasizing enhanced patient comfort after septoplasty if the suturing technique is practised.[12] Likewise, in our study , trans-septal suturing significantly reduced the incidence of post surgical nasal pain, headache, difficulty in breathing, post-nasal drip, sleep disturbance, and alleviated the concerns faced by the patient during pack removal.

Naik too, confirmed that intranasal packing lead to increased incidence of synechiae and adhesions when compared with trans-septal through-and-through splint suturing in the Indian demographic.[13] Our study, on the contrary, was conducted with the intent to propose the efficacy of a more cost-effective procedure and still gives the desired result. Hence, for our study, trans-septal suturing was chosen as the parameter of comparison with packing, instead of more expensive alternatives available in the market, such as splints. We did not encounter any post-operative complications in either of the groups. Hence, we recommend the practice of placing sutures to positively improve the quality of life of the patients while also relieving the financial burden on the patients presenting to a government setup.

Another drawback of nasal packing is that it tends to restrict the nasal component of respiration and thereby the overall respiratory function, rendering the quality of sleep to be impacted negatively.[12] This is implicated to reduced PaO_2 levels after bilateral nasal packing and the inadequate oral breathing during sleep, which causes hypoxia to be appreciated by the patient more in the nocturnal phase.[12] This is especially highlighted in elderly patients with underlying ischaemic heart diseases accompanying respiratory conditions such as obstructive sleep apnoea or chronic obstructive pulmonary disease.[12] Patients undergoing suturing suffered significantly reduced incidence of respiratory problems ($p < 0.001$) along with a higher comfort score.[14] Likewise, in our study, sleep disturbance was significantly lesser ($p < 0.004$) in trans-septal suturing group as compared to nasal packing. Patients with nasal packs confirmed that the presence of nasal obstruction, anxiety and headache caused them to develop sleep disturbance. Hence we effectively conclude that trans-septal

suturing alleviates the concerns and anxiety of patients and helps them to recuperate faster.

In a study done by Certal et al, packing and non-packing methods seemed to give equal incidence of postoperative risk of bleeding.[15] However, suturing techniques implicated a significant reduction in incidence of post-operative nasal pain as well as headache.[15] Similarly, post-operative bleeding in both groups were comparable in our study. Correction of grossly deviated septum and prominent spurs resulted in more incidence bleeding post-procedurally, irrespective of the group that the patient belonged to. However, nasal pain and headache were significantly relieved in trans-septal suturing group with $p<0.001$, proving yet again the benefit of suturing over packing.

Though epiphora is mainly the result of nasolacrimal duct obstruction caused by packing, Abdulkhaliq et al proved in their study that there was no statistical difference between both the groups.[16] Even in our study, though epiphora was higher in the packing group, it was not found to be statistically significant on comparing with the suturing group.

What makes our study of great value is that not only does it extensively cover multiple parameters in its post-operative symptom assessment than ever done before in a single study; but it also evaluates the occurrence of post-nasal drip in relation to these two techniques, which has never previously been reported with respect to Indian population in literature. This renders our study pivotal as a benchmark for future studies of comparison in this aspect, eliciting a highly significant reduction of post-nasal discharge in patients who underwent trans-septal suturing (p value - 0.002). This also resulted in reduced hawking, throat irritation and coughing in patients leading to a symptom-free post-operative period.

Thus, while the long term impact and complications of the two methods are similar, we can conclude from our study that trans-septal suturing offers better quality of life to patients in the weeks following septoplasty due to its undeniable superiority in relieving symptomatic troubles. Hence, our study promotes its routine use in patients undergoing septoplasty, rather than following traditional methods of packing, especially in the poor strata of the Indian society.

Conclusion

Nasal pain, headache, dyspnoea, sleep disturbance, post – nasal drip, crusting, excessive lacrimation were reduced in the trans – septal suturing group as compared to packing. However, nasal bleeding was present in both the groups. Trans-septal suturing also alleviates pack removal problems like anxiety, pain, bleeding in a patient. It also proves to be a cost-effective modality and gives a better post-operative quality of life to patients. Hence, we recommend trans-septal suturing as the preferred method over anterior nasal packing in patients after septoplasty.

References

1. Dubin MR, Pletcher SD. Postoperative packing after septoplasty: Is It Necessary? Otolaryngol Clin North Am. 2009; 42: 279-85
2. Von Schoenberg M, Robinson P, Ryan R. Nasal packing after routine nasal surgery -is it justified? J Laryngol Otol. 1993; 107: 902-5
3. Lemmens W, Lemkens P. Septal suturing following nasal septoplasty. A valid alternative for nasal packing? Acta Otorhinolaryngol Belg. 2001; 55:215-21
4. Yilmazer C, Sener M, Yilmaz I et al. Pre-emptive analgesia for removal of nasal packing: A double-blind placebo controlled study. Auris Nasus Larynx 2007; 34: 471-5
5. Samad I, Stevens HE, Maloney A. The efficacy of nasal septal surgery. J Otolaryngol. 1992; 21: 88-91
6. Taasan V, Wynne JW, Block AJ. The effect of nasal packing on sleep-disordered breathing and nocturnal oxygen desaturation. Laryngoscope 1981; 91: 1163-72
7. Sessions R. Membrane approximation by continuous mattress sutures following septoplasty. Laryngoscope 1984; 94: 702-3
8. Lee IN, Vukovic L. Hemostatic suture for septoplasty: how we do it. J Otolaryngol 1988;17:54-6
9. Hari C, Marnane C, Wormald Peter-John. Quilting sutures for nasal septum. J Laryngol Otol. 2008; 122(5):522-3
10. Daneshrad P, Chin Gregory Y, Rice Dale H. Fibrin glue presents complications of septal surgery: findings in a series of 100 patients. Ear Nose & Throat J. 2003; 82:196-8
11. Huang IT, Podkomorska D, Murphy MN, Hoffer I.

Toxic shock syndrome following septoplasty and partial turbinectomy. J Otolaryngol. 1976; 15:310-2

12. Cukurova I, Cetinkaya EA, Mercan GC, Demirhan E, Gumussoy M. Retrospective analysis of 697 septoplasty surgery cases: packing versus trans-septal suturing method. Acta Otorhinolaryngol Ital. 2012; 32(2):111-4
13. Naik K. A Novel way of Trans-Septal Splint Suturing Without Nasal Packing for Septoplasty. Indian J Otolaryngol Head Neck Surg. 2015; 67(1):48-50 doi: 10.1007/s12070-014-0763-x. Epub 2014 Aug 10.
14. Ali AP, Mahbobeh O, Shahrokh K. Comparison results of nasal septum suturing and nasal packing following septoplasty. Romanian Journal of Rhinology. 2019; 9(34): 97-99. DOI: 10.2478/rjr-2019-0012
15. Certal V, Silva H, Santos T, Correia A, Carvalho C. Trans-septal suturing technique in septoplasty: a systematic review and meta-analysis. Rhinology 2012; 50(3):236-45
16. Abdulkhaliq KA, Dashti AH, Ammar MSJ. Trans-septal suture versus intranasal silicone splint in septoplasty; International Journal of Technical Research and Applications. 2015; 3(3):159-65.

Main Article

Clinical Assessment of Sensorineural Hearing Loss among Diabetes Mellitus Patients

https://doi.org/10.47210/bjohns.2020.v28i2.297

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ABSTRACT

Introduction

Hearing impairment is known to hamper the quality of life among patients, especially among diabetics due to the association of neuropathy with diabetes mellitus (DM). However, the prevalence and degree of the sensorineural hearing loss (SNHL) depends upon different factors, such as age, gender, disease duration of DM, family history and glycaemic status of the patients. Therefore, this study aimed to assess the association of SNHL with DM duration and familial DM and gender preponderance among SNHL–DM patients.

Materials and Methods

140 patients with DM were assessed for hearing impairment using Rinne, Weber and Absolute Bone Conduction Tests along with pure tone audiometry. Patients' glycaemic status was determined by estimating fasting blood glucose (FBG) and post prandial blood glucose (PPBG) levels. Independent t-test, chi-square, ANOVA and Pearson's correlation tests along with linear regression model were used to find association and correlation using R software.

Results

Out of 140 patients, 60 were suffering from SNHL and majority was suffering from bilateral minimal hearing loss. SNHL was significantly associated with family history, age, duration of DM. FBG and PPBG levels were (p values:1.79E08, 4.41E-06 and 0.02), however, significantly correlated with duration of DM, FBS and PPBG level (r value:0.14–0.41). Furthermore, significant SNHL at 500 and 8000Hz was observed in the present study (p value:0.002).

Conclusion

A conclusive proof was drawn that family history of DM serves as a valuable variable in assessing the SNHL among DM patients.

Keywords

Hearing; Hearing Loss, Sensorineural; Diabetes Mellitus, Type 2; Audiometry, Pure-Tone

Diabetes mellitus (DM) is a non-communicable, metabolic and chronic disease, marked by the high blood glucose level due to either absolute or relative insulin hormone deficiency. According to the International Diabetes Federation report, 7.5 billion people were recorded to have DM worldwide in 2017 with 8.8% prevalence among adult population. In the Indian population, approximately 72.9 million individuals have been diagnosed with DM.[1] Frequently encountered complications of DM include hearing loss, tinnitus, cardiovascular disease, neuropathy and retinopathy.[2,3] Among these, hearing impairment affects mostly the quality of life of affected individuals by impacting their personal and social lives. However, hearing impairment is also frequently associated with age-related degenerative changes of auditory organs in

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elderly persons.[4]

Researchers have reported hearing loss of low, mid and high frequencies[5,6] which can be due to either neuropathy[7] or micro vascular complications.[8] Hearing loss complications in DM can be bilateral (B/L) and sensorineural (SN)and may be gradually progressive. Sensorineural hearing loss (SNHL) is a disorder of the inner ear, vestibulocochlear nerve or central brain processor unit impairment that can be congenital or acquired. Cochlear changes, such as increased thickness of basal membrane and stria vascularis vessel walls, internal auditory artery sclerosis[9] and inner ear neural system degeneration[10] are responsible for hearing impairment among DM patients. Hearing impairment can be due to either combined effect of neural and vasculature system impairment or they can independently cause auditory loss among DM patients. However, there is a persistent disagreement among researchers on DM being a possible cause for SNHL.[5, 10]

Researchers studied the association of SNHL with age,[11] duration of DM,[11] HbA1c,12 gender,13 hypertension,[13] hyperlipidemia,[14] obesity[13] and socioeconomic status,[13] along with drinking and smoking habits. Hawang et al.[13] reported that central obesity, hypertension alcohol intake and male gender are positively associated with high frequency SNHL. However, Parmar et al.[14] pointed out that patients with DM and hyperlipidemia have significantly higher hearing threshold at mid- and high frequencies as compared to normal subjects (control). However, few studies explored the association of family history of DM with SNHL.[15,16] Therefore, the present study is projected to assess SNHL and its pattern in DM patients with family history of DM.

Materials and Methods

The present study was conducted from January 2012 to December 2012 in the Department of Otorhinolaryngology after taking prior approval from Institutional Ethics Committee. A total of 140 patients with DM were enrolled and informed consents were obtained from them. Patients of both genders with symptoms of DM were included in the study. However, patients with history of noise exposure and ototoxic drug intake, hearing difficulty caused by other diseases and debilitated patients were excluded. The diabetic status of the patients was confirmed by analysing their blood sugar levels. Patients with Fasting Blood Glucose (FBG) level >100 mg/dL, first hour Post Prandial Blood glucose(PPBG) level > 200 mg/dL and random blood glucose (RBG) level >200 mg/dL were included in the present study. A detailed history was collected pertaining to DM, such as its duration, type and family history, history of other comorbid conditions along with demographics (age and gender) through an interview. Laboratory estimation of FBS and PPBS were carried out followed by ear; nose and throat (ENT) examination and findings were recorded on a predesigned and pretested Performa. Blood glucose assay was done using routine biochemical method.

Assessment of Hearing Impairment:

The patients were subjected to different tests, such as tuning fork and audiometric test to analyse their hearing status. The tuning fork test comprised of three tests, namely Rinne (to compare bone and air conduction)[17] Weber[17] and Absolute Bone Conduction (ABC; to identify SNHL)[18] tests for initial screening, followed by audiometric testing.[19] Rinne and Weber test are usually combined to identify the type of hearing loss. However, pure tone Audiometry was used to measure the hearing threshold at different frequencies, and to confirm the type of hearing loss.[21] Tests were done using 256 Hz, 512 Hz and 1024 Hz tuning forks. The audiometric tests consisted of Pure tone audiometry, Pure tone Air Conduction (AC) Threshold Audiometry, Pure Tone Bone Conduction (BC), speech reception and test for recruitment in order to measure ABC, speech reception threshold values and Short Increment Sensitivity Index (SISI).[19,22]

The pure-tone audiometry was conducted with ALPS Pure Tone Audiometer(Model AD 2000) at frequencies of 125 Hz, 250 Hz, 500 Hz, 1000 Hz, 2000 Hz, 4000 Hz and 8000 Hz for each ear, which were categorized as low (125 and 250 Hz), mid (500-2000 Hz) and high-frequencies (4000 and 8000 Hz).6 Hearing loss scale defined by the American Speech-Language

Table 1: Prevalence and association of SNHL among patients of DM as per demographics of the patients

PARAMETERS		FREQUENCY	STATUS OF SNHL		P VALUE
			PRESENT	ABSENT	
Gender	Male	88 (62.86%)	35(58.33%)	53(66.25%)	0.34
	Female	52(37.14%)	25(41.67%)	27(33.75%)	
Family history of DM	Positive	47 (33.57%)	28(46.67%)	19(23.75%)	0.004**
	Negative	93 (66.43%)	32(53.33%)	61(76.25%)	

Note: SHNL: sensorineural hearing loss and DM: diabetes mellitus

*** signifies p values<0.01*

Hearing Association (ASHA), was used to categorize DM patients as normal (-10 to 15 dB) and SNHL with varying degree of hearing loss (16 to 91+).[23]

Statistical Analysis:

Statistical analysis was performed using R software v 3.6.0 for evaluating frequency distribution, percentage, independent t-test, chi-square, ANOVA and Pearson's correlation tests and p values of <0.05, considered as statistically significant. The results of continuous data were presented as mean ± standard deviation while mean difference between different groups were compared using independent t-test. The correlation of various factors with SNHL was assessed using Pearson's correlation test.

Results

140 DM patients enrolled for the study aged between 20 to 50 years with the mean age being 40±9.2 years and duration of the DM being 4.87±3.64 years. The mean FBS and PPBS levels were 124.71±37.79 mg/dL and

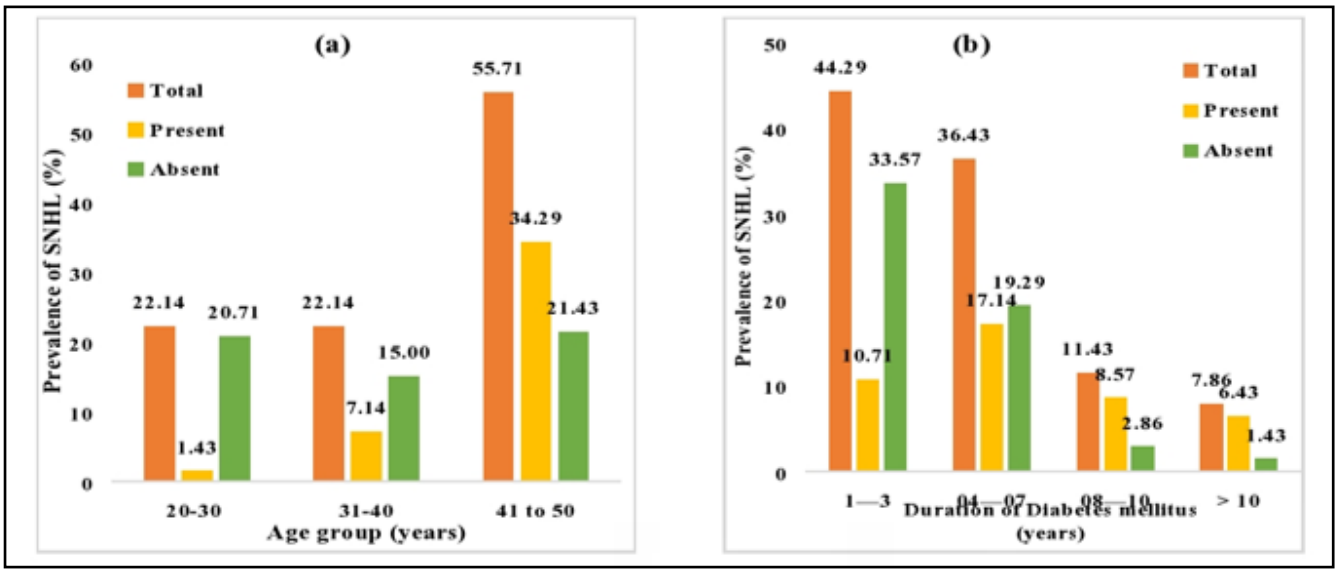

Fig. 1. Sensorineural hearing loss patients distribution based on (a) age and (b) duration of diabetics

Table II: Prevalence of SNHL different categories among different groups

SNHL DIFFERENT CATEGORIES		B/L MIN	B/L MILD	B/L MODERATE	B/L MODERATELY SEVERE	L MIN	R MILD & L MIN	R MIN	R MIN & L MILD	TOTAL
Total		**26**	**19**	**5**	**4**	**2**	**1**	**1**	**2**	**60**
Age Group (Years)	**20-30**	**1**	**0**	**0**	**0**	**1**	**0**	**0**	**0**	**2**
	31-40	**5**	**1**	**3**	**0**	**0**	**0**	**0**	**1**	**10**
	41-50	**20**	**18**	**2**	**4**	**1**	**1**	**1**	**1**	**48**
Duration of diabetes (Years)	**1–3**	**9**	**1**	**1**	**0**	**1**	**1**	**1**	**1**	**15**
	4–7	**12**	**10**	**2**	**0**	**0**	**0**	**0**	**0**	**24**
	8–10	**2**	**7**	**1**	**0**	**1**	**0**	**0**	**1**	**12**
	Above 10	**3**	**1**	**1**	**4**	**0**	**0**	**0**	**0**	**9**
Positive family history cases		**14**	**9**	**2**	**1**	**1**	**0**	**0**	**1**	**28**

Note: SNHL: sensorineural hearing loss, B/L: bilateral, Min: minimum, L:left and R: right

188.98±53.47 mg/dL, respectively. (Table 1 and Fig. 1) The patients' distribution based on the tuning fork test and pure tone Audiometry revealed that 12 patients with reduced ABC (3 patients had reduced ABC and normal SISI) and 12 patients with reduced SISI (3 patients had reduced SISI and normal ABC level).

Out of 140, sixty patients were diagnosed with SNHL; rest eighty had normal hearing levels. Male dominance of 62.86% (88) among the subject population was observed, out of which 35 were suffering from SNHL. Among female patients, 25 were suffering from SNHL (Table I). Age-wise distribution of patients revealed majority of patients (78) belonged to age group of 41-50 years, out of which 48 were suffering from SNHL (Fig. 1-a). Highest numbers of SNHL cases (24) were observed in the group having diabetic history of 4-7 years; however, majority of SNHL cases (32) were observed in the group which did not have any family history of DM (Fig. 1-b).

Out of 60 cases of SNHL, there were 26 cases of B/L minimum(16–25 dB),19 cases of B/L mild (26–40 dB), 5 cases of B/L moderate (41–55 dB) and 4 cases of B/L severely moderate (56–70 dB). 2 cases had left min. (Table II) Right mild with left min, right min and right min with left mild, respectively had 1 case each. However, none of the patients suffered from severe hearing loss (71–90 dB) or profound hearing loss (91+). The prevalence of B/L min cases were found to be higher among all other categories. The age group of 41–50 years had highest number of B/L minimum hearing loss cases (20). Similarly, patients having 4–7 years of DM disease duration (12) or patients with positive family history of DM (14) were also most frequently suffering from B/L minimum hearing loss (Table II).

The association of SNHL with various variables, such as age, disease duration, gender, family history, FBG and PPBG is presented in Table I and III. Age, disease duration, PPBG and family history were found to be significantly associated with SNHL (P value of1.79E-08, 4.41E-06, 0.02 and 0.004, respectively) using independent t-test and chi square tests for continuous and non-continuous variables. In case of patients with positive history of DM, 28 patients had SNHL while 19 patients hadn't. Patients having SNHL belonged to higher age group (44.57±6.05 years) compared to patients without SNHL (36.56±9.69). Similar scenarios

Table III: Association of SNHL with various parameters among DM patients

VARIABLES		SNHL		P VALUE
		ABSENT	PRESENT	
		MEAN±SD	MEAN±SD	
Age (Years)		36.56±9.69	44.57±6.05	1.79E-08**
Duration of DM (Years)		3.6±2.56	6.57±4.15	4.41E-06**
GlycemicStatus	FBS (mg/dL)	120.04±37.48	130.93±37.61	0.09
	PPBS (mg/dL)	179.9±54.02	201.08±50.68	0.02*

Note: SNHL: sensorineural hearing loss, SD: Standard deviation, DM: Diabetes mellitus, FBS: Fasting blood sugar and PPBS: Post prandial blood sugar
** and ** indicates statistically significant, P values of <0.05 and <0.01*

were observed in case of disease duration and PPBG.

The mean values of disease duration and PPBG among SNHL patients were found to be 6.57±4.15 and 201.08±50.68 respectively. Furthermore, the SNHL was also correlated with duration of diabetics, FBG and PPBG (Pearson's correlation). Slight positive correlation was found between SNHL and FBG (r value=0.14). Similar results were observed while correlating PPBG with SNHL (r value=0.2). However, moderate positive correlation was found between duration of diabetes and SNHL (r value=0.41). Furthermore, the results of frequency-wise assessment of SNHL with fasting glucose level are tabulated in Table IV. Significant increase in hearing threshold was observed among the patients with varying FBS concentration in either left or right across low, medium and high frequencies (P value: 0.02–0.002), except 1000–8000Hz for left ear and 125 Hz for right ear, based on ANOVA analysis. However, SNHL for both ears were observed with low (250 Hz) and mid-frequency (500 Hz) with P values of 0.02–0.002. (Table IV)

Discussion

DM is frequently associated with hearing damage as it affects both microscopic and large sized blood vessels. The underlying cause of nerve damage can be micro vessel diseases, neural tissue hypoxia, changes due to atherosclerotic and metabolic disorders, especially in DM. Among DM patients, majority of hearing loss cases belong to high frequency SNHL, therefore, either they go unreported or overlooked. Hence, the present research is focused on clinically assessing the SNHL among DM patients, with respect to family history along with other variables, namely demographics and glycaemic status.

This study demonstrated the prevalence of 42.85% SNHL cases among 140 DM patients that was not in accordance with other studies, which showed varying degree of SNHL.[24,25] Rajamani et al. reported the SNHL prevalence of 51.3%.[24] Conversely, a prospective study done by Harkare et al. reported higher incidences (74.07%) of SNHL among DM patients.[25] Majority of SNHL patients were suffering from bilateral minimum SNHL (26/60) similar to results reported by Dadhich et al. (48/73).[11] Although gender did not significantly affect SNHL, male diabetics (58.33%) had slightly higher incidence of SNHL as compared to females (41.67%) that accorded with results of Harkare et al.[25]

We observed that age, disease duration, PPBG and family history was significantly associated with occurrence of SNHL on evaluating the status of SNHL among DM patients. Among these variables, age is usually associated with hearing loss in elderly person. Age-linked hearing loss is classified as hearing loss that starts at mid to late adulthood, which can be bilateral and progressive SNHL devoid of any underlying causes, such as loud noise exposure, otological diseases and toxic drugs and other medical conditions

Table IV: Comparative account of SNHL with blood glucose levels

FREQUENCY (HZ)	EAR	"FASTING BLOOD GLUCOSE LEVELS (MG/DL) MEAN ± SD				P VALUE
		<100	100-150	151-200	>200	
125	Left	14.71±12.85	14.80±12.82	23.75±16.96	23.33±8.76	0.019*
	Right	14.71±12.91	14.67±14.20	23.96±21.31	20.83±9.70	0.054
250	Left	14.12±12.46	15.33±12.26	23.75±17.71	23.33±13.29	0.02*
	Right	14.26±12.98	14.87±13.78	25.21±20.88	29.17±12.42	0.004**
500	Left	12.65±10.89	14.87±13.24	25.21±17.35	22.50±10.84	0.002**
	Right	11.76±10.79	14.93±12.66	23.96±16.94	25.00±13.04	0.002**
1000	Left	12.35±11.89	14.08±14.46	20.63±15.83	19.17±13.93	0.124
	Right	12.94±11.62	13.64±13.55	22.50±19.89	22.50±14.05	0.028*
2000	Left	14.56±14.05	16.78±17.08	23.54±17.72	20.00±12.65	0.204
	Right	13.68±12.02	15.39±15.44	26.25±19.80	20.00±14.14	0.013*
4000	Left	16.03±15.70	17.11±19.92	22.71±19.17	29.17±16.56	0.25
	Right	14.56±14.69	16.18±19.63	29.58±27.18	30.83±19.85	0.0097**
8000	Left	18.82±18.30	17.96±21.36	26.25±25.16	39.17±21.78	0.06
	Right	21.76±17.96	17.76±20.93	31.04±28.28	41.67±28.40	0.012*

*Note: SNHL: Sensorineural hearing loss, SD: Standard deviation and Hz: Hertz * and ** indicates statistically significant, P values of <0.05 and <0.01*

(hypertension and atherosclerosis). According to the Dadhich et al., SNHL prevalence increases with age among DM patients that accorded with our results, which showed that elderly adult patients (41-50 years) had significantly increased incidences of SNHL with p value of 1.79E-08.[11] Furthermore, such hearing loss still persists after senile deafness correction.[26] We used linear model to check the confounding effect of age on the relationship of FBS and hearing loss, which showed a strong association between age and FBS (p value = 0.00934), indicating that age has a strong relation with FBS. However, no such relation was observed between FBS, age and SNHL patients with DM due to unequal distribution of samples along the age groups. Therefore, conclusion could not be drawn that SNHL was due to either age or DM.

Another association probed was between SNHL and disease duration, i.e. duration of DM. We found that significant association existed between the duration of DM and SNHL (p value: 4.41E-06) and our results were in accordance with findings of Srinivas et al[12] and Bhasker et al.[27] In contrast, Harekare et al reported existence of insignificant association between duration of DM and SNHL (P value: 0.29).[25] The increased hearing threshold may be owing to the neurological and microvascular complications associated with DM that results in either microangiopathy or neuropathy.[28] These changes can be evident from the autopsy of DM patients which showed internal auditory artery thickening, spiral ganglion atrophy or cranial nerve degeneration, especially 8th cranial nerve.[29] We also found that there was moderate correlation between duration of diabetes and SNHL (r value=0.41), similar to the Pemmaiah & Srinivas;[30]

however, they reported significant correlation between SNHL and disease duration at two different frequencies, namely 2000Hz and 4000Hz with r values of 0.561 and 0.727, respectively.

In the present study, the glycaemic status of the patients, i.e. only PPBG levels were significantly associated with the SNHL (Pvalue:0.02) and accorded with the findings of Harkare et al.[25] They reported 100% incidence of SNHL among patients having blood glucose level 301 mg/dL. We found a slight positive correlation between SNHL and blood glucose levels (FBG with r value=0.14 and PPBG with r value=0.2), however, insignificant association were noted with FBG levels.

We also studied the comparative account between SNHL and FBG with respect to low-, mid- and high-frequencies that demonstrated the significant SNHL among all the frequencies and in accordance with findings of Ren et al.[5] and Weng et al.[6] However, Ren et al.[5] reported only low and high-frequency SNHL whereas Weng et al.[6] noticed low- and mid-frequency loss in DM patients with sudden SNHL. Another variable which was tested for association was family history of DM that was found to be significantly associated with SNHL (P value: 0.004) which is in accordance with the results of Bhavitha and Simha.16 We observed 46.67% SNHL prevalence among DM patients with positive family history, however, they reported a significant higher prevalence (58.70%) of SNHL with DM family history with p value of 0.002. A genetic study was done by Moteki et al., where they pointed out that there is coexistence of mutations, i.e., 3243 A>G (mitochondrial) and P2X2 gene mutations, which associated with DM and SNHL, respectively.[15]

Many researchers highlighted the association of different variables with SNHL,[11-14,32] however, few studies assessed the family history of the patients.[15,16] Therefore, the present study has focused on the association of SHNL with family history of DM among adult population along with age, duration of DM and glycaemic status. The present study concluded that all these parameters were significantly associated with SNHL among DM patients and majority of the patients suffered from B/L minimal SNHL only. However, the limitation of the present study is the non-age-matched sample. Hence, the future studies are recommended using equal sample distribution with age matched DM and normal control samples to assess the SNHL.

References

1. International Diabetes Federation. IDF Diabetic Atlas 8th Edition. [Homepage on the internet], [cited 2019 Jul 25]. Available from: https://www.idf.org/e-library/epidemiology-research/diabetes-atlas/134-idf-diabetes-atlas-8th-edition.html.
2. Sugimoto S, Teranishi M, Fukunaga Y, Yoshida T, Sugiura S, Uchida Y, et al. Contributing factors to hearing of diabetic patients in an in-hospital education program. Acta Otolaryngol. 2013;133:1165-72
3. Oh IH, Lee JH, Park DC, Kim M, Chung JH, Kim SH, et al. Hearing loss as a function of aging and diabetes mellitus: a cross sectional study. PLoS One 2014; 9:e116161
4. Nelson EG, Hinojosa R. Presbycusis: a human temporal bone study of individuals with flat audiometric patterns of hearing loss using a new method to quantify stria vascularis volume. Laryngoscope 2003; 113:1672-86
5. Ren H, Wang Z, Mao Z, Zhang P, Wang C, Liu A, et al. Hearing Loss in Type 2 Diabetes in Association with Diabetic Neuropathy. Arch Med Sci. 2017; 48:631-7
6. Weng SF, Chen YS, Hsu CJ, Tseng FY. Clinical features of sudden sensorineural hearing loss in diabetic patients. Laryngoscope 2005; 115:1676-80
7. Horikawa C, Kodama S, Tanaka S, Fujihara K, Hirasawa R, Yachi Y, et al. Diabetes and risk of hearing impairment in adults: a meta-analysis. J Clin Endocrinol Metab. 2013;98:51-8
8. Frisina ST, Mapes F, Kim S, Frisina DR, Frisina RD. Characterization of hearing loss in aged type II diabetics. Hear Res. 2006; 211:103-13
9. Fukushima H, Cureoglu S, Schachern PA, Paparella MM, Harada T, Oktay MF. Effects of type 2 diabetes mellitus on cochlear structure in humans. Arch Otolaryngol Head Neck Surg. 2006;132:934-8
10. Xipeng L, Ruiyu L, Meng L, Yanzhuo Z, Kaosan G, Liping W. Effects of diabetes on hearing and cochlear structures. J Otolaryngol. 2013; 8:82-7
11. Dadhich S, Jha SG, Sinha V, Samanth TU. A prospective, observational study of incidence of sensory neural hearing loss in diabetes mellitus patients. Indian J Otolaryngol. 2018; 24:80-2
12. Srinivas CV, Shyamala V, Kumar BS. Clinical study to evaluate the association between sensorineural hearing loss and diabetes mellitus in poorly controlled patients whose HbA1c> 8. Indian J Otolaryngol Head Neck Surg. 2016; 68:191-5
13. Hwang JH, Wu CC, Hsu CJ, Liu TC, Yang WS. Association of central obesity with the severity and audiometric configurations of age-related hearing impairment. Obesity.2009; 17:1796-

1801. doi:10.1038/oby.2009.66

14. Parmar SM, Khare P, Chaudhary M. Evaluation of effects of diabetes mellitus type 2 and hyperlipidemia on hearing. Indian J Otol. 2017; 23:155-61
15. Moteki H, Azaiez H, Booth KT, Hattori M, Sato A, Sato Y, et al. Hearing loss caused by a P2RX2 mutation identified in a MELAS family with a coexisting mitochondrial 3243AG mutation. Ann Otol Rhinol Laryngol. 2015;124(1_suppl):177S-178S
16. Bhavitha R, Simha V. Clinical Evaluation of Sensoneural Hearing Loss in Pateints with Diabetes Mellitus. Scholars Journal of Applied Medical Sciences 2018; 6: 2950-4
17. Shargorodsky J, Curhan SG, Eavey R, Curhan GC. A prospective study of cardiovascular risk factors and incident hearing loss in men. Laryngoscope 2010; 120:1887-91.
18. Hinchcliffe R. Recommended procedure for Rinne and Weber tuning fork tests. British Journal of Audiology 1988; 22:153-6
19. Bhat KV, Naseeruddin K. Combined tuning fork tests in hearing loss: explorative clinical study of the patterns. J Otolaryngol. 2004; 33:227-34
20. Wahid N, Attia, M. Weber Test. In StatPearls[Internet]. StatPearls Publishing,[cited 2019 Jul 25]. Available from:https://www.ncbi.nlm.nih.gov/books/NBK526135/
21. National Health and Examination survey. [Internet]. Audiometry procedures manual, 2003. [Cited 2019 Jul 25]. Available from: https://www.cdc.gov/nchs/data/nhanes/nhanes_03_04/AU.pdf.
22. American Speech-Language-Hearing Association. Guidelines for manual pure-tone threshold audiometry [Guidelines]. [Homepage on the internet]. [cited 2019 Jul 25]. Available from from https://www.asha.org/policy/gl2005-00014.htm
23. Clark JG. Uses and abuses of hearing loss classification. Asha1981;23:493-500
24. Rajamani S, Senniappan S, Radhakrishnan S. Prevalence and factors influencing sensorineural hearing loss among type II diabetes mellitus patients. International Journal of Advances in Medicine. 2018; 5:732-7
25. Harkare VV, Deosthale NV, Khadakkar SP, Dhoke PR, Dhote KS, Gupta A. SNHL in Diabetics: A Prospective Study. People's Journal of Scientific Research 2014; 7:38-42
26. Axelsson A, Sigroth K, Vertes D. Hearing in diabetics. Acta Otolaryngol Suppl. 1978; 356:1-23
27. Bhaskar KN, Chalihadan S, Vaswani R, Abdul Rehaman CP. Clinical and audiometric assessment of hearing loss in diabetes mellitus. International Journal of Scientific Study 2014; 2:1-4
28. Bainbridge KE, Hoffman HJ, Cowie CC. Diabetes and hearing impairment in the United States: audiometric evidence from the National Health and Nutrition Examination Survey, 1999 to 2004. Ann Intern Med. 2008; 149:1-10
29. Cullen JR, Cinnamond M. Hearing loss in diabetics. J Laryngol Otol. 1993; 107:179-82
30. Pemmaiah KD, Srinivas DR. Hearing loss in diabetes mellitus. International Journal of Collaborative Research on Internal Medicine. 2011;3:725-731.

