

Original Article

Otological Presentation of Obstructive Nasal Lesions: A Study of 50 Cases

Md. Zahidul Islam¹, Md. Shafiul Akram², Mashuque Mahmud³, Md. Shahjahan Sarker⁴, Mahmudul Hasan⁵, Md. Abdur Rahman⁶, Md. Sirajul Islam Mahfuz⁷

Abstract:

Purpose: To analyze and realize the importance of the Eustachian tube dysfunction (ETD) in the pathogenesis of the middle ear infections as to raise the awareness amongst the Otolaryngologists consider the tubal dysfunctions in the management of the otitis media.

Materials and methods: 50 patients of aural discharge, aural fullness, aural pain, hearing loss and tinnitus along with nasal obstruction were selected from July 2013 upto December 2013 in the outpatient department (OPD) of Otorhinolaryngology - Head and Neck Surgery dept. of Dhaka Medical College Hospital under a specific prospective study protocol. All patients of aural fullness, hearing loss, aural pain, tinnitus, aural discharge along with nasal obstruction with or without sore throat were included; patients of sensorineural hearing loss, primary external ear pathology, ear malignancy and congenital ear diseases were excluded.

Results: 58% patients were below 20 years, 50% were children, 64% were male and 36% female. 62% patients got aural fullness, 60% hearing loss (40% mild, 14% moderate, 6% severe conductive and 8% mixed), 26% tinnitus, 32% aural discharge and 30% aural pain and amongst the patients of otitis media with effusion (OME), 100% got hearing loss, 73% aural fullness, 33% tinnitus, 27% aural pain and 13% dizziness. 78% patients got septal deviation, 58% hypertrophied inferior turbinates (HIT), 32% enlarged adenoids (EA), 62% allergic rhinitis, 26% acute upper respiratory tract infection (URTI), 4% nasal polyps and 48% sinusitis/mucosal thickening and amongst the children, 64% got enlarged adenoids, 56% HIT, 76% septal deviation (SD), 24% acute URTI, 4% antrochoanal polyp, 20% adenoid with HIT and 44% adenoid with SD. Regarding findings in the patients of enlarged adenoid, 37.5% patients got eustachian tube dysfunction (ETD) and 62.5% got hearing loss due to OME as well as acute otitis media (AOM), chronic otitis media (COM) and it showed that mild adenoid caused ETD occasionally on URTI but mild adenoid with HIT caused OME.

Conclusion: The obstructive nasal lesions are intermingled with otitis media, so this association should be sought out and underlying nasal lesions must be managed if any as a part of treatment of the otitis media.

Key words: Eustachian tube dysfunction, Acute otitis media, Chronic otitis media, Otitis media with effusion.

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1. Associate Professor (ENT), National Institute of ENT, Tejgaon, Dhaka
 2. Research Officer, National Institute of ENT, Tejgaon, Dhaka
 3. Assistant Professor (ENT), Cumilla Medical College, Cumilla
 4. Assistant Professor (ENT), National Institute of ENT, Tejgaon, Dhaka
 5. Assistant Professor (ENT), National Institute of ENT, Tejgaon, Dhaka
 6. Professor (ENT), National Institute of ENT, Tejgaon, Dhaka
 7. Professor (ENT), National Institute of ENT, Tejgaon, Dhaka

Address of Correspondence: Dr. Md. Zahidul Islam, Associate Professor (ENT), National Institute of ENT, Tejgaon, Dhaka, Bangladesh. Cell: +8801918185926, E-mail: rois87dc@gmail.com

