Preoperative predictors of ossicular necrosis in chronic suppurative otitis media, tubotympanic disease

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A dissertation submitted in part fulfillment of MS Branch IV, ENT examination of the Tamil Nadu Dr. MGR Medical University, to be held in March 2009

Department of Otorhinolaryngology
Christian Medical College, Vellore

Certificate

This is to certify that the dissertation entitled 'Preoperative predictors of ossicular necrosis in chronic suppurative otitis media, tubotympanic disease' is the bonafide original work of Dr Jareen Ann Ebenezer submitted in fulfillment of the rules and regulations for the MS Branch IV, ENT examination of the Tamil Nadu Dr. MGR Medical University, to be held in March 2009.

Guide:

Dr Rupa Vedantam
Professor and Head
Dept of Otorhinolarygology
Christian Medical College
Vellore, India

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AIMS AND OBJECTIVES OF THE STUDY

To study the relationship of preoperative clinical, audiological and radiological factors with the status of the ossicular chain at surgery in patients with chronic suppurative otitis media (CSOM), tubotympanic disease.

PRESENT KNOWLEDGE AND REVIEW OF LITERATURE

Introduction:

Chronic suppurative otitis media (CSOM) refers to chronic infection (more than 6 weeks) of the middle ear cleft (Eustachian tube, middle ear and mastoid) in the presence of a persistent tympanic membrane perforation. (World Health Organization, 1998). There may be intermittent or constant purulent otorrhoea and mastoiditis[1]. CSOM has been an important cause of middle ear disease since prehistoric times. Its prevalence depends to a great extent on race and socio-economic factors[2]. Otologists, well aware of the potentially dangerous and life-threatening risks of otitis media, concentrate on treating the disease and rendering the ear safe. Hearing reconstruction often takes second priority[3,4]. Patients, however, are more concerned about the hearing loss caused by this disease because the other subjective symptoms of CSOM may be minor or lacking[4].

Chronic Suppurative Otitis Media : Clinical features :

a) Symptoms :

The classic symptoms of chronic suppurative otitis media are otorrhoea and deafness. The ear discharge may be intermittent or continuous and may affect one or both ears. It may be serous, mucoid or purulent in nature, often greatly increasing in quantity during an upper respiratory infection or due to water contamination from the external canal after bathing or swimming. The presence of granulation tissue or an aural polyp may cause blood staining of the discharge; this may also be frequently associated with cholesteatoma or even bone erosion without cholesteatoma. Less common otologic symptoms which are more indicative of a complication include earache, vertigo, deviation of angle of mouth and periauricular swelling[5].

b) Otoscopic features :

The tympanic membrane perforation is the hallmark of the safe type of otitis media. The defect in the tympanic membrane may range from the size of a pin-hole to a subtotal defect where only the annulus may be left intact. There may also be ear discharge, which indicates the state of activity of the disease.

The state of the middle ear mucosa, the presence of an aural polyp, ossicular abnormalities, the Eustachian tube, the round window niche and the promontory can be seen and assessed depending on the size and location of the perforation[6].

In the atticoantral type of disease, the otoscopic appearances include retraction of the postero-superior quadrant of the pars tensa, or attic retraction, with accumulation of keratin debris and cholesteatoma. There may or may not be associated granulation tissue and scutum erosion[7].

Conductive Hearing Loss in CSOM :

Hearing loss in CSOM may be conductive, sensorineural or mixed. The auricle, external auditory canal, tympanic membrane and ossicular chain participate in localization, amplification and transmission of sound to the cochlea. The malleus, incus and stapes along with the tympanic membrane

are a vital part of the impedance matching mechanism of the middle ear. Disruption of this conductive mechanism at any point till the stapes footplate may result in conductive hearing loss[8,9].

Chronic suppurative otitis media is commonly associated with conductive hearing loss. A large central perforation of the tympanic membrane greatly decreases the effective vibratory area of the tympanic membrane. It may reduce the 'baffle effect' of the round window and thus cause a more severe deafness. Impairment of the mobility of the ossicular chain, either through bone erosion or ossicular fixation may also increase the degree of hearing loss. Granulation tissue, mucosal oedema and other middle ear pathology may influence the sound conducting mechanism.