Conventional versus Flap Tracheostomy: Operative Simplicity, Stomal Care, Complications

https://doi.org/10.47210/bjohns.2020.v28i2.328

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ABSTRACT

Introduction

Tracheostomy is a life saving procedure and its operative principle has withstood the test of time although the operative techniques have evolved. Inferiorly based flap on the anterior tracheal wall (Bjork flap) was demonstrated in 1952. The present study aims to compare conventional tracheostomy with flap tracheostomy.

Materials and Methods

A Randomized Control Trial was conducted in a tertiary care teaching medical institute comparing conventionally tracheostomised patients (Group A) with the group where flap based method was followed (Group B).

Results

The study population was comprised of 110 patients with equitable distribution in the groups. Per-operative time to establish an airway was measured and statistically correlated. 9.26% patients of Group A had difficult tube change, with none in Group B. Ease of stomal care by the patient and family members (Visual Analogue Scale), depicted it to be "very easy" in 76.36% of the patients in Group B and 16.36% in Group A. Stomal healing in first week was proper in 61.82% of patients in Group A and 80% of the patients in Group B. Tracheo-cutaneous fistula (TCF) developed in 18.18% of the adult and geriatric patients of Group A undergoing stomal closure. None of the patients in Group B had TCF following stomal closure.

Conclusion

Establishing Bjork flap tracheostomy is equally quick making the process suited for emergency situations. Flap based tracheostomy has early stomal maturation so, care of the stoma by health assistants and family members is easy.

Keywords

Tracheostomy; Surgical Flap, Anterior Tracheal Wall

Tracheostomy has been a life saving procedure practiced since time immemorial.[1] Chevalier Jackson in the twentieth century defined the principles of the operation.[2] Since then the indications have evolved. So has evolved the operative techniques sticking to the same operative principles. Literature has documented procedures ranging from making a surgical opening in the trachea via an incision on the tracheal wall (tracheotomy), excision of portion of the anterior wall cartilage (tracheostomy) to facilitate the stoma, flaps of the tracheal wall for ostomy to percutaneous dilatation methods under endoscopic guidance. Amongst the various flaps of the anterior tracheal wall that has been advocated, inferiorly based tracheal flap demonstrated by Bjork in 1952 has stood the test of time.[3] Specific complications have been attributed to Bjork tracheostomy though the same has been a matter of debate.[4]

The present study aims to determine the operative simplicity in establishing a flap tracheostomy as compared to conventional method. Assess the post operative ease of stomal care delivered by trained

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nurses and family members. Evaluate compliance of Bjork Flap Tracheostomy with respect to conventional Tracheostomy.

Materials and Methods

A Randomized Control Trial was conducted in a tertiary care teaching medical institute of Kolkata, West Bengal, from August 2014 to July 2016. Patients requiring tracheostomy for establishing the airway were considered as the universe. Among them, those patients undergoing tracheostomy in a fixed surgical unit of the institute under the same senior surgeon were only included in the study. The study population were divided into two groups (i.e. Group A and Group B) by Block Randomisation with a Block of 4. Single blinding was done where the patient consented for both the procedures but didn't know, to which group they will be allotted to.

The study population was comprised of 110 patients (n=110), 55 were allotted in either group. The age range of the study population was 4 to 75 years.

Patients undergoing tracheostomy with neoplasm on the tracheal wall, intra-luminal growth, deviated trachea, prior tracheostomised state were excluded from the study.

In Group A, the adult patients underwent conventional tracheostomy. Following a midline vertical skin incision ranging from the level of the lower border of the cricoids cartilage to the suprasternal notch, soft tissue was dissected and strap muscles retracted to expose the trachea. Thyroid isthmus was retracted if required. Anterior wall of the tracheal cartilage was excised ranging from the level of the 3rd tracheal ring to the 4th tracheal ring. In prior intubated patients the endotracheal tube was removed before high volume low pressure cuffed Portex™ tracheostomy tube was placed. In paediatric patients undergoing tracheotomy, no tracheal cartilage was excised. A vertical midline incision was placed on the anterior tracheal wall after placing stay suture on the either sides. Age appropriate proper size tracheostomy tube was placed across the incision. Local dressing was done as per protocol.

In Group B, Bjork flap tracheostomy was done. Vertical midline incision was placed similar to conventional method. Further dissection was done to expose the trachea. An inferiorly based flap (inverted 'U' shapped) was fashioned on the anterior tracheal wall. The superior limit of the flap was an inter cartilaginous horizontal incision between the second and the third tracheal rings. On either side, the horizontal incision was turned down cutting through the third and the fourth rings, hinging across a base of around 12-15 mm. In paediatric patients the similar inferior based anterior tracheal flap was made with base width of around 5mm.[5] The upper lip of the flap was secured to the lower border of the skin incision with 2-0 synthetic, monofilament, non-absorbable polypropylene suture. Age appropriate proper size tracheostomy tube as used in group A was placed. Local dressing was done.

Per operatively parameters such as, ease of the procedure, time required to establish airway, difficulties encountered and complications resulted were noted.

The patients were followed up for one year. Institution based protocol for tracheostomy care was observed. First tube change was performed at 48hours. Next change at 7 days or as and when required. During the follow-up period, documentation was made as to the ease of stomal care when done by the trained nursing personnel during the first tube change and subsequently by the patients or family members. A Visual Analogue Scale was utilised to evaluate the same. Post operative complications during the follow-up period were noted. All patients including those undergoing stomal closure went through flexible endoscopic evaluation of trachea during the closure procedure and subsequently at 4 months interval to exclude tracheal stenosis. For conventional tracheostomy closure (Group A), the stoma was refashioned. Adjacent neck muscle layer was dissected, isolated and closed in midline followed by midline skin closure. In Group B, the inferiorly based flap was released to ensure closure. Tracheal cartilage flap was repositioned but not sutured to the tracheal wall. The muscle and the skin were closed in midline separately.

Results

The demographic analysis of the study population

Table I: Distribution of study population in various age groups

	GROUP A	GROUP B
PEDIATRIC (4-12yrs)	1	3
ADULT (13-60yrs)	27	25
GERIATRIC (61-75yrs)	27	27

revealed, 72.72% of the study population was male and the rest female. Both the genders were equally distributed in either group. Paediatric (4-12years), adult (13-60years) and geriatric population (60-75years) in Group A was 1.81%, 49.09% and 49.09% respectively. The distribution of ages in Group B was 5.45%, 45.45% and 49.09% respectively. (Table I)

In Group A, planned tracheostomy was done in 23.63% patients and the rest underwent emergency tracheostomy. In Group B, 30.91% underwent planned surgery and the rest emergency intervention. In Group A and B, 7 and 12 patients respectively had received radiation therapy prior to tracheostomy. (Fig. 1)

Per-operative time required to establish an airway was measured in both the groups. It was classified as 'under 4 minutes', '4-8 minutes' and 'more than 8 minutes'. In Group A, airway was established in 4 patients under 4minutes, 44 patients in 4-8 minutes and 7 patients required more than 8 minutes. In Group B, 5 patients had established airway in 4 minutes, 49 patients in 4-8 minutes and 1 required more than 8 minutes. Statistical correlation was done amongst Group A and B. Chi square value was found to be 4.879. p value of 0.087 (>0.05, i.e. not significant).

During the post operative period the first tracheostomy tube change is done by trained nursing personnel along with an ENT surgeon 2 days after the surgery. Any prior tube change is done solely by team of surgeons. The first protocol based tube change at 48 hours post operative was termed difficult if the nursing personnel required active intervention by the ENT surgeon during such procedure. In Group A, the single paediatric patient who underwent tracheotomy had a difficult first tube change. 5 adult patients (9.26%) of Group A had difficult tube change. None of the patients in Group B required active intervention of an ENT surgeon during the first tube

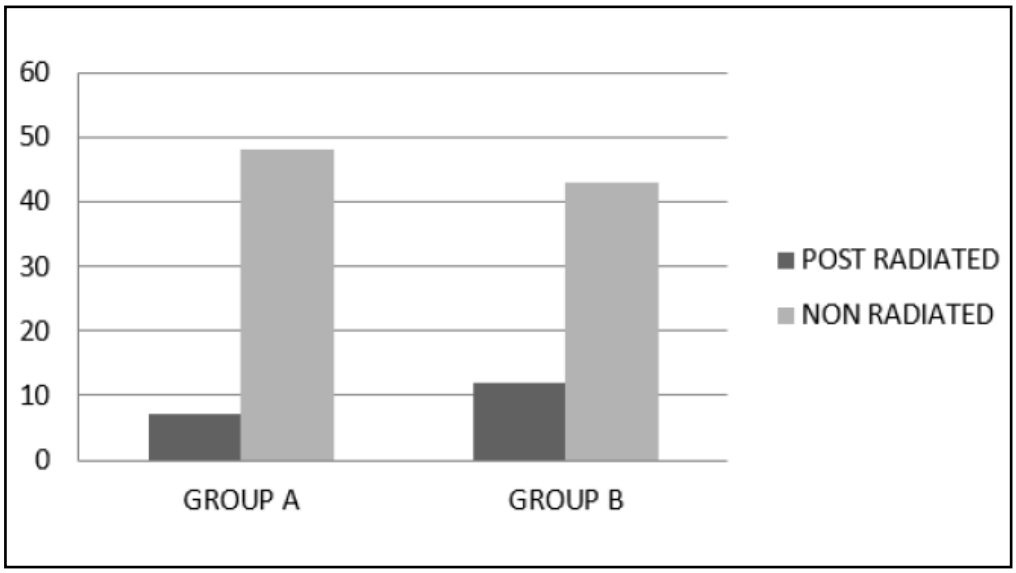

Fig. 1. Distribution of patients according to pre operative radiation exposure

Table II: Distribution of patients in either group in matter of stomal care ease by patients and family members, assessed by Visual Analogue Scale score

	GROUP A	GROUP B	TOTAL
VAS 0	9 (16.363%)	42 (76.363%)	51
VAS 1 to 10	46	13	59
TOTAL	55	55	110

Chi - 39.8106
P - <0.00001 Significant difference

change.

Further the ease of tracheostomy care by the patients and family members was assessed by a Visual Analogue Scale, ranging from "0"- Very easy to "10"- Very difficult. In Group A 16.36% of the patients reported stomal care to be "very easy". 76.36% of the patients in Group B reported it to be "very easy". Statistical correlation showed Chi square value of 39.81. p value of <0.00001. (<0.05= significant difference). (Table II)

Stomal healing within first week was proper in 61.82% of patients in Group A and 80% of the patients in Group B.

Closure of tracheal stoma was tried in all the paediatric patients belonging to both the groups. In adult and geriatric population it was tried in 40.74% patients in Group A and 55.77% patients in Group B. Average time of stomal closure was 2 weeks following tracheostomy. In Group A, the only child undergoing tracheotomy had resulted Tracheo-Cutaneous Fistula (TCF) following stomal closure. 18.18% of the adult and geriatric patients of Group A undergoing stomal closure had TCF. None of the patients in Group B had TCF following stomal closure.

As mentioned, tracheal stenosis was evaluated in all the patients, specifically those undergoing stomal closure. Stenosis was evaluated at the level of the stoma and at the level of the tip of the tracheostomy tube. In either group no cases of tracheal stenosis was found during the follow up period.

Other complications encountered both per operatively and post operatively was evaluated. Primary haemorrhage was found in 5 patients of Group A and 4 in Group B. Secondary haemorrhage, 2 in Group A and nil in Group B. 4 patients had surgical emphysema in Group A and only 1 patient in Group B. Dysphagia was complained by 2 patients in Group A and 1 patient in Group B. Peristomal granulation, stomal stenosis, trachea-esophageal fistula and trachea-cutaneous fistula was found in 4, 6, 1 and 5 patients respectively in Group A. None of these complications were present in any of the patients of Group B. Tracheostomy related death didn't occur in either group. (Fig. 2)

Discussion

Tracheostomy is a common procedure in an ENT operating room and intensive care units. The planned tracheostomies were performed prior to major head neck and maxilla facial surgeries. Few patients of laryngeal carcinoma put to radiotherapy were tracheostomised considering impending airway obstruction following radiation induced oedema. Patients requiring prolong mechanical ventilation were also tracheostomised. All the paediatric patients in the study population underwent tracheostomy as an indication for prolong mechanical ventilation. Emergency tracheostomies were performed in cases of laryngeal neoplasm with stridor, maxillofacial and airway trauma, endolaryngeal foreign body.

Midline vertical skin incision has been used in both the groups undergoing tracheostomy. This elaborate incision offers better access, adequate visualisation of deeper structures, prevents injury to structures lateral to the trachea e.g. the recurrent laryngeal nerves. Cosmetically disfiguring scar is not an issue with an adequate but small vertical incision. This incision renders the procedure simple for residents in training. Malata et al in their audit of Bjork flap tracheostomies in

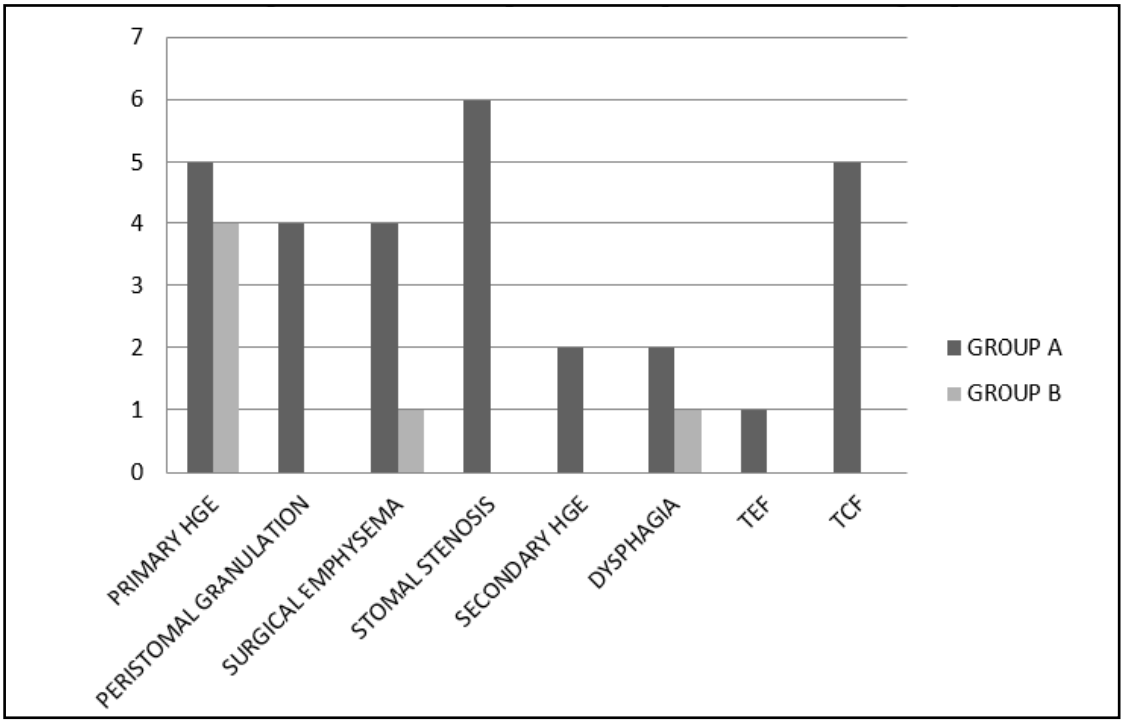

Fig. 2. Per Operative and Post Operative complications faced in either group

head and neck plastic surgery had similar opinion with regards to vertical skin incision access.[6]

There lies no statistical difference in the time required to access the airway in either of the groups (p value = 0.087). So it can be opined that Bjork flap tracheostomy can be safely undertaken in emergency situations. Weissler et al advocated Bjork flap based tracheostomy via a vertical skin incision in emergency situation.[7]

Flap tracheostomy advocates a flap of anterior tracheal in place of excising anterior tracheal wall window. The flap is anchored to the inferior margin of the soft tissue incision by a non absorbable suture. This ensures that the flap does not mobilise and fall back in place occluding the stoma. The tract thus created has early maturity. Its inferior surface contributed by the endotracheal mucosal lining over the flap ensures proper curved smooth passage. This enables easy tube placement. As has been the protocol in our institute the first tube change at 48 hours post operative period is done by a trained nursing personnel along with an ENT surgeon. In our study the nursing personnel could change the tube with ease without requiring any assistance from the surgeon in all the adult patients belonging to the Flap tracheostmy group i.e. Group B. But in 9.26% of the adult population in Group A has been reported to have poorly matured stoma at 48 hours and so tube changing was difficult.

Active intervention of the ENT surgeon was required to ensure tracheostomy tube placement. The trained staff had opinion that the smooth inferior wall of the matured stoma ensured that there was no false passage of the tube. The tube glided into the trachea following the endotracheal mucosal path. Considering paediatric tracheotomy (only a small incision over the anterior tracheal wall in place of excision of anterior tracheal window) there is every possibility of collapse of the tracheotomy, poor stomal maturation; flap based tracheostomy ensures preservation of tracheal wall along with prevention of the aforesaid complications. None of the paediatric patients in Group B required intervention by the surgeon during the first tube change. In Group A the single tracheostomised child required an intervention by the surgeon as the trained staff failed to cannulate the stoma on her own.

Patients discharged with tracheostomy tube, has not only them but also their family members undergo a basic training programme for home based tracheostomy tube care and acute emergency management, before they are discharged. The family members are encouraged to care for the tracheostoma even when the patient is in hospital. This ensures supervised intervention and does away with any fear associated with home based management of tracheostomised patients. At a follow up period of one month the family members or the patients caring for the stoma were put through an assessment of the ease of care based on a Visual Analogue Scale. The '0' end denoted 'Very easy' to "10"- Very difficult. In Group A 16.36% of the patients reported stomal care to be "very easy". 76.36% of the patients in Group B reported it to be "very easy". Statistical correlation showed Chi square value of 39.81. p value of <0.00001. (<0.05= significant difference).

So the stomal care is significantly easy in Group B as compared to Group A. Problems faced by patients or their family members in Group A include- small stoma with difficulty to put in the canula, reddish tissue growth around the opening, occasional bleeding from the tissue when the tube was forced against it, passage of the tube in place other than the airway that required replacement of the tube, need to call the local health care assistant to help place the tube. In Group B patients and family members reported easy tube placement even if accidental decannulation occurred. Heffner et al. reports that the Bjork flap provides a bridge of tracheal conduit that guides the tracheostomy tube and avoids creation of false passage and is therefore safer to use.[8]

Stomal healing with proper tract formation within first week is 80% in Group B as compared to 61.82% in Group A. Flap based procedure with better mucosalisation of the tract ensures early maturity of the stoma.

Complications are an important part of surgical intervention. Surgeons are faced with a larger burden of complications in emergency operative procedures like urgent airway management. In this study population, per operative haemorrhage has been an important complication. 5 and 4 patients in Group A and B respectively had primary haemorrhage. This can be attributed to manner of surgical dissection, anatomical alterations in neck, coagulopathies the patient might suffer from, the disease condition itself, or prior radiation therapy rendering the field fibrotic and difficult to work on.

Tracheo stomal closure was tried in all the paediatric patients belonging to both the groups. In adult and geriatric population it was tried in 40.74% patients in Group A and 55.77% patients in Group B. Average time of stomal closure was 2 weeks following tracheostomy. In Group A, the only child undergoing tracheotomy had resulted Tracheo-Cutaneous Fistula (TCF) following stomal closure. No child developed TCF in Group B. Due to small sample size of paediatric population, statistically significant difference amongst paediatric patients in the Groups can't be commented upon. In a study by Colman et al of 172 paediatric patients who underwent tracheostomy during a period of 4 years, no relationship between stomal maturation (as in flap tracheostomy in our study) and tracheostomy related complication- TCF.[9] 18.18% of the adult and geriatric patients of Group A undergoing stomal closure had TCF. None of the patients in Group B had TCF following stomal closure. So there is obvious significant difference between the 2 groups. This can be attributed to the proper repositioning of the vascularised anterior tracheal wall flap back in position following decannulation ensuring prevention of TCF. According to Joseph et al age at tracheostomy and duration of tracheostomy are important determing factors for formation of Tacheo-cutaneous fistula.[10]

Tracheal steonsis at the soma, tube cuff and at the site of tubal tip is a dreaded complication of tracheostomy. Arola et al demonstrated 0-21% incidence of tracheal stenosis.[11] All patients in the study population underwent evaluation of Tracheal stenosis including those who were decannulated. The evaluation was done with flexible endoscopes. In either group no cases of tracheal stenosis was found. No statistical correlation is thus impossible between the groups. Proper surgical method compounded with adequate flap width and length in Group B seems to have prevented the complication. Tracheostomy tube of adequate length, curvature and high volume low pressure cuff has also prevented the injury to tracheal mucosa along the length of the tube at the pressure points. Flap tracheostomy was studied in 25 adult canines by Lulenski et al. They used inverted U

flap. Histological examinations were made, and tracheal dimensions were calculated on sections through the stomal area. Tracheal dimensions were found to be maintained. Additional support for flap tracheostomy has been found from this animal experiment.[12] Stomal stenosis had been a notorious complication in Group A although none has been reported in Flap Tracheostomy. The reason behind the finding is easy and early maturation of the tract in Group B. Inferior lip ensures easy tube placement and less chances of stoma injury in Group B as compared to A. Preistomal granulation chances are less in Group B. Thus granulations less often occlude the flap based matured stoma. Healing of immature stoma by fibrosis, traumatisation of peristomal tissue, repeated injury to the granulation during tube placement further accentuates the risk of stomal stenosis.

Complications that occurred equivocally in both the groups include- surgical emphysema and dysphagia. Complications that occurred only in Group A include peristomal granulation, secondary haemorrhage, trachea-esophageal fistula. There were no tracheostomy related deaths in either group.

Conclusion

Midline vertical soft tissue approach to any format of tracheostomy renders the process simple and is not very cosmetically disfiguring in the long run. Establishment of the airway in Bjork flap tracheostomy is equally quick making the process suited for emergency situations. Flap based tracheostomy has early stomal maturation so, care of the stoma by health assistants and family members is easy. Although there are various attributes to the formation of tracheo-cutaneous fistula, repositioning of the vascularised flap of inferior tracheal wall reduces such complications during decannulation in case of Bjork tracheostomy. Stomal stenosis which is notorious complication of conventional method is found to be alleviated by flap tracheostomy. Rate of occurrence of other complications like peristomal granulation, surgical emphysema, and secondary haemorrhage is reduced in flap based method.

References

1. Frost EAM. Tracing the tracheostomy. Ann Otolaryngol. 1976; 85: 618-24
2. Jackson C. Tracheostomy. Laryngoscope 1909; 19: 285-90
3. Bjork VO, Engstorm CG. The treatment of ventilator insufficiency after pulmonary resection with tracheostomy and prolong artificial ventilation. J Thorasic Surg. 1955; 30:356-67
4. Kinley CE. A Technique of Tracheostomy. Can Med Assoc J. 1965; 92(2): 79–81
5. Dukes HM. Tracheostomy. Thorax 1970; 25(5): 573-6
6. Malata CM, Foo ITH, Simpson H, Batchelor AG. An audit of Bjork flap tracheostomies in head and neck plastic surgery. British J Oral Maxillofac Surg. 1996; 34: 42-6
7. Weissler MC, Couch ME. Tracheostomy and intubation. Byron. Bailey Head and Neck Sugery Otolaryngology 2006; 3: 677-89
8. Heffner JE, Miller KS, Sahn SA. Tracheostomy in the intensive care unit. Part 2: Complications. Chest 1986; 90(3): 430-6
9. Colman KL, Mandell DL, Simons JP. Impact of stoma maturation on paediatric tracheostomy related complications. Archives of Otolaryngology- Head Neck Surg. 2010; 136(5): 471-4
10. Joseph HT, Jani P, Preece JM, Bailey CM, Evans JN. Paediatric tracheostomy: persistant trachea-cutaneous fistula following decannulation. Inter J Ped Otorhinolaryngol. 1991; 22(3): 231-6
11. Arola MK. Tracheostomy and its complications. A retrospective study of 794 tracheostomised patients. Ann Chir Gyneocol. 1981; 70: 96-106
12. Lulenski GC, Batsakis JG. Management of the flap tracheostomy: An experimental study. Arch of Otolaryngol. 1979; 105(5): 260-3.

Cochlear Otosclerosis: An Undiagnosed Cause of Progressive Sensorineural Hearing Loss

https://doi.org/10.47210/bjohns.2020.v28i2.329

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ABSTRACT

Introduction

Otosclerosis presents as conductive or mixed hearing loss depending upon the stage of the disease. Isolated sensorineural hearing loss though known to occur has been rarely reported. Pure cochlear otosclerosis presenting as sensorineural hearing loss should be considered as a differential diagnosis in young patients presenting with progressive hearing loss. The aim of this article is to note the prevalence of cochlear otosclerosis in young patients presenting with progressive sensorineural hearing loss.

Materials and Methods

Retrospective chart analysis of 19 patients who presented with progressive SNHL with unknown etiology was done.

Results

Otosclerosis involving the pericochlear region was diagnosed in 27 ears which was identified by high resolution CT scan of temporal bone. In four ears, in addition, there was demineralization just anterior to oval window without clinical evidence of stapes fixation.

Conclusion

In young patients with progressive SNHL, high resolution CT scan of temporal bone should be done to identify progressive cochlear otosclerosis. Medical management should be initiated in these patients to halt the progression of disease.

Keywords

Otosclerosis; Hearing Loss, Sensorineural; Tomography, X-Ray Computed

Otosclerosis (OS) is a progressive hereditary disease of collagen which involves foci of bone resorption along with new bone formation in the enchondral bone of the otic capsule. During the active stage a reddish blush may be visible on the promontory and seen through an intact tympanic membrane known as the Schwartze sign.[1]

Fixation of the stapes due to OS focus results in conductive hearing loss (CHL). Expansion of the focus causes involvement of the cochlear endosteum and it manifests as sensorineural hearing loss (SNHL). At this stage the patient may present with mixed hearing loss.[1] Guild was the first to differentiate between clinical OS and histologic OS.[2] In clinical OS (fenestral) the disease process begins just anterior to the oval window in an area known as fissula ante fenestrum, resulting in fixation of the stapes and clinically presents as CHL. It has a prevalence rate of 0.3%.[3] In histologic OS one or more foci are located in the bony labyrinth without involvement of cochlear endosteal layer or stapes fixation.[4] It is an asymptomatic condition and is only detected in serial sectioning of the temporal bones during autopsies. About 12-15% bones with histologic OS have demonstrated stapedial fixation.[3] Cochlear OS has also been described in literature when the histologic focus involves the cochlear endosteum and results in atrophy of sensory, neural and supporting tissues of the cochlea

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without fixation of the stapes.[5]

According to Shambaugh, otosclerotic focus involving the cochlear endosteum can result in SNHL and this can occur without fixation of stapes.[6] Balle and Linthicum also believed that many of the cases where presbycusis is assumed to be the cause of SNHL are in fact due to cochlear OS without stapes fixation.[7] However, according to Guild, atrophy of neural structures of the cochlea occurs with same frequency in ears with cochlear otosclerosis as in ears without it.[2] Thus it is debatable whether cochlear otosclerosis presenting as pure SNHL exists as a clinical entity.

Various other conditions can present as progressive SNHL. These include post meningitis cochlear ossification, post-concussion, CNS syphilis, Meniere's disease, noise exposure and endocrinopathy.[8] Early identification of any possible cause of progressive SNHL helps in initiation of treatment. High resolution computed tomography (HRCT) scan can be used to make a definitive diagnosis of cochlear OS when it is suspected to be the cause of progressive SNHL. The OS lesion can be identified based on the variation in density from the normal otic capsule or altered contour, double ring effect due to demineralization or new bone formation in the bony labyrinth.[9]

The aim of this article is to emphasize that cochlear OS should be considered as a possible cause of progressive SNHL, especially in young subjects. The patient should be evaluated by HRCT of the temporal bone. Early detection would help institute appropriate medical therapy which may halt the progression of disease.

Materials and Methods

Retrospective chart analysis of patients presenting with history of progressive hearing loss in the otolaryngology outpatient department of our tertiary care hospital from 2012-16 was done. Patients with obvious cause of hearing loss such as Meniere's disease, chronic ear disease, history of exposure to loud sounds or to ototoxic medication and presbycusis were excluded from the study. Data pertaining to otoscopy, tuning fork tests, pure tone audiometry (PTA) and impedance audiometry was extracted from the medical records. The pure tone threshold average at 500 Hz, 1KHz, 2KHz, 4KHz for bone conduction (BC) and air conduction (AC) was calculated. Routine blood investigations such as complete blood counts, erythrocyte sedimentation rate (ESR), C reactive protein (CRP), VDRL, fasting and post prandial blood sugar levels were also analysed to rule out any systemic disease associated with SNHL.

HRCT scan of temporal bones was evaluated to identify any likely cause of progressive SNHL. The multidetector CT scanner with following technical parameters is routinely used to identify cochlear otosclerosis in our institution : Kv- 140, mA-120, slice thickness – 0.625mm, pixels – 0.562:1, rotation time 1 sec, DFOV 25 cm, algorithm – bone +, retro reconstruction 0.31 mm, window width 3000 and window level 500. Coronal reformation is performed following the acquisition of submillimetric (0.625mm) slices in the axial plane. Images are evaluated by optimizing the window width and window level to specifically evaluate areas of demineralization in the middle and inner ear with special attention to cochlear turns, area anterior to footplate of stapes, round window (RW) and internal auditory canal (IAC). In addition, all cases are evaluated for round window obliteration, anatomy of facial nerve canal, jugular bulb foramen, cochlear duct and ossicular fixation. The HRCT scans are reported by single experienced neuroradiologist.

Results

Nineteen (19) patients with history of progressive hearing loss were identified over a period of four years. Twelve patients were male (63.2%) and seven patients were female (36.8%). The mean age of patients was 37±12 years at the time of presentation. The mean age of onset of hearing loss was 31.6±11.9 years. The AB gap was <10dB in all but three ears (patient no 5, 11, 12). Details of all 19 patients is as per Table I. Of the 38 ears evaluated with HRCT scan, a circumferential rim of hypolucency in the cochlear turns was identified in 27 ears (71.1%). In the remaining ears no other pathology was identified. The final diagnosis of cochlear OS in these 27 ears was made on the basis of history, audiometry findings and HRCT. The mean age of patients diagnosed to have cochlear OS was 37.1±11.2 years.

Table I: Demographic profile, radiologic findings and hearing status of all 19 patients

PATIENT NO	AGE (YEARS)/ SEX	HL TYPE	LATERALITY	HRCT FINDINGS	PTA	PTA THRESHOLD AV: AC, L (DBHL)	ABG (R) (DB)	ABG (L) (DB)
1	38/M	SNHL	B/L	B/L PC lucency	68.75	38.75	7.5	3.75
2	35/ F	SNHL	B/L	(L) PC lucency	32.5	57.5	6.25	15
3	27/ M	SNHL	B/L	Normal	61.25	66.25	7.5	10
4	35/ F	SNHL	B/L	B/L PC lucency	71.25	60	5	10
5	20/ M	SNHL	B/L	B/L PC lucency	61.25	56.25	13.75	8.75
6	53/ F	SNHL	R	B/L PC lucency more prominent on (R)	62.15	23.75	1	5
7	49/ M	SNHL	B/L	B/L PC lucency	41.25	60	2.5	5
8	33/ M	SNHL	B/L	B/L PC lucency, B/L	80	75	BC no response at 2 & 4 KHz	BC no response at 2 & 4 KHz
9	70/ F	SNHL	B/L	Normal	70	75	7.5	7.5
10	17/ M	SNHL	B/L	Normal	40	50	1.25	8.75
11	31/ M	(R) mixed HL, (L) SNHL	B/L	B/L PC lucency. B/L	51.25	47.5	12.5	10
12	55/ F	SNHL	B/L	B/L PC lucency	55	58.75	8.75	12.5
13	31/ M	SNHL	B/L	B/L PC lucency	78.75	81.25	7.5	10
14	43/F	SNHL	B/L	Normal	48.75	51.25	7.5	7.5
15.*	52/M	SNHL	B/L	B/L PC lucency	18.75	22.5	3.75	2.5
16	20/M	SNHL	B/L	B/L PC lucency	48.75	51.25	2.5	8.75

Contd. Next Page

Table I: Demographic profile, radiologic findings and hearing status of all 19 patients (Contd.)

PATIENT NO	AGE (YEARS)/ SEX	HL TYPE	LATERALITY	HRCT FINDINGS	PTA	PTA THRESHOLD AV: AC, L (DBHL)	ABG (R) (DB)	ABG (L) (DB)
17	32/M	SNHL	B/L	Normal	30	42.5	2.5	8.75
18	34/M	SNHL	B/L	B/L PC lucency	65	63.75	7.5	5
19	34/F	SNHL	B/L	B/L PC lucency	73.75	52.5	6.25	6.25

M= male, F= female, HL= hearing Loss, L= left, R= right, B/L = Bilateral, SNHL= sensorineural hearing loss, PC= pericochlear, FP= footplate, AC= air conduction, BC= bone conduction, ABG= air-bone gap, dBHL= decibel hearing level.

*** Patient no 15 thresholds at 2-8KHz (R) - 20-40dBHL, (L) - 10-75dBHL (B/L high frequency hearing loss).**

Fig.1A shows the circumferential rim of hypolucency seen in the cochlear turns of patient no 11 on HRCT. Fenestral otosclerosis in addition to a pericochlear focus was also identified in this patient (Fig 1B). This patient had a mixed type of hearing loss (AB Gap 25 dB) at lower frequencies and pure SNHL at other frequencies on (R) side.

Similar HRCT findings were seen in patient no 8, though he had no evidence of CHL. HRCT scan of patient no 6 showed a faint rim of demineralization in cochlear turns on (L) side as well, even though patient had presented with (R) sided SNHL (Fig. 2A,B).