Introduction

The small Eustachian tubes (ET) with tiny canals, responsible for proper vibration of the tympanic membrane (TM) by equalizing middle ear pressure and draining fluid from the middle ear, on getting plugged for a variety of reasons cause not only aural pain, aural discomfort but also muffled hearing or partial hearing loss, feeling of fullness /plugging in one or both ears, ringing or clicking or popping or ticking sounds in the ears, even tenderness around the ear and trouble with balance - a phenomenon referred to as ETD^{1,2}. This results in the pathogenesis of the middle ear infections or inflammation viz. AOM, COM and otitis media with effusion (OME). Nasal obstruction is probably the most common symptom of the diseases of nasal and paranasal sinuses and it is due to septal deviation, adenoidal hypertrophy with nasopharyngitis, allergic and infective rhinitis, rhinosinusitis and nasopharyngitis, HIT and nasal polyps³, amongst them allergies and acute coryza and influenza viral infections of nose, sinuses and nasopharynx causing severe nasal obstruction are the most common causes of ETD. Allergies by provoking edema and clogging with mucus, viral infections by provoking edema, clogging with mucus and destroying ciliary apparatus (viral infection probably being the most common cause of ciliary damage), adenoidal hypertrophy and nasal polyps, particularly the antrochoanal variety, by physical compression and nasopharyngeal neoplasia by invasion disrupt the proper tubal functions and give rise to aural symptoms of which the most frequent one is hearing loss by OME secondary to ETD^{1,2,4}. People with sinusitis are more likely to develop plugged ET and it is the smoking causing ciliary damages in throat and middle ear, obesity causing fat deposition around the tubes, allergies causing increased mucus and congestion, childhood having much smaller tubes and still

developing immune system rendering them prone to frequent colds and sinus infections and some activities associated with altitude changes suffer some people more regularly than the others^{1,2}. Sometimes ETD may be mistaken for patulous ET, and nasal polyps rather than the cystic fibrosis or allergic fungal sinusitis that obstruct nasal cavities and nasopharynx or both affect the tubes^{1,5}. When the air pressure in the nasopharynx is more than that in the middle ear pressure simply the tubal blockage commences, gradual pressure difference by one atmospheric pressure blocks the tube totally - thus the length and severity of ETD symptoms depend upon their initial causes, mostly it settles by itself within a couple of weeks which is especially true for children with common cold, hearing returns to normal within a week or two once the cold has gone as well as for minor bothersome symptoms in patients with altitude change which goes off on forcing a yawn or with chewing gum swallowing but when the symptoms are severe and persist for more than two weeks should be attended by doctors; children also should be attended since they are at an overall higher risk of getting ear infection or their symptoms mimic an ear infection^{1,2,6}. For chronic ETD (when the symptoms persist for more than six weeks) which is common in some adults - URTI, allergies, nasal obstruction by other cause viz. EDs in old children, nasal polyp, rarely nasopharyngeal tumour must be searched out; sometimes muffled hearing or aural fullness does not go away even the original cause (usually a bad cold) has gone away, sometimes symptoms persist despite trying all the usual treatments⁶. Chronic ETD does not get any genetic predisposition and constant ear pain, tinnitus and dizziness alongside muffled hearing usually are the harbingers of the complications of ETD like recurring symptoms and AOM, OME, COM, retracted membrane⁶.

Treatment for ETD also depends upon both the severity and cause of the condition, thus may include home remedies (chewing, swallowing, suckling, saline nasal spray), OTC medications (antihistamines for allergies, NSAIDs) and prescribed drugs (oral and nasal steroid) and supplements (pressure equalization implant, balloon dilator in eustachian tube) for severe cases, even myringotomy with or without tympanotomy and surgery for nasal obstruction for long-lasting severe cases; regarding the treatment of or prevention of the complications the restoration of eustachian tube patency is a mandatory since pressure equalization help with chronic middle ear infections and treating the underlying causes is the best way treat and prevent recurring symptoms hence the otitis media-thus children or adult getting frequent ear infections should be treated with the thinking of ETD^{1,2}.