The tympanic membrane and ossicular chain : Their function in hearing :

During the course of evolution, as animals left the seas and took over dry land, there was a need to overcome the mechanical air-water sound barrier. The middle ear mechanism was adapted from the now useless branchial apparatus. Thus, air-borne sound vibrations of a large amplitude but small force are transformed to fluid-borne sound vibrations of small amplitude but larger force[10].

The middle ear acts as an 'acoustic transformer' by coupling sound vibrations from the air column in the external canal to the cochlea. An acoustic transformer increases either pressure or volume velocity while decreasing the other, thereby equalizing the sound power at the input and output. This is mainly accomplished through the action of the tympanic membrane and the ossicular chain. The major transformer mechanism within the middle ear is the ratio of the tympanic membrane area to the area of the stapes footplate, called the 'areal ratio'. The other transformer is the 'ossicular lever', the lever action which results from the different lengths of the rotating malleus and incus arms around the axis of rotation of the ossicles. In humans, the lever arms of the malleus and incus are nearly the same length, hence their ratio of 1.3 contributes a 2dB gain in sound pressure. If these sound transformers acted ideally, the theoretical middle ear sound pressure gain would be 28dB. In reality, this is decreased due to the fact that portions of the tympanic membrane vibrate differently and that a part of the force is used up in overcoming the inertia of the ossicles and their suspensory ligaments[9].

Defects in the ossicular chain :

a) The importance of ossicular chain defects :

Defects of the ossicular chain may occur in all chronic middle ear disease, with or without cholesteatoma[11]. However discontinuity of the ossicular chain is typically confirmed only during an operation. Another method of defining the middle ear and identifying ossicular discontinuity is by high resolution three-dimensional computed tomography. This is not routinely done for patients with uncomplicated CSOM as it contributes little to the actual surgical management of these patients[12]. The cost of the study is also a limiting factor. Knowing before surgery whether the patient has an ossicular discontinuity is important because it allows the surgeon to plan ahead regarding the need for an ossiculoplasty and the type of ossiculoplasty required for that particular patient[13]. Carrillo et al suggested that a larger air-bone gap at higher frequencies pointed towards the presence of ossicular discontinuity[14]. The patient, anxious about his hearing improvement following surgery, could also be provided with a realistic explanation and accurate information about the status of his/her ossicular chain, the probability of performing an ossiculoplasty, possible surgical complications and benefits[13].

b) The prevalence of ossicular defects in similar studies :

A comparison of details from previous studies on ossicular necrosis has been listed in Table I. One of the earliest thorough studies on ossicular discontinuity was carried out in 1979 by Mirko Tos[11], who studied a series of 1,100 consecutive ears, 426 with cholesteatoma. He described the various defects of the ossicular chain and related them to the nature of the disease and the site of the perforation. He found that an isolated defect of the long process of the incus was the commonest ossicular defect in all types of ear disease and that ossicular erosion was by far more common in ears with cholesteatoma or the presence of squamous epithelium[11].

Table I : A comparison of incidence of ossicular discontinuity in previous studies

Author	**Diagnosis**	**Number**	***Ossicular discontinuity***	**Malleus**	**Incus**	**Stapes**
Austin, 1971	Chronic ear disease	not specified	50%			
Sade et al, 1977	Simple chronic otitis	75	42%			
Tos M, 1979	Cholesteatoma, granulating otitis, sequelae of otitis, adhesive otitis	1100	8.63%	25	74	21
Jeng FC et al, 2003	Non cholesteatoma ears	153	11.11 %		17	
Carrillo RJC et al, 2007	Both cholesteatoma & non cholesteatoma ears	not specified	58.3%			

c) Factors predicting the defects in the ossicular chain :

The various defects of the ossicular chain are related to the nature of the disease and the site of the perforation[11]. Thomsen et al[15] and Tos[11] observed that the long process of the incus and the stapes suprastructure are the most frequently affected ossicles[11,16]. Tos[11] found that isolated defects of the malleus handle were more common in total perforations, or in cholesteatoma of the pars tensa[11]. He also found that total or posterior perforations are associated with necrosis of the ossicles more often than anterior or inferior perforations. Destruction of the body of the incus and head of the malleus usually showed the presence of squamous epithelium in close proximity[11].