On the contrary in patient no 2, no OS focus was seen radiologically in (R) ear even though SNHL was bilateral (Fig. 3). This patient, however, had a higher degree of hearing loss in the (L) ear (PTA average 57.5 dBHL) compared to (R) ear (PTA average 32.5 dBHL). In none of the ears obliteration of the round window (RW), focus in the IAC ossicular fixation, or enlarged vestibular aqueduct was identified. Fig. 4 shows the normal cochlea of patient no 14. Details of patients who were diagnosed with cochlear OS is mentioned in Table

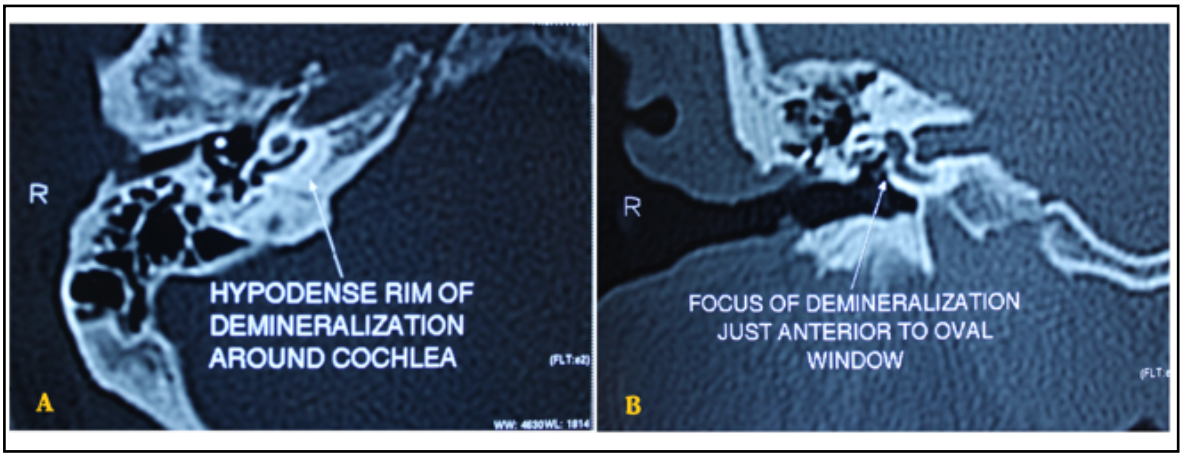

Fig. 1. Axial HRCT image of patient no 11 showing circumferential rim of hypolucency in pericochlear region on (R) side (A). Coronal HRCT image of patient no 11 showing focus of demineralization just anterior to stapes footplate (B).

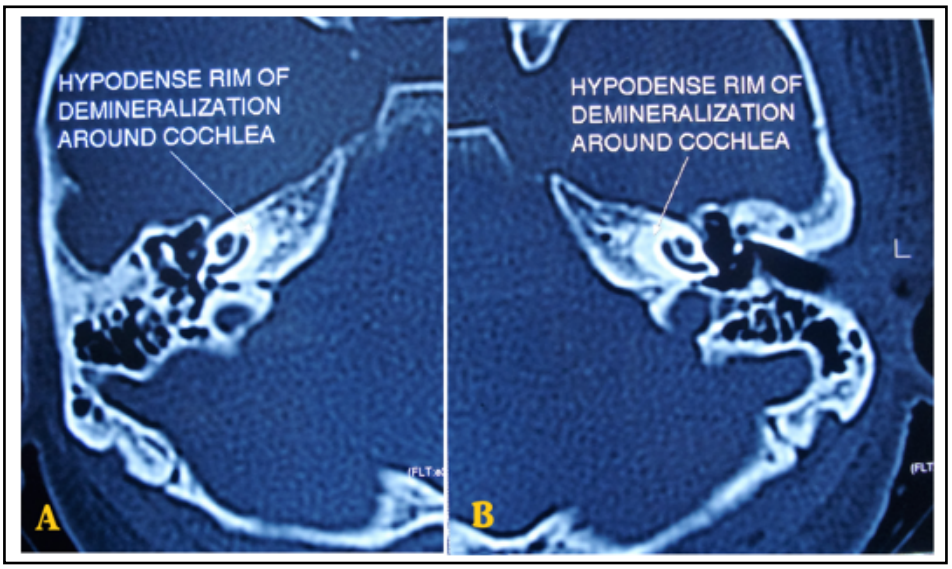

Fig. 2. Axial HRCT image of patient no 6 showing focus of demineralization in pericochlear region on (R) side (A). Axial HRCT image of patient no 6 showing focus of demineralization in pericochlear region on (L) side (B).

II. Most of the patients diagnosed to have cochlear OS had moderate or severe degree of hearing loss (Table III).

Overall, associated symptom in the form of tinnitus was present in 78.9 % ears. Vertigo was present in only three patients (patient no. 14, 15, 19). Blood investigations were negative in all but one (patient no. 10). This patient had elevated inflammatory markers such as ESR and CRP indicative of a possible immune mediated cause of SNHL. This patient had been advised to attend rheumatology outpatient department to rule out any coexisting systemic autoimmune disease.

Patients who were diagnosed with cochlear OS were started on oral sodium fluoride (NaF) in the dose of 20 mg twice daily along with oral calcium and vitamin D3. The dose was increased (20 mg thrice daily) in patient no 15 as he subjectively reported some deterioration of hearing, even though other associated symptoms had

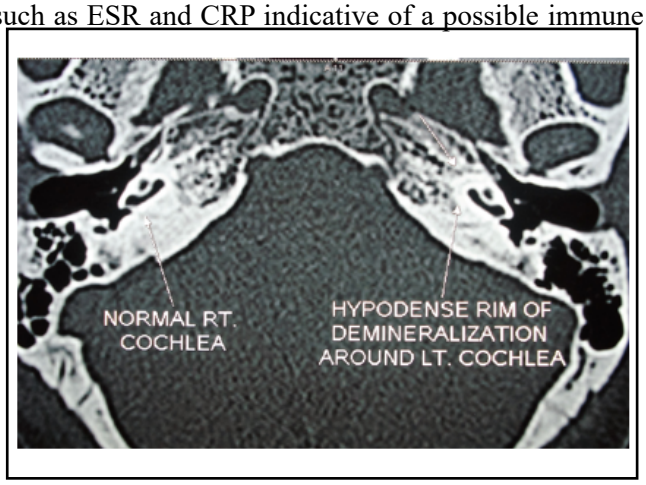

Fig. 3. Axial HRCT image of patient no 2 showing circumferential rim of hypolucency in pericochlear region on (L) side only

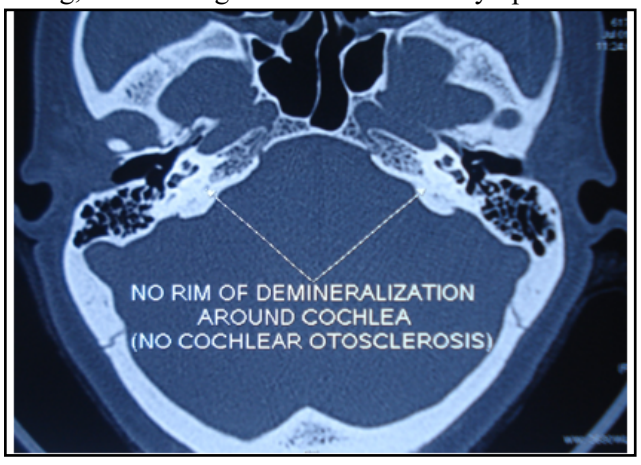

Fig. 4. Axial HRCT image of patient no 14 showing normal cochlea.

Table II: Characteristics of cases of sensorineural hearing loss found to have cochlear otosclerosis.

SERIAL NO.	FEATURE	N=14 CASES (28 EARS)
1	Age at onset (years ± SD)	32.5 ± 11.4
2	Positive family history	2 of 14 subjects (14.3%)
3	History of tinnitus	23 of 28 ears (82.1%)
4	History of vertigo	2 of 14 subjects (14.3%)
5	Schwartze sign	2 of 28 ears (7.1%)
6	Association with pregnancy	1 of 5 female patients

SD= standard deviation

Table III: Degree of hearing in ears diagnosed with cochlear otosclerosis

S.NO	DEGREE OF HEARING	NO OF EARS (N = 27)
1	Normal (0-25)	03*
2	Mild (26-40)	1
3	Moderate (41-60)	12
4	Severe (61-80)	10
5	Profound (>80)	1

**Patient no 06 - (L) ear thresholds were normal even though there was radiologic evidence of cochlear OS. Patient no 15 - pure tone average was normal, elevated thresholds at 2-8KHz.*

*** WHO Grades of hearing impairment*

disappeared. Skeletal X Rays were done which excluded any pre-existing skeletal flourosis in these patients. Duration of therapy, change in subjective symptoms of patients who were started on therapy is as per Table IV.

Record of hearing thresholds after at least six months of NaF therapy was available only for six patients (Fig. 5). The median follow up period was 11 months (range 6 to 16). PTA average thresholds improved in five ears (mean, 10.5 ± 7.27 dB) and seven ears showed slight progression (mean, 4.46 ± 4.07 dB). When comparing mean BC thresholds of these 12 ears pre and post therapy at individual frequencies, mean improvement at 0.5 KHz, 1KHz and 4KHz was -1.25 dB (95% CI, -1.29 to -1.21), -1.25 dB (95% CI, -1.40 to 1.10), -1.67 dB (95% CI, -1.88 to -1.45) respectively. There was deterioration at 2 KHz (mean, 2.5 dB; 95% CI, 2.30 to 2.70).

Discussion

OS is unique to the human temporal bone. In 1735, Valsalva first described bony ankylosis of the stapes to oval window (OW) margins in temporal bone dissection of a man who had been deaf during his lifetime. In 1852, Toynbee found fixation of stapes in 39 bones out of 1659 temporal bone dissections and subsequently considered it to be the commonest cause of deafness. The term otosclerosis, however, was first used by von Troltsch in 1861. The first account of histology of this disease was given by Politzer. It was his work that established OS as a primary disorder of otic capsule and not secondary to middle ear catarrh as was believed at that time.[10]

Various theories describe the causation and pathogenesis of this entity which include metabolic disorders, vascular disease, infection, trauma, developmental disorder, immune mediated causes and viral infection. Exacerbation of symptoms during period of increased hormonal activity such as pregnancy has been documented suggesting a possible hormonal mechanism. It is predominantly a disease of the Caucasian race.[2,3] It is extremely rare in oriental and black races.[1] It is inherited as an autosomal dominant trait with incomplete penetrance. The reported incidence of sporadic cases is about 30%.[1]

The most common site of occurrence of OS is anterior aspect of OW. This results in fixation of the stapes which may be fibrous initially and later becomes bony ankylosis. Other areas of predilection are RW, apical medial wall of cochlea, anterior and posterior wall of

Table IV: Summary of subjective change in symptoms of patients started on sodium flouride therapy.

PATIENT NO	INITIAL SYMPTOMS	DURATION OF THERAPY (MONTHS)	CHANGE IN SYMPTOMS POST TREATMENT
1	HL, tinnitus	6	HL stabilized, no tinnitus, discontinued treatment after six months due to gastric irritation
2	HL, tinnitus	<06	Gastric irritation, Lost to follow up
4	HL	16	(R)Slight deterioration, (L) improved thresholds
5	HL, tinnitus	15	HL stabilized, no tinnitus
6	HL, tinnitus	6	Slight progression of HL, tinnitus persistent
7	HL, tinnitus	<06	Lost to follow up
8	HL, tinnitus	<06	Lost to follow up
11	HL, tinnitus	7	Lost to follow up
12	HL, tinnitus	<06	Lost to follow up
13	HL, tinnitus	6	HL stabilized, no tinnitus
15	HL, tinnitus, discomfort to loud sounds, vertigo	10	Discomfort to loud sounds and vertigo relieved, slight progression of HL
16	HL, tinnitus	12	Improved thresholds, no tinnitus
17	HL	4	Progression of HL, lost to further follow up
18	HL, tinnitus, vertigo	2	Tinnitus stopped, persistence of dizziness after two months, lost to further follow up

HL= Hearing loss

IAC and within the stapedial footplate.[1] Conductive deafness does not occur till the lesion involves the oval window area.[1] Overall, the incidence of conductive deafness due to OS in adult white population is 3 to 10 per 1000.[2] OS is more common in females. Positive family history is found in two third of cases.[1] In our study though, 35.7% of the patients diagnosed to have cochlear OS were females and positive family history of deafness was present only in two subjects.

The exact incidence of various types of OS is not known. Guild has reported a higher proportion of histologic OS (85%) compared to clinical OS (15%) in his series.[2] In another series by Schuknecht and Barber, clinical OS was found in 75%, histologic OS in 25% and cochlear OS only in 3% of the bones studied.[11] The most likely difference as noted by Schuknecht and Barber is that Guild's series included bones from unselected routine autopsy cases whereas their series included bones of otologic interest.

According to Shambaugh, occurrence of progressive SNHL in a patient with family history of OS, presence of Schwartze sign in an ear with pure SNHL, insidious onset of SNHL in early life or pure SNHL documented in one of the ears of a patient already known to have stapes fixation in the contralateral ear, are some of the features suggestive of cochlear OS.6 In our series, the mean age of onset in patients who were diagnosed to have cochlear OS was 32.5±11.4 years and mean age of presentation was 37.1±11.2 years. Only two patients had a first degree relative with early onset of hearing loss (patient no. 1 & 15). Schwartze sign was seen bilaterally only in one patient (patient no. 5). Beales has postulated that OS should be considered as having two components – a cochlear component and a mechanical one which

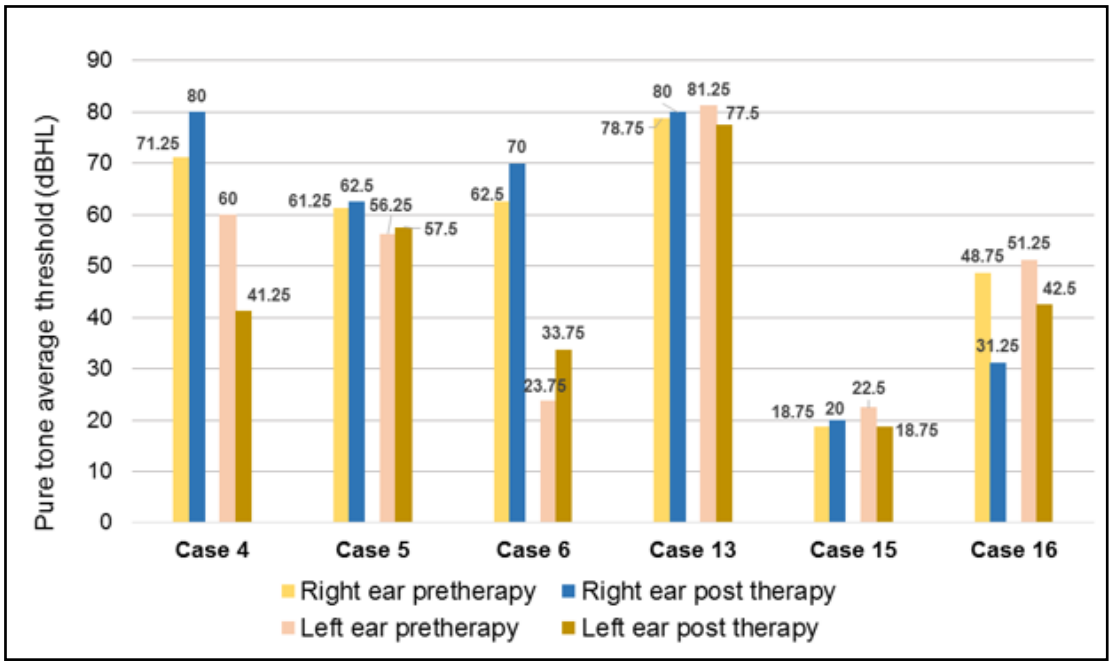

Fig. 5. PTA average threshold pre and post therapy (for patients who continued sodium flouride for >6 months).

involves fixation of stapes. The two components may occur independently or progress in combination producing a variety of clinical presentation ranging from pure cochlear type to obliterative type.[10]

SNHL occurring in cochlear OS has been attributed to release of enzymes such as trypsin, alpha-1 antitrypsin, ribonuclease which are toxic to the Organ of Corti. Causse and Chevance were able to demonstrate lysosome containing histiocytes in the advancing edge of otosclerotic lesion.[12] According to Parahy and Linthicum, SNHL can also occur in OS without the involvement of cochlear endosteum. In such cases it is believed that certain proteolytic enzymes that are harmful to the cochlea reach the cochlear endosteum through small bony channels present in the focus resulting in atrophy of stria vascularis and hyalinization of spiral ligament.[13] Lowering of the perilymph pH due to release of hydrogen ions from the spongiotic focus is also known to have deleterious effect on the cochlear structures.[14]

The existence of cochlear OS as a definite entity has been controversial. Schuknecht and Kirschner have strongly refuted the concept of cochlear OS presenting as pure SNHL. They postulated that when the otosclerotic lesion is severe enough to involve cochlear endosteum resulting in atrophy of sensory, neural and supporting structures, it involves the stapes as well.[4,5] They have also stated that severe OS can cause SNHL, yet there is no conclusive evidence that OS of lesser magnitude can cause pure SNHL.[5]

Balle and Linthicum on the other hand have postulated that cochlear OS can be a cause of pure SNHL. They have reported on histologic features of cochlear OS in seven temporal bones documented to have moderate to profound degree of hearing loss. The histologic findings included atrophy of the stria vascularis, hyalinization of the spiral ligament, hair cell and ganglion cell loss, hydrops and distortion of cochlear walls. They were also able to correlate the severity of histologic findings to the degree of hearing loss. According to them histologic studies of temporal bones cannot reflect the true incidence of pure cochlear OS.[7]

A definitive diagnosis of pure cochlear OS in a live patient is not possible.[6] Earlier Valvassori reported use of polytomography as a reliable tool for identifying lesions of cochlear otosclerosis.[15] In the past, Freeman

also had emphasized the importance of polytomography as he could identify 53 cases of cochlear OS in his series of 100 consecutive cases of progressive SNHL.[8] In the present era, HRCT scan of temporal bone is considered to be of immense value in diagnosing cochlear OS.[16,17] Radiologically, it appears as a hypodense lesion or an area of irregularity or increased thickness in the otic capsule.[18] The area of lucency can be single, multiple or confluent.[19] The CT grading system for OS proposed by Rotteveel is well accepted classification system. The classification is based on location and type of lesion. Fenestral type is classified as Grade 1, retrofenestral type as Grade 2A: double ring or halo effect, Grade 2B: narrowing of basal turn, Grade 2C: both, Grade 3: diffuse confluent lesions.20, 21 In our study all lesions were Grade 2A with the exception of two patients who also had a coexisting Grade 1 lesion. Lagleyre et al have reported that HRCT has a sensitivity of 95.1% and specificity of 99.5% in diagnosing OS. Using their protocol, they were able to pick up otosclerotic hypodensities anterior to the OW, in pericochlear region, RW and in the IAC. A definitive diagnosis of OS was established at the time of surgery in this study. In their series of 200 patients, 51 patients also had a pure SNHL in the contralateral ear. HRCT was positive in 68%, negative in 22% and doubtful in 10% of the 51 ears with pure SNHL.22 Foci less than 2-3 mm or inactive lesions where the density of lesion has become similar to that of surrounding bone are likely to remain undetected in HRCT.[17,18]

In our series we have used HRCT scan to evaluate the temporal bones of young patients who presented with progressive hearing loss. Otosclerotic focus could be identified in 27 out of 38 ears evaluated. We could also identify cochlear OS in the contralateral ear of a patient which had yet not manifested with hearing loss. We did not correlate the site of OS focus with the audiologic frequencies involved though there are studies in which correlation of abnormal densitometric points with the audiologic frequencies involved has been demonstrated.[18] We believe that HRCT scan is an essential tool in evaluating patients with unilateral or bilateral progressive SNHL which is inappropriate for age. It helps in establishing a diagnosis as well as early initiation of treatment which can halt further progression of hearing loss.

Medical management of cochlear OS is in the form of NaF along with oral calcium and vitamin D3. It promotes the calcification of the otospongiotic lesion. Otosclerotic bone due to its high metabolic activity selectively takes up fluoride compared to normal skeletal bone resulting in formation of fluorapatite which is hard and resists bone resorption.[23] The bone resorption and vascularity of the spongiotic bone is reduced resulting in maturation of the focus.[14,23] NaF also inhibits the release of enzymes by the otosclerotic focus which are known to cause SNHL.[13,23] It is used in the dosage of 40 mg per day for two years. Once the hearing loss stabilizes a maintenance dose of 20 mg per day for one's lifetime can be given.[10] NaF has been extensively used by Shambaugh and he found that with its prolonged use the re-calcification of the focus increases which is apparent radiologically, however, after a variable period of time.6 Biphosphonates which are potent anti-bone resorption agents have also been advocated in management of OS in recent literature.[24,25]

Joint pains, fluorosis of lumbar spine, allergic dermatitis, hair loss, dental mottling, gastric irritation are some of the side effects reported after prolonged use of NaF.[23,26] Gastric discomfort can be reduced by simultaneous intake of calcium carbonate though it can reduce absorption due to formation of calcium fluoride.[23,27] In our study two patients (Patient no. 1 & 2) developed gastric irritation which was severe enough to discontinue treatment. Patient no 1 restarted therapy after a gap of one year. Another patient (Patient no. 13) discontinued treatment as he developed elevated serum uric acid levels while on NaF therapy. Skeletal X Ray should be done prior to starting the treatment as well as during the treatment to watch for developing skeletal fluorosis.[10]

There is paucity of clinical evidence supporting the role of sodium fluoride in treating OS.[26,28] In a randomized controlled trial using NaF in patients with OS (20 mg twice daily for two years) statistically significantly greater deterioration of hearing was seen in the untreated arm.[29] Derks et al demonstrated improved BC thresholds for low and high frequencies in the ears treated with NaF compared to untreated ears which had a statistically significant progression of SNHL.[30] Forquer et al also found that hearing loss in patients with clinical

as well as cochlear OS stabilized or progressed slowly when treated with NaF.[31] In our study, we found benefit either in the form of improved or stabilized thresholds in a small group of patients whose follow up audiogram was available, or disappearance of associated symptoms such as tinnitus with NaF therapy. (Table IV, Fig. 5)

We have presented data of 19 patients who presented with progressive SNHL. HRCT scan helped in establishing diagnosis of cochlear OS in 14 patients who were started on NaF therapy. As most of the patients in our series had moderate or severe degree of hearing loss, our intent of starting medical therapy was to prevent the progression of hearing to profound disabling levels. Limitation of this study is lack of objective assessment of the response to therapy in all the patients as some of them were lost to follow-up.

We feel that absence of immediate benefit and need for prolonged therapy limits the compliance. Possible role of NaF therapy in stopping further progression of hearing loss and associated symptoms must be emphasized during counseling. Also increase in dosage can be considered in those patients who continue to have progressive disease as recommended by Shambaugh.[6]

Conclusion

There are no conclusive studies which can prove or refute the concept of cochlear OS causing pure SNHL. However, cochlear otosclerosis has been considered as a cause of pure SNHL in literature. It is not possible to histologically prove cochlear otosclerosis in life. In our clinical practice we do come across young patients with SNHL which is progressive. Identifiable causes of SNHL such as cochlear otosclerosis should always be suspected in these patients. In most instances the cause of SNHL is assumed to be secondary to noise, ototoxic medication, head trauma or even presbycusis if the patient's age and history so permits. HRCT of temporal bones in these patients is seldom asked for. Hence, cochlear OS often remains undiagnosed. We recommend that all patients with early onset unilateral or bilateral progressive SNHL should undergo HRCT temporal bone in addition to the routine investigations. Early detection of cochlear OS will help institute medical therapy which may halt the further progression of hearing loss.

Authors' Note: This paper was presented in the Indian Society of Otology Conference in November, 2016 at Calicut, Kerala (India).

References

1. MN Wali. Otosclerosis. Can Fam Physician. 1987; 33: 1491-3
2. Guild SR. Histologic otosclerosis. Annal Otol Rhinol Laryngol. 1944; 53: 46
3. Declaua F, Spaendonck MV, Timmermans JP, Michaels L, Liang J, Qiu JP, et al. Prevalence of otosclerosis in an unselected series of temporal bones. Otol Neurotol. 2001; 22: 596-602
4. Schuknecht HF. Cochlear otoslerosis- A continuing fantasy. Arch Otolaryngol Head Neck Surg. 1979; 222: 79-84
5. Schuknecht HF, Kirschner JC. Cochlear otosclerosis: fact or fantasy. Laryngoscope 1974; 84: 766-82
6. Shambaugh G, Jr. The therapy of cochlear otosclerosis. Ann Otol Rhinol Laryngol. 1966; 75: 579-83
7. Linthicum FH, Balle V. Histologically proven cochlear otosclerosis with pure SNHL. Annal Otol Rhinol Laryngol. 1984; 93: 105-11
8. Freeman J. Progressive Sensorineural hearing loss and cochlear otosclerosis: A prospective study. Laryngoscope 1979; 89: 1487-521
9. Franceschini SS, Ravecca F, De Vito A, Berrettini S. Progressive sensorineural hearing loss in cochlear otosclerosis. Acta Otorhinolaryngol Ital. 1998; 18: 59-65
10. Beales PH. Otosclerosis – past and present. J. R. Soc. Med. 1979; 72: 553-61
11. Schuknecht HF, Barber W. Histologic variants in otosclerosis. Laryngoscope 1985; 95: 1307-17
12. Causse JR, Chevence LG. Sensorineural hearing loss due to cochlear otospongiosis: etiology. Otolaryngol Clin North Am. 1978; 11: 125-34
13. Parahy C, Linthicum FH Jr. Otosclerosis: relationship of spiral ligament hyalinization to sensorineural hearing loss. Laryngoscope 1983; 93: 717-20
14. Petrovic AG, Stuzmann JJ, Shambaugh GE Jr. Experimental studies on pathology and therapy of otospongiosis. Am J Otol. 1985; 6: 43-50
15. Valvassori GE. Radiologic diagnosis of cochlear otosclerosis. Laryngoscope. 1965; 75: 1563-71
16. Veillon F, Riehm S, Emachescu B, Haba D, Roedlich MN, Greget M, et al. Imaging of the windows of the temporal bone. Semin Ultrasound CT MR. 2001; 22: 271-80
17. Mafee MF, Henrikson GC, Deitch RL, Norouzi P, Kumar A, Kriz R, et al. Use of CT in stapedial otosclerosis. Radiology 1985; 156: 709-14

18. Guneri EA, Ceryan K, Ada E, Guneri A. High resolution computed tomographic evaluation of cochlear capsule in otosclerosis: relationship between densitometry and sensorineural hearing loss. Ann Otol Rhinol Laryngol. 1996; 105: 659-64
19. Youssef O, Rosen A, Chandrashekhar S, Lee HS. Cochlear otosclerosis: The current understanding. Ann Otolol Rhinol Laryngol. 1998; 107: 1076-9
20. Rotteveel LJ, Proops DW, Ramsden RT, Saeed SR, van Olphen AF, Mylanus EA. Cochlear implantation in 53 patients with otosclerosis: Demographics, Computed tomographic scanning, surgery and complications. Otol Neurotol. 2004; 25: 943-52
21. Merkus P, van Loon MC, Smit CF, Smit C, de Cock Adrianus FC, Hensen EF. Decision making in advanced otosclerosis: An evidence based strategy. Laryngoscope. 2011; 121: 1935-41
22. Lagleyre S, Sorrentino T, Calmels MN. Shin YJ, Escude B, Deguine O, et al. Reliability of High Resolution CT Scan in Diagnosis of Otosclerosis. Otol Neurotol. 2009; 30: 1152-9
23. Causse JR, Causse JB, Uriel J, Berges J, Shambaugh GE Jr, Bretlau P. Sodium fluoride therapy. Am J Otol. 1993; 14: 482-90
24. Kennedy DW, Hoffer ME, Holliday M. The effects of etidronate disodium on progressive hearing loss from otosclerosis. Otolaryngol Head Neck Surg. 1993; 109: 461-7
25. Brookler K. Medical treatment of otosclerosis: rationale for use of bisphosphonates. Int Tinnitus J. 2008; 14: 92-6
26. Hentschel MA, Huizinga P, van der Velden DL, Bittermann AJN, van der Heijden GJM, Grolman W. Limited evidence for the effect of sodium fluoride on deterioration of hearing loss in patients with otosclerosis: A systematic review of literature. Otol Neurotol. 2014; 35: 1052-7
27. Deka RC, Kacker SK, Shambaugh GE Jr. Intestinal absorption of fluoride preparations. Laryngoscope 1978; 88:1918-21
28. Cruise AS, Singh A, Quiney RE. Sodium fluoride in otosclerosis treatment: review. J Laryngol Otol. 2010; 124: 583-6
29. Bretlau P, Salomon G, Johnsen NJ. Otospongiosis and sodium fluoride. A clinical double-blind, placebo-controlled study on sodium fluoride treatment in otospongiosis. Am J Otol. 1989; 10: 20-2
30. Derks W, De Groot JA, Raymakers JA, Veldman JE. Fluoride therapy for cochlear otosclerosis? An audiometric and computerized tomography evaluation. Acta Otolaryngol. 2001; 121: 174-7
31. Forquer BD, Linthicum FH, Bennett C. Sodium fluoride: Effectiveness of treatment for cochlear otosclerosis. Am J Otol. 1986; 7: 121-5.

Main Article

Outcome of Post-Operative Mastoid Cavity Obliteration with Autologous Adipose Tissue

https://doi.org/10.47210/bjohns.2020.v28i2.331

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ABSTRACT

Introduction

Mastoid operations have been in practice for over four centuries for suppurative conditions of the ear. Intact canal wall mastoidectomy has the advantage of better functional results while canal wall down mastoidectomy offers excellent exposure for disease eradication and post operative monitoring but is associated with significant cavity problems. In order to overcome the problems associated with canal wall down procedure while retaining its advantages the concept of mastoid cavity obliteration was introduced. This study analysed the outcomes of mastoid cavity obliteration and to assess the outcomes of mastoid cavity obliteration with autologous adipose tissue.

Materials and Methods

A prospective, experimental, randomized study was conducted over a period of 18 months among patients presenting with active squamous variety of Chronic Otitis Media. The patients were randomly allocated to two groups, A and B. Both groups underwent canal wall down Mastoidectomy followed by obliteration with autologous adipose tissue in Group B .

Results

In group A, the mean duration required for complete epithelialization was 10.8 weeks. In group B, the average time taken for complete epithelialization was 5.6 weeks. All cases had their graft intact at the end of 12 weeks. Debris was present in group A for a mean duration of 9.47 weeks. In group B, debris was found for a mean duration of 3.33 weeks. Patients from group A complained of discharge from their ears for a mean duration of 7.47 weeks. In group B the same symptom persisted over 3.33 weeks.

Conclusion

Cavity problems encountered is considerably less in the group obliterated with adipose tissue.

Keywords

Mastoidectomy; Adipose Tissue

Chronic otitis media is an inflammatory process in the middle-ear space that results in long-term, or more often, permanent changes in the tympanic membrane including atelectasis, dimer formation, perforation, tympanosclerosis, retraction pocket development or cholesteatoma. Nadol classified chronic otitis media as chronic active otitis media with or without cholesteatoma; chronic inactive otitis media with perforation, with retraction pocket, adhesive otitis media, with ossicular fixation or resorption; chronic inactive otitis media with frequent reactivation.[1,2] Another proposed classification is based on classifying the disease into mucosal(active/inactive); squamous(active/inactive) and healed.[3]

Mastoid operations have been in practice for over four centuries for suppurative conditions of the ear. Jansen

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described canal wall up mastoidectomy in 1953. The main advantage of canal wall up mastoidectomy is that normal anatomy can be preserved along with clearance of disease process.[4]

But in cases of unresectable cholesteatomas extending up to petrous apex, unreconstructible posterior canal wall, failure of first stage canal wall up procedures owing to poor Eustachian tube function, a canal wall down mastoidectomy is done which requires the removal of posterior wall of external auditory canal.[5]

Intact canal wall mastoidectomy has the advantage of rapid wound healing and also avoids frequent cleaning of the cavity. But the chances of residual disease as well as recurrent cholesteatoma is high with this procedure.[6]

With canal wall down mastoidectomy there is excellent exposure for disease eradication and post operative monitoring; also there is less chance of leaving behind residual disease with low rates of recurrence.[7] But it is associated with patient's intolerance to water exposure; requirement of frequent cleaning of cavity; calorically and barometrically induced vertigo; aesthetic inconvenience due to too large meatoplasty; recurrent infection; difficulty in wearing traditional hearing aids. Also the final hearing gained after staged ossiculoplasties in patients who have undergone canal wall down mastoidectomy is usually 5-10dB worse than those who underwent canal wall up tympano-mastoidectomy due to ineffective sound transmission.[6,7,8.9]

In order to overcome the problems associated with canal wall down procedure while retaining its advantages the concept of mastoid cavity obliteration was introduced by Mosher in 1911.[6] Over the course of this century, there have been numerous reports detailing a variety of techniques of obliterating the mastoid cavity.

In developing countries like India where proper health care services is still not available to a sizeable chunk of the society, mastoid obliteration reduces the number of hospital visits for cavity management.

Keeping this in mind, the following study has been conducted with the aims and objectives to study the outcomes of mastoid cavity obliteration and to assess the outcomes of mastoid cavity obliteration with autologous adipose tissue as obliterating material for mastoid obliteration.

Materials and Methods

A prospective, experimental, randomized study was conducted over a period of 18 months amongst the patients attending the Otorhinolaryngology out-patients wing of a peripheral referral institute from January 2017 to June 2018. All cases of chronic otitis media with cholesteatoma, with extensive granulations, with attic or postero-superior retraction pockets where the fundus is not visible were included in the study. Known cases of chronic otitis media with complications and those who didn't wish to participate in the study were excluded.

After proper selection, the patients were counselled about the disease process and the treatment modalities with the expected outcome and the complications of the procedures. They were then subjected to pure tone audiometry, X-ray mastoids, Examination under microscope, routine pre operative investigations followed by pre anaesthetic check-up. The patients were then divided into two groups by systematic random sampling-

Group-A: Canal Wall Down (CWD) Mastoidectomy without obliteration

Group-B: CWD Mastoidectomy and obliteration with adipose

Operative procedure: All the surgeries were done under general anaesthesia. Lignocaine(2%) with 1:100000 adrenaline was infiltrated for haemostasis. Temporalis fascia graft was harvested. Post auricular incision was made. A Canal Wall Down mastoidectomy was done to remove any disease process in the mastoid and middle ear. It was followed by ossicular reconstruction if required, followed by tympanoplasty. Then, according to pre-determined protocol, the mastoid cavity was left as such (Group A) or obliterated using autologous adipose tissue (Group B). (Fig. 1)

Post operative treatment and follow up: Each patient was given an antibiotic, analgesic and antihistaminic medication for one week. Patients were discharged on the second postoperative day. Ear pack was removed two days after surgery. Patients were then asked to follow-up at two weeks after pack removal, one and half months post-operative and 3 months postoperative for assessment of hearing by pure tone audiogram.