Childhood deafness is a common health problem worldwide and in the developing countries, the greater proportion of childhood deafness is due to infection and in a study it was 38%⁷. Eustachian tube is traditionally assumed to be the main route for organisms reaching the middle ear and studies showed that shorter, straighter and more patulous tubes are more prone to AOM but research has found no difference in tubal dimensions in otitis prone and non-prone children rather significantly poorer active tubes are more prone to OM⁶. OME is virtually universal in children. Both infection and allergies cause adenoidal enlargement and major risk factor for OME is the eustachian tube blockage by this enlarged adenoid superadded by infection⁸. The important question is whether the allergic children are more prone to OME and if they do so whether their OME is more protracted than normal and several studies in different age group concluded that allergies not as a risk factor for occurrence or

persistence of OME⁹⁻¹¹. Risk factors for AOM in young children are prematurity, frequent URTI and no breast feeding¹²; and recurrent episodes of AOM is likely to be the single factor in developing their OME⁶. Important middle ear disease COM is one of the most common ear diseases in South East Asia having a prevalence rate about 5.2% in general population, which is 12.44%, 7.39% in Bangladesh; studies in Bangladesh, India, some countries in Africa and amongst certain underprivileged ethnic groups have shown that COM prevalence rate is 2-17% among children¹²⁻¹⁵. Recurrent attack of AOM and OME in children renders degeneration of outer and inner fibrous layers of lamina propria as well as submucosal layer of tympanic membrane resulting chronic perforation or retraction. Adult COM, which is not uncommon, results from episodes of AOM and some risk factors for AOM and OME are also common for COM like ETD, URTI and socioeconomic status cleft palate etc¹⁶. Rather than the racial variation in eustachian tube anatomy, repeated acute URTI, ultimately nasopharyngitis, with nasopharyngeal colonization by a wide variety of otological pathogens became recognized as a pivotal risk factor hence it prevents resolution of OM^{4,17}. Early nasopharyngeal colonization is associated with early onset of AOM¹⁸, which is in turn with the early onset of COM- higher rate of tympanic membrane perforation is seen in the 02-04 years age group which is roughly three times the rate seen in the adult^{19,20} and this implies the higher prevalence of childhood rhinitis and nasopharyngitis⁴. URTI produce transient ETD in healthy individuals may result OM-specifically repeated episodes of AOM results in chronic mucosal OM if the TM fails to heal after the first episode whereas OME predisposes to chronic squamous OM²¹. Adult-onset OME is less than childhood OME since its prevalence rate has been

reported as only 6% in a population aged 15 years or over and adults account for about 15% of OME cases²². Directly related to OME in adults is ET obstruction and indirectly related pathologies are URTI, nasal and nasopharyngeal allergies, nasal obstruction, nasopharyngeal pathologies, barotrauma, poorly pneumatized mastoid, prolonged intubation, autoimmune disease-CSF otorrhoea may present with the symptoms²¹; sinusitis being the main correlating disease since 63% patients of adult- OME got a prior URTI regarding which *S. pneumoniae* and *H. influenza* (15 specimen out of 19), *M. catarrhalis*, adenovirus were as common as in childhood-OME and OME is highly prevalent in HIV- infected adults (18%, in a series, were having COM, mostly OME)²³⁻²⁷ and regarding allergies, 57% had a positive skin prick test in a study with 53 adult-onset OME cases²⁸ and in another series with 48 non-acute OME, 97% got presence of allergy by using IgE level, RAST and skin test of which 89% got clinical corroboration and 88% eosinophilic effusions¹¹ and regarding nasopharyngeal lesions, 20% of adult-OME had enlarged adenoids, branchial cysts and Wegener's Granulomatosis were also reported to be the causes^{21,23} and in the endemic zones of nasopharyngeal carcinoma (NPC), adult-OME incidence were also high as to be 85% before and 48% after irradiation-

and however, position of the tube is less important than invasion of the tube by NPC²⁹⁻³¹ and barotrauma as well as HBO therapy render the ET unable or difficult to equalize negative middle ear pressure –out of 33 adult underwent HBO therapy, 52% got OME and 24% of them required ventilation tube³² and poorly pneumatized mastoid exhibited higher chance of OME following URTI in comparison with normal mastoid²⁶. Clinical diagnoses of NPC are confirmed by tympanometry, MRI should be the more routine investigation in endemic zone of NPC, aspiration of fluid during myringotomy was considered to be the confirm diagnosis in some studies^{24,33,34} but it is emphasized that absence of fluid does not imply an absence of OME since one-third patients in the same MRI study had fluid in the mastoid rather than in the mesotympanum.