Jeng et al[13] demonstrated that the air-bone gap was larger in ears with ossicular discontinuity than those without. However, it was not a good parameter for predicting ossicular discontinuity because the cholesteatoma or granulation tissues might serve as transmission bridges and reduce the ABG. Poor mastoid pneumatization as seen on X-ray, presence of ear discharge, subtotal perforation, polypoidal middle ear mucosa and presence of granulations were all present more often in ears with ossicular discontinuity. Adhesion of the perforation edges to the promontory showed a statistically significant association with ossicular discontinuity. In 2007,

Carrillo et al[14] studied the possibility of predicting ossicular discontinuity using the ABG. They concluded that narrow ABG at lower frequencies suggested absence, and larger ABG at higher frequencies suggested the presence of ossicular discontinuity.

Histopathological features of CSOM :

The severity of the disease is a deciding factor in the histopathological changes that occur in the ossicles. The degree of inflammation is also related to the duration of otorrhoea.

The tympanic cleft is lined by a layer of cuboidal or columnar epithelium, which may be ciliated in places and may have goblet cells in the region below the horizontal segment of the facial nerve[6].

In chronic suppurative otitis media without cholesteatoma, the middle ear epithelium begins to resemble classical respiratory epithelium. Mucosal oedema occurs due to increased capillary permeability of the lamina propria of the middle ear mucosa. There is also a collection of cells of chronic inflammation, namely, lymphocytes, plasma cells and histiocytes. An increase in the number of goblet cells may occur and their secretions contribute the mucoid component to the ear discharge[6].

Chronic inflammation predisposes to the formation of granulation tissue, aural polyps and sometimes, cholesterol granuloma. As the disease progresses, there is a gradual decrease in vascularity, leading to fibrosis and tympanosclerosis[6].

a) Manner in which CSOM affects the ossicles :

The bony resorption seen as a result of CSOM is due to an increase in osteoclastic activity or avascular necrosis. The state of chronic inflammation and granulation which occurs in CSOM is responsible for the increased osteoclastic activity. The long process of the incus and the stapes suprastructure are most susceptible to avascular necrosis. Fixation of the head of malleus and incus can also occur due to new bone formation[6]. A variety of necrosed ossicles can be seen in Figure 1.

b) Bone Resorption :

Although cholesteatoma is a major factor contributing to ossicular pathology, there is bone erosion is CSOM without cholesteatoma. Earlier theories of bone 'necrosis' have now been replaced by bone 'resorption' which suggests the presence of live cells and demineralization of bone. The anoxia-necrosis theory of Ruedi and Tumarkin 1958 has been replaced by

the inflammation-enzymatic theory, though it has been suggested by Tos that pressure also does play a part[11]. Schachern[16] has reported that granulation tissue may be trapped in non-draining spaces of the middle ear cleft. Chole[17] observed that granulation tissue can lead to bone erosion; also that it is the presence of infection and inflammation rather than just the presence of cholesteatoma which leads to bone resorption. Chole[17] has given ultrastructural evidence in human and experimental cholesteatomas that bone resorption is primarily a result of the action of multinucleated osteoclasts on bone. These osteoclasts release enzymes (acid phosphatases, collagenases, acid proteases) into their immediate microenvironment. The osteoclast activity is mediated by certain cytokines and interleukins which act as cellular recruiters and promoters. They often need a 'physical trigger factor', e.g. pressure caused by cholesteatoma or granulations. Thomsen et al[15] have shown that bone resorption can occur in those with CSOM with or without cholesteatoma.