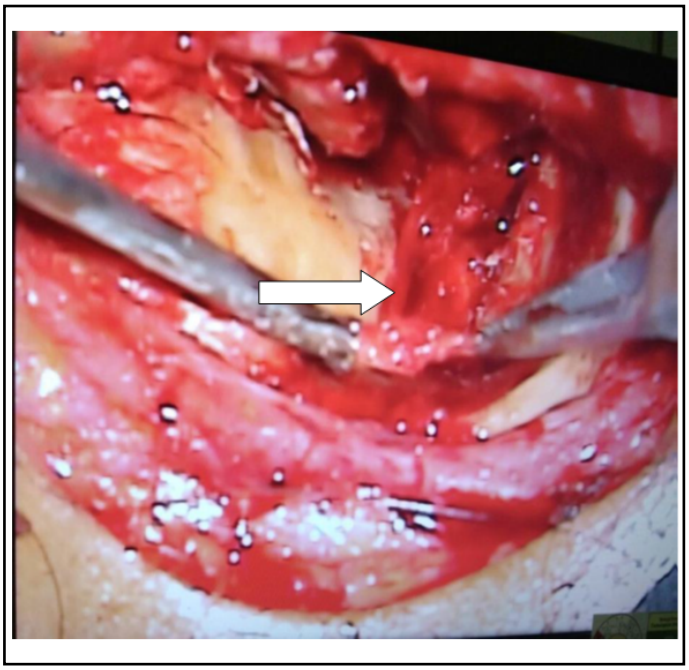

Fig. 1. Intra-operative picture showing Canal Wall Down Mastoidectomy with obliteration with adipose tissue

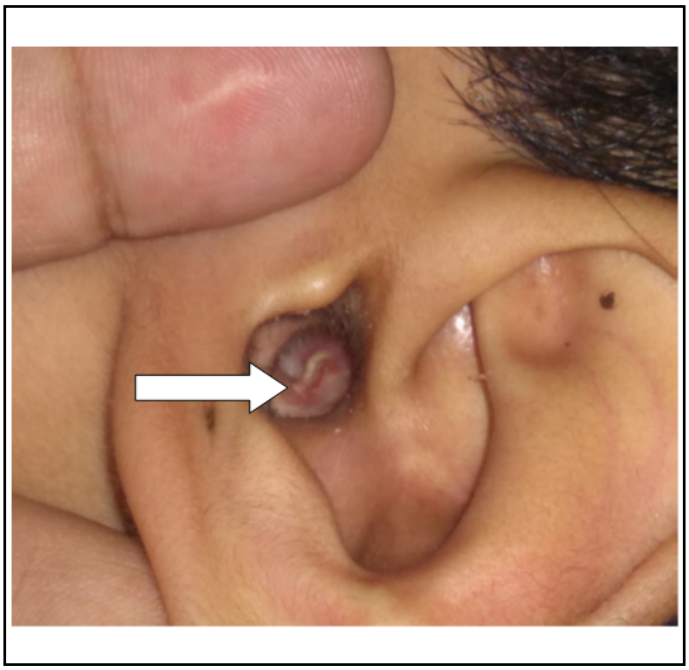

Fig. 2. Post operative photograph showing a well epithelialized, obliterated mastoid cavity at 12th week

Results

The impact of the surgical procedures undertaken were assessed on the basis of (a) healing of mastoid cavity in terms of epithelialisation, (b) post-operative problems and (c) hearing gain.

Each group comprised of 15 individuals. The individuals were divided into 4 age groups: 5-15 years, 16-25 years, 26-35 years and 36-45 years. A total of 9 males and 6 females were in group A, 7 males and 8 females in group B. The mean age of the individuals in group A was 25.47 years with a standard deviation of 8.89 years; in group B it was 24.4 years with a standard deviation of 10.13 years.

The individuals in each group underwent Examination under microscope (EUM) prior to surgery. Individuals with attic pathology comprised the majority (5 cases in each group) followed by postero-superior retraction pockets (PSRP) and adhesive otitis media.

Conductive deafness was prevalent in both the groups with mixed deafness detected only in 2 patients in group A and 1 in group B.

The pre-operative mean pure tone average in group A was 47.6 dB with a standard deviation of 10.34 dB. The mean Air-bone gap (A-B gap) in group A was 25.87 dB with a standard deviation of 9.99 dB. The mean pure tone average in group B was 46.6 dB with a standard deviation of 10.81 dB. The mean A-B gap in group B was 25.93 dB with a standard deviation of 9.6 dB. In both groups, individuals mostly had moderate degree of hearing loss followed closely by individuals with mild degree of loss (9 moderate and 4 mild in group A; 10 moderate and 4 mild in group B).

On classifying the study subjects based on the type of tympanoplasty, Type III tympanoplasty was most commonly performed in both the groups followed by type IV tympanoplasty.

The study subjects have been followed up in the post operative period according to the protocol as previously determined. Examination under microscope was carried out during each post-operative visit. The following parameters were noted, namely, epithelialization of the mastoid cavity; presence of debris in the mastoid cavity; duration of otorrhoea; common postoperative complications like pain, vertigo, granulation tissue,

morbidity of donor site etc; graft integrity at post-operative visits of 2 weeks, 4 weeks, 6 weeks and 12 weeks. The post-operative pure tone audiometry was performed at 12 weeks and the hearing outcome evaluated based on A-B gap closure.

Epithelialization of the mastoid cavity was noted as partial or complete during each of the post-operative visits. In group A, the mean duration required for complete epithelialization was 10.8 weeks with a standard deviation of 2.48 weeks. In group B, the average time taken for complete epithelialization was 5.6 weeks with a standard deviation of 2.03 weeks. The time taken for epithelialization amongst the 2 groups have been assessed with unpaired t-test and it has been found that the difference in time taken for epithelialization is highly significant when group A (CWD Mastoidectomy without obliteration) is compared against group B (CWD Mastoidectomy with obliteration with adipose). (Fig. 2)

As far as graft integrity is concerned, all cases had their graft intact at the end of 12 weeks. Post-operative pain was complained by 4 patients in the group without obliteration; 3 in case of obliteration with adipose. There was no further complaint during the study period. Presence of debris in the mastoid cavity was then evaluated amongst the groups. Debris was present in group A for a mean duration of 9.47 weeks with a standard deviation of 3.25 weeks. In group B, it has been found for a mean duration of 3.33 weeks with a standard deviation of 1.23 weeks. Collection of debris in the mastoid cavity has been assessed using the same unpaired t-test as above with similar statistical significance being obtained between obliterated versus unobliterated group.

Vertigo in the post-operative period was complained mainly by patients in the group which did not undergo any obliteration. 8 cases complained of vertigo in the immediate 2nd week of post-operative visit, of which 5 cases had their symptoms resolved by the end of 4 weeks whereas another 2 individuals had persistent symptoms till the end of 6 weeks. By the end of 12 weeks no one complained of vertigo. In group B only 1 patient complained of vertigo at 2nd week which resolved subsequently with no such complaints during any of the subsequent post-operative visits.

Otorrhoea following canal wall down procedures being the most frequent complaint during the post-operative period has been evaluated next. Patients from group A complained of discharge from their ears for a mean duration of 7.47 weeks with a standard deviation of 2.88 weeks. In group B the same symptom persisted over 3.33 weeks with a standard deviation of 1.23 weeks. The duration of otorrhoea was evaluated using the same principles with no different statistical outcome, i.e., obliteration groups showing better results compared to group A.

At the end of 12th post-operative week, all individuals were subjected to pure tone audiogram. In group A, the mean PTA was 36.06 dB with a standard deviation of 10.05 dB. The mean A-B gap in group A was 17.07 dB with a standard deviation of 10.59 dB. In group B, the mean PTA was 36.2 dB with a standard deviation of 10.42 dB. The mean A-B gap in group B was 15.2 dB with a standard deviation of 10.06 dB. Each group showed statistically significant improvement in pre and post-operative pure tone audiometry and A-B gap when tested by paired t-test but the hearing gain in the individual groups when tested against each other failed to produce any statistical significance when tested by unpaired t-test.

Discussion

The global burden of illness from COM involves 65–330 million individuals with draining ears, 60% of whom (39–200 million) suffer from significant hearing impairment. Over 90% of the burden is borne by countries in the south-east Asia and western Pacific regions, Africa, and several ethnic minorities in the Pacific rim. COM is uncommon in the Americas, Europe, the Middle East, and Australia. Among the South-East Asian countries, prevalence rate of COM in Thailand ranged from 0.9 to 4.7% while the Indian prevalence is 7.8%.[10]

The primary goal of middle ear surgery, whether or not the patient has a cholesteatoma, is extirpation of the disease, anatomical preservation of the middle ear, and functional restoration.[11] The type of mastoidectomy is based on the extent of the disease, preoperative health of the patient, the status of the opposite ear, and both the surgeon's and patient's preference.[10]

The canal wall up mastoidectomy involves removing the mastoid air cells lateral to the facial nerve and otic capsule bone while preserving the posterior and superior external auditory canal walls. It preserves the normal anatomy of the ear canal, thus avoiding the risk of bowl problems and the necessity for periodic cleaning, leading to reduced healing time and, possibly, better hearing results. However, a high residual and recurrence rate continue to be major drawbacks to canal wall up surgery.[10]

The canal wall down technique creates an open cavity after removal of the bony posterior canal wall, providing excellent exposure for cholesteatoma removal. Recurrence rate is lowered to 5–10%. The main disadvantages of this technique are related to the necessity for periodic cleaning of the mastoid cavity with persistent otorrhea. Other problems with open cavity include difficulty in the use of a hearing aid, water intolerance due to susceptibility to infection, and proneness to vertigo by a caloric stimulus such as warm/cold air or water. CWD when compared to CWU has lower risk of recurrence (2-10%) but higher risk of otorrhoea (20-60%). These problems can be minimized by different obliterative procedures.[10]

The concept of mastoid cavity obliteration was first introduced by Mosher in 1911. Mastoid reconstruction and obliteration procedures involve free grafts (which are further sub-divided to biologic and non-biologic) and local flaps.

With the advent of numerous techniques for mastoid obliteration, the debate continues till date regarding the best material that can be used for obliteration. Each method has some distinct benefits over the others. Although these techniques offer the major advantage of eliminating the cavity problem, there can be long-term problems. For example, some of the obliterated tissues can shrink over time, minimizing the effectiveness of the mastoid obliteration.[11]

Obliteration with fat has been of particular interest due its inherent properties, which may be the cause for low complication rate associated with use of fat. Fat tissue is not inert; it stores macrophages, which participate in inflammation and the immune reaction. Fat tissue also stores a high proportion of lymphocytes which, along with macrophages, can lead to immune regulation. Also fat provides a ready contrast medium in postoperative fat suppression and diffusion sequences on the MRI scan, which provides a very adequate method to detect recurrence.[12]

The mean age of the individuals in our study- in group A was 25.47 years with a standard deviation of 8.89 years; in group B it was 24.4 years with a standard deviation of 10.13 years. The mean age of individuals in the study by Bhandary et al. was 26.95 + 9.525 years which is coinciding with our study.[13]

On examination under the microscope, 62% subjects in our study had cholesteatoma (comprising both PSRP with cholesteatoma as well as attic cholesteatoma), nearly 9% had granulation and 29% comprised the remainder. In the study by Deshmukh et al, 80% of patients had cholesteatoma, 13.3% had granulations and 6.6% comprised the remainder.[14] The findings of our study are in tune with the above study where individuals with cholesteatoma formed the majority followed by granulation tissue.

In the present study nearly 89% individuals had mild-moderate degree of hearing loss. This finding is slightly different from that of the study mentioned above, where 33.3% had hearing loss of <30 dB, 50% had hearing loss between 30 and 60 dB and only 16.6% had hearing loss >60 dB.[14]

The mean time required for complete epithelialization of un-obliterated mastoid cavities in this study was around 3-6 months which correlates well with that of Deshmukh et al where 94% cases showed epithelialization at the end of 6 months.[14] The mean duration for complete epithelialization of cavities obliterated by adipose tissue was 6-8 weeks in the present study. This correlates with the study conducted by Ravi and Kumar[15] but does not agree with the study by Bhandary et al[13] where the mean duration was around 17 weeks.

The time required to obtain a dry cavity in un-obliterated cases was around 8-10 weeks in our study which correlates well with the study by Desmukh et al.[14] In cases obliterated with adipose tissue the time required to obtain a dry cavity was around 4-5 weeks in our study. None of the other studies (Ravi and Kumar,[15] P. Montandon et al1,[6]) had any other complaints regarding

otorrhoea.

Post-operative vertigo was found to be significantly higher in groups that did not undergo any obliteration compared to groups that were obliterated with adipose.

Post-operative hearing was found to improve in both the study groups. However only a modest improvement in post-operative hearing is seen in studies where no obliteration was done.[11,16]

Conclusion

Obliteration of the mastoid cavity offers possible remedy to the various cavity problems associated with a large mastoid cavity. This study, conducted with the aim to assess the outcomes of mastoid cavity obliteration using autologous adipose tissue demonstrated shorter duration of accumulation of debris and early achievement of dry ear resulting in early healing of mastoid cavity in terms of epithelialization is obliterated cavities.Hearing gain showed no difference when compared amongst the two groups though each group showed significant statistical improvement post-operatively.

Thus, it can be seen that cavity obliteration offers a simple solution to avoid the cavity problems and also promotes early healing of mastoid cavity. Adipose being easily available, can be readily used for obliteration. However, larger studies are needed to conclusively prove or refute the hypothesis statistically.

References

1. Gopen Q. Pathology and clinical course of the inflammatory diseases of the middle ear. In: Gulya AJ, Minor LB, Poe DS, editors. Glasscock-Shambaugh Surgery of the Ear. 6th ed. Shelton, Connecticut: People's Medical Publishing House-USA; 2010
2. Nadol JB. The chronic draining ear. Current therapy in Otolaryngol Head Neck Surg. 1987;18:18-22
3. Browning GG. Merchant SN, Kelly G, Swan IR, Canter R, McKerrow WS. Chronic otitis media. In: Gleeson M, editor. Scott-Brown's Otorhinolaryngology, Head and Neck Surgery. 7th ed. Great Britain. Edward Arnold (Publishers) Ltd; 2008
4. Haynes DS, Wittkopf J. Canal-wall-Up Mastoidectomy. In: Gulya AJ, Minor LB, Poe DS, editors. Glasscock-Schambaugh Surgery of the Ear. 6th ed. Shelton, Connecticut: People's Medical Publishing House-USA; 2010
5. Kveton JF. Open Cavity Mastoid Operations. In: Gulya AJ, Minor LB, Poe DS, editors. Glasscock-Schambaugh Surgery of the Ear. 6th ed. Shelton, Connecticut: People's Medical Publishing House-USA; 2010
6. Rao U.S. Obliteration of mastoid cavity and reconstruction of attic area using conchal cartilage. Asian Pac. J. Health Sci. 2016; 3(1):140-3
7. Cho SW, Cho YB, Cho HH. Mastoid Obliteration with Silicone Blocks after Canal Wall Down Mastoidectomy. Clinical and Experimental Otorhinolaryngology 2012; 5(1):23-7
8. Uçar C. Canal wall reconstruction and mastoid obliteration with composite multifractured osteoperiosteal flap. European Archives of Oto-Rhino-Laryngology 2006; 263(12):1082-6
9. Alves RD, Cabral Junior F, Fonseca AC de O, Bento RF. Mastoid Obliteration with Autologous Bone in Mastoidectomy Canal Wall Down Surgery: a Literature Overview. International Archives of Otorhinolaryngology 2016; 20(1):76-83
10. Singh M, Jain S, Rajput R, Khatua RK, Sharma D. Retrospective and Prospective Study of Singapore Swing Method on Healing of Mastoid Cavity. Indian J Otolaryngol Head Neck Surg. 2010; 62: 365-71
11. Lee HJ, Chao JR, Yeon YK, Kumar V, Park CH, Kim HJ, Lee JH. Canal Reconstruction and Mastoid Obliteration Using Floating Cartilages and Musculoperiosteal Flaps. Laryngoscope 2017 May;127(5):1153-60
12. Preetam C, Verma R, Thakar A, Sikka K. Tympanomastoid obliteration for recalcitrant attico-antral chronic suppurative otitis media. Indian J Otol. 2011;17:26-9
13. Bhandary S, Paudel D, Chettri ST, Natesh VN, Shilpakar SL, Sah BP, Pokharel A. Obliteration of Mastoid Cavity with Temporalis Muscle Pedicle and Abdominal Fat Graft and Compare the Efficacy of Each Method: A Prospective Randomized Clinical Trial. Otolaryngology Online Journal 2015; 5(3)
14. Deshmukh S, Sharma A, Dabholkar J. Mastoid cavity obliteration: Our experience. Otolaryngologia Polska 2012; 66:379-81
15. Ravi K, Kumar SK. Mastoid cavity obliteration in open cavity type (MRM) with fibro fatty tissue and subcutaneous periosteal flaps study conducted at RIMS college Ongole ENT department 2015-2016. Journal of Dental and Medical Sciences. 2016;15(6):63-70
16. Montandon P, Benchaou M, Guyot JP. Modified canal wall-up mastoidectomy with mastoid obliteration for severe chronic otitis media. ORL J Otorhinolaryngol Relat Spec. 1995; 57(4):198-201.

Main Article

Manifestations of Tuberculosis in Ear, Nose, Throat, Head and Neck Region – A Retrospective Study

https://doi.org/10.47210/bjohns.2020.v28i2.332

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ABSTRACT

Introduction

Tuberculosis can involve any organ or site. Otorhinolaryngologist may encounter tuberculosis affecting lymph nodes, ear, larynx, deep neck spaces, salivary glands etc. which can mimic other chronic granulomatous conditions or malignancy. To ensure early diagnosis, it is important to recognize its cardinal signs and symptoms and to be aware of potential pitfalls in diagnosis. This study was done to learn the clinical presentation of tuberculosis in ear, nose, throat and head and neck region, and to assess the effectiveness of various investigations and treatment done for the same.

Materials and Methods

A retrospective study done in our institution involving 120 patients suffering from tuberculosis in ear, nose, throat and head and neck region who attended pulmonary medicine or ENT OPD or ward between January 2008 to December 2017 that is, 10 years. Study period for data collection and analysis was 1 month.

Results

Total 120 patients-69 males and 51 females. Most common site was cervical lymph nodes(77.5% patients), followed by larynx(8.3%),middle ear(7.5%),deep neck spaces(2.5%) and salivary glands and nose(1.7% each). Histopathology was highly sensitive(99. 1%).All except one patient responded to first-line antitubercular drugs, the other patient was given treatment for MDR-TB to which he responded.

Conclusion

Tuberculosis can involve any site in the head and neck region, most common being cervical lymph nodes mainly presenting as neck swelling. Variable nature of manifestations of tuberculosis makes it essential to have high degree of suspicion for early diagnosis.

Keywords

Tuberculosis; Otorlaryngology; Lymph Nodes; Larynx; Ear, Middle; Neck; Retrospective Study

Tuberculosis (TB) is one of the oldest diseases of mankind and it is very common in developing nations such as India. There are nearly 9 million new patients and 2 million deaths from tuberculosis worldwide every year.[1] India has about 23 % of world's share.[1] Tuberculosis is a chronic infectious granulomatous disease caused by Mycobacterium tuberculosis. M. Tuberculosis is an acid fast bacillus. Increased incidence of tuberculosis in India is due to increased prevalence of immunodeficiency through HIV infection, malignancies, drug addiction, poverty and overcrowding.

Health care givers, TB contacts, prisoners, smokers, elderly, slum dwellers form the other socially vulnerable groups. Although TB mainly affects the lungs, extrapulmonary tuberculosis is also a significant disease entity. Among the total 1.68 million patients of TB notified in the year 2014, 2.75 lacs patients were of extrapulmonary tuberculosis.[1] Tuberculosis can affect any organ in the body except nail, hair and teeth.[2] In the head and neck region, TB can involve the cervical lymph nodes, larynx, middle ear, nose, oral cavity, salivary

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Table I: Site of tuberculosis

SITE OF TUBERCULOUS LESION	NUMBER OF PATIENTS	PERCENTAGE
Cervical lymph nodes	93	77.50%
Larynx	10	8.30%
Middle ear	7	5.83%
Nose	2	1.67%
Parotid gland	2	1.67%
Thyroid gland	1	0.83%
Deep neck spaces	3	2.50%
Cervical lymph nodes + middle ear*	2	1.67%

**Two patients of middle ear tuberculosis also had cervical lymphadenopathy.*

and thyroid glands, deep neck spaces and pharynx. Tuberculosis of lymph node is a common cause of lymph node enlargement. TB can affect middle ear and presents as painless recurrent otorrhoea, presence of abundant pale granulations and severe hearing loss sometimes with facial nerve palsy.[3] Head and neck tuberculosis is also an interesting field of research because of the varied presentations and different sites of involvement. It may simulate malignancy and may be misdiagnosed.[2] The various diagnostic methods are direct microscopy, standard culture methods, rapid culture method and polymerase chain reaction test also known as cartridge based nucleic acid amplification test (CB-NAAT). CB-NAAT is a rapid diagnostic test which provides results within 2-3 hours and also gives result on rifampicin susceptibility.[4] Government has proposed an END TB strategy. The important components of this strategy are early diagnosis with latest diagnostic tools, complete treatment with standard antiTB regimen, treatment of comorbidities and providing patient support.[5] The purpose of this study are to study the clinical presentations of tuberculosis in ear, nose, throat and head and neck regions and to assess the investigations and outcome of anti-Koch's treatment for the same.

Materials and Methods

It is a retrospective study, the study source being patients attending ENT OPD and ward, pulmonary medicine OPD and ward at our hospital between January 2008 and December 2017, that is, last 10 years. All patients who were diagnosed with tuberculosis of ear, nose, throat and head neck region were included in the study.

Table II: Age distribution

AGE (YEARS)	NUMBER OF PATIENTS	PERCENTAGE
0-20	31	25.8%
20-30	37	30.8%
30-40	19	15.8%
40-50	25	20.8%
50-60	02	1.67%
›60	06	5%

Institutional Ethics Committee (IEC) permission was obtained prior to the commencement of the study. We collected patient's data from medical record section and computerized data system. We studied patient's complete history, findings of ear, nose, throat, along with systemic and general examinations. We also reviewed investigations done, e.g., fine needle aspiration cytology and ultrasonography of neck swelling, Ziehl-Neelsen staining and culture of ear discharge, if present; biopsy and histopathological examination and also the management of these patients. We then analysed the whole data and compared it with standard observations.

Results

Total 120 patients of tuberculosis of ear, nose, throat and head-neck region were found and were analysed. (Table I) Most common site was cervical lymph nodes.

Most of the patients of tuberculous lymphadenitis were in age group of 7-25 years with about 64 patients being In this age group; while laryngeal tuberculosis was seen frequently in 40-60 years of age. (Table II)

Out of 120 patients, 69 patients were males and 51 were females.

The chief complaint with cervical lymph node tuberculosis (93 patients) was neck swelling (Fig.1A &1B) which was unilateral in 86 and bilateral in 7 patients. Level II was most commonly affected (57 patients) followed by level V (26 patients). These patients also had other complaints including fever (22 patients), weight loss (17 patients) and cough with expectoration (8 patients). Fine needle aspiration cytology (FNAC) and ultrasonography USG) were done in all patients. All except four showed tuberculous lymphadenitis on FNAC. In these 4 patients, excisional biopsy of lymph node was done for histopathological diagnosis. Contrast enhanced computed tomography (CECT) neck showed presence of nodal abscess with nodal mass in few patients (Fig. 2). Montoux test was positive in majority of patients (88 patients). Erythrocyte sedimentation rate (ESR) was raised in 75 patients. Eight of these 93 patients had concomitant pulmonary tuberculosis. Six patients were HIV positive.

Patients with laryngeal tuberculosis (10 patients) mainly presented with hoarse voice. All these patients had pulmonary tuberculosis as well. Diagnosis was done by laryngoscopy and biopsy of the lesion for histopathological confirmation. Most common sites in larynx were inter arytenoid area, vocal cords and arytenoids. No patient of laryngeal tuberculosis was found to be HIV positive.

All patients with tuberculosis of middle ear (9 patients) presented with painless profuse otorrhea and 5 of these patients had severe to profound hearing loss at presentation. Two patients had developed ipsilateral mastoid abscess, 1 of which also developed ipsilateral lower motor neuron facial nerve palsy(Fig.1A &1B). One patient of middle ear tuberculosis had ipsilateral lower motor neuron facial nerve palsy without mastoid abscess. Three patients had multiple perforations (Fig.3), while rest had total perforation. In one patient, the middle ear tuberculosis complicated into tuberculous meningitis. Two patients of middle ear tuberculosis also had cervical lymphadenopathy. Pulmonary tuberculosis was present in all the patients. Ear swab for acid fast bacillus was positive in 7 patients. The 2 patients with mastoid abscess required mastoid exploration and while in 1 patient with facial nerve palsy without mastoid abscess, transmastoid facial nerve decompression was done. Histopathological diagnosis of tuberculosis could be made in all these 3 patients. Sample for histopathology was pale granulations collected while doing mastoid exploration.

Patients with nasal tuberculosis (2 patients) presented with blood stained nasal discharge and nasal blockage. They were diagnosed by nasal endoscopy and biopsy of inflammatory nasal tissue. One of these two patients had pulmonary tuberculosis as well.

Patients of parotid gland tuberculosis (2 patients) presented with swelling over parotid region (Fig.4). Among these 2 patients, one developed spontaneous rupture of the parotid resulting in parotid fistula and purulent discharge from that site. Both the patients had tuberculous changes in lungs.

Patient of thyroid gland TB (1 patient) presented with thyroid swelling and the diagnosis was done by FNAC. Patient had tuberculosis of lungs and intestine too. Deep neck space tuberculosis patients (3 patients)

Fig. 1 A & B. Tubercular mastoid abscess with lower motor neuron in facial palsy with tubercular cervical lymphadenitis

Fig.3. Multiple perforations in tympanic membrane

came with complaints of dysphagia and neck pain and TB was diagnosed by microbiological and cytological examinations of the aspirated pus. One of these three patients had pulmonary TB.

Among all these 120 patients of extrapulmonary tuberculosis,32 patients had pulmonary tuberculosis as

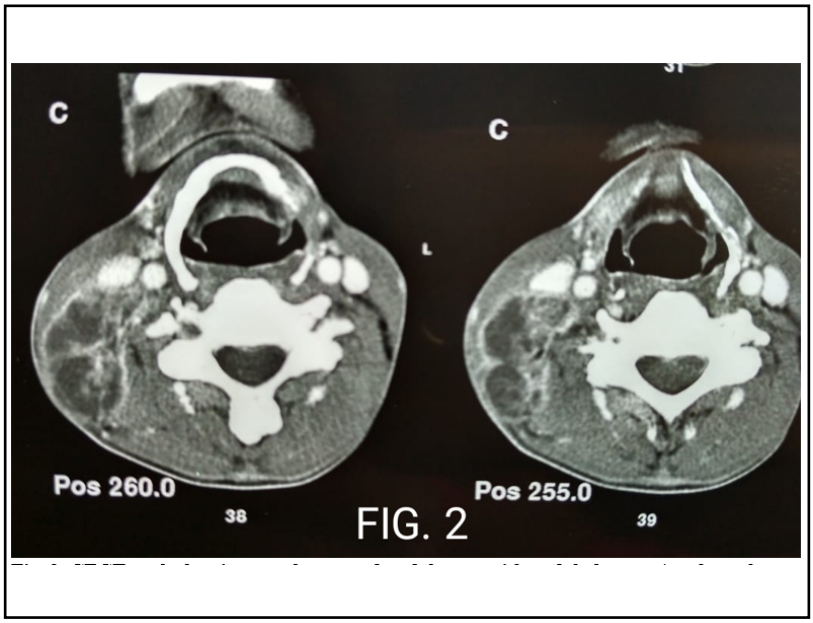

Fig. 2. CECT neck showing conglomerated nodal mass with nodal abscess s/o tuberculous cervical lymphadenopathy

Fig.4. Parotid gland swelling

Table III: Comparison of the present study with other studies

	PRESENT STUDY (2008-17)	PANDURANG ET AL. (2011-13)[18]	HAFEEZ ET AL. (2005-06)[19]	KHAN ET AL. (1987-88)[3]	SRIRAM ET AL. (2012-14)[15]	DAS ET AL. (STUDY PERIOD: NA)[20]
No. of patients	**120**	**70**	**100**	**135**	**104**	**63**
Lymph node	**77.50%**	**77%**	**97%**	**79.70%**	**86.50%**	**90.50%**
Deep neck spaces	**2.50%**	**10%**	**2%**	**6.80%**	**3.80%**	
Larynx	**8.30%**	**8.50%**	**1%**	**8.30%**	**4.80%**	**7.90%**
Salivary glands	**1.70%**	**3%**			**3.80%**	
Nose	**1.70%**			**2.90%**		
Middle ear	**7.50%**			**1.96%**		**1.60%**
Pharynx	**0**	**1.50%**			**1%**	
Thyroid gland	**0.83%**					
Presence of pulmonary TB	**26.67%**	**33%**		**28.90%**	**16.30%**	
Presence of HIV[a] infection	**5%**	**12.80%**			**26%**	

[a]*HIV=Human Immunodeficiency Virus*

well. Chest x-ray changes for pulmonary tuberculosis was seen in all these 32 patients (26.67%) while sputum for acid fast bacillus was positive in 27 patients (22.5%). All patients were started on anti-Koch's treatment (AKT) according to Revised National Tuberculosis Control Program guidelines and all of them responded well to the first line antituberculous drugs; except 1 patient who had to be given treatment for Multi drug resistant-TB to which he responded. Patients who were positive for HIV were started on anti-retroviral therapy, if not already taking it. No patient died in 1 year follow up period.

Discussion

Tuberculous cervical lymphadenopathy is the most common presentation of head and neck tuberculosis.[6] Tuberculous bacilli are carried to the lymph nodes commonly by lymphatics and occasionally by blood route. In lymphatic spread, tubercles are first formed in the cortex while in hematogenous spread, medulla of lymph node is the first part to be affected. Hence periadenitis and early matting is seen in lymphatic spread. Microscopically, it presents as a central zone of caseation necrosis which stains pink with eosin surrounded by the zone of epitheloid cells with few langhan's type of giant cells. In the outermost zone, few deeply staining lymphocytes with fibrous tissue can be seen.

Baskota et al. found that level V lymph node was most commonly involved (51%); while Jha et al. found upper jugular nodes (level II) to be most commonly affected, similar to our study.[7,8] FNAC was found to be the most efficient technique used for diagnosis similar to findings of study by Nalini and Vinayak.[9] Malakar et al. also found in their study that FNAC was sensitive to detect tuberculous lymphadenopathy in 79% patients.[10]

Laryngeal TB occurs mostly secondary to pulmonary

tuberculosis or may be primarily involved too.[11] In our study, it was the second most common lesion found. This agrees with the study by Choudhury et al.[12]

TB of middle ear cleft is very rare. Occurs primarily by hematogenous spread or secondary to pulmonary tuberculosis due to entry of bacteria through eustachian tube during cough or sneeze.[13]

Tuberculosis of nose is mainly secondary to pulmonary TB and presents with nasal discharge, nasal blockage, nasal ulcers or friable nasal masses which bleed easily and attached to the septum or inferior turbinate. Diagnosis of nasal TB is by histopathological examination of nasal masses.[14] TB of parapharyngeal space occurring without cervical spine caries is rare. In such patients, the focus of sepsis is usually in tonsil or pharynx. The infection can affect either the prestyloid or poststyloid or both the compartments. Definitive diagnosis is by isolation of M tuberculosis bacterium on smear or culture. CT scan is of great help in assessing the extent of the abscess.[15] Involvement of major salivary glands is uncommon and is usually secondary to pulmonary TB, but the tonsils or oral cavity may be the source of primary infection. Presentation is with local swelling, multiple fistula intraorally or extraorally, irregular low-grade fever and pain during mouth opening. Investigations for salivary gland TB include FNAC, USG, ZN staining and culture of discharge if any and salivary flow. Open biopsy is to be avoided because of danger of chronic fistula.[16]

TB of thyroid gland is extremely rare due to probable bactericidal action of the colloid and good vasculature of the gland. Symptoms are nonspecific and it may present with solitary thyroid nodule. Similarly, TB of thyroid gland can be diagnosed with ultrasonography, FNAC and polymerase chain reaction tests.[17]

Conclusion

Tuberculosis can affect almost every organ individually or in combination. The variable nature of manifestations of tuberculosis and the fact that it can mimic malignancy or other chronic granulomatous conditions make it essential to have high degree of suspicion for early diagnosis and prompt antituberculous treatment. As and when needed, appropriate investigations should be done to diagnose TB. AntiTB treatment for extra-pulmonary TB carries a good response rate.

References

1. Global tuberculosis report 2015 [Internet]. Geneva World Health Organization 2015. Available at: http://www.who.int/tb/publications/ global_report/en/. Accessed Mar 01, 2016
2. Aisenberg GM,Jacobson K,Chemaly RF, Rolston KV, Raad II, Safdar A. Extrapulmonary tuberculosis active infection misdiagnosed as cancer:mycobacterium tuberculosis disease in patients at a Comprehensive cancer centre(2001-2005). Cancer 2005;104(12):2882-87
3. Khan KA, Khan NA, Maqbool IM. Otorhinolaryngological manifestations of tuberculosis. JK Sci. 2002; 4(3):115-8
4. Two hour detection of MTB and resistance to rifampicin. Cepheid International 2011. https://www.cepheid.com/en. Accessed Jul 2018
5. RNTCP national strategic plan 2017-2025. NSP draft 2017-2025
6. Sharma S, Sarin R, Khalid U.K, Singla N, Sharma PP, Behera D. Clinical profle and treatment outcome of tuberculous lymphadenitis in children using DOTS strategy. Indian J Tuberc. 2010; 57:4-11
7. Baskota DK, Prasad R, Sinha BK, Amatya RC. Distribution of lymph nodes in the neck in patients of tuberculous cervical lymphadenitis. Acta Otolaryngologica 2004;124(9):1095-98
8. Jha BC, Das A, Nagarkar NM, Gupta R, Singhal S. Cervical tuberculous lymphadenopathy: changing clinical pattern and concepts in management. Postgrad Med J. 2001; 77:185-7
9. Nalini B, Vinayak S. Tuberculosis in ear,nose,and throat practices :its presentation and diagnosis. Am J Otolaryngol. 2006; 27(1):39-45.doi:10.1016/j.amjoto.2005.07.005
10. Malakar D, Jajoo I, Swarup K, Gupta OP, Jain AP, Pofee VW. A clinical evaluation of fine needle aspiration cytology in the diagnosis of lymphadenopathy. Ind J Tub. 1991;38: 17-9
11. Ling L, Zhou SH, Wang SQ. Changing trends in the clinical features of laryngeal tuberculosis: a report of 19 patients. Int J Infect Dis. 2010;14(3):230–5
12. Choudhury N, Bruch G, Kothari P, Rao G, Simo R. 4 years experience of head and neck tuberculosis in a South London Hospital. JR Soc Med. 2005; 98:267–9
13. Sahn SA, Davidson PT. Mycobacterium tuberculosis infection in the middle ear. Chest 1974; 66(1):104-06
14. S Bahadur, A Thakar. Specific chronic infections. In: Michael Gleeson, editor. Scott Brown's otorhinolaryngology,head and neck surgery, 7th Ed. England: Hodder Arnold; 2008; 1458-60
15. Sriram R, Bhojwani KM. Manifestations of tuberculosis in Otorhinolaryngology practice: A retrospective study conducted in a coastal city of South India. Indian J Otolaryngol Head Neck

Surg. 2017; 69(2):210-5

16. Dixit R, Sharma S, Nuwal P. Tuberculosis of oral cavity. Indian J Tuberc. 2008; 55(1):51-3
17. Yin TD, Wu W, Cao S, Li H. Analysis Of Misdiagnosis Of 4 Patients Of Tuberculosis Of Thyroid And Literature Review. Case Reports in Endocrinology (Internet). 2012 [cited 2015 Nov21]; 4 pages. doi:10.1155/2012/862595
18. Pandurang K, Sheno VS, Bhojwani K, Alva A, Prdead.V, et al. Tuberculosis in head and neck in India:down but not yet dead. .J Mycobac Dis. 2014; 4(2):148.doi:10.4172/2161-1068.1000148
19. Hafeez M, Inayatullah, Ahmad I, Zakirullah. Otorhinolaryngological manifestations of tuberculosis. Pak J Med Sci. 2011; 27(4):855–7
20. Das S, Das D, Bhuyan U, Saikia N. Head and neck tuberculosis:Scenerio in a tertiary care Hospital of North Eastern India. Journal of Clinical and Diagnostic Research, 2016; 10(1):MC04-MC07.