Methods:

Type of study: Prospective study

Place of study: Department of ENT, Dhaka Medical College Hospital.

Period of study: 6 months from July 2013 to December 2013.

Number of patients: 50 patients of aural fullness/hearing loss, aural pain, tinnitus, aural discharge along with nasal obstruction with or without sore throat were selected randomly.

Results:

Table I :
Distribution of the otological symptoms (n=50)

symptomes	No. of the patients	Diagnosis	Percentage
Aural fullness	31(intermittent-20;often,disappears on swallowing/valsalva-11)	ETD/OM	62% (40%+22%)
Hearing loss	30(mild-20,moderate-07,severe-03; mixed-04)	OM	60%
Tinnitus	13	ETD/OM	26%
Aural discharge	16(AOM-03,OME-01,COM-often 03; on cold attack-09)	OM	32%
Aural pain	15(AOM-03,mild pain OME-04, discomfort ETD-08)	ETD/OM	30%

Table II :
Symptoms of provocative obstructive nasal lesions (n=50)

Symptomes	No. of the patients	Percentage
SD	39(mild-20,moderate-12,gross-07)	78%
HIT	29(only HIT-07,along with septal dev.-22)	58%
EAs	16(only adenoid-06,along with HIT-05,along with septal dev.-11)	32%
Allergic rhinitis	31	62%
Acute URTI	13(children-06,adoloscent-04,adult-03)	26%
Nasal polyp	02(ethmoidal-01,antrochoanal-01)	04%
Sinusitis/Mucosal thickening	24(with allergic rhinitis-20,with URTI-02,with polyp-02)	48%

Table III :
Otological findings (n=50)

Side	Tinnitus	Aural		Tympanic membrane (n=50)					Tunning fork test (n=50)		
		fullness/ ETD	Dull	Mild retraction	Mod retraction	Gross retraction	Perfo- ration	Ragged, sodden, featu.	Normal	Weber lateral	Rinne nega. on
Right	04	04	03	02	03	01	03	-	20	05	04
Left	05	05	04	04	04	02	05(01 -AOM)	01 (AOM)	17	08	05
Bila- teral	04	11	05	04	02	01	06 (01- AOM)	-	17		11
Total	13 (26%)	20 (40%)	12 (24%)	10 (20%)	09 (18%)	04 (8%)	14 (28%)	01 (2%)	17 (34%)	13 (26%)	20 (40%)

Table IV :
Otological investigations (n=50)

Side	Hearing loss(n=30)					Tympanogram type(n=50)				
	Mild	Moderate	Severe	Mix.-COM	A	As	C	B	Flat	
Right	04	02	-	01	20	05	15	06	-	
Left	06	03	02	02	18	02+03	02+12	03+04	02+03	
Bilateral	10	02	01	01	18	03	12	04	01	
Total	20(67%)	07(23%)	03(10%)	04(13%)	20(40%)	05(10%)	15(30%)	06(12%)	04(8%)	

Table V :
Provocative obstructive nasal lesions-findings (n=50)

Side	SD	HIT	EA	URTI	Nasal Polyps	EAs with HIT	EAs with SD	Bilateral nasal obstruction
Left	17	HIT-03, HIT+SD-06	-	06	01	02	05	
Bilateral	10	HIT- 02,HIT+SD -06	16	03	01	02	03	27
Total	39(78%)	29(58%)	16(32%)	13(26%)	02(4%)	05(10%)	11(22%)	27(54%)

Table VI :
Childhood distribution of the obstructive nasal lesions (n=25)

Lesions	No. of the patients	Percentage
EAs	16	64%
HIT	14	56%
SD	19	76%
URTI	06	24%
Polyp(antrochoanal)	01	4%
EAs with HIT	05	20%
EAs with SD	11	44%

Discussion:

A full history of middle ear disease in childhood, earache, symptoms of nasal obstruction and fullness, cigarette smoking, any profession related with altitude change and barotrauma, a family history of middle ear disease specially for NPC and whether onset of symptoms are associated with episodic URTI or AOM should be carried out^{21,24} and to assess the functional and organic obstruction of the ET, transnasal endoscopy of the pharyngeal orifice revealed edema of the orifice 27%, blockage by mucopurulent discharge 23%, atrophy of the orifice 10% and normal appearance 40% in a study of 78 adult-OME cases³⁵ and tubotympano-aerodynamography revealed 52% functional and 46% organic obstruction in a study of 162 ears with OME³⁶. In our study, 58% patients were below 20 years of age and 50% were children, and 64% were male and 36% female. In a study on COM prevalence in one district level hospital in Bangladesh, maximum (36.5%) patients were of 11-20 years age group and 43.5% were below 20; in another study on degree and pattern of hearing impairment in national institute of ENT of Bangladesh, 61.27% patients were male- so they almost mimic our age and sex distribution.

In our study, 62% patients got aural fullness (40% intermittent-due to ETD, 22% often-due to OM), 60% hearing loss (40% mild, 14% moderate, 6% severe conductive and 8% mixed), 26% tinnitus, 32% aural discharge (6% of AOM, 2% of OME, 24% of COM-6% often and 18% on cold attack) and 30% aural pain (6% marked pain in AOM, 8% mild pain in OME, 16% just discomfort in ETD); and amongst the patients of OME, 100% got hearing loss, 73% aural fullness which is often and only disappears on effort of swallowing / valsalva, 33% tinnitus, 27% aural pain and 13% dizziness. In a study on hearing impairment degree and pattern in Bangladesh, 40.9% patients got mild hearing loss which mimics our result and another study on symptoms of OME revealed hearing loss- 97%, aural fullness- 77% and earache, dizziness were also often reported by the subjects which also almost mimics our result²¹. From the sincere history taken, the patients of ETD make air thrashings open the eustachian tube by frequent swallowing or hawking or yawing or even valsalva but when the tube is no more ready to open mechanically the OME is invited; the AOM is invited if any superadded infection-they simply narrate that on a cold attack or on an exposure to an allergen their ear/ears starts/ start discharge or weep or being full, the ear on the side of more nasal obstruction starts first or do more or both and even a mild cold attack affects the ears on the side of nasal obstruction; more on the side of more obstruction.

Distribution of the causative or correlating factors in adult-onset OME varies in the literatures-in an Israel study, they were as sinus disease 66%, enlarged adenoid (19%-some of which were related to smoking), NP tumours 13% and unidentifiable 2% but in endemic zone for NPC, it is relatively high^{23,37} and in another series, as URTI 22%,

chronic sinusitis 14%, NPC 6%, another causes 14%²¹ In our study, 78% patients got septal deviation (40% mild, 24% moderate, 14% gross), 58% got HIT, (14% only HIT, 44% with SD), 32% got EAs (12% only adenoid, 10% with HIT, 22% with SD), 62% got allergic rhinitis, 26% got acute URTI (12% children+8% adolescent, 6% adult), 4% got nasal polyps (2% antrochoanal, 2% ethmoidal) and 48% got sinusitis/mucosal thickening (40% with allergic rhinitis, 4% URTI, 4% polyposis) which were provocative for OM/ETD and amongst the children, 64% got enlarged adenoids, 56% HIT, 76% SD, 24% acute URTI, 4% antrochoanal polyp, 20% adenoid with HIT and 44% adenoid with SD. Adenoiditis alone as well as adenotonsillitis which is a common ENT problem bearing 20% of all throat infection cause the tubal blockage³⁸- one study showed that the more the size of enlarged adenoid the more is the chance of occurring OME (about 73% with severe adenoids), having more hearing loss (about 38% OME patients with severe adenoids got moderate deafness) and more negative middle ear pressure (63% patients with severe adenoids got pressure ranging from -201 to -400 daPa)⁸ and another study with adenoid surgery showed that 51% patients were of 4-10 years and sufferings like nasal obstruction with mouth breathing was 46%, nasal obstruction with ETD was 7% and associated tonsillitis was 30%³⁸ –so adenoid is not always responsible to develop OME but in most OME patients usually have enlarged adenoid and in this study, 42% patients got no hearing loss audiometrically⁸.