The Air-Bone Gap and its relevance to ossicular discontinuity or fixation :

The difference between the levels of air conduction and bone conduction in an audiogram is termed the 'Air-Bone Gap'(ABG). In cases

where the ABG is 15dB or greater, it implies that there is a significant disruption of the conductive pathway[18]. This may be due to a number of causes, ranging from wax impacted in the ear canal, a perforation in the tympanic membrane, ossicular discontinuity or fixity of the ossicles.

Carrillo et al[14] reviewed 276 patients undergoing tympanoplasty for CSOM without cholesteatoma and found a significant co-relation between ABG and ossicular discontinuity. As the efficiency of the ossicular chain varies at different frequencies, a frequency-specific analysis would better reflect a relationship between air-bone gap and the status of the ossicles. They stated that a 20dB or less ABG at 500Hz predicts an absence of ossicular discontinuity and an ABG of 30dB or greater at 2000Hz predicts the presence of ossicular discontinuity. This difference may be due to the varying efficiencies of the ossicular chain in transmitting sound of different frequencies. The ABG was not a reliable indicator in ears with cholesteatoma. They concluded that narrow ABG at lower frequencies suggested an absence of ossicular discontinuity while wide ABG at higher frequencies indicated the presence of ossicular discontinuity.

Those patients with a small chance of ossicular pathology need to be taken up only for a simple tympanoplasty, while those with higher chances of disruption of the ossicles would require a mandatory exploration of the

ossicular chain and reconstruction. The presence of cholesteatoma would be a definite indication of mastoid exploration. They concluded that a combination of frequency specific ABG and clinical findings would provide a good estimate of the degree of ossicular disruption[14].

Radiology

Today, the use of conventional radiology is limited to the evaluation of gross anatomical landmarks and the assessment of mastoid pneumatization. The development of the mastoid varies from person to person and from one side to the other side of the same patient. Pneumatization may be limited to a single antral cell, or it may extend into the mastoid tip, the squama of the temporal bone and even invade the adjacent zygoma and occipital bone. The non-pneumatized mastoid process may consist of solid bone or diploic spaces filled with fatty marrow[19].

It has been established by numerous studies that there is a correlation between ear disease and the size of the mastoid air cell system. Several methods exist to measure the size or volume of the mastoid, the main ones being direct volumetric measurement and a planimetric measurement of area on a standard lateral projection. Diamant in 1940, described the distribution of the mastoid air cell system and measured the roentgenological area of the

mastoid planimetrically. This method is still the simplest and the most used technique for measurement of the size of the mastoid air cell system. In 1965, Flisberg and Zsigmond[20] demonstrated that there is good agreement between a planimetric determination of the area of the mastoid air cell system and a direct clinical determination based on volumetric measurements. Normally, the area of the shadow of a spherical object has an exponential relation to the volume of that object. However, the mastoid pneumatization area resembles that of a cone with its base oriented laterally. In this orientation, the area of the shadow of the base of a cone has an almost linear correlation with its volume[21].

When a well aerated mastoid cavity communicates well with the middle ear, it acts as a reservoir of air to buffer the negative pressure effects of intermittent Eustachian tube dysfunction[22].

Successful treatment of CSOM :

Three major factors can influence the pathologic condition to be treated and the outcome. The first is the continuum of otitis media including silent otitis media, secondly the characteristics and location of pathologic conditions whether localized or generalized and third the pathologic anatomical narrowing or obstructive sites along the middle ear cleft.

Obstruction of the aditus ad antrum contributes to the pathogenesis and accentuates pathologic conditions in otitis media. These factors require the tailoring of surgical treatment of CSOM so that they may be correctly identified and treated sequentially and systematically[16]. Success of surgery for CSOM requires expert preoperative and intra-operative management decisions, meticulous microsurgical technique and individualized post operative care[3]. Ability to predict the ossicular status, plan the surgery and discuss the possible outcome with the patient will lead to better results and patient satisfaction.