Outcomes of Swallowing Rehabilitation in Patients with Dysphagia: A Retrospective Study

https://doi.org/10.47210/bjohns.2020.v28i2.334

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ABSTRACT

Introduction

Swallowing, as the first phase of digestion, is one of the most complicated neuromuscular processes of the central nervous system. The presence of dysphagia can itself cause medical, psychosocial, and economic complications. A medical complication of dysphagia includes aspiration pneumonia, malnutrition, significant weight loss, and dehydration.

Materials and Methods

It was a retrospective study. In information regarding speech and swallowing therapeutics were studied from the routine clinical records by speech pathologist.

Results

The present retrospective study was focused on highlighting the outcomes of swallowing rehabilitation in patients with dysphagia in various aetiologies. It was observed that swallowing compensatory strategies, postural changes, rehabilitative manoeuvres, thermal stimulation, indirect and direct therapy showed tremendous improvement in patients with dysphagia with various aetiologies.

Conclusion

This study provides further information regarding the severity of swallowing physiology in disorders and its improvement subsequent to traditional swallowing therapy which will contribute to literature in Indian context.

Keywords

Deglutition Disorders; Rehabilitation; Outcomes

Swallowing, as the first phase of digestion, is one of the most complicated neuromuscular processes of the central nervous system. It involves multiple areas of the brain and a series of voluntary and involuntary muscular contractions. Oropharyngeal dysphagia is a highly prevalent clinical condition among stroke patients, but the prevalence of dysphagia is different in various studies, because of differences in the definition of dysphagia, the method of assessing swallowing function, the timing of swallowing assessment after stroke, and the number and type of stroke patients studied.[1,2] Overall, swallowing disorders (dysphagia with or without aspiration) are seen in about half (55%) of all stroke patients admitted to hospital.[3,4]

A medical complication of dysphagia includes aspiration pneumonia, malnutrition, significant weight loss, and dehydration[5] along with psychosocial because eating is a pleasurable and social activity, and inability to eat normally may affect patient morale and quality-of-life.6 Complications due to dysphagia especially include pneumonia, and managing infection also increases healthcare costs by increasing the length of hospital stay and increasing the need for expensive respiratory and nutritional support.[3,4]

To prevent and minimize these complications, diagnosis and management of dysphagia must be done

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as soon as possible by a speech-language pathologist.[5] Various rehabilitation techniques are utilized by a speech pathologist for management dysphagia. The traditional swallowing therapy includes Compensatory strategies, postural changes, rehabilitative manoeuvres, thermal stimulation, indirect and direct therapy.[6]

Compensatory medical approaches include: enteral feeding by means of a nasogastric tube or by percutaneous endoscopic gastrostomy, modification of food consistency, postural correction to facilitate bolus transition, reducing rate of eating and ensuring oral hygiene by conventional oral care.[7,8]

Other approaches are rehabilitative methods, including oral motor exercises; airway-protecting maneuver, thermal-tactile stimulation, and Shaker exercises.[6-10] Recently, neuromuscular electrical stimulation, biofeedback, and transcranial magnetic stimulation have been used as techniques for swallowing therapy.[11]

Some studies have only focused on early intervention, and do not consider the time at which swallowing rehabilitation should be initiated for optimal recovery.[12-15] Current state of the art in the management of dysphagia includes a wide variety of multidisciplinary practices. These practices range from simple diet adjustment to more invasive surgical interventions, depending on the nature and severity of the disorder. Because management of dysphagia is a relatively new specialization, the direct therapeutic interventions offered by the speech language pathologist have consisted largely of compensatory mechanisms by which the patient compensates for disordered swallowing with adjustments in diet consistency or behaviorally based manoeuvres.

The role of speech language pathologist is significant in the rehabilitation of swallowing disorders. It is the need at present to emphasize the importance of traditional therapy which helps in improvement in swallowing mechanism. There are a lot of studies presented in the western context emphasizing on swallowing therapy. Hence, there arises a need to focus on the same in Indian context. The present retrospective study documents outcomes of swallowing rehabilitation in a group of patients with dysphagia. This study provides further information regarding the severity of swallowing physiology in disorders and its improvement subsequent to traditional swallowing therapy which will contribute to literature in Indian context. Hence, the aim of the study is to evaluate the outcomes in swallowing rehabilitation in the patients with dysphagia.

Materials and Methods

It was a retrospective study. This has been approved by the Ethics committee of the hospital.

The subjects of this retrospective study observational study included adults and children from the age groups of 0 to 80 years. These were divided as follows: Group I- 0 to 10 years, Group II- 10 to 20 years, Group III- 21 to 40 years, Group IV- 41 to 60 years, Group V- 61 to 80 years. Individuals reporting or referred to the department of Audiology and Speech Pathology with neurological speech, language swallowing disorders, articulation, fluency, voice disorders for therapy were studied. The Clinical speech language swallowing proforma was used to evaluate patients and to diagnose the patients for speech language swallowing disorders. The proforma was to assess basically the above mentioned domains and for assessing the severity of dysphagia. Nair hospital bedside assessment and severity scale was used which is freely available for use. The severity is determined by adding all scores and the total score is compared with the severity scale.[16]

The information about the speech language and swallowing disorders therapeutics was considered for this study which was exclusively performed by the Speech Language Pathologist. The therapy outcomes of patients, who were diagnosed and referred by doctors from IPD and OPD of Neurology, Medicine, ENT, oncology and other departments of the hospital, were studied. This included patients from past 2 years (October 2016- October 2018) of speech language and swallowing therapy. The information based on therapeutics was studied in terms of the diagnosis, age groups gender, severity of dysphagia and rehabilitative outcomes. The severity of dysphagia was assessed pre and post therapy and compared. This further helped to determine if patients could be started with oral feeds and alternative mode of nutrition i.e. Ryle's Tube or Nasogastric Tube or PEG tube can be eliminated. The

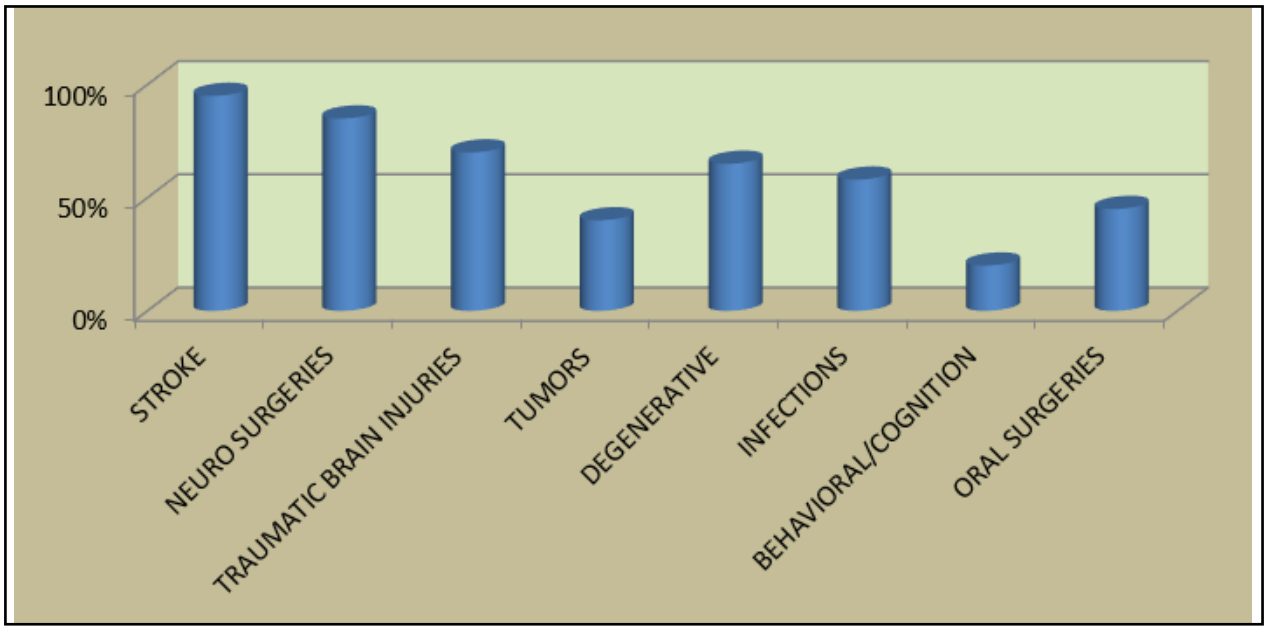

Fig. 1. Etiologies causing Dysphagia

analysis was done using percentage and non parametric tests Chi-square.

Results

All patients were individually evaluated as per the proforma and clinical swallowing, speech and language evaluation were done using bedside evaluation scales before initiating therapy. This study was based on outcomes of swallowing rehabilitation in patients with dysphagia in a tertiary care multidisplinary hospital. It was observed that the total number of dysphagia cases was 185 and sessions required were 690 to rehabilitate patients with dysphagia. This included various types of etiologies and severities of dysphagia. (Fig. 1) All individuals received therapy on one to one basis either bedside on or on OPD basis. The number of sessions for each individual was unequal depending on the recovery status. The recovery was based on the post therapy assessment using the severity scale of swallowing for each. (Table I) Those cases which were drop outs were not included in the study; rest were discharged based on recovery status.

The most common aetiology of dysphagia was stroke. (Fig. 1) Other causes of dysphagia were neurological surgeries, traumatic brain injuries, tumors, infections in central and peripheral nervous systems, degenerative neurogenic disorders, oral and neck surgeries and behavioral/cognitive etiologies. Behavioral or cognitive problems lead to lesser amount of dysphagia/ feeding issues. These etiologies can lead to dysphagia in 3 different phases i.e. oral preparatory, oral phase and pharyngeal phase.

Oral or head and neck surgeries due to oral cancers involving any oral structure i.e. tongue, buccal cavity, hard or soft palate, thyroid/parathyroid. Some patients

Table I: Pre and Post therapy Outcome measures

	SEVERITY RANGE OF DYSPHAGIA	SCORES
Pre therapy severity	**Severe to complete**	**5-6**
Post therapy severity	**Normal to slight**	**0-1**

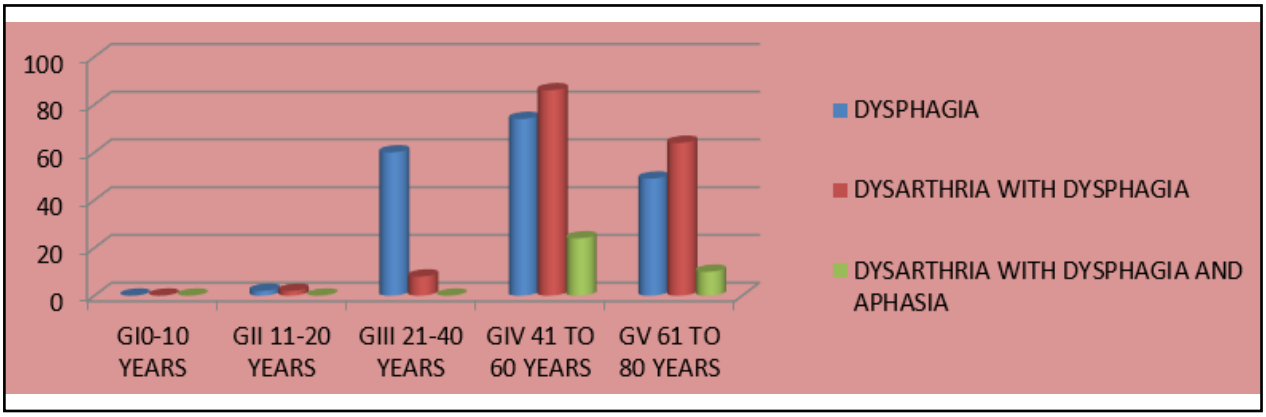

Fig. 2. Disorders as per age groups

undergoing mandibullectomy, glossectomy had oral preparatory phase dysphagia. Most of these etiologies are mechanical causes of dysphagia can affect the oral preparatory, oral and pharyngeal phase of swallow eg. Post thyroidectomy leading to vocal cord palsy. Few patients with Dementia had behavioral issues while feeding despite not having difficulty with swallowing mechanism. Most of the patients in the acute stage of dysphagia were on alternative mode of swallowing for nutrition i.e. through Nasogastric Tube/ Ryle's Tube; only few had Percutaneous Endoscopic Gastrostomy (PEG).

When results were studied as per age groups it was seen there was significant difference (Chi square<0.001) in all groups, while no difference as per gender. It was observed (Fig. 2) that maximum cases had dysphagia in age groups more than 21 years to 80 years of age. In the age groups of 41 to 60 years and Group 61 to 80 years it was observed patients had dysphagia, and also had dysphagia with other disorders i.e. dysarthria and aphasia. With increasing age groups there was more occurrence of dysphagia with other neurocommunication and motor speech disorders.

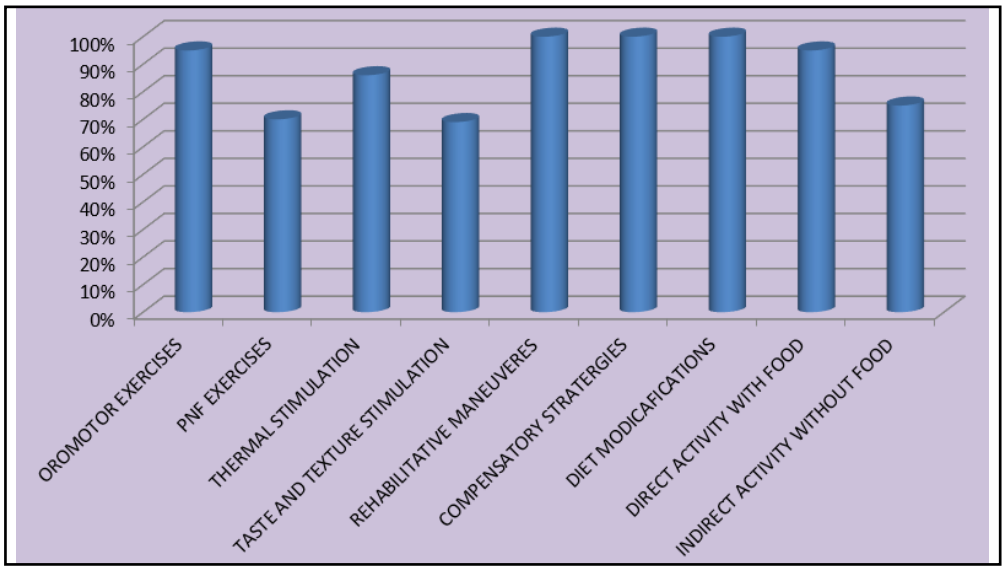

Fig. 3. Therapeutic Rehabilitative Management

Various therapeutic management options are utilized by Speech Pathologist to rehabilitate patients with dysphagia. The current treatment of dysphagia in patients with stroke or other etiologies is the traditional swallowing therapy by a speech pathologist. Compensatory strategies, postural changes, rehabilitative maneuvers, thermal stimulation, indirect and direct therapy were included in this therapy.[6] The therapeutic rehabilitative management included compensatory strategies, rehabilitative manoeuvres, thermal, taste and texture stimulation which is proprioceptive neuromuscular functioning exercises (PNF), oromotor exercises, diet modifications. All these were planned and used as indirect and direct methods in dysphagia rehabilitation. (Fig. 3)

Discussion

Rehabilitative maneuvers such as Masako, Guided Mendelson's, supraglottic and effortful swallow contributed almost 95% in rehabilitation. Compensatory strategies such as head positioning using chin tuck, chin up, head rotation to better side or head tilts were utilized as per different etiologies and phase of dysphagia. These behaviorally based maneuvers include techniques designed to improve airway protection, such as the Supraglottic swallow, Mendelson's, Masako and other manoeuvres designed to facilitate transfer of the bolus through the pharyngeal cavity by maximizing forces and minimizing resistance to the bolus, such as chin-tuck posturing.[9,10] These were used in patients with neurological disorders such as cortical strokes, brainstem stokes like lateral meduallary syndrome, cerebellar strokes, progressive disorders such as Parkinson's disease, Progressive supranuclear palsy, Amyotropic lateral sclerosis etc.[17,18]

These interventions, although serving an important role, provide an immediate but only transient approach to the underlying physiologic deficit. These are compensatory in nature. Development of rehabilitative exercises and substantiation of efficacy are needed to address long-term changes in swallowing physiology.19 Several techniques have been proposed as compensatory in intent, but clinical experience suggests that these techniques, when repeated in the context of an exercise regime, may facilitate overall change in swallowing physiology. The effortful swallow consists of a normal swallow in duration and sequence but with greater effort or force during the muscle contraction.[20-22]

This technique was initially conceptualized to facilitate bolus clearance from pharyngeal recesses as a result of increased tongue-base approximation to the posterior pharyngeal wall. The Mendelsohn manoeuvre was designed to address deficits associated with reduced opening of the upper oesophageal sphincter.[23-25] Execution involves the prolongation of a pharyngeal swallow when the thyroid cartilage is at its point of highest excursion, with the intent of maintaining opening of the upper oesophageal sphincter.[23,24] More recently, a tongue-holding manoeuvre, or the Masako manoeuvre, was developed based on the observation that patients with base of tongue resection demonstrate spontaneous increase in posterior pharyngeal wall excursion to compensate for the base of the tongue. Execution of this manoeuvre significantly facilitates posterior pharyngeal wall movement and thus may provide a direct rehabilitative exercise to address pharyngeal contraction.[26-29]

With the use of various techniques in this study, eventually diet modification was done to initiate direct swallowing therapy. This was done using thin, thick liquids, semisolids blended, soft and hard solids consistencies of food. While doing so, patient's oral intake was monitored 3 times a day in proportions. Also while they were all on oral feeds for nutrition; symptoms of dysphagia was observed such as chocking, gurgle voice, breathing difficulties, aspiration, coughing during and after swallow. As they developed and could manage adequate oral feed quantities without any difficulties, they were recommended to have complete oral feed.

Conclusion

Through this study it was seen that there is a major role of a Speech Pathologist in dysphagia management in a hospital setup. This helps us to learn the effectiveness of traditional dysphagia therapy and it outcomes in various etiologies leading to dysphagia. This study also helps to create awareness amongst other medical fraternity

about the role and importance to initiate therapeutic management at the earliest for patient benefits.

References

1. Martino R, Foley N, Bhogal S, Diamant N, Speechley M, Teasell R. Dysphagia after stroke: incidence, diagnosis, and pulmonary complications. Stroke 2005; 36 (12):2756-63
2. Mann G, Hankey GJ, Cameron D. Swallowing disorders following acute stroke: prevalence and diagnostic accuracy. Cerebrovasc Dis. 2000; 10(5):380-6
3. Langdon C, Blacker D. Dysphagia in stroke: a new solution. Stroke Res Treat. 2010;2010:570403. doi:10.4061/2010/570403
4. Langdon, PC, Lee AH, Binns CW. Dysphagia in acute ischaemic stroke: severity, recovery and relationship to stroke subtype. J Clin Neurosci. 2007; 14(7):630-4
5. Nilsson H, Ekberg O, Olsson R, Hindfelt B. Dysphagia in stroke: a prospective study of quantitative aspects of swallowing in dysphagic patients. Dysphagia 1998; 13(1):32-8
6. Vesey S. Dysphagia and quality of life. Br J Community Nurs. 2013 (Suppl: S14, S16, S18-S14, S16, S19)
7. Davis LA. Quality of Life Issues Related to Dysphagia. Topics in Geriatric Rehabilitation 2007; 23(4):352-65. doi: 10.4172/2329-9096.1000340
8. Murry T, Carrau RL. Clinical Management of Swallowing Disorders. 3rd ed. San Diego, CA: Plural Pub; 2012
9. Speyer R, Baijens L, Heijnen M, Zwijnenberg I. Effects of therapy in oropharyngeal dysphagia by speech and language therapists: a systematic review. Dysphagia 2010; 25(1):40-65
10. Logemann JL. Management of the patient with disordered oral feeding. In: Logemann JA, Ed: Evaluation and Treatment of Swallowing Disorders. Austin: Pro-Ed, 1983, 127-59
11. Huckabee ML. Outcomes of Swallowing Rehabilitation in Chronic Brainstem Dysphagia: A Retrospective Evaluation. Dysphagia1999; 14:93-109
12. Khedr EM, Abo-Elfetoh N. Therapeutic role of rTMS on recovery of dysphagia in patients with lateral medullary syndrome and brainstem infarction. J Neurol Neurosurg Psychiatry 2010; 81(5):495-9
13. Carnaby G, Hankey GJ, Pizzi J. Behavioural intervention for dysphagia in acute stroke: a randomised controlled trial. Lancet Neurol. 2006; 5(1):31-7
14. Takahata H, Tsutsumi K, Baba H, Nagata I, Yonekura M. Early intervention to promote oral feeding in patients with intracerebral hemorrhage: a retrospective cohort study. BMC Neurol. 2011; 11:6
15. Hagg M, Larsson B. Effects of motor and sensory stimulation in stroke patients with long-lasting dysphagia. Dysphagia 2004; 19(4):219-30
16. Babani N, Hattangadi GA. Bedside assessment protocol and grading scale for dysphagia in adults. Journal of Indian Speech Language & Hearing Association 2014; 28(1):10-24
17. Crary MA, Carnaby-Mann GD, Groher ME. Initial psychometric assessment of a functional oral intake scale for dysphagia in stroke patients. Arch Phys Med Rehabil. 2005; 86:1516-20
18. Kahrilas PJ, Logemann JA, Krugler C, Flanagan E. Volitional augmentation of upper esophageal sphincter opening during swallowing. Am J Physio. 1991; 260:G450-6
19. Kahrilas PH, Logemann JA, Lin S, Ergun GA. Pharyngeal clearance during swallow: a combined manometric and videofluoroscopic study. Gastroenterology 1992; 103:128-36
20. Kahrilas PJ, Lin S, Logemann JA, Ergun GA, Facchini F. Deglutitive tongue action: volume accommodation and bolus propulsion. Gastroenterology 1993; 104:152-62
21. Fujiu M, Logemann J. Effect of a tongue-holding maneuver on posterior pharyngeal wall movement during deglutition. Am J Speech Lang Pathol. 1996; 5:23-30
22. Fujiu M, Logemann J, Pauloski B. Increased Post-operative posterior pharyngeal wall movement in patients with anterior oral cancer: preliminary findings and possible implications for treatment. Am J Speech Lang Pathol. 1995; 4:24-30
23. Gupta H, Banerjee A. Case Report Recovery of Dysphagia in Lateral Medullary Stroke.Hindawi Publishing Corporation, Case Reports in Neurological Medicine 2014; Volume 2014, Article ID 404871, 4 pages http://dx.doi.org/10.1155/2014/404871
24. K Govathi N, Akanksha G, Sushant B, Arun G. Recovery Pattern of Dysphagia in Lateral Medullary Infarction- A Case Report. GlobOto 2017; 3(2): 555610. DOI: 10.19080/GJO.2017.03.555610
25. Rey-Matias R, Froilan C, Leochico D. Rehabilitation Techniques in Dysphagia Management among Stroke Patients: A Systematic Review. Int J Phys Med Rehabil. 2016; 4:340. doi:10.4172/2329-9096.1000340
26. Smithard DG, O'Neill PA, England RE, Park CL, Wyatt R, Martin DF, et al. The natural history of dysphagia following a stroke. Dysphagia 1997; 12(4):188-93
27. Waxman MJ, Durfee D, Moore M, Morantz RA, Koller W. Nutritional aspects and swallowing function of patients with Parkinson's disease. Nutr Clin Pract. 1990; 5:196-9
28. Shaker R, Kern M, Bardan E, Arndorfer RC, et al. Effect of isotonic/isometric head lift exercise on hypopharyngeal intrabolus pressure. Presented at the Fifth Annual Dysphagia Research Society Meeting [abstract]. Dysphagia 1997; 12:107
29. Shaker R, Kern M, Bardan E, Taylor A, et al. Augmentation of deglutitive upper esophageal sphincter opening in the elderly by exercise. Am J Physiol. 1997; 272:G1518-22.

Impact of Adenotonsillectomy on Weight Gain in Children

https://doi.org/10.47210/bjohns.2020.v28i2.335

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ABSTRACT

Introduction

'Failure to thrive' is considered to be an indication for adenotonsillectomy in children. There is contrasting evidence regarding weight gain in children following adenotonsillectomy. The objective of this study is to evaluate weight change post adenotonsillectomy.

Materials and Methods

A prospective study conducted on 45 children of age between 5 to 15 years, who underwent adenotonsillectomy in our hospital. Weight is calculated at three different period i.e. preoperative, postoperatively at 3 and 6 months and compared with standard Indian Academy of Paediatrics (IAP) weight for age.

Results

Out of 45 children 62.2% were less than 10 years of age with a male preponderance of 60%. 55.6% had normal birth weight. Preoperatively the average weight of the participants was 25.69 kg when compared to IAP normal weight for age which was 29.67 kg. Postoperatively at 6 months it was 28.19 kg as opposed to the expected weight of 31.88 kg.

Conclusion

The weight gain post adeno-tonsillectomy failed to meet the expected IAP value and was below the expected value. Hence there was no significant weight change following adenotonsillectomy.

Keywords

Tonsillectomy; Adenoidectomy; Weight Gain

The tonsils and adenoids are the secondary lymphoid organs located in the pharynx. They are part of the inner Waldeyer's ring. Due to their typical location i.e. at the entrance of the respiratory and the alimentary tracts, they are considered as a first line of defense against the infections. Hence the recurrent and chronic infection leads to enlargement of the tonsils and adenoids.

The defence function of the tonsils and adenoids are much greater in children than in adults. Thus, adenotonsillar hypertrophy is normal in early childhood, as an index of immunological activity. They usually increase in size throughout childhood and tend to regress in adolescence. But a chronic or massive hypertrophy of adenoids and tonsils can lead to upper airway obstruction and dysphagia respectively and its consequences.[1,2,3]

Adenotonsillectomy (T&A) is one of the common surgical procedures performed in the paediatric age group. Its indication has been changing from recurrent infection to hypertrophy and OSAS, due to the widespread usage of the effective oral antibiotics at the primary care level.[4,5,6]

In fact, failure to thrive has been an indication for T&A, because it was noted that there was a favourable postoperative weight gain especially in underweight children, which was attributed to reduced upper airway obstruction and dysphagia.[7,8,9]

Recently two retrospective studies reported a significant increase in BMI percentile after adenotonsillectomy, particularly in children younger

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than 6 years, although no significant increase in rates of obesity was observed.[10,11] There are reports which show that only tonsillectomy had a positive influence on somatic growth postoperatively, while others found that adenoidectomy is as effective as tonsillectomy or adenotonsillectomy.[12]

Owing to the lack of proper conclusion from the previous studies and as there is a paucity of research in India with respect to weight gain following T&A, we decided to conduct a study on influence of T&A on weight in children between 5 to 15 years.

Materials and Methods

All consenting patients aged between 5 to 15 years attending outpatient department of ENT in our hospital diagnosed with Adenotonsillar Hypertrophy, during the study period of 2 years were included. Patient with concurrent diseases that may affect growth were excluded.

All the patients underwent clinical examination, pre-operative routine haematological investigations, measurement of weight, X ray nasopharynx or flexible Nasopharyngoscopy to look for adenoid size preoperatively. Irrespective of type of adenotonsillectomy, postoperatively. Patients were followed up after 3 months and 6 months after surgery and weight measurement was done. Children were weighted in their indoor clothing and without shoes. The measurement was plotted in Indian Academy of Pediatrician (IAP) weight for age growth chart in percentile (for boys and girls) and the IAP 50th percentile was taken as median value. The results were correlated after applying statistical tests.

The data collected was entered in Microsoft excel and analysed using IBM SPSS Version 22. Qualitative data was represented in the form of frequency and percentage. Association between qualitative variables was assessed with Fisher's exact test. Mean & SD value was calculated for continuous variables. Inter variable comparison was done with unpaired t test, and within the group comparison were done with paired t test. P value of <0.05 was considered statistically significant.

Results

We studied 45 children between 5 to 15 years of age. We classified them into two groups by dividing them according to their age for analytical purposes. First group was children aged 5 to 10 years and the other group had children aged 11 to 15 years. 28 belonged to the age group of 5 to 10 years and rest 17 were between 11 to 15 years i.e. 62.2% and 37.8% respectively. In our study, mean age at the time of surgery was 9.32 years. Out of 45 patients, 60% (27) were males, 40% (18) were females.

On analyzing preoperative weight distribution 7 children were in normal weight group and 38 were in below normal weight i.e. 15.6% and 84.4 % respectively. Postoperative distribution of weight after 6 months showed, 6 children were in normal weight group and 39 were in below normal weight group i.e. 13% and 87 % respectively. The percentage of below normal weight in preoperative (84.4%), and postoperative (86.7%) period were more or less same, hence there was no significant change in weight. (Fig. 1)

There was no significant change in weight between two age groups, during the preoperative and postoperative period.

Results showed that preoperatively children were below the expected weight for the age. At postoperative 3months and 6 months following T&A children failed to reach the expected weight for the age. (Table I)

Discussion

The present study was undertaken to determine whether adenotonsillectomy during childhood is associated with change in weight. This topic is still controversial, because many studies were showing positive results i.e. weight gain after adenotonsillectomy but some of the studies conclude that there was no weight gain following adenotonsillectomy.

Majority of the children at the time of surgery belonged to younger age group i.e. less than ten years which was comparable to other similar studies.[11,13,14] On reviewing the literature for the effect of adenotonsillectomy surgery on weight gain in children,

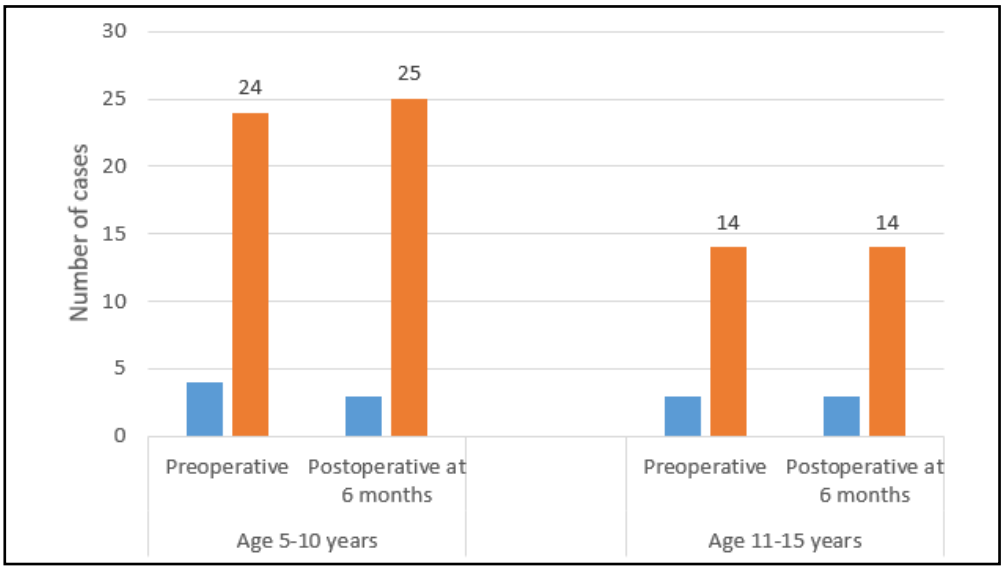

Fig. 1. Correlation of age and weight

there was contrasting evidence noted. Few studies mentioned favourable results in terms of weight gain following adenotonsillectomy.[10,11,12] And in some studies the results failed to show any significant weight gain following adenotonsillectomy.[15,16] In our study, there was no difference in weight changes between the two arbitrary age groups.

Also, in our study 84.4% of the children were below 50th percentile of the expected weight. This can be attributed to the rampant malnourishment in Indian paediatric population. Similar findings were noted in other studies.[17,18] Czechowicz and his colleagues in their study noted that Asian children had lower weight percentiles compared to their African and European counterparts and that intervention in the form of adenotonsillectomy at a younger age group was associated with significant weight gain.[10] This was especially true for children with weight percentiles in the lower range. Also, of particular importance mentioned in the above study is the point that the weight gain did not plateau until after 1 year of surgery. The immediate post-operative period is associated with decreased weight owing to the pain associated with surgery. Hence even though there is conflicting evidence regarding the role of adenotonsillectomy in children with failure to thrive, the findings of this study do not document any significant weight changes at the end of 6 months follow-up.

Conclusion

Majority of children in our study had below normal weight during preoperative period based on IAP weight for age chart median percentile. There was no significant weight gain noted after adenotonsillectomy in both normal and below normal group based on median

Table I: Mean weight of study group and IAP 50th percentile

WEIGHT WITH RESPECT TO AGE (KILOGRAMS)	PREOPERATIVE	AFTER 3 MONTHS	AFTER 6 MONTHS
According to IAP Weight	**29.67**	**30.75**	**31.88**
Actual Weight	**25.69**	**26.81**	**28.19**

IAP- Indian Academy of Pediatrics

percentile of IAP weight for age chart. Hence, we conclude that there is no impact of adenotonsillectomy on weight gain in children.