Regarding findings in the patients of enlarged adenoid, 38% patients got ETD and 62% got hearing loss due to OME as well as COM and it showed that mild adenoid caused ETD occasionally on URTI but mild adenoid with HIT caused OME; 6% patients with mild adenoid caused 11% OME in which hearing

loss was 100% mild and 100% got -100 to -200 middle ear pressure, 19% with moderate adenoid caused 33% OME in which hearing loss was 100% mild and 75% got -101 to -200 and 25% -201 to -400 pressure and 31% with severe adenoid caused 56% OME in which 60% hearing loss was mild and 40% moderate and 20% got -101 to -200 and 80% -201 to -400 pressure and rest 6% with severe adenoid got COM having -201 to -400 pressure –in a study on effect of enlarged adenoid in the development of OME in Bangladesh revealed 42% normal hearing and 58% hearing loss; mild adenoid caused 14.2% OME in which hearing loss was 100% mild and 100% got -100 to -200 middle ear pressure, moderate adenoid caused 57.9% OME in which hearing loss was 83% mild, 17% moderate and 75% got -101 to -200 and 25% -201 to -400 pressure and severe adenoid caused 72.7% OME in which 62.5% hearing loss was mild and 37.5% moderate and 37.5% got -101 to -200 and 62.5% -201 to -400 pressure⁸ and another study with adenoid surgery showed 7% ETD by nasal obstruction with enlarged adenoid and 42% case got normal hearing audiometrically^{8,38} which almost mimic our result; another audiological study also revealed OME related hearing loss in the children being 18-35 dB³⁹ –another also revealed fluctuating hearing loss ranging from 15-40 dB with a mean of 27 dB found in most children having middle ear effusion resulted from OME or AOM; after resolution of symptoms middle ear effusion may persists even for weeks to months following AOM, although this condition is indistinguishable from OME, it is the volume rather than the viscosity/quality of the fluid that matters on hearing threshold and duration of the effusion can be acute (< 3 weeks), sub acute (3 weeks-3 months) and chronic (≥ 3 months)⁴⁰ but our study revealed 78% hearing loss being mild in the adenoid induced OME and this small variation is since our study is

not only on adenoids rather on all the benign obstructive nasal lesions. Clinically OME is more apparent in adult than in young children who cannot easily express themselves with mild to moderate hearing loss and 42-69% adult- OME were reported to be bilateral²⁹. ETD is more common in patients of COM than in normal individuals and incidence of COM in cleft palate patients followed upto 10 years of age is around 20%, with 2% of them having cholesteatoma and hypoplastic tensor veli palatine muscle in cleft palate predispose to ETD^{41,42}.

In our study, regarding aural fullness (20-40%- due to ETD) it was 4 in right, 5 in left, 11 bilaterally; regarding tinnitus 13-26% it was 4 in right, 5 in left, 4 bilaterally; regarding OM (30-60%)-amongst the OME 15-50% it was 3 in right, 4 in left, 8 bilaterally, amongst the COM (12-40%) it was 3 in right, 3 in left, 6 bilaterally and amongst the AOM 3-10% it was 1 in right, 2 in left; regarding tympanic membrane status, amongst dull membrane (12-24%) it was 3 in right, 4 in left 5 bilaterally, amongst mildly retracted membrane 10-20% it was 2 in right, 4 in left, 4 bilaterally, amongst moderately retracted membrane 9-18% it was 3 in right, 4 in left, 2 bilaterally, amongst grossly retracted membrane 4-8% it was 1 in right, 2 in left, 1 bilaterally, amongst COM perforation 12-24% it was 3 in right, 4 in left 5 bilaterally and amongst AOM 3-6%, perforation was 1 in left, 1 bilaterally and in the rest, the membrane was ragged sodden featureless; regarding tuning fork test, amongst 20(40%) patients 17 got bilaterally normal test of which 3 got Weber lateralization to left being added to another 5, lateralization also to right was 5(10 got Weber lateralization), amongst Rinne negative (20-40%) 4 was on right, 5 on left, 11 bilaterally; regarding hearing loss, amongst the mild loss (20-67%) 4 was in right, 6 in left, 10 bilaterally, amongst the moderate loss