Surgery for CSOM :

The American Academy of Ophthalmology and Otolaryngology, Subcommittee on the Conservation of Hearing 1965, defined 'Tympanoplasty' as a procedure to eradicate disease in the middle ear and to reconstruct the hearing mechanism, with or without tympanic membrane grafting. This surgery can be combined with either a Canal wall up or a Canal wall down mastoidectomy to eradicate disease from the mastoid. The same report defined 'Myringoplasty' as an operation in which the reconstructive procedure is limited to the repair of a tympanic membrane perforation. 'Ossiculoplasty' is an operation performed to repair or

reconstruct the ossicular chain. This can be achieved by using the available ossicle remnants themselves, if they are healthy, or artificial biomaterial prostheses like the TORP (total ossicular replacement prosthesis) or PORP (partial ossicular replacement prosthesis) [10,23]. Figures 2 and 3 show an ossiculoplasty being performed.

Summary :

Defects of the ossicular chain may occur in CSOM, with or without cholesteatoma. However discontinuity of the ossicular chain is typically confirmed only during an operation. Knowing before surgery whether the patient has an ossicular discontinuity is important because it allows the surgeon to plan ahead regarding the need for an ossiculoplasty and the type of ossiculoplasty required for that particular patient. Ability to predict the ossicular status, plan the surgery and discuss the possible outcome with the patient will lead to better results and patient satisfaction.

MATERIALS AND METHODS

a) Study Design:

This was a prospective, non randomized, non controlled, descriptive study.

b) Operational Definitions:

For the purposes of this study, 'CSOM' is defined as recurrent or persistent bacterial infection of the ear, lasting 6weeks or more, with conductive hearing loss and a persistent perforation of the tympanic membrane.

'Ossicular discontinuity' was defined as bone erosion as a result of chronic suppurative otitis media in non cholesteatomatous ears, which caused a discontinuity of the ossicular chain as visualized under the operating microscope.

c) Subjects:

This was a prospective, non randomized, non controlled study, including all patients aged five years and above, who presented to the Out Patients Clinic of the Department of Otorhinolaryngology, Christian Medical College, Vellore, with a clinical diagnosis of chronic suppurative otitis media, tubotympanic disease, who were candidates for cortical mastoidectomy with tympanoplasty.

d) Exclusion criteria:

Those patients with atticoantral disease, cholesteatoma or marginal perforations were excluded from the study. Also, those patients who had undergone surgery previously for the same ear were excluded.

e) Informed Consent:

Informed consent was taken from all patients being enrolled in the study. The consent form is attached as Appendix A.

f) Methods:

A pre-surgical evaluation included a clinical examination using an otoscope, Plain X-ray mastoids Law's view and a Pure Tone Audiogram. Each patient was subjected to a detailed pre-operative clinical examination using an otoscope on the day before the surgery. Within a period three months prior to surgery, all patients underwent a pure tone audiogram. The Air-Bone Gap between 0.5 and 4KHz was noted. Figures 4 and 5 are examples of pure tone audiograms of patients with incus necrosis.

Plain X-ray of the mastoids was also done. Mastoid pneumatization was measured using the planimetric technique. A tangent was drawn to the postero-superior aspect of the posterior canal wall and it was made to

intersect the dural plate and the sinus plate. The mastoid cells behind this line were traced out and the area covered was measured in mm^2. Figures 6 and 7 illustrate the lines used to make the planimetric measurements.

Intraoperatively, the otomicroscopic findings were documented. Findings after tympanomeatal flap elevation, including ossicular defects, discontinuity, the presence of granulations, tympanosclerosis and status of the aditus were noted.

Data was collected preoperatively using a detailed proforma, which has been attached as Appendix B. The Data spreadsheet has been attached as Appendix C.

g) Calculation of Sample Size:

Sample size for this descriptive study was calculated using the following formula

$$\mathbf{n = 4pq / d^2}$$

where n = sample size

p = proportion/ percentage/ incidence of the disease

q = compliment of the incidence

d = percentage difference

For this study, consider

p = 10% (as derived based on study by Jeng F et al, 2003 : 17 out of 153 cases)

q = 90 % (i.e.- 100-10)

d = 5%

Thus n = $\frac{4 \times 10 \times 90}{5 \times 5}$ = 144 cases

h) Statistical Analysis:

Categorical variables are presented using frequencies and percentages. Association between categorical variables was assessed using Chi-square test with Yates continuity correction or by Fisher's exact test and multivariate logistic regression analysis. *P-value < 0.05* is considered statistically significant. All statistical analyses were performed using SPSS 11.0 for Windows.