There are few drawbacks in our study. We have not taken control group of similar age and gender for comparison. Only weight of the children was taken for growth development, other parameters were not evaluated. Long term follow-up needed for definitive conclusion on effect of adenotonsillectomy on weight. The effect of adenotonsillectomy on parameters other than weight like, height, BMI, growth hormone levels, will give more valuable conclusion. The effect of adenotonsillectomy on growth can be studied more comprehensively if we have case-cohort groups who are matched for other parameters to eliminate selection bias.

References

1. Baradaranfar MH, Dodangeh F, Atar ST. Humoral and cellular immunity parameters in children before and after adenotonsillectomy. Acta Medica Iranica 2007;345-50
2. Gray LP. The T's and A's problem-assessment and reassessment. J Laryngol Otol. 1977; 91(1):11-32
3. Böck A, Popp W, Herkner KR. Tonsillectomy and the immune system: a long-term follow up comparison between tonsillectomized and non-tonsillectomized children. European archives of Oto-rhino-laryngology 1994; 251(7):423-7
4. Grundfast KM, WittichJr DJ. Adenotonsillar hypertrophy and upper airway obstruction in evolutionary perspective. The Laryngoscope 1982; 92(6):650-6
5. Lind MG, Lundell BP. Tonsillar hyperplasia in children: a cause of obstructive sleep apneas, CO2 retention, and retarded growth. Archives of Otolaryngology 1982;108(10):650-4
6. Deutsch ES. Tonsillectomy and adenoidectomy: changing indications. Pediatric Clinics of North America 1996; 43(6):1319-38
7. Schiffmann R, Faber J, Eidelman AI. Obstructive hypertrophic adenoids and tonsils as a cause of infantile failure to thrive: reversed by tonsillectomy and adenoidectomy. Int J Pediatr Otorhinolaryngol. 1985; 9(2):183-7
8. Williams III EF, Woo P, Miller R, Kellman RM. The effects of adenotonsillectomy on growth in young children. Otolaryngol Head Neck Surg. 1991 Apr;104(4):509-16
9. Hodges S, Wailoo MP. Tonsillar enlargement and failure to thrive. British Medical Journal (Clinical research ed.) 1987; 295(6597):541
10. Czechowicz JA, Chang KW. Analysis of growth curves in children after adenotonsillectomy. JAMA Otolaryngol Head Neck Surg. 2014; 140(6):491-6
11. Smith DF, Vikani AR, Benke JR, Boss EF, Ishman SL. Weight gain after adenotonsillectomy is more common in young children. Otolaryngol Head Neck Surg. 2013; 148(3):488-93
12. Vontetsianos HS, Davris SE, Christopoulos GD, Dacou-Voutetakis C. Improved somatic growth following adenoidectomy and tonsillectomy in young children. Possible pathogenetic mechanisms. Hormones (Athens) 2005; 4(1):49-54
13. Selimoğlu E, Selimoğlu MA, Orbak Z. Does adenotonsillectomy improve growth in children with obstructive adenotonsillar hypertrophy? Journal of International Medical Research 2003; 31(2):84-7
14. Ahlqvist-Rastad J, Hultcrantz E, Melander H, Svanholm H. Body growth in relation to tonsillar enlargement and tonsillectomy. Int J Pediatr Otorhinolaryngol. 1992; 24(1):55-61
15. Roche AF. The influence of tonsillectomy on growth and caloric intake. J Pediatr. 1964; 65(3):360-7
16. Mills RP, Hibbert J. The effects of recurrent tonsillitis on growth and cervical lymphadenopathy in children. Int J Pediatr Otorhinolaryngol. 1983;6(1):77-82. doi:10.1016/s0165-5876(83)80105-0
17. Aydogan M, Toprak D, Hatun Ş, Yüksel A, Gokalp AS. The effect of recurrent tonsillitis and adenotonsillectomy on growth in childhood. Int J Pediatr Otorhinolaryngol. 2007; 71(11):1737-42
18. Conlon BJ, Donnelly MJ, McShane OP. Tonsillitis, tonsillectomy and weight disturbance. Int J Pediatr Otorhinolaryngol. 1997; 42(1):17-23.

Diagnostic Accuracy of FNAC Diagnosed Benign Thyroid Lesions Compared with Post-Operative Histopathology Results

https://doi.org/10.47210/bjohns.2020.v28i2.336

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ABSTRACT

Introduction

Fine Needle Aspiration Cytology (FNAC) is one of the important diagnostic tools in diagnosing the thyroid diseases, however incidental thyroid malignancies among patients operated for a benign thyroid disease is not infrequent. The incidence ranges between 3 and 16%. The clinical significance of these tumours cannot be ignored once a correct staging has been carried out. The objectives of this study were to assess the diagnostic accuracy of FNAC of Thyroid gland and to know the incidence of incidental thyroid malignancy.

Materials and Methods

This was a prospective study conducted from August 2018 to December 2019, after taking institutional ethical clearance, with a study population of 72 patients with thyroid swelling of either sex between age group (18-72yrs). Pre operative FNAC was done and underwent thyroid surgery in our hospital. Post operative thyroid specimen were sent for histopathological examination. All reports were collected and analyzed statistically.

Results

Total 72 patients were included within age group of 18-72yrs among them, 8 were males and 64 were females having biologically normal thyroid function test. FNAC was showed 91% sensitivity and 68% specificity in diagnosing the colloid goiter and also 62% sensitivity and 75% specificity in diagnosing the nodular goitre. However, 8(11.1%) patients were showing incidental thyroid malignancy postoperatively.

Conclusion

FNAC is an effective tool in diagnosing the thyroid diseases; however incidental thyroid malignancy should also be considered and correlated with clinical diagnosis.

Keywords

FNAC; Thyroid Neoplasms; Sensitivity and Specificity; Data Accuracy

The burden of thyroid disease in the general population is enormous. Thyroid disorders are the most common among all the endocrine diseases in India. Thyroid nodules are most often be only benign or can be associated with malignancy or develop malignancy over a period of time. Most of the thyroid nodules are benign and fewer than 5% of them are actually malignant. Of all patients with grave's disease, approximately 30% presents with palpable or radiological nodules. Out of which malignant potential is present in 17% as compared to incidence of 5% usually found in the general population. Many cases treated as Hashimoto's thyroiditis may have an associated malignancy. FNAC of the thyroid gland is safe, inexpensive, minimally invasive, and highly accurate in the diagnosis of nodular thyroid disease.

Though FNAC has revolutionized the management of thyroid nodule, the accuracy of cytological diagnosis is highly dependent on the person performing the aspiration and the person interpreting it. Analysis of

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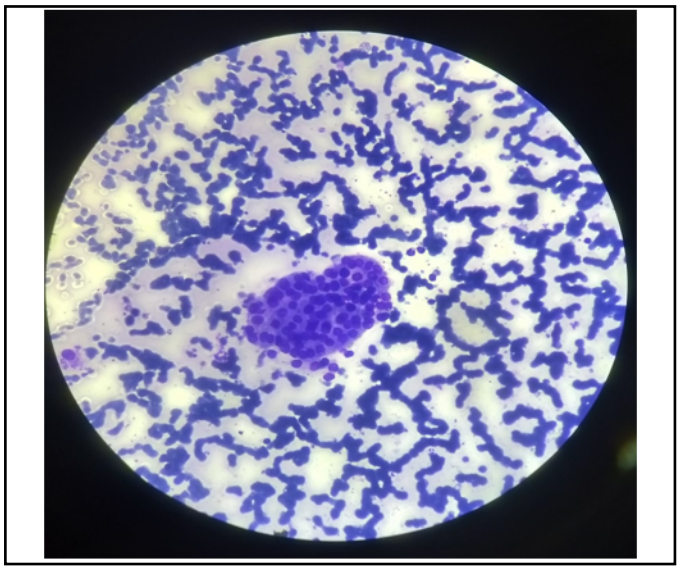

Fig.1. FNAC showing nodular goitre of a patient with thyroid swelling (H&E, 40x)

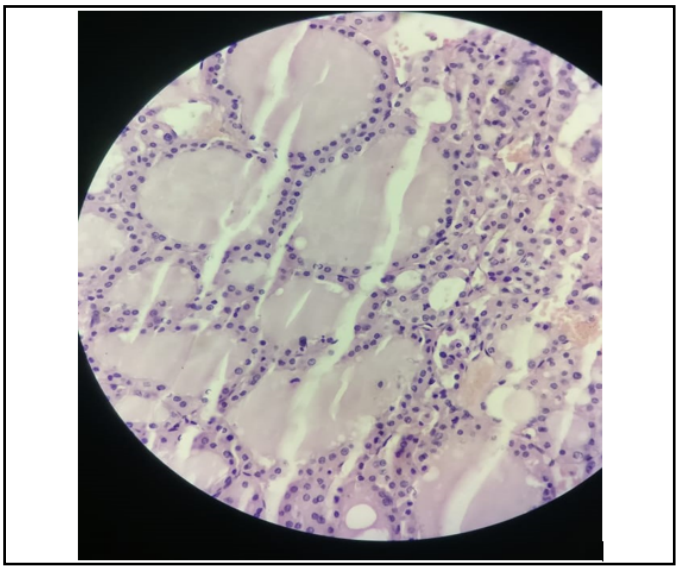

Fig.2. Postoperative histopathology showing nodular goitre of the Fig.1 (H&E, 40x)

recent data suggests a false-negative rate of 1-11%, a false-positive rate of 1-8%, a sensitivity of 65% to 98%, and a specificity of 72% to 100%.[1] Studies show that 2–15% of FNAC are insufficient to diagnose, 50–70% benign, 15– 30% suspicious and 5–10% malignant.[2] Limitations of FNAC are related to the skills of the aspirator, the experience of the cytologist, and the difficulty in distinguishing some benign cellular adenomas from their malignant counterparts. This is a study for assessing the diagnostic accuracy of fine needle aspiration cytology diagnosed benign thyroid lesions with post operative histopathology results and to

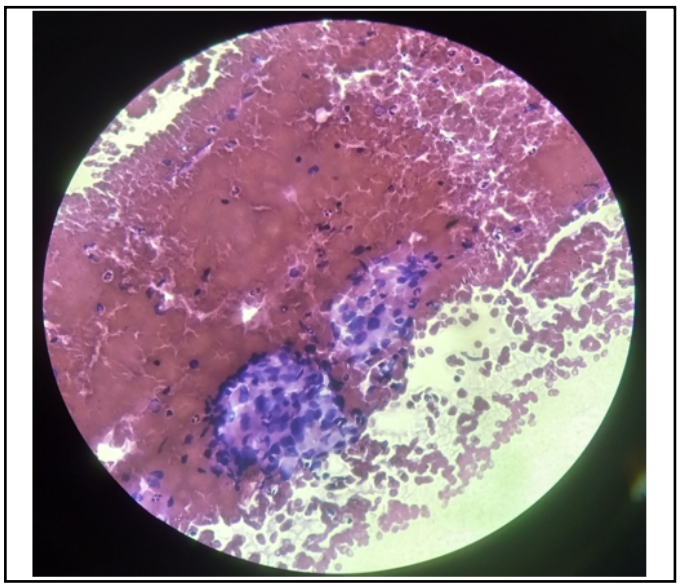

Fig. 3. FNAC showing colloid nodular goitre of a patient with thyroid swelling (H&E, 40x)

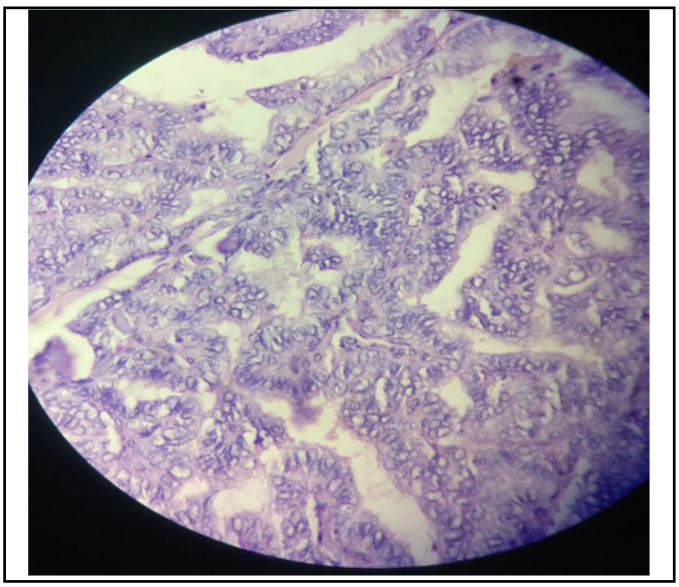

Fig.4. Postoperative Histopathology Showing Micro Papillary Carcinoma (H&E, 40x)

Table I: Diagnostic Accuracy of FNAC of Nodular Goitre compared with HPE

FNAC	HPE		TOTAL
	COLLOID GOITRE	OTHERS	
Colloid Goitre	20(91%)	16	36
Others	2	34(68%)	36
Total	22	50	72

identify the incidence of incidental thyroid malignancy post operatively in patients treated surgically for FNAC proved benign thyroid disease.

Materials and Methods

This prospective study was carried out on patients with thyroid swelling, of either sex between age group (18-72 years) who, considering the inclusion and exclusion criteria, agreed to participate in study were included. Inclusion criteria includes, all the patients with thyroid swelling undergoing thyroid surgery, where FNAC, prior to thyroid surgery, showed benign thyroid disease. Exclusion criteria excludes, all the patients undergoing thyroid surgery, where FNAC prior to surgery showed malignant thyroid disease. This study was done from August 2018 to December 2019 with a sample size of 72 and sampling method being convenience sampling. After detailed history and examination of the patients, pre-operative investigations and FNAC was done and consent for surgery was taken and underwent Thyroidectomy. All patients underwent intracapsular thyroidectomy. Specimen sent for Histopathological examination and reports were collected and statistical analysis was done. Categorical data is represented in the form of percentages. Statistical tests applied are Correlation, Sensitivity, Specificity, Positive Predictive Value, Negative Predictive Value, Accuracy.

Results

Data analysis was done with the software IBM SPSS Version 22.0 for Windows.

In our study, 72 patients were included of which, 8 patients were male and 64 were female with age group between 18-72 years. The mean age of the study patients was 39.9±12.3 years. All patients were having normal biochemical thyroid function tests. Fine needle aspiration cytology (FNAC) of thyroid was done in all patients; of 72 patients' specimens, 36(50%) were having colloid goitre, 30(42%) were having nodular goitre, 2(2.81%) were having Hashimato's thyroiditis and 4(5.63%) patients were having Adenomatous nodule. After the final histopathology, of 72 patients' specimens, 22(30.5%) specimens showed Colloid goitre, 28(39%) specimens showed Nodular goitre, 4(5.63%) specimens showed Hashimato's thyroiditis, 5 had lymphocytic thyroiditis, 3 specimens showed follicular adenoma, 2

Table II: Diagnostic Accuracy of Fnac of Nodular Goitre compared with HPE

FNAC	HPE		TOTAL
	NODULAR GOITRE	OTHERS	
Nodular Goitre	13(62%)	13	26
Others	8	38(75%)	46
Total	21	51	72

specimen showed adenomatous nodule and 8(11.1%) patients specimen showed malignancy features, 6 had papillary carcinoma and 1 each had follicular and medullary carcinoma. FNAC accuracy in diagnosing the nodular goitre and colloid goitre was calculated statistically. (Table I & II) Incidental thyroid malignancy was 11.1% which can be comparable with other studies.

Discussion

Though thyroid gland is a superficial organ easily identifiable in the neck, at times it will be difficult to differentiate a malignant gland from a benign thyroid gland. There is a possibility of malignancy associated with benign disorders.[2] Multinodular goitre, nodules in hypothyroid patient, cystic thyroid nodule may all possess a focus of papillary carcinoma and by cytology alone, it may not possible to differentiate these nodules into benign and malignant. It becomes difficult for the surgeon to make a correct decision on treatment modality, thus making the role of surgery important in doubtful thyroid swellings. On the other hand, if patients not diagnosable by FNAC are treated medically, it is possible the tumour in them may progress leading to delay in the management of thyroid cancer. Current indications of total thyroidectomy are toxic and non-toxic multinodular goiter, Hashimoto's thyroiditis, Graves's disease and malignancies either with a less aggressive clinical course (e.g., papillary thyroid carcinoma) or a rapidly progressive course (e.g. anaplastic thyroid cancer).

FNAC was done for 247 patients by Harshwardhan et al.[2] Cytopathological analysis showed 132 cases of papillary thyroid cancer, 7 cases of Medullary thyroid carcinoma and 2 cases of Anaplastic thyroid carcinoma. Out of 106 FNAC proven benign cases, 90 cases had undergone total thyroidectomy, 51 cases were found malignant in final HPR. The incidence of malignancy was 48 % in overall benign thyroid lesions in which association of malignancy was highest for follicular neoplasm (71%) followed by adenomatous hyperplasia (57%).

In another study with FNAC proved 1054 (64.3%) benign cases, 128 (7.8%) malignant, 306 (18.66%) suspicious and 151 (9.2%) inadequate for diagnosis, of the 192 cases, which underwent surgery, FNAC reports were compared with histopathologic results and stastical indices were calculated. The sensitivity, specificity, and accuracy were 91.6%, 100% and 97% respectively.[3]

Muthu and Saravanakumar (2019) examined 54 specimens of solitary thyroid nodules histopathologically. The incidence of malignancy in the present series is 18.51%.

In the series of Faisal et al., 211 patients underwent thyroidectomy. The female (n=179) to male (n=32) ratio was 5.6 : 1, and the age ranged from 18 to 75 years. The peak age incidence was in the fifth decade of life (60 patients). The incidental carcinoma was found in 22 (10.4%) patients. The frequency of papillary carcinoma was 8.5% of all thyroid diseases and 81.8% of the incidental thyroid malignancies (18/22). Moreover, the frequency of follicular carcinoma was 1.4% of all thyroid swewllings (three cases) and 13.6% of incidental thyroid malignancies.[5]

Out 72 patients in this series, 8(11.1%) patients were having incidental thyroid malignancy. Among 8 patients 6 patients were papillary malignancy (8.33% of all thyroid swellings and 75% of all incidental thyroid malignancies), 1 patient was diagnosed with medullary carcinoma (1.38% of all thyroid swellings and 12.5% of all incidental thyroid malignancies), 1 patient was diagnosed with follicular carcinoma (1.38% of all thyroid swellings and 12.5% of all incidental thyroid malignancies).

Conclusion

FNAC is an effective tool in diagnosing the thyroid diseases with specificity 68% and sensitivity of 91% in diagnosing colloid goitre and specificity of 75% and sensitivity of 62% in diagnosing nodular goitre, however incidental thyroid malignancy should also be considered and correlated with the clinical diagnosis.

References

1. V.Kopperundevi. Histopathological review of thyroid swellings a retrospective study. Volume 2; Issue 5. International Journal of Medical and Health Research 2016; 2(5):10-3
2. Harshwardhan D, Pokharkar, Neharika. Thyroid cancer

in benign thyroid diseases: Incidence and role of total thyroidectomy. International Journal of Applied Research 2018; 4(7):1-5

3. Nyyef A, Hassan AJ, Aljaoha RK. The role of FNAC in diagnosis of papillary carcinoma of thyroid. Volume 6, Issue 1. IOSR Journal of Dental and Medical Sciences 2013; 6(1):7-13
4. S. Muthu, R.Saravanakumar. A prospective study of incidence of malignancy in solitary nodule of thyroid. International Journal of Contemporary Medical Research 2019; 6(5):E24-6
5. Faisal M, Fathy H, Risk A, Atwa MM. V37 I3 P(361-367) Incidental thyroid carcinoma after thyroidectomy for benign thyroid disease in Suez Canal region. The Egyptian Journal of Surgery 2018; 37(3):361-7.

Analysis of Microbes and their Sensitivity Patterns in Chronic Otitis Media in West Bengal

https://doi.org/10.47210/bjohns.2020.v28i2.337

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ABSTRACT

Introduction

Chronic otitis media (COM) is a commonly encountered condition in India because of socio-economic factors. Empirical antimicrobial therapy is crucial till definitive surgical management can be done. Periodic updating of prevalence and antibiogram of the etiological microorganisms of COM is thus important.This hospital based study aimed to detect the ongoing trend of microbes associated with chronic otitis media in eastern India and determination of antibiotic sensitivity patterns of bacteria.

Materials and Methods

A prospective study was conducted wherein ear swabs were taken from discharging ears of selected patients and sent for culture and antibiotic sensitivity tests.

Results

One hundred and forty two (142) samples were collected from 104 patients. 124 samples revealed monomicrobial involvement while 5 samples did not reveal any pathological organism. Pseudomonas aeruginosa and Staphylococcus aureus were the most common isolates comprising 31.33 % and 30.67 % respectively. Most organisms were susceptible to fluoroquinolones and aminoglycosides along with imipenem, meropenem followed by penicillin group of antibiotics.

Conclusion

The huge burden of chronic otitis media patients in India makes it essential to have an evidence-based protocol for initiation of empirical treatment. Hence, an idea about the microbes commonly responsible for disease and their antibiotic sensitivity patterns is helpful in clinical practice.

Keywords

Otitis Media; Microbial Sensitivity Tests

Chronic otitis media (COM) is a commonly encountered condition in India because of socio-economic factors like malnutrition, overcrowding and poor hygiene, inadequate healthcare, and recurrent upper respiratory tract infection.[1]

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Though the main aim of treatment of COM lies in surgical repair, it is necessary to eliminate the infection before surgery or to combat complications of COM with the help of antibiotics. If we fail to manage cases with empirical antibiotics, culture and sensitivity of the ear discharge are usually done to know about the causative organisms, their antibiotic susceptibility patterns. Most cases of complicated COM do not give us ample time for culture and antibiotic sensitivity tests. In those cases, we must administer some broad-spectrum antibiotic to combat the situation promptly to avoid further aggravation of the condition. So, the choice of antibiotics always plays a crucial role in managing COM.

The prevalence and antibiogram of causative organisms of COM have been reported to vary with

time and geographical areas. This is probably due to recurrent mutations of the causative microorganisms as a result of indiscriminate use of antibiotics. Hence, periodic updation of prevalence and antibiogram of the etiological microorganisms of COM would help to manage those cases more effectively.

In different studies three to four decades back, Pseudomonas and Proteus were the most common microbial isolates in patients with COM.[2,3] The incidence of community-acquired methicillin-resistant Staphylococcus aureus (MRSA) has increased over the years. It is now the most common isolate reported, followed by methicillin-sensitive S. aureus (MSSA).[4,5] Besides, different studies show there is a gradual increase in the involvement of gram negative bacteria like Enterobacteriaceae family along with the involvement of different anaerobic bacteria and fungi. The causative organisms also differ between mucosal and squamous varieties of COM. Squamous variety of diseases are almost thirty times more likely to have mixed infections compared to the mucosal variety of disease, which is usually monomicrobial.[6]

This study aimed to detect the ongoing trend of microbes associated with chronic otitis media in Eastern India and the determination of antibiotic sensitivity patterns of bacteria. Due to the absence of a recent data, this study was designed to see the recent trend and make empirical treatment of COM more effective.

Materials and Methods

This prospective hospital based study was conducted in the Department of Otorhinolaryngology – Head and Neck Surgery, in collaboration with the Department of Microbiology, in a tertiary care hospital in Kolkata between September 2012 to August 2014. Patients attending the outpatient clinic and diagnosed to have active chronic otitis media were considered for inclusion and a sample selected at random using random number tables. Patients who had already received topical or systemic antimicrobials in the past three months were excluded from consideration.

Ear swabs were taken with a sterile swab from discharging ears of the selected patients. Care was taken to ensure that the swab was taken from the deeper part of the external auditory canal using sterile ear speculum to avoid contact between the swab stick and outer part of the external auditory canal. The swab stick was then placed inside a sterile test tube and sent immediately to the microbiology department for culture and antibiotic sensitivity test.

After ear swabs were taken, wet mopping was done to clean the external auditory canal and clinically classify the ear as a mucosal or squamous variety of COM. Data was collected and subsequently analysed using commercially available software.

Results

A total of 104 patients were selected as per the previously described methods. Among them, 66 patients had unilateral ear discharge, while the remaining 38 patients had bilateral ear discharge. The total number of samples studied was 142.

There was almost equal gender distribution among the patients with 49 (47.11%) male patients and 55 (52.89%) female patients. The age of the patients ranged from 1 year to 60 years. The maximum number of cases were within the 2nd decade, that is in the age group of 11-20 years (n=35, 33.65%).

Among 142 discharging ears, 98 ears had active mucosal variety comprising 69.01% while 44 ears had active squamous type comprising 30.99%.

Out of the 142 samples, 124 samples revealed monomicrobial involvement. Polymicrobial involvement was seen in 13 samples, with each sample having two types of bacterial or fungal species. (Table I) 5 samples did not reveal any pathological organism. Therefore, the total number of isolates for sensitivity testing from 142 samples was 150.

Pseudomonas aeruginosa and Staphylococcus aureus were the most common isolates comprising 31.33 % and 30.67 % respectively. Klebsiella and Proteus sp. were found in 8.67 % and 7.33% cases, respectively. Some fungal species like Aspergillus and Candida sp. were also detected. (Table II)

Among 150 isolates, only five samples (3.33%) revealed anaerobic bacilli as a causative organism. It

Table I: Distribution of different organisms isolated from ear discharge samples

ISOLATED ORGANISMS	MONOMICROBIAL	POLYMICROBIAL	CULTURE NEGATIVE
Pseudomonas aeruginosa	45	2	
Staphylococcus aureus	35	11	
Streptococcus pneumoniae	3	0	
Klebsiella sp.	9	4	
Proteus sp.	6	5	
Acinetobacter sp.	13	0	
E. coli	2	0	
Aspergillus sp.	5	1	
Candida sp.	3	1	
Anaerobes	3	2	
No growth or only commensals			5
Total number of samples (n=142)	124	13	5

was noted that all the 5 samples showing anaerobic organisms were drawn from an active squamous variety of ear disease. In three cases, anaerobic bacteria were isolated as a monomicrobial involvement while in the other two cases, it was isolated along with Staphylococcus aureus. Streptococcus pneumoniae and E. coli were detected only in 3 and 2 samples, respectively. Among 13 samples where there was mixed flora, the most common gram-positive organism found was Staphylococcus aureus, the second microbe usually being Klebsiella, Proteus or anaerobes.

All the causative bacteria except anaerobes had undergone antimicrobial susceptibility testing using Mueller Hinton agar. Fungal isolates were also excluded

Table II: The distribution of different organisms

ISOLATED ORGANISMS	NUMBERS	PERCENTAGE (%)
Pseudomonas aeruginosa	47	31.33
Staphylococcus aureus	46	30.67
Streptococcus pneumoniae	3	2
Klebsiella sp.	13	8.67
Proteus sp.	11	7.33
Acinetobacter sp.	13	8.67
E. coli	2	1.33
Aspergillus sp.	6	4
Candida sp.	4	2.67
Anaerobes	5	3.33
Total	150	100

from antimicrobial sensitivity patterns. We found most of the organisms were susceptible to fluoroquinolones and aminoglycosides along with imipenem, meropenem followed by penicillin group of antibiotics. (Table III)

Discussion

COM is a significant public health problem, and India is one of the countries with high prevalence where urgent attention is needed.[7] It is a significant cause of preventable hearing loss, particularly in the developing world,[8] and a serious concern in children because it may have long-term effects on early communication, language development, auditory processing, educational process, and physiological and cognitive development.[7] Early microbiological diagnosis ensures prompt and specific treatment to avoid such complications.

Predominant bacterial aetiology (aerobic) of COM in this region is Pseudomonas aeruginosa (31.33%), and this observation was very close to the views by other researchers like studies in Nagpur (41.5%),[9] in DHQ (45.9%), in Hyderabad (54%).[10] In contrast, other studies from different areas showed different trends as Staphylococcus aureus was the most prevalent organism in studies in Uttarakhand (48.69%),[11] in Kathmandu (32.2%),[12] in County Hospital, Bosnia (30.6%)[13] and this could be due to the variation in the prevalence of microorganisms or effects of local conditions. In our study, we could isolate Staphylococcus aureus in 30.67% of cases which is remarkably close to the isolates of Pseudomonas. Therefore, as per our research, both Pseudomonas aeruginosa and Staphylococcus aureus predominate the clinical prevalence.

Pseudomonas aeruginosa was more susceptible to antibiotic like ciprofloxacin, levofloxacin, ceftazidime, cefoperazone + sulbactam, amikacin, gentamicin, imipenem, meropenem while most resistant against amoxicillin, amoxicillin + clavulanic acid, ceftriaxone, ceftriaxone + sulbactam, erythromycin, azithromycin. It was noticeably clear from our study that the drugs which were being used for gram negative organisms in our in-patient department, like ceftriaxone or ceftriaxone + sulbactam, were losing the fight against resistant bacteria like Pseudomonas aeruginosa. In our study, Pseudomonas was sensitive to piperacillin + tazobactam in only 8 cases (17.02%) which was quite contrary to a survey held in DHQ Teaching Hospital and Microbiology Department where it was sensitive in all the cases (100%).[14]

Staphylococcus aureus was susceptible to linezolid, vancomycin, tazobactam + piperacillin, amikacin, gentamicin, imipenem, ceftriaxone + sulbactam, levofloxacin, ciprofloxacin but mostly resistant to cotrimoxazole, amoxicillin, erythromycin, which are commonly used to eradicate upper respiratory tract infections and associated infective conditions. Like most studies, in our research, we found amikacin to be effective against both Staphylococcus aureus and Pseudomonas aeruginosa, which was in contrast to a study conducted in Hyderabad,[10] where amikacin was effective against Pseudomonas aeruginosa in 55% and against Staphylococcus aureus in 72% cases.

When the results of our study were compared with results of other studies, it was clear that the microbial profile and AST pattern of COM has been changing over time. Indiscriminate and irrational antibiotic use, as well as patient noncompliance, are the factors usually responsible for the changes. The advent of sophisticated synthetic antibiotics has increased the relevance of reevaluation of the modern-day flora in COM and their in vitro AST patterns to assist efficacious empirical treatment.

Conclusion

The massive burden of chronic otitis media patients in India makes it essential to have an evidence-based protocol for initiation of empirical treatment. Geographical variation of antibiotic sensitivity patterns and even microbes presents a challenge to the clinician initiating empirical treatment till formal microbiological confirmation is obtained. Often, due to lack of resources or sheer numbers, the clinician is forced to omit culture sensitivity tests totally. Hence, an idea about the microbes commonly responsible for disease and their antibiotic sensitivity patterns is helpful in clinical practice.