(7-23%) 2 was in right, 3 in left, 2 bilaterally, amongst the severe loss (3-10%) 2 in left, 1 bilaterally and amongst mixed loss (4-13%) 1 in right, 2 in left, 1 bilaterally and regarding tympanogram in the concerned aural fullness and hearing loss, 18 out of 20 (40%) of ETD got bilateral A type, rest 2 got As type in left being added to another 3 (total 5-10%; 5 in right-3 bilaterally), this rest 2 got C type in left being added to another 12 (total 15-30%; 15 in right-12 bilaterally), this rest 3 got B type in left being added to another 4 (total 6-12%; 6 in right-4 bilaterally), this rest 2 got flat type in left being added to another 3 (total 4-8%; 3 in left, 1 bilaterally) having a left preponderance and in the context of provocative obstructive nasal lesions, regarding SD (out of 39- it was 17 towards left, 12 towards right, 10 bilaterally); regarding HIT (out of 29- it was 3 only HIT, 6 along with SD in left; 2 only HIT, 10 along with SD in right; 2 only HIT and 6 along with SD bilaterally); Enlarged adenoids were 16 causing bilateral obstruction and adenoid with HIT were 2 in left, 1 in right 2 bilaterally and adenoid with SD were 5 in left, 3 in right, 3 bilaterally; regarding URTI (out of 13- it was obstruction 6 in left, 4 in right, 3 bilaterally in children); regarding nasal polyp (out of 2-1 was antrochoanal in left, 1 ethmoidal bilaterally)- thus implying that both provocative obstructive nasal lesions as well as otological lesions and findings got preponderance in the left side and proving as well as establishing that there is a sound and profound association between obstructive nasal lesions and middle ear pathologies via Eustachian tube involvement which is also supported by other previous studies viz. an Israeli study showed sinus diseases as 66% cause, enlarged adenoid as 19% cause and another study showed URTI as 22% cause of adult-OME, another study declaring the sinusitis being the main correlating disease for OME showed 63% patients of adult-OME got a prior

URTI^{23,24,37}; our study also showed sinusitis/ mucosal thickening in 48% cases, enlarged adenoid in 32% cases and URTI in 26% cases being related to OME in both children and adult- and regarding URTI in enlarged adenoid, it is the removal of a chronic source of nasopharyngitis rather than removal of mechanical obstruction of ET which is achieved by adenoidectomy. According to the statement given by the patients, they being used to make repeated swallowing, hawking, yawning or even valsalva as an effort open the blocked eustachian tube suffer from eructation, belching and this is the relation with GERD which needs further research. MRI showed tensor veli palatini destruction and eustachian tube erosion, localised effusion in mastoid or tympanum or both in NPC⁴¹; study revealed that 33% such OME resolved after radiotherapy although radiation to ears, ET and NP for NPC and other head-neck cancers itself is also a causative factor in OME⁴⁴ but luckily or unluckily we did not have any NPC in our study. Overall, reduction or alleviation of the nasal obstructive lesions should lessen the magnitude of the otological lesions or even heal up them totally but leaving the obstructive nasal lesions as it is may not improve/heal up the otological lesions even after surgical procedures which we found clinically and it will explore the field of further study. Both medical as well as surgical addressing of the obstructive nasal lesions aided the clinical improvement of our cases; on the contrary, failing of this addressing resulted recurrence or no marked improvement in the treatment in some of our patients.

Conclusion:

This study implies that there is a sound and profound association between obstructive nasal lesions and middle ear pathologies via Eustachian tube involvement. If the study could be performed in a large scale,

eustachian tube involvement in the pathogenesis of the otitis media would be more established; hence its management will carry more credit in the management of otitis media. So commencement of treatment or finalization of treatment plan for otitis media should not ignore the checking for or exclusion of the underlying causes as well as risk factors for eustachian tube dysfunction, specially the nasal obstructive lesions.

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