RESULTS

Table 1
Age and sex distribution

Age	Males (%)	Females (%)	Total
5-15yr	3 *(2.0%)*	5 *(3.3%)*	8 *(5.3%)*
16-25yr	32 *(21.3%)*	13 *(8.7%)*	45 *(30.0%)*
26-50yr	44 *(29.3 %)*	37 *(24.7%)*	81 *(54.0%)*
>50yr	9 *(6.0%)*	7 *(4.7%)*	16 *(10.7%)*
Total	88 *(58.7%)*	62 *(41.3%)*	150

The mean age of patients studied was 32, ranging from 9 to 65. The majority of patients studied fell within the 26-50 year group. This was probably because it was patients in this age group who chiefly sought surgery for a discharging ear. Male patients outnumbered females except in the youngest age group, as can be seen in Figure 8.

Table 2

Analysis of the symptoms related to ear discharge

Symptom	N	%
Discharge		
Unilateral	70	46.6
Bilateral	80	53.3
Duration of discharge		
<1yr	9	6
1-5 yrs	33	22
5-10yrs	32	21.3
>10yrs	76	50.6
Amount of discharge		
Copious	94	62.6
Scanty	56	37.3
Blood staining of discharge		
Always	0	0
Occasionally	26	17.3
Never	124	82.6
Most recent episode of ear discharge		
Presently discharging	24	16
<6wks	66	44
6wks to <3m	26	17.3
3m to 1yr	29	19.3
>1yr	5	3.3

The majority of patients studied had history of either unilateral or bilateral ear discharge for more than 10years. Most of them had copious, non blood-stained discharge, which was consistent with a clinical picture of tubotympanic disease. Sixteen percent of patients had active ear discharge at the time of surgery, while 44% had active discharge within 6 weeks of surgery. (Refer Figures 9 and 10)

Table 3
Analysis of the symptoms related to hearing loss

	N	%
Hearing loss		
Unilateral	66	44
Bilateral	84	56
Duration of hearing loss		
<1yr		
1-5 yrs	10	6.7
5-10yrs	35	23.3
>10yrs	31	20.6
	74	49.3
Degree of Hearing loss		
Inability to hear conversational speech	30	20%
Inability to hear even shouted words	8	5.3%

Fifty-six percent of patients in this study had bilateral hearing loss. The majority of them had long standing hearing loss; with 40 patients reporting hearing loss for 20 years or more. Seven patients reported hearing loss of 40 years' duration. Mild hearing loss was more common. Only 20% patients complained of inability to hear conversational speech and 5.3% were unable to hear even shouted speech. The results suggest that patients seek surgical treatment more often when the disease is bilateral, and hearing loss is long standing.

Table 4
Analysis of the Otoscopic findings

	N	%
Status of ear		
Discharging	35	23.3
Moist	98	65.3
Dry	17	11.3
Size of perforation		
1 quadrant	21	14
2 quadrants	51	34
3 quadrants	39	26
4 quadrants	39	26
Site of perforation		
Antero-inferior	139	92.6
Antero-superior	71	47.3
Postero-inferior	127	84.6
Postero-superior	59	39.3
Blood stained discharge	2	1.33
Round Window exposure	103	68.6
TM edge adhesion	24	16
IS joint area exposure	55	36.6
Middle Ear Mucosa		
Normal	66	44
Oedematous	82	54.6
Polypoidal	2	1.3
Granulations	3	2
Handle of Malleus		
Bare	85	56.6
Foreshortened	38	25.3
Tip eroded	16	10.6

On otoscopy, most of the ears studied had moist, oedematous middle ear mucosa. Fifty-two percent of patients had large perforations, involving 3 or 4 quadrants of the pars tensa, 39.3% of which involved the postero-superior quadrant also. Two patients happened to have blood stained discharge, one

had myringitis and the other had traumatized her canal while cleaning it. The incudo-stapedial joint area was visible in 36.6% of cases, through a postero-superior quadrant perforation. The tympanic membrane edges were adherent to middle ear mucosa in 16% of patients. Three patients had granulations on the middle ear mucosa visible through the perforation. One of them had florid granulations in the external auditory canal, on the tympanic membrane and on the middle ear mucosa. The handle of malleus was bare in the majority of cases, but only 10.6% had erosion of the tip.