Table III: Antibiotic sensitivity pattern of most common microorganisms

ANTIBIOTICS	P. AERUGINOSA (47 CASES)		S. AUREUS (46 CASES)		S. PNEUMONIAE (3 CASES)		KLEBSIELLA (13 CASES)		PROTEUS SP. (11 CASES)		ACINETOBACTER SP. (13 CASES)		E. COLI (2 CASES)	
	SENSITIVE	RESISTANT	SENSITIVE	RESISTANT	SENSITIVE	RESISTANT	SENSITIVE	RESISTANT	SENSITIVE	RESISTANT	SENSITIVE	RESISTANT	SENSITIVE	RESISTANT
AMO	3 (6.38%)	44 (93.62%)	14 (30.43%)	32 (69.57%)	2 (66.67%)	1 (33.33%)	5 (38.46%)	8 (61.54%)	3 (27.27%)	8 (72.73%)	1 (7.69%)	12 (92.31%)	0 (0.00%)	2 (100%)
AMC	7 (14.89%)	40 (85.11%)	23 (50.00%)	23 (50.00%)	3 (100%)	0 (0.00%)	6 (46.15%)	7 (53.85%)	4 (36.36 %)	7 (63.64%)	2 (15.38%)	11 (84.62%)	0 (0.00%)	2 (100%)
ERY	2 (4.26%)	45 (95.74%)	28 (60.87%)	18 (39.13%)	2 (66.67%)	1 (33.33%)	1 (7.69%)	12 (92.31%)	1 (9.09%)	10 (90.91%)	NA	NA	0 (0.00%)	2 (100%)
AZY	5 (10.64%)	42 (89.36%)	30 (65.22%)	16 (34.78%)	2 (66.67%)	1 (33.33%)	2 (15.38%)	11 (84.62%)	1 (9.09%)	10 (90.91%)	NA	NA	0 (0.00%)	2 (100%)
COT	11 (23.40%)	36 (76.60%)	4 (8.70%)	42 (91.30%)	0 (0.00%)	3 (100%)	6 (46.15%)	7 (53.85%)	2 (18.18%)	9 (81.82%)	0 (0.00%)	13 (100%)	1 (50.00%)	1 (50.00%)
CLA	4 (8.51%)	43 (91.49%)	25 (54.35%)	21 (45.65%)	1 (33.33%)	2 (66.67%)	1 (7.69%)	12 (92.31%)	0 (0.00%)	11 (100%)	NA	NA	0 (0.00%)	2 (100%)
CIP	33 (70.21%)	14 (29.79%)	37 (80.43%)	9 (19.57%)	2 (66.67%)	1 (33.33%)	8 (61.54%)	5 (38.46%)	6 (54.55%)	5 (45.45%)	7 (53.85%)	6 (46.15%)	2 (100%)	0 (0.00%)
LEVO	41 (87.23%)	6 (12.77%)	43 (93.48%)	3 (6.52%)	3 (100%)	0 (0.00%)	10 (76.92%)	3 (23.08%)	10 (90.91%)	1 (9.09%)	8 (61.54%)	5 (38.46%)	2 (100%)	0 (0.00%)
CEFPO	19 (40.43%)	28 (59.57%)	27 (58.70%)	19 (41.30%)	2 (66.67%)	1 (33.33%)	6 (46.15%)	7 (53.85%)	7 (63.64%)	4 (36.36%)	4 (30.77%)	9 (69.23%)	2 (100%)	0 (0.00%)
CEFU	21 (44.68%)	26 (55.32%)	31 (67.39%)	15 (32.61%)	3 (100%)	0 (0.00%)	7 (53.85%)	6 (46.15%)	6 (54.55%)	45 (45.45%)	5 (38.46%)	8 (61.54%)	2 (100%)	0 (0.00%)
CFT	35 (74.47%)	12 (25.53%)	32 (69.57%)	14 (30.43%)	3 (100%)	0 (0.00%)	7 (53.85%)	6 (46.15%)	7 (63.64%)	4 (36.36%)	3 (23.08%)	10 (76.92%)	1 (50.00%)	1 (50.00%)
LIN	NA	NA	46 (100%)	0 (0.00%)	3 (100%)	0 (0.00%)	NA	NA	NA	NA	NA	NA	NA	NA
CEF+S	36 (76.60%)	11 (23.40%)	38 (82.61%)	8 (17.39%)	2 (66.67%)	1 (33.33%)	8 (61.54%)	5 (38.46%)	5 (45.45%)	6 (54.55%)	10 (76.92%)	3 (23.08%)	2 (100%)	0 (0.00%)
CEFT	3 (6.38%)	44 (93.62%)	35 (76.09%)	11 (23.91%)	3 (100%)	0 (0.00%)	7 (53.85%)	6 (46.15%)	4 (36.36%)	7 (63.64%)	6 (46.15%)	7 (53.85%)	2 (100%)	0 (0.00%)
CEFT+S	5 (10.64%)	42 (89.36%)	40 (86.96%)	6 (13.04%)	3 (100%)	0 (0.00%)	8 (61.54%)	5 (38.46%)	8 (72.73%)	3 (27.27%)	8 (61.54%)	5 (38.46%)	2 (100%)	0 (0.00%)
CEFI	19 (40.43%)	28 (59.57%)	37 (80.43%)	9 (19.57%)	3 (100%)	0 (0.00%)	6 (46.15 %)	7 (53.85%)	5 (45.45%)	6 (54.55%)	4 (30.77%)	9 (69.23%)	1 (50.00%)	1 (50.00%)
CFZ	27 (57.45%)	20 (42.55%)	39 (84.78%)	7 (15.22%)	3 (100%)	0 (0.00%)	9 (69.23%)	4 (30.77%)	6(54.55%)	5 (45.45%)	5 (38.46%)	8 (61.54%)	2 (100%)	0 (0.00%)
AMI	42 (89.36%)	5 (10.64%)	45 (97.83%)	1 (2.17%)	2 (66.67%)	1 (33.33%)	11 (84.62%)	2 (15.38%)	9 (81.82%)	2 (18.18%)	9 (69.23%)	4 (30.77%)	2 (100%)	0 (0.00%)
GEN	40 (85.11%)	7 (14.89%)	43 (93.48%)	3 (6.52%)	2 (66.67%)	1 (33.33%)	12 (92.31%)	1 (7.69%)	10 (90.91%)	1 (9.09%)	10 (76.92%)	3 (23.08%)	2 (100%)	0 (0.00%)
IMI	45 (95.74%)	2 (4.26%)	39 (84.78%)	7 (15.22%)	3 (100%)	0 (0.00%)	13 (100%)	0 (0.00%)	10 (90.91%)	1 (9.09%)	13 (100%)	0 (0.00%)	1 (50.00%)	1 (50.00%)
MER	47 (100%)	0 (0.00%)	11 (23.91%)	35 (76.09%)	3 (100%)	0 (0.00%)	12 (92.31%)	1 (7.69%)	9 (81.82%)	2 (18.18%)	12 (92.31%)	NA	2 (100%)	0 (0.00%)
TAZ+PIP	8 (17.02%)	39 (82.98%)	40 (86.96%)	6 (13.04%)	2 (66.67%)	1 (0.00%)	NA	NA	NA	NA	10 (76.92%)	3 (23.08%)	NA	NA
VAN	NA	NA	45 (97.83%)	1 (2.17%)	3 (100%)	0 (0.00%)	NA	NA	NA	NA	NA	NA	NA	NA

(AMO - Amoxicillin, AMC - Ampicillin, ERY - Erythromycin, AZY - Azithromycin, COT - Cotrimoxazole, CLA - Clarithromycin, CIP - Ciprofloxacin, LEVO - Levofloxacin, CEFPO - Cefpodoxime, CEFU - Cefuroxime, CFT - Cefotaxime, LIN - Lincomycin, CEF+S - Cefuroxime + Sulbactam, CEFT - Ceftriaxone, CEFT+S - Ceftriaxone + Sulbactam, CEFI - Cefixime, CFZ - Ceftazidime, AMI - Amikacin, GEN - Gentamicin, IMI - Imipenem, MER - Meropenem, TAZ+PIP - Tazobactam + Piperacillin, Van - Vancomycin)

References

1. WHO. Child and adolescent health and development. Prevention of blindness and deafness. Chronic suppurative otitis media. Burden of illness and management options. Geneva, Switzerland: WHO; 2004 http://www.who.int/pbd/deafness/activities/hearing_care/otitis_media.pdf (Accessed on September 17, 2012).
2. Eason RJ, Harding E, Nicholson R, Nicholson D, Pada J, Gathercole J. Chronic suppurative otitis media in the Solomon Islands: a prospective, microbiological, audiometric and therapeutic survey. N Z Med J. 1986; 99(812):812-5
3. Kenna MA, Bluestone CD. Microbiology of chronic suppurative otitis media in children. Pediatr Infect Dis. 1986; 5:223-5
4. Ahn JH, Kim MN, Suk YA, Moon BJ. Preoperative, intraoperative, and postoperative results of bacterial culture from patients with chronic suppurative otitis media. Otol Neurotol. 2012; 33:54-9
5. Park MK, Jung MH, Kang HJ, et al. The changes of MRSA infections in chronic suppurative otitis media. Otolaryngol Head Neck Surg. 2008; 139:395-8
6. Brook I. The role of anaerobic bacteria in chronic suppurative otitis media in children: implications for medical therapy. Anaerobe 2008; 14(6):297-300. doi:10.1016/j.anaerobe.2008.12.002
7. Acuin J. Geneva: World Health Organisation; 2004. Global burden of disease due to chronic suppurative otitis media: Disease, deafness, deaths and DALYs Chronic Suppurative Otitis Media–Burden of Illness and Management Options; pp. 9-23
8. Berman S. Otitis media in developing countries. Pediatrics. 1995; 96:126-31
9. Microbiological profile of Chronic Suppurative Otitis Media. International Journal of Biomedical Research 2014; 5(3):204-6
10. Prayaga N, Moorthy S, Lingaiah J, Katari S, Nakirakanti A. Clinical Application of a Microbiological Study on Chronic Suppurative Otitis Media. Int J Otolaryngol Head Neck Surg. 2013; 02:290-294
11. Prakash R, Juyal D, Negi V, et al. Microbiology of chronic suppurative otitis media in a tertiary care setup of Uttarakhand state, India. N Am J Med Sci. 2013;5(4):282-7
12. AZHARI SI, MULYATI S. Gambaran X-Foto Schuller Pada Pasien Otitis Media Kronis Di Rumah Sakit Angkatan Laut Dr. Ramelan Surabaya Periode 2015-2016. Hang Tuah Medical journal [S.l.] 2018; 16(1):69-80
13. Kristo B, Buljan M. Microbiology of the chronic suppurative otitis media. Medicinski glasnik : official publication of the Medical Association of Zenica-Doboj Canton, Bosnia and Herzegovina 2011; 8:284-6
14. Iqbal K, Ismail Khan M, Satti L. Microbiology of chronic suppurative otitis media: experience at Dera Ismail Khan. Gomal Journal of Medical Sciences. 2020; 9(2):189-93.

Case Series

Eagle Syndrome - An Entity Often Misdiagnosed

https://doi.org/10.47210/bjohns.2020.v28i2.333

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ABSTRACT

Introduction

Throat pain is one of the most common complaints that an otolaryngologist encounters in daily practice, there can be numerous aetiologies to it. Eagle Syndrome is one such entity which is characterized by chronic throat pain due to elongation of the styloid process or mineralization of the stylohyoid ligament. The vague symptomatology of this condition often leads to delayed diagnosis and treatment.

Case Reports

We report 5 cases of Eagle Syndrome. All the patients presented to us with complaints of chronic throat radiating to neck which did not relieve on taking medications, for the same they have been consulting multiple specialities. The diagnosis of Eagle Syndrome was confirmed by palpation in tonsillar fossa and orthopantomogram revealed enlarged styloid process measuring more than 30mm. Upon confirmation, all the 5 patients underwent tonsillo-styloidectomy and on consequent follow ups, they were symptomatically improved.

Discussion

Eagle Syndrome is a diagnosis of exclusion and should raise high index of suspicion in patients with nonspecific throat and neck pain not responding to any conservative treatment. With an increasing incidence of side effects following injudicious treatment given in such cases, this condition requires attention, as it a rare entity and often misdiagnosed.

Keywords

Eagle Syndrome

Eagle syndrome is a clinical entity that is often misdiagnosed. It is a constellation of signs that occurs due to elongated styloid process or as a result of mineralization of the stylohyoid or stylomandibular ligament, also known as Long Styloid Process Syndrome or Styloid Process Neuralgia.[1] The term was coined by W. Eagle, an otolaryngologist in the year 1937.

The symptoms often vary from throat pain to chronic neck pain that doesn't relieve on medications, thus becomes one of the main differential diagnoses of chronic throat and neck pain. Affects approximately 4% of population out of which only 4% are symptomatic thus diagnosing it becomes challenging and is often misdiagnosed owing its vague symptomatology.[2]

As a result, it becomes troublesome for the patient as he pays multiple visits to multiple specialities which leads to delayed diagnosis and treatment. Patient is prescribed unnecessary analgesics and antibiotics which predisposes him to laryngopharyngeal disease. However, palpable styloid process in tonsillar fossa with radiological evidence of the elongated styloid process accomplishes the diagnosis.

Tonsillo-styloidectomy remains the main stay of treatment for Eagle Syndrome with a high success rate as there are no recurrances.[3]

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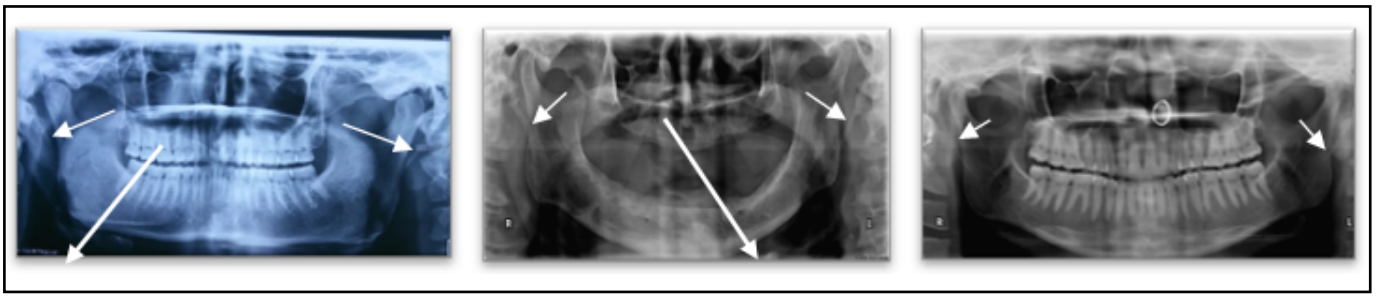

Fig. 1. Othropantamogram showing elongated styloid process of our patients

Case Reports

Case 1

A 30-year-old male presented to us with complaints of intermittent throat pain associated with pain in the neck for 1 year, with history of low-grade fever. Patient gave history of visiting multiple specialities for the same complaints before he came to us. ENT examination revealed bilateral grade 1 tonsillar hypertrophy, on bimanual examination bilateral bony prominences were palpable in tonsillar fossa. Orthopantomogram revealed bilateral enlargement of styloid process measuring 40.5mm on right side and 60.2mm on left side. (Fig 1) Diagnosis of stylalgia was confirmed and was started on analgesics and preoperative evaluation was done. Following which the patient underwent bilateral tonsillo-styloidectomy (Fig. 2) by an intra oral approach and bilateral enlarged styloid process were removed. (Fig. 3) On regular follow ups on day 7, day 14, and 1month post-surgery, the patient symptomatically improved.

Case 2

38-year-old female presented to us with complaints of right sided dull aching throat pain radiating to neck, right ear and foreign body sensation in throat for 6 months. ENT examination revealed tenderness over bilateral tonsillar fossa with palpable bony prominences. Orthopantomogram revealed bilateral elongated styloid process measuring right side 46.4mm, left side 48.8mm (Fig. 1). The diagnosis of Eagle Syndrome was thus accomplished. Initially patient was treated conservatively with analgesics but showed no improvement. Following which she underwent bilateral tonsillo-styloidectomy (Fig. 2) which showed bilateral enlarged styloid process (Fig. 3). Patient was discharged

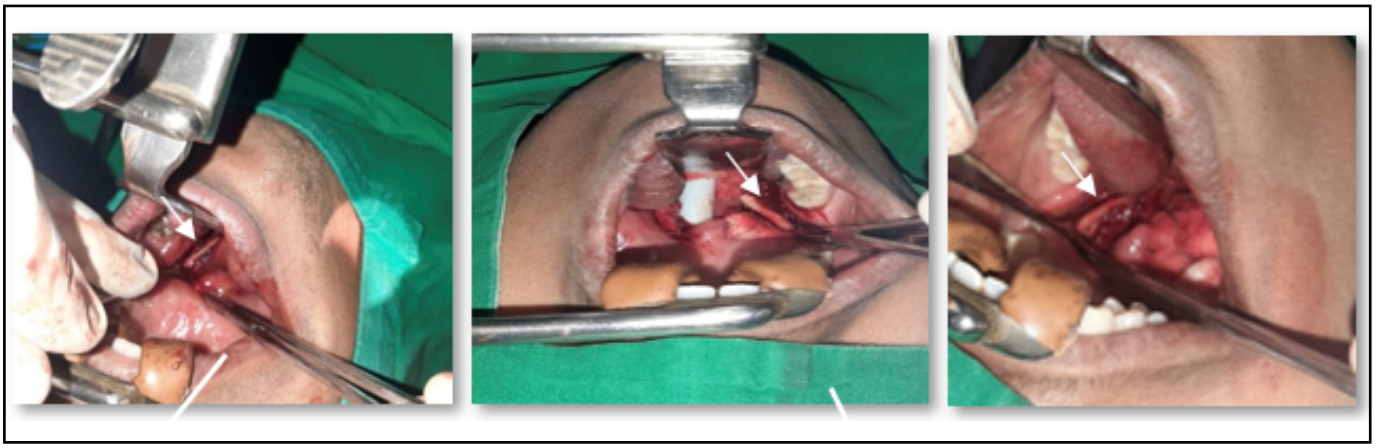

Fig. 2. Intra operative picture showing enlarged styloid process

Fig.3. Post operative specimen of styloid process after tonsillo-styloidectomy

with oral antibiotics and analgesics, followed up on day 7, day 14 and 1-month following surgery and she showed prompt relief of symptoms.

Case 3

A 42-year-old female patient came to us with complaints of persisting nagging pain over the right side of the throat radiating to face and ipsilateral ear for the last 2 years and not relieved by any analgesics and antidepressants. She was examined by Oro-maxillo-facial surgeon and Neurologist elsewhere and found to be clinically normal. ENT examination here showed normal oropharynx on inspection. But a bony mass was palpable in bilateral tonsillar fossa and was tender, which again confirmed the site of pain. The diagnosis of stylalgia was confirmed by taking an orthopantomogram which revealed bilateral enlarged styloid process measuring 40mm on right and 42mm left side respectively. A course of conservative management with carbamazepine was tried but only temporary relief was noticed. She underwent tonsillo-styloidectomy (Fig. 3) after pre-operative evaluation. Patient was regularly followed up on day 7, day 14, and 1-month post-surgery was found to be symptom free.

Case 4

A 34-year-old male presented to us with complaints of intermittent dull aching throat pain associated with pain in the neck for 1 year. Patient has been visiting various doctors for the same complaints before he came to us. On ENT examination bimanual examination bilateral tenderness was present in bilateral tonsillar fossa. Orthopantomogram revealed bilateral enlargement of styloid process measuring 30.5mm on right side and 31.6mm on left side. (Fig.1) He was started on analgesics and preoperative evaluation was done, once diagnosis was confirmed. Following which the patient underwent bilateral tonsillo-styloidectomy (Fig. 2) by an intra oral approach and bilateral enlarged styloid process were removed (Fig. 3). Following surgery patient had regular follow ups on day 7, day 14 and 1 month and had symptomatic relief of symptoms.

Case 5

A 40-year-old female presented to us with complaints of dull aching throat pain radiating to neck, both ears and foreign body sensation in throat for 8 months which did not relieve on taking medications. ENT examination revealed tenderness over bilateral tonsillar fossa with palpable bony prominences. Orthopantomogram was done and the findings were: bilateral elongated styloid process measuring right side 35.4mm, left side 32 mm (Fig. 1). Based on the imaging findings and clinical examination, the diagnosis of Eagle Syndrome was thus accomplished. Initially patient was treated conservatively with analgesics but showed no improvement. Following which she underwent bilateral tonsillo-styloidectomy which showed bilateral enlarged styloid process (Fig.

3). Patient was discharged with oral antibiotics and analgesics, followed up on day 7, day 14- and 1-month post-surgery and she showed drastic relief of symptoms.

Discussion

Eagle Syndrome refers to a constellation of signs and symptoms that occurs due to elongated styloid process or as a result of mineralization of the stylohyoid or stylomandibular ligament, also known as Long Styloid Process Syndrome or Styloid Process Neuralgia.[1]

The styloid process is an elongated projection which originates from the petrous part of temporal bone, medially and anteriorly to the stylomastoid foramen, between the internal and external carotid arteries, and laterally to the tonsillar fossa. Embryologically derived from Reichert's cartilage from the second branchial arch.3 The normal length of the styloid process is 20–30 mm and it is said to be elongated when it is 30 mm or longer. 4% of world population is affected out of which only 4 % are symptomatic thus diagnosing it becomes challenging. It is frequently seen in females between 30-50 years.[2]

First described in literature by Lucke and Weinlecher in 1872 while in 1937 American otorhinolaryngologist W. Eagle described it as a syndrome complex mainly in two varieties. The classical variety that presents as throat pain with referred otalgia and foreign body sensation in the throat. The carotid artery variety in which styloid process compresses the carotid artery leads which to carotidynia, headache and dizziness.[4]

Several theories have been postulated regarding the etiology of Eagle Syndrome, the most accepted one being the growth of the osseous tissue along stylohyoid ligament.

Symptomatically it presents with throat pain, chronic neck pain, facial pain, odynophagia, throat pain radiating to ear, foreign body sensation in throat and neuralgic pain. Owing to such vague and varied symptoms the diagnosis is often delayed, misdiagnosed and it makes it troublesome for the patient as they undergo unnecessary investigations. It also makes the patient vulnerable for laryngopharyngeal reflex and allergic rhinitis symptoms.[5]

Differential diagnosis of Eagle Syndrome should include trigeminal neuralgia, migraine, temper-mandibular joint disorders, temporal arteritis, unerupted or impacted molar teeth and faulty dental prostheses. As we proceed towards the treatment the differential diagnosis has to be kept in mind and thus making it a diagnosis of exclusion and should be considered as one of the important causes in the differential diagnosis of orofacial pain.

This syndrome is also known as glossopharyngeal neuralgia as it compresses the glossopharyngeal nerve along its course over the elongated styloid process leading to glossopharyngeal neuralgia. Thus, when we evaluate cases referred for glossopharyngeal neuralgia, the diagnosis of Eagle Syndrome should be kept in mind.[5]

Accomplishment of diagnosis of this syndrome thus necessities a thorough clinical examination that includes proper history, bimanual palpation of tonsillar fossa for elongated styloid followed by radiological evaluation-Orthopantomogram or X-ray Towne's view.[6]

The treatment options for this syndrome includes both surgical and non-surgical approach. Conservatively in mild to moderate cases the patient can be treated with analgesics, anti-convulsant, anti-depressants and local injection of steroids in the tonsillar fossa but often there is temporary relief of symptoms with this line of management. And it thus necessitates a surgical approach, tonsillo-styloidectomy which remains the gold standard treatment modality as it provides prompt relief of symptoms with a high success rate.[7,8,9]

In our current case series, all the 4 patients presented to us with complaints of chronic throat pain radiating to neck, the condition was initially misdiagnosed and the patients were referred to different specialites which not only was troublesome but also affected the quality of their life. Following the confirmation of the diagnosis of Eagle Syndrome by palpation in tonsillar fossa and radiographically, we planned surgical excision because the patient's symptoms were severe and did not respond to medical management. As both elongated styloid processes were quite enlarged in all 4 cases, we performed tonsillo styloidectomy. All the patients responded well to the procedure and were symptom free upon repeated

follow up. Thus surgical management remains the gold standard of treatment for this syndrome.

Conclusion

Eagle Syndrome is a condition that is rare but often it is misdiagnosed owing to this vague and varied symptomology and that leads to delayed diagnosis and prompt treatment. It is a diagnosis of exclusion and should raise high index of suspicion in patients with nonspecific throat and neck pain not responding to any conservative treatment.

References

1. Malik JN, Monga S, Sharma AP, Nabi N, Naseeruddin K. Stylalgia revisited: clinical profile and management. Iran J Otorhinolaryngol. 2018;30(6):335-40
2. Sharma P, Shekhar S, Bajpayi BB, Kumar S, Pal PNP. Eagle's syndrome - A rare entity but not so uncommon alleviating pain and symptomatology. IOSR J Dental Med Sci. 2017;16(5):31-2
3. Moon CS, Lee BS, Kwon YD, et al. Eagle's syndrome: a case report. J Korean Assoc Oral Maxillofac Surg. 2014; 40(1):43-7. doi:10.5125/jkaoms.2014.40.1.43
4. K. C. Prasad, M. P. Kamath et al. Elongated styloid process (Eagle's syndrome): A clinical study. J Oral Maxillofac Surg. 2002; 60(2):171-5
5. Slavin KV. Eagle syndrome: entrapment of glossopharyngeal nerve. Journal of Neurosurgery 2020; 97 (1) :216-8
6. Mishra A, Dabholkar J, Lodha J, Sharma A, Mhashal S. Stylalgia: A Missed Diagnosis. Otolaryngol Pol. 2015;69(2):34-37. doi:10.5604/00306657.1131148
7. Taheri A, Firouzi-Marani S, Khoshbin M. Nonsurgical treatment of stylohyoid (Eagle) syndrome: a case report. J Korean Assoc Oral Maxillofac Surg. 2014; 40:246-9
8. Han MK, Kim DW, Yang JY. Non Surgical Treatment of Eagle's Syndrome - A Case Report -. Korean J Pain. 2013; 26(2):169-172. doi:10.3344/kjp.2013.26.2.169
9. Naik SM, Naik SS. Tonsillo-Styloidectomy for Eagle's Syndrome: A Review of 15 Cases in KVG Medical College Sullia. Oman Medical Journal 2011; 26(2): 122-6.

Hyperkinetic Dysarthria with Spasmodic Torticollis

https://doi.org/10.47210/bjohns.2020.v28i2.319

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ABSTRACT

Introduction

Spasmodic Torticollis is a condition in which tonic or clonic spasm in the Sternocleidomastoid and Trapezius muscles, cause the head to be deviated to the right or left and has also been reported to disrupt speech production. Spasmodic Torticollis is a part of unknown etiology of Hyperkinetic Dysarthria.

Case Report

We report a case of 30 years old male diagnosed as Hyperkinetic Dysarthria who was treated with speech therapy and without Botulinum toxin. Post therapy status was recorded after 12 sessions (frequency twice a week) and it shows improvement in speech characteristics.

Discussion

Management of the patients with Hyperkinetic Dysarthria with Spasmodic Torticollis is a long term and often a lifelong process. Speech therapy is offered at intervals in order to maintain communicative competence. Reported studies suggest the use of Botulinum toxin, but in the present study treatment efficacy was proven without the use of Botulinum toxin.

Keywords

Dysarthria, Hyperkinetic; Torticollis, Spasmodic; Speech Therapy

Dysarthria is a speech disorder caused by muscle weakness due to brain damage.[1] Hyperkinetic Dysarthria is usually caused due to pathologies and/or lesion in the Basal Ganglia, the related portion of the Extrapyramidal System, or sometimes the Cerebellar control unit.[2] Spasmodic Torticollis is a condition in which tonic or clonic spasm in the Sternocleidomastoid and Trapezius muscles causes the head to be deviated to the right or left and has also been reported to disrupt speech production3. It is also known as Cervical Dystonia[3] and is found to be associated with Hyperkinetic Dysarthria of unknown etiology (21%).[4]

Case Report

We report a case of 30 years old male patient with paresis of left hand and presented to us with symptoms of getting stuck during conversation and having effortful speech. Previous medical report revealed to have variable diagnoses such as Focal Dystonia, Spasmodic Dysphonia and Orofacial Dyskinesia. He reported with a history of Hepatitis and Seizures at the age of 9 yrs. He took various medications for recovery from Dyskinesia but it was not benefitted. We followed an assessment protocol for the subject as given by American Speech Language Hearing Association.[5] Assessment involves the use of both formal and informal tests. Pre-therapy assessment involves non-speech assessment, speech production assessment and other assessments include language, cognitive-communication and dysphagia assessment. (Table I)

As the management, speech therapy was provided to establish intelligible communication skills in all the communicative situations. Specific targets were selected as the goal for treatment to improve respiratory support, articulation, oral resonance, prosodic features, alternate

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Table I: Pre-therapy assessment of the case

1. Non speech assessments:
a. Oral peripheral mechanism (OPM) examination showed normal appearance of all the articulators except for tongue which is short. Functions of articulators were assessed and it revealed restricted movement of soft palate. Gag reflex was found to be hypoactive.
b. Maximum phonation duration (MPT) was found to be 15 seconds with the average of 3 trials.
c. Alternate Motion Rate (AMR) and Sequential Motion Rate (SMR) were found to be affected.
d. Observation of facial and neck muscle tone:
i. At rest: Hypertonia
ii. During non-speech activities: Hypertonia
e. Cranial nerve examination (CN V, VII, IX, X, XI, XII) — shows VII, IX, X, XII are affected.
2. Speech Production Assessment:
a. Articulation assessment was done using Bengali Articulation Test5 and findings revealed devoicing of Stop sounds.
b. 7-point Intelligibility rating scale6 was used and client's speech was rated as 3, which indicates that the speech could be understood with concentration and effort especially by sympathetic listener (where 0 is denoted as normal and 6 as unintelligible).
c. Through informal assessment prosodic features of the client was evaluated and it was found to be affected.
d. Frenchay Dysarthria Assessment7 revealed all the domains were affected with varying degree except for Reflex (figure 1).
e. Acoustic analysis involved both perceptual and instrumental measurements. Perceptual assessments was done using GRBAS8 scale which revealed Grade 1, Roughness 2, Breathiness 0, Asthenic 0, and Strain 1. Instrumental assessment of voice analysis was done using Dr. Speech software by Tiger electronics version 4.0 and Nasometer-II version 2.6 and findings revealed hoarse voice and Hypernasal voice respectively.
3. Other assessments include Language, Cognitive-Communication and Dysphagia assessment.
a. Language assessment was done using Aphasia screening and language was found to be intact.
b. Cognitive-Communication assessment was done using Mini Mental State Examination (MMSE)9 and cognition was found to be intact with a score of 30.
c. Dysphagia assessment was done using Mann Assessment of swallowing ability (MASA)10 and it revealed no difficulty in swallowing with a score of 187.
d. Differential Diagnosis was done and the Final diagnosis for the case was Hyperkinetic Dysarthria.

motion rate, sequential motion rates, and vocal function and to reduce rate of speech.[11] (Table II)

Treatment method used while treating the patient was devoid of Botulinum Toxin-A (BOTOX). Post therapy status was recorded after 12 sessions (frequency twice a week) and it shows improvement in speech characteristics. (Table III)

Discussion

The aim of the therapy given in this case study was to achieve "compensated intelligible speech" rather than "normal speech" and through speech therapy this aim was achieved successfully. Although, pharmacological therapy with Botulinum toxin is used more commonly,[13]

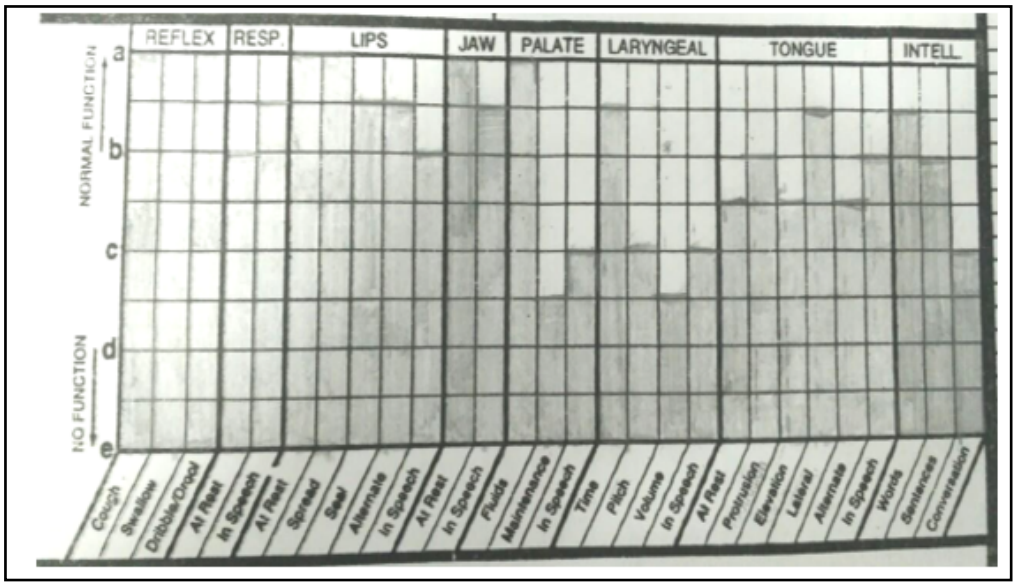

Fig. 1. Pre Therapy Frenchay Dysarthria Assessment

Table II: Therapy plan illustrating the goals and activities

GOALS	ACTIVITIES
To improve respiratory support	Client was instructed to take breathe deeply before initiation of speech and exhale slowly while onset of speaking2.
Rehabilitate and improve vocal function.	Techniques:- Relaxation1[2]:
	· Relax the shoulders and using a mirror for visual feedback.
	· Discrimination between tensed and relaxed muscles.
	To improve Reduced loudness (Change of loudness technique1[2]):
	· Client was instructed to raise his shoulders, inhale and phonate a high pitch "humm". And then was instructed to lower the shoulders and exhale and produce "humm" sound.
	· Client was made to produce inhalation phonation and exhalation phonation.
To facilitate oral resonance and improve articulation	Client was instructed to speak in an increased loudness with mouth and jaw- open
To elicit gag reflex	Palpation of the soft palate was administered as a therapeutic management.
To improve prosodic features	Stress and emphasis were used to convey different meanings and at a reduced rate of speech by increasing the pauses between words.
To improve alternate motion rate (AMR)	The client will be asked to take a breath and repeat "Puh-Puh-Puh " as fast and as steadily as possible for 3 to 5 secs2. This will be followed by similar repetitions of "Tuh-Tuh-Tuh----" and "Kuh-Kuh-Kuh----". These AMR tasks permit judgments of rate, rhythm, precision and range of motion and rapid movements of the lips, jaw and tongue.
To improve sequential motion rate (SMR)	The client will be instructed to take a breath and produce "Puh-Tuh-Kuh" repeatedly for 3 to 5 seconds2.
To reduce the rate of speech,	Client was counseled to speak at a slower rate, and Hand tapping12 was also demonstrated to the client as a self-monitoring skill for the same.

Table III: Showing the post therapy improvement

DOMAINS	PRE THERAPY				POST THERAPY			
Acoustic analysis (instrumental) [Nasometer II Normative values for Bengali speakers Zoo passage: 11.25+5.63 Nasal passage: 59.25+7.96 PB passage:31.47+6.65]	Hoarse voice (as measured using Dr. Speech software). Hypernasality was found (as measured using Nasometer-II)				Normal voice (as measured using Dr. Speech software) and Hypernasality was reduced (as measured using Nasometer-II).			
	Passage	Mean %	Min %	Max %	Passage	Mean %	Min %	Max %
	ORAL	22	4	98	ORAL	20	4	96
	NASAL	80	16	98	NASAL	62	11	97
	PB	87	45	99	PB	38	7	96
Acoustic analysis (Perceptual)	GRBAS scale showed G1R2B0A0S1				GRBAS scale showed G1R0B0S1			
FDA	Revealed all the domains were affected with varying degree except for Reflex				Showed improvements in all the domains as illustrated in figure [2]			
Gag reflex	Absent				Present			
MPT (average of 3 trials)	18 seconds				22 seconds			
AMR	/pa/- 20 times in 7 seconds				/pa/- 20 times in 5 seconds			
	/ta/- 20 times in 9 seconds				/ta/- 20 times in 6 seconds			
	/ka/- 20 times in 10 seconds				/ka/- 20 times in 7 seconds			
SMR	/pataka/- 20 times in 14 seconds				/pataka/- 20 times in 9 seconds			
Cranial nerve examination (CN V, VII, IX, X, XI, XII)	Shows CN VII, IX, X, XII are affected				Shows CN XII is affected			
Speech Intelligibility (measured using 7-point intelligibility rating scale where '0' indicates normal and '6' indicates unintelligible)	Client's speech was rated as 3, which indicates that the speech could be understood with concentration and effort especially by sympathetic listener.				Speech intelligibility was also improved to achieve point 1 rating in which indicates "speech could be understood without difficulty, however still feel that speech is not normal"			
Rate of speech	Cannot be measured due to unintelligible speech				140 words/min			
Bangla Articulation test	Devoicing of speech sounds: /b/, /g/, /gh/.				Correct production of all speech sounds			

much research is still needed to establish the findings of this case study.

The treatment for Hyperkinetic Dysarthria with Spasmodic Torticollis is basically based on the Client-Oriented approach.[2] Here in this case report, compensatory speaker strategies[2] were used to improve speech intelligibility and it was noted that rate reduction resulted in improvement in the speech intelligibility. Another study[14] also reported similar findings that reducing the speech rate resulted in improved speech intelligibility. Also enhancement in the voice quality[15] results in improvement of speech intelligibility as seen in the present case. Although some people with Hyperkinetic Dysarthria inadvertently discover their own tricks, which they use to inhibit certain movements and facilitate speech and these may be further explored with the speech pathologist to develop communication.

Basically the client's presenting Hyperkinetic Dysarthria with Spasmodic Torticollis (Laterocollis) might not be fully cured[16] with speech therapy and if speech impairment prevails in further Dysarthria assessment, then BOTOX or Botulinum Toxin-A injection might be required as advised by the Neurologist. In the present case, the patient was not willing to go for BOTOX treatment. Thus it might be suggested that speech therapy might improve symptoms of these clients

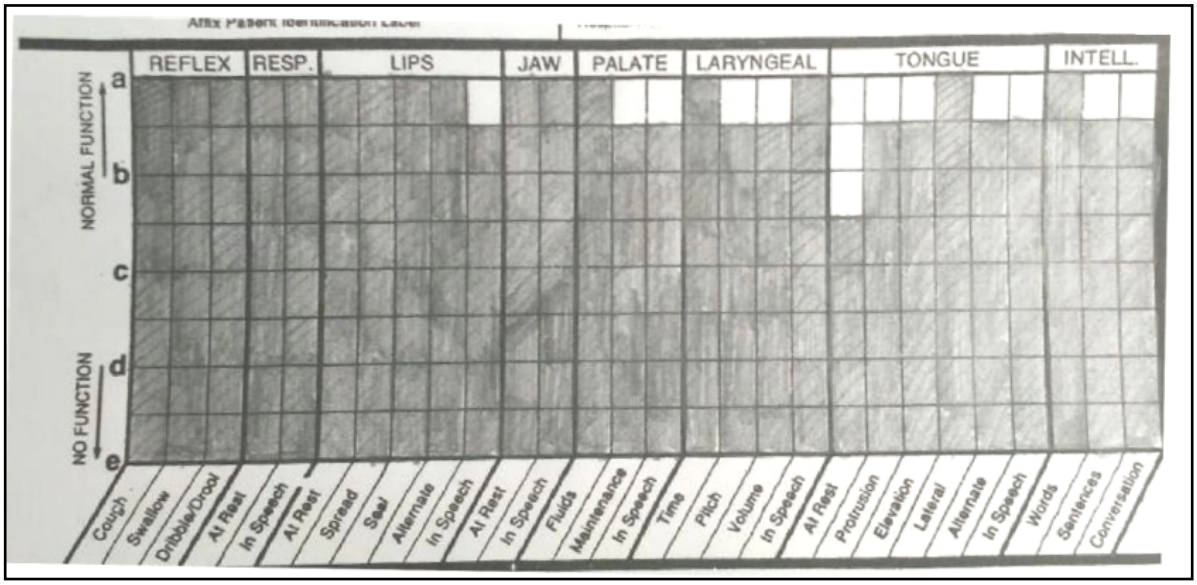

Fig. 2. Post Therapy improvement in FDA (after 12 sessions of speech therapy)

thereby reducing the dependency on medications.

References

1. Dysarthria [Internet]. Asha.org. 2019 [cited 30 August 2019]. Available from: https://www.asha.org/public/speech/disorders/dysarthria/
2. Duffy, JR. Motor Speech Disorders: Substrates, Differential diagnosis and Management. 3rd ed. United States: Elsevier Mosby; 2013
3. Murdoch B. Acquired Speech and Language Disorders. 2nd ed. New York, NY: Springer; 2013
4. Botox injections - Care at Mayo Clinic - Mayo Clinic [Internet]. Mayoclinic.org. 2019 [cited 1 June2019]. Available from https://www.mayoclinic.org/tests-procedures/botox/care-at-mayo-clinic/pcc-20384660-
5. Dysarthria in Adults: Assessment [Internet]. Asha.org. 2019 [cited 1 June 2019]. Available from: https://www.asha.org/PRPSpecificTopic.aspx?folderid=8589943481&section=Assessment
6. Home | Ali Yavar Jung National Institute of Speech and Hearing Disabilities (Divyangjan), Mumbai. [Internet]. Ayjnihh.nic.in. 2019 [cited 1 June 2019]. Available from: http://www.ayjnihh.nic.in
7. Enderby P. Frenchay Dysarthria Assessment. International Journal of Language & Communication Disorders. 1980; 15(3):165-73
8. Hirano, M. Clinical examination of the voice. New York, NY: Springer-Verlag; 1981
9. Kabir ZN, Herlitz A. The Bangla adaptation of Mini-Mental State Examination (BAMSE): an instrument to assess cognitive function in illiterate and literate individuals. Int J Geriatr Psychiatry. 2000;15(5):441-50. doi:10.1002/(sici)1099-1166(200005)15:5<441::aid-gps142>3.0.co;2-o
10. Mann G. MASA : the Mann assessment of swallowing ability. Australia ; Clifton Park, NY : Singular/Thomson Learning; 2002
11. Shipley, KG, Mcafee, JG. Assessment in speech-language pathology: a resource manual. (2nd ed.). San Diego: Singular publishing group; 1998
12. Hegde M. Pocket guide to treatment in speech language pathology. San Diego: Singular Publ. Group; 1990
13. Spasmodic Torticollis | American Association of Neuromuscular & Electrodiagnostic Medicine [Internet]. Aanem.org. 2019 [cited 1 June 2019]. Available from: https://www.aanem.org/Patients/Muscle-and-Nerve-Disorders/Spasmodic-Torticollis
14. Nishio M, Tanaka Y, Sakabibara C, & Abe N. (2011). Effectiveness of speech rate conversion software for patients with dysarthria. Journal of Communications Research 2011; 2(1):1-12
15. Will L, Ramig LO, Spielman JL. Application of the Lee Silverman Voice Treatment (LSVT) to Individuals with multiple sclerosis, ataxic dysarthria, and stroke. In Seventh International Conference on Spoken Language Processing 2002
16. Fabbro F, Asher R. Concise encyclopedia of language pathology. New York: Elsevier; 1999.

Case Report

Synchronous Buccal Carcinoma and Laryngeal Carcinoma- A Rare Presentation

https://doi.org/10.47210/bjohns.2020.v28i2.325

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ABSTRACT

Introduction

Second primary malignancy as squamous cell carcinoma of larynx is rare after squamous cell carcinoma of buccal mucosa.

Case Report

A 47 year old male patient presented with a painless, progressive ulcer of left buccal mucosa near the angle of mouth for the last 3 months who also developed alteration in the quality of voice and respiratory distress 2 months later. On biopsy buccal lesion came out to be moderately differentiated squamous cell carcinoma. Direct laryngoscopy revealed a glottic growth and guided biopsy from the lesion revealed well differentiated keratinising squamous cell carcinoma which was managed successfully by total laryngectomy and buccal carcinoma by wide local excision followed by primary closure and supraomohyoid neck dissection in the same sitting.

Discussion

Having both index primary tumour and second primary tumour in head and neck region is a rare occurrence more so glottic carcinoma occurring synchronously with buccal carcinomas. Surgery is the mainstay of treatment in both the conditions.

Keywords

Neoplasms, Second Primary; Carcinoma, Squamous Cell; Larynx; Mouth Mucosa

Head and neck region are a harbour of malignancies with global incidence between 400,000 and 600,000 new cases per year and 223,000 and 300,000 yearly deaths.[1] Head and neck squamous cell carcinomas (HNSCC) incidence wise are the sixth leading human cancer worldwide.[2] More than 90% of these cancers arise from the mucosal surfaces of the oral cavity, oropharynx and larynx with oral cancer being by far more common in the Indian subcontinent.[3,4]

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Second primary malignancy (SPM) is a common entity in the head and neck region. Multiple primaries are seen in about 9.7% of head and neck cancer patients.

Multiple malignancies can be of two types; synchronous when the second primary neoplasm (SPN) is diagnosed within 2-6 months of diagnosis of index tumour or metachronous, if the second primary tumour is diagnosed 6 months following diagnosis of primary tumour.[5,6] Out of the multiple primary malignancies in the head and neck region, 46.9% present as synchronous.[7] The criteria used for diagnosis of multiple primary cancers was first given by Warren and Gates, which are:

1. Each of the tumours must be malignant, being confirmed on histology.
2. Each must be geographically separate and distinct. The lesions should be separated by normal mucosa.
3. Probability of one being the metastasis of the

Fig. 1. Appearance of the buccal lesion before treatment

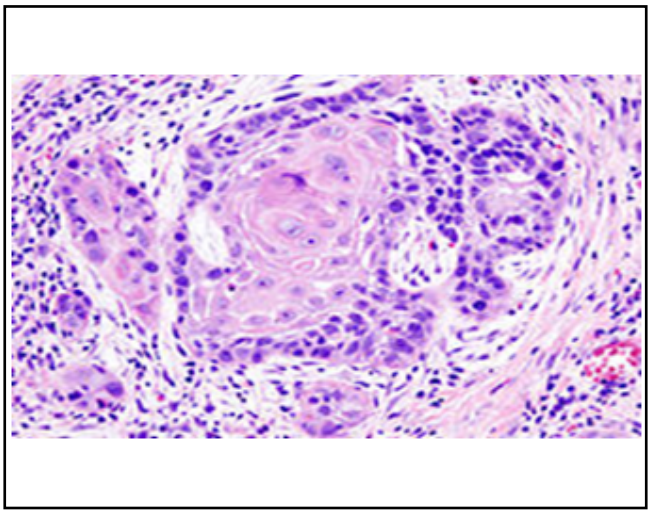

Fig. 2. Histopathological picture of moderately differentiated squamous cell carcinoma of buccal mucosa (H & E, 10X)

other must be excluded.

It was modified later by Moertel et al (1961), Curtis and Ries (2006) and Morris et al (2010).[8] The most common site of second malignancy following index primary tumour in head and neck is breast and gastrointestinal system followed by lungs.[9] Our case in this report has both the tumours in the head and neck region with the index tumour in the oral cavity and the SPT in the larynx.

Case Report

A 47year old male presented to ENT department with complaints of an ulcerative lesion in the left buccal mucosa, near the angle of mouth for 3 months. The ulcer was insidious in onset, gradually increasing in size and painless. The patient had a history smoking twenty bidis a day for last twenty years. On examination the ulcerative lesion being 2.5×3 cm. in size and had rolled out, discontinuous margins, indurated base with melanoplakia of surrounding mucosa. Skin of the cheek was free on palpation and easily pinchable. No enlarged neck node could be palpated. Thus overall TNM stage becomes T2N0M0. The patient was then planned for wedge biopsy from the lesion including its margin. The histopathological examination (HPE) report came out to be moderately differentiated squamous cell carcinoma. The patient was then planned for wide local excision of the buccal carcinoma with 1.5 cm margin followed by primary repair in collaboration with department of plastic surgery as the presumed need for commisuroplasty and left supraomohyoid neck dissection in this clinically N0 neck.

In the meantime when the patient was being prepared for that planned surgery he started to develop hoarseness of voice, episodes of respiratory distress and aspiration of food materials for 2 months following the appearance of the oral ulcer. Fibreoptic Laryngoscopic (FOL) findings using a 8 mm 70° rigid laryngoscope, revealed a growth involving the right vocal cord, right aryepiglottic fold, anterior commissure, right false cord, anterior part of the left vocal cord. Right hemi-larynx was fixed. Glottic chink was narrow. CECT scan of neck including larynx revealed a growth involving right vocal cord, right aryepiglottic fold, anterior commissure with compromised airway. Breach was seen in the inner lamina of the thyroid cartilage with no involvement of the neck structures and no lymphadenopathy. Metastatic workup was negative and hence the TNM stage was Stage-III (T3N0M0). The patient was planned for direct laryngoscopy and biopsy from representative areas of growth in the department of ENT.

Direct laryngoscopy was done after a high tracheostomy and the HPE result of biopsy specimen

Fig. 3. CECT scan of neck and larynx at the level of thyroid ala showing cartilage breach on right side

Fig. 4. Postoperative follow up showing healed permanent tracheostome and buccal lesion after excision

came out to be well differentiated keratinizing squamous cell carcinoma. The patient was then planned for total laryngectomy as in this T3 glottic disease with dysfunctional larynx in the same sitting with excision of buccal carcinoma followed by repair and neck dissection and adjuvant radiotherapy and the patient party counselled likewise with informed consent taken. Total laryngectomy was done by conventional technique using Gluck-Sorenson incision and the usual postoperative period was uneventful. The patient was discharged after two weeks. The patient was followed up in the OPD after three weeks of surgery with HPE report of the specimens of total laryngectomy, excised buccal mucosa and supraomohyoid neck dissection. The HPE report of excised buccal mucosa specimen was-moderately differentiated non keratinizing squamous cell carcinoma with depth of invasion less than 5mm and margins were free from tumour, laryngectomy specimen was-well differentiated keratinizing squamous cell carcinoma, neck dissection specimen was-deep fascia and no lymph node sample detected. The patient during the postoperative period had normal oral stoma with adequate mouth opening and the neck wound was healthy with patent airway(adequate permanent tracheostoma). The patient was then referred for external beam radiation therapy and he was thereafter followed up every 1 month for 3 months and thereafter every 6 months. The postoperative period till now has been uneventful.

Discussion

The first case of synchronous cancers was reported by Billroth in 1889. That was a case of stomach cancer and second primary in external ear. Development of multiple malignant lesions can be explained by the phenomenon of "field of cancerization", that occurs in the aerodigestive tract mucosa at this level when exposed to the same type of carcinogens if they act consistently for a prolonged period of time.[10] Tobacco is a common risk factor responsible for the occurrence of both laryngeal carcinoma and buccal carcinoma and our reported case can be explained by theory of the "field of cancerization".

Buccal carcinoma arises from the overlying epithelium with its higher rate attributed to the widespread practice of betel nut chewing. It generally presents as a slow growing mass on the buccal mucosa. Associated symptoms are pain, bleeding, sensory deficit, dysphagia, odynophagia, trismus, facial palsy. The neck and parotid gland are palpated for lymphadenopathy. According to Diaz et al, 27 percent patients presented with clinically positive neck nodes in their study.[11] A meta-analysis by Chhetri et al involving four studies on

223 patients whereby in most T-stage were either T2 or T3.[12] Early lesions are managed by wide local excision by transoral route with at least 1 cm margin, whereas advanced lesions are excised and reconstructed with a cheek flap.

Min et al from their study on Korean population found standardised incidence ratio of SPN among oral cancer survivors was 1.47. Index oral cancer subsite was mainly floor of mouth (1.95) followed by gingiva (1.30). Most of the second cancers were associated with radiation history (1.94). The incidence of SPN was highest between 6 and 23 months after index oralcancers.[13] In a study by Sassi et al on Brazilian population most common index tumour site was the floor of mouth (43.2%) followed by buccal mucosa (2.7%). Most primary tumours were diagnosed at an advanced stage (stage 4 > stage 3). The most common site of SPN was oral cavity (40.5%) followed by oesophagus (16.2%) and larynx (8.1%). The average interval between diagnosis of first primary tumour and SPN was 65 months.14 Rajani et al in their study on a group of Indian people showed the most common subsites of oral cancer leading to SPN were lower gingivobuccal sulcus followed by palate, tongue and buccal mucosa.[15]

Hoarseness of voice is a usual presenting feature of early stage glottic carcinoma while dysphagia and aspiration present late in the course. Early stage disease does not have lymph node metastasis whereas, metastasis to pretracheal lymph nodes occur following subglottic extension. Early glottic carcinomas are managed by radiotherapy, endoscopic laser excision or conservative laryngeal surgery. Moderately advanced lesions(T3) are usually treated with chemoradiation until and unless there are features of laryngeal dysfunction.T4 (T4a) lesions are treated by total laryngectomy with central compartment neck dissection. Ghosh et al in such a rare case report opined that if surgery is needed for both the tumours, it can be done in a single stage in majority of the cases with low mortality and morbidity.[16] In our case too, we performed surgeries for both conditions in a single sitting.

The case being reported here is unusual as: (a) In our case the index tumour is in the buccal mucosa which isn't the most common site when compared with other studies. (b) Most of the SPNs present after 6 months as described in the above studies whereas our case is that of a synchronous secondary tumour. (c) Most of the cases of SPN have been found to be diagnosed at an advanced stage, whereas in our case the index buccal cancer belongs to early stage. (d) Most commonly reported SPN for oral carcinoma is the oral cavity itself but here second malignancy was in the larynx which is rare. (e) SPN for oral carcinomas have mostly been found in previously irradiated cases whereas in our case had no such history. (f) Most common laryngeal cancer associated with oral carcinoma is supraglottic carcinomas whereas our case it was glottic carcinoma.

References

1. Chaturvedi A K, Anderson W F, Lortet-Tieulent J, Curado M P, Ferlay J, Franceschi S, Rosenberg P S, Bray F, and Gillison M L. Worldwide Trends in Incidence Rates for Oral Cavity and Oropharyngeal Cancers. J Clin Oncol. 2013; 31(36):4550-4559
2. Jemal A, Siegel R, Ward E, Murray T, Xu J, Thun M J. Cancer statistics. CA Cancer J Clin.2007;57(1):43-66
3. Vigneswaran N, Michelle D. Williams. Epidemiological Trends in Head and Neck Cancer and Aids in Diagnosis. Oral Maxillofac Surg Clin North Am. 2014; 26(2): 123-41
4. Lambert R, Sauvaget C, de Camargo Cancela M, Sankaranarayanan R. Epidemiology of cancer from the oral cavity and oropharynx. Eur J Gastroenterol Hepatol. 2011; 23:633
5. Mehdi I, Shah AH, Moona MS, et al. Synchronous and metachronous malignant tumours expect the unexpected. J Pak Med Assoc. 2010; 60(11):905-9
6. Morris L G, Sikora A Gg, Patel S G, Hayes R B, Ganly I. Second primary cancers after an index head and neck cancer: subsite-specific trends in the era of human papillomavirus-associated oropharyngeal cancer. J. Clin.Oncol.2011; 29(6):739-46
7. Krishnatreya M, Rahman T, Kataki A C, Das A, Das A K, Lahkar K. Synchronous primary cancers of the head and neck region and upper aero digestive tract: Defining high-risk patients. Indian J Cancer 2013;50:322-6
8. Warren S, Gates O. Multiple primary malignant tumours: A survey of the literature and statistical study. Am J. Cancer.1932; 16:1358-414
9. Bagri P K, Singh D, Bardia M R. Double Primary Malignancies: A Clinical & Pathological Analysis Report from Regional Cancer Institute in India.Iran J Cancer Prev. 2014 Spring7(2)
10. Slaughter D P, Southwick H W, Smejkal W. Field cancerization in oral stratified squamous epithelium; clinical implications of multicentric origin. Cancer 1953; 6(5):963-8
11. Diaz E M Jr, Holsinger F Cc, Zungia E R, et al. Squamous cell

carcinoma of the buccal mucosa: one institution's experience with 119 previously untreated patients. Head Neck. 2003; Apr. 25(4):267-73

12. Chhetri D K, Rawnsley J D, Calcaterra T C. Carcinoma of the buccal mucosa. Otolaryngol Head Neck Surg. 2000; 123(5):566-71
13. Min SK, Choi SW, Lim J, Park JY, Jung KW, Won YJ. Second primary cancers in patients with oral cavity cancer included in the Korea Central Cancer Registry. Oral Oncol. 2019; 95:16-28. doi:10.1016/j.oraloncology.2019.05.025
14. Sassi L M, Cervantes O, Schussel J L, Stramandinoli R T, Guebur M I, Ramos G H A. Incidence of second primary oral cancer tumours: a retrospective study. Rev. Oconto ciênc. 2010; 25(4):367-70
15. Rajani B C, Hoda N , Dikhit P S, Roy S. Incidence of Second Primary Tumor in Patients with Oral Squamous Cell Carcinoma: Experience from a Tertiary Cancer Centre. IOSR Journal of Dental and Medical Sciences. (IOSR-JDMS) 2019; 18(8):72-4
16. Ghosh D, Sannigrahi R, Basu S K, Basu P. Synchronous squamous cell carcinoma of external acoustic meatus following pigmented basal cell carcinoma of cheek -a rare occurrence. Bengal Journal of Otolaryngology and Head Neck Surgery 2015; 23(3):123-8.

A Novel Account of the 'Forgotten Disease'

https://doi.org/10.47210/bjohns.2020.v28i2.324

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ABSTRACT

Introduction

Septic thrombophlebitis of Internal Jugular Vein (IJV) secondary to head neck infection, demonstrated by Lemierre in 1936, is a rare although known entity.

Case Report

A 35 year old known diabetic female patient presented in the ENT OPD with complaints of excruciating pain and swelling on the left side of the neck for past three weeks. A tender neck mass of around 8cm x 5cm in the left side of neck along the anterior border of sternocleidomastoid was palpable. A computed tomographic (CT) scan of neck revealed a thrombosed left internal jugular vein along with adjoining left sided level 2 and 3 suppurative cervical lymphadenopathy. She was treated conservatively with broad spectrum antibiotics and the patient improved after prolonged treatment.

Discussion

Lemierre syndrome has been appropriately termed as a forgotten disease. This has been an important cause of missed cases. High suspicion of IJV thrombosis in head neck infections, more so in the oropharyngeal infection is the key to diagnosis.

Keywords

Lemierre Syndrome; Thrombophlebitis; Neck

Septic thrombophlebitis of Internal Jugular Vein (IJV) secondary to head neck infection is a rare although known entity. Demonstrated by Lemierre in 1936, the disease still continues to have fatal natural course.[1] In the past few decades there has been a significant decrease in the incidence of the disease, probably due to the advent of a vast spectrum of antibiotics.[2] Lemierre syndrome affects mainly healthy people in the second and third decades of life.[3]

Fusobacterium necrophorum, a Gram-negative non-spore-forming obligate anaerobe is the main culprit organism.[3] Fusobacterium being a oropharyngeal commensal leads to Lemierre's syndrome following oropharyngeal and dental infections which accounts for 85% of the cases of the disease.[4] This article describes one such rare case.

Case Report

A 35 year old known diabetic female patient presented in the ENT OPD in a tertiary care hospital of Eastern India with complaints of excruciating pain and swelling on the left side of the neck for past three weeks. She had fever with highest recordable temperature of around 103° F. There was associated complaint of lethargy. The patient was a follow up case in the same department for chronic rhinosinusitis. She used to present with recurrent episodes of nasal blockage, cough and cold, headache, running nose. On physical examination the patient was febrile, tachypnoeic, tachycardic. A neck mass of around

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Fig. 1. Arrow points to the mass on the left side of the neck

8cm x 5cm in the left side of neck along the anterior border of sternocleidomastoid was palpable.(Fig.1) It was tender, local temperature was raised, firm in consistency, margins ill-defined and fixed to underlying structures. Nasal examination revealed mucopus in the right nasal cavity.

The patient was admitted in the indoor unit of the same department and was managed conservatively. Baseline work up revealed uncontrolled glycaemic status. A computed tomographic (CT) scan of neck (Fig. 2) revealed a thrombosed left internal jugular vein along with adjoining left sided level 2 and 3 suppurative cervical lymphadenopathy. CT scan of the paranasal sinuses (Fig. 3) showed hypodense shadows in the ethmoids, right sphenoid sinuses and a non contrast enhancing mass extending from the right nasal cavity into the nasopharynx. CT scan of chest did not demonstrate any significant abnormality. In Colour Doppler study, extra-cranial part of left IJV showed echogenic thrombus in its lumen with no flow, suggestive of IJV thrombosis. Haematological investigations showed increased leucocyte count with polymorphonuclear leucocytosis.

A blood culture specimen in both aerobic and anaerobic medium was collected aseptically and sent for microbiological assessment. Broad spectrum antibiotics were started in the form of injection Clindamycin. The culture sensitivity report obtained thereafter failed to demonstrate any significant pathological flora. So the same antibiotics were continued for three weeks. In consultation with cardio thoracic and vascular surgeons anticoagulant therapy was started in the form of heparin and warfarin. Heparin administration was tapered off in the next three days. Warfarin was continued for two weeks. A detailed recording of the patient's haemo-

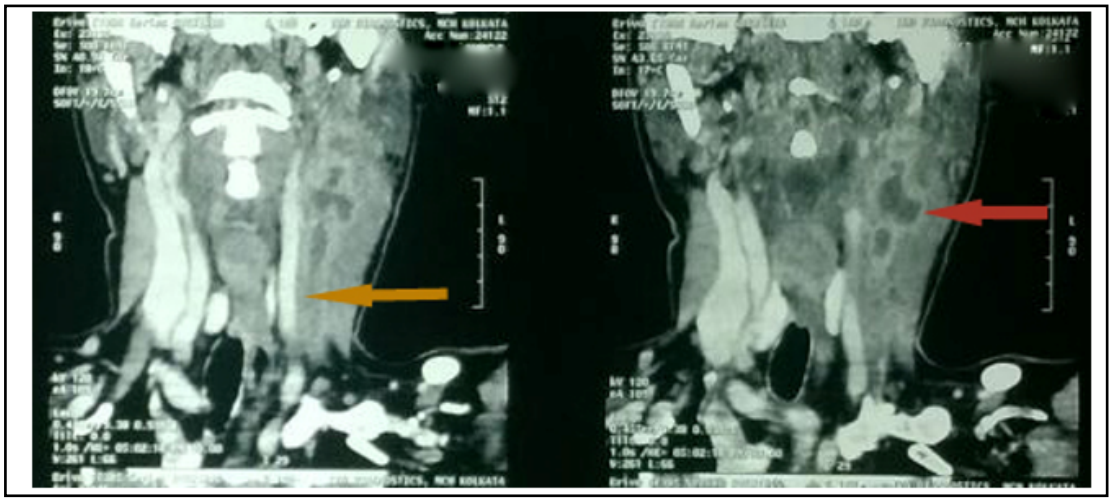

Fig. 2. CT scan of neck. Yellow arrow points to the thrombosed left internal jugular vein. Red arrow points to adjoining left sided level 2 and 3 suppurative cervical lymphadenopathy with central necrosis

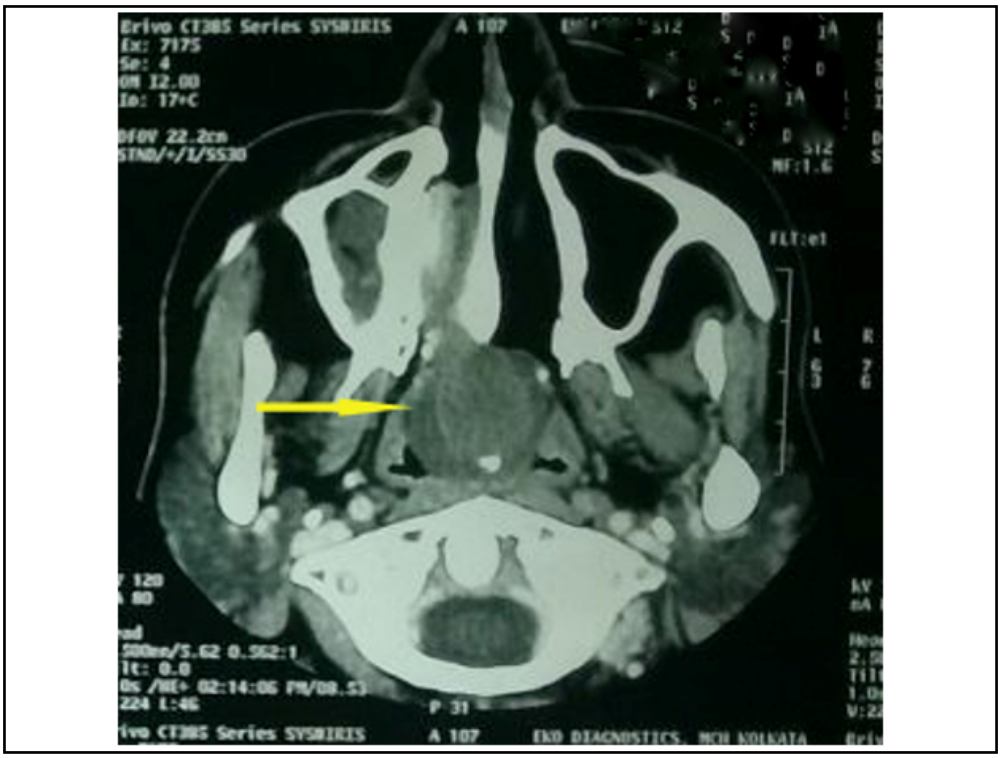

Fig. 3. Non contrast enhancing mass in left nasal cavity extending to nasopharynx

dynamic status was ensured. The stay in hospital was uneventful with the patient getting discharged in afebrile state with subsided neck swelling, after 3 weeks of injectable antibiotic administration. No surgical intervention had to be resorted to for the neck mass. Intranasal corticosteroid spray and oral antibiotics consisting of amoxycilin clavulanate was prescribed for three weeks along with probiotic capsules. Subsequent diagnostic nasal endoscopies showed that the nasal passage had cleared considerably due to reduction in size of the nasal polyp. Endoscopic excision of the nasal polyp was planned two months later after optimization of the glycemic status of the patient but she did not give consent for surgery and opted for medical management of rhinosinusitis.

Discussion

Septic thrombophlebitis of IJV secondary to head neck infection was taken as a case definition to diagnose Lemierre Syndrome.[1]

Postanginal sepsis was the term used by many authors in the first 40 years of the 20th century.[5,6] Vogel and Boyer in 1980 first included "Lemierre" in the title of their case report.[7] Shannon et al. in 1983 have been the first authors to use the term "Lemierre syndrome", in the title of their paper.[8]

Differences exist as to whether Lemierre syndrome relates to infection arising in the oropharynx caused by F. necrophorum or should it include infections arising in any site other than the oropharynx, or infections by non Fusobacterium group.[9] F. necrophorum although thought to be an unusual commensal of the oral flora can exist in other sites of the body. Lemierre reported that infection could arise from the gastrointestinal tract, the female genital tract, or the urinary tract.[10] Alston et al identified 21 published cases of F. necrophorum in the United Kingdom, of which six were directly related to tonsillar infection and would be compatible with Lemierre syndrome, seven infections arose from the female genital tract, including three cases that were postabortion or postpartum.[10]

There is dearth of data regarding the presence of this bacterium in the lymphoid organs other than the Waldeyer's ring. This case reports IJV thrombosis secondary to suppurative lymphadenitis of neck which in turn is secondary to an acute exacerbation of a chronic rhinosinusitis in an immunocompromised patient. There had been no case reports of IJV thrombosis from suppurative lymphadenitis as of now.

IJV thrombophlebitis at the early stage might be asymptomatic, so there is considerable dependence on imaging techniques. Radiological demonstration of IJV thrombosis might be the first evidence to suggest the diagnosis of Lemierre syndrome.[11] Extensive thrombophlebitis may be an indication for anticoagulation therapy or even surgical ligation. The standard method of diagnosis of deep venous thrombosis is catheter venography. However, this is invasive and carries the risk of propagating a septic process.[12] So it is rarely performed. Ultrasound scanning has been advocated for imaging jugular venous thrombosis. The technique is rapid, low cost, and noninvasive. But ultrasound cannot image beneath the clavicle or the mandible. Ultrasound may fail to indicate small thrombus. So it's sensitivity is impaired.[12] Several studies have demonstrated failure of ultrasonography to demonstrate internal jugular vein thrombosis that was readily demonstrable by contrast CT.[13] It is, however, of particular value for serial scanning after initial imaging.

Blood culture remains the standard for identification of Fusobacterium. But reliable identification to species level, additional tests are required. F. necrophorum is the only Fusobacterium sp. that ferments lactate to propionate, a fact that that is used for confirmation of F. necrophorum by gasliquid chromatography. However, for most routine laboratories, confirmation would probably be best achieved by referral of significant isolates, such as from blood culture, to a reference laboratory.[14] Non identification of Fusobacterium in blood culture in our case causes a diagnostic dilemma. It makes us ponder whether Lemierre syndrome can be caused by other pathogens. According to Riodan et al. the evidence is inconclusive as to whether F. necrophorum is the sole cause of Lemierre's syndrome.[14]

Since Lemierre Syndrome involves IJV thrombosis and abscess lacunae in the lungs the antibiotic choice and regimen has to be appropriately tailored. Amoxicillin and clavulanic acid or clindamycin, together with metronidazole remain the first line of defense before further wide spectrum antibiotics are used, such as vancomycin or imipenem.[15] Clindamycin is not susceptible to the "Eagle effect" as is penicillin. Empirical use of clindamycin has thus been advocated in severe infections.[16] The use of anticoagulants has been controversial, and no controlled studies exist.

Other treatment modalities include IV immunoglobulin, activated protein C, and hyperbaric oxygen in order to improve the immune response against the bacterial super-antigens. This modalities are still in experimental stage.[17,18]

Conclusion

Lemierre Syndrome has been appropriately termed as a forgotten disease. This has been an important cause of missed cases. High suspicion of IJV thrombosis in head neck infections, more so in the oropharyngeal infection is the key to diagnosis. Contrast enhanced CT of the neck is a tool to identify the thrombus. Demonstration of causative organism should not restrict the clinician to start antibiotic therapy. Clindamycin is the empirical antibiotic of choice. Long term antibiotic regimen is advocated. Need for anticoagulant therapy is debated. Early identification and appropriate intervention reduces the mortality.

References

1. Lemierre A. On certain septicaemias due to anaerobic organisms. Lancet. 1936; 227:701-3
2. McMullan R, McConville C, Clarke JC, Adams DA, Hedderwick S. Lemierre syndrome: Remember the forgotten disease. Ulster Med J. 2004;73:123-5
3. Righini CA, Karkas A, Tourniaire R, et al. Lemierre syndrome: study of 11 cases and literature review. Head Neck 2014; 36(7):1044-51. doi: 10.1002/hed.23410
4. Moore BA, Dekle C, Werkhaven J. Bilateral Lemierre's syndrome: A case report and literature review. (238-40). Ear Nose Throat J. 2002; 81:234-6
5. Abt, I. A. 1932. Postanginal sepsis. J Pediatr. 1:8-15
6. Boharas, S. Postanginal sepsis. Arch. Intern. Med. 1943; 71:844-53

7. Vogel, LC, Boyer KM. Metastatic complications of Fusobacterium necrophorum sepsis. Am J Dis Child 1980; 134:356-8
8. Shannon GW, Ellis CV, Stepp WP. Oropharyngeal Bacteroides melaninogenicus infection with septicemia: Lemierre's syndrome. J Fam Pract. 1983; 16:159-66
9. Chirinos JA, Lichtstein DM, Garcia J, Tamariz LJ. The evolution of Lemierre syndrome. Report of 2 cases and review of the literature. Medicine 2002; 81:458-65
10. Alston JM. Necrobacillosis in Great Britain. Br Med J. 1955; 1524-8
11. Screaton NJ, Ravenel JG, Lehner PJ, Heitzman ER, Flower CDR. Lemierre syndrome: forgotten but not extinct—report of four cases. Radiology 1999; 213:369-74
12. Braun IF, Hoffman JC Jr, Malko JA, Pettigrew RI, Daniels W, Davis PC. Jugular venous thrombosis: MR imaging. Radiology 1985; 157:357-60
13. Hong P, MacCormick J, Lamothe A, Corsten M. Lemierre syndrome: presentation of three cases. J. Otolaryngol. 2005; 34:352-8
14. Riordan T. Human infection with Fusobacterium necrophorum (Necrobacillosis), with a focus on Lemierre's syndrome. Clin Microbiol Rev. 2007; 20:622-59
15. Hughes CE, Spear RK, Shinabarger CE, Tuna IC. Septic pulmonary emboli complicating mastoiditis: Lemierre's syndrome revisited. Clin Infect Dis. 1994; 18:633-5
16. Pillai A, Thomas S, Williams C. Clindamycin in the treatment of group G beta-haemolytic streptococcal infections. J Infect. 2005; 51:e207-11
17. Hodgson R, Emig M, Pisarello J. Hyperbaric oxygen (HBO2) in the treatment of Lemierre syndrome. Undersea Hyperb Med. 2003; 30:87-91
18. Tewfik TL, Husein M, Shapiro RS, Oudjhane K. Lemierre syndrome in an immunocompromised patient. Int J Pediatr Otorhinolaryngol. 1999; 51:195-9.