Table 5

ASHA Classification of degrees of hearing loss, 1974.

Adapted from the Goodman Scale 1965

PTA Average of 500, 1000, 2000 KHz

Hearing loss (dB)	
0-25	Normal
26-40	Mild
41-55	Moderate
56-70	Moderately-severe
71-90	Severe
>91dB	Profound

Table 6

Distribution of degree of hearing loss according to the Goodman Scale

Hearing loss (dB)	N	%
0-25	10	6.7
26-40	57	38
41-55	42	28
56-70	28	18.7
71-90	8	5.3
>91dB	5	3.3
Total	150	100

Sixty-six percent of cases studied fell within the mild and moderate categories. Thirty-eight percent of patients had a mild hearing loss, 28% of patients had a moderate hearing loss, 18.7 % had moderately-severe loss and 5.3% had severe hearing loss. Only 5 patients had a profound loss in our study.

Table 7
Distribution of Air-Bone Gap

Air-Bone Gap (dB)	500Hz	1000Hz	2000Hz	3000Hz	4000Hz
<20 dB	12	16	42	36	33
%	*(8)*	*(10.7)*	*(28)*	*(24)*	*(22)*
20-39dB	67	75	89	96	89
%	*(44.7)*	*(50)*	*(59.3)*	*(64)*	*(59.3)*
40-59dB	65	51	19	18	28
%	*(43.3)*	*(34)*	*(12.7)*	*(12)*	*(18.7)*
≥60dB	6	8	0	0	0
%	*(4)*	*(5.3)*	*(0)*	*(0)*	*(0)*

The majority of patients studied had an air bone gap of 20-39dB. A larger air bone Gap of 40-59dB was more evident in the lower frequencies. Only 14 patients had an air bone gap greater than 60dB. Larger air bone gaps were manifested more frequently at the lower frequencies, as seen in Figure 11.

Table 8

Findings of ossicular necrosis on intra-operative examination under the operating microscope

	N	%
Incus necrosis	*24*	
Long process	10	41.6
Lenticular process	24	100
Body (incl LP & lentic P)	1	4.16
Malleus Necrosis	*24*	
Handle	24	
Head	0	
Stapes necrosis	*5*	
Only Head	0	
Suprastructure	5	

During surgery, the ossicles were examined under the operating microscope. The lenticular process of the incus was found to be necrosed in 24 ears. Both the lenticular process and the long process were eroded in 10 cases, and the body was also eroded in one patient. The handle of malleus was eroded in 24 cases and the stapes suprastructure in 5 cases. These results support the theory that the region of the incudo-stapedial joint, the lenticular process in particular, is the most vulnerable to erosion in chronic suppurative otitis media.

Table 9

Correlation between presence of incudal necrosis and degree of hearing loss

Hearing loss (dB)	Necrosis of lenticular process	Necrosis of long process	Necrosis of body
0-25	1	0	0
26-40	1	0	0
41-55	9	4	1
56-70	7	3	0
71-90	5	3	0
>91dB	1	0	0
Total	24	10	1

Necrosis of the lenticular process of the incus was the most frequent finding, the next being erosion of the long process of the incus, as illustrated in Figure 12. Hearing loss due to incus necrosis most commonly manifested as a loss of 41-55dB or moderate hearing loss.

Table 10
Correlation between presence of incudal necrosis and Air-Bone Gap

Air-Bone Gap (dB) at 500Hz	Necrosis of lenticular process	Necrosis of long process	Necrosis of body
<20	1	0	0
20-39	5	2	0
40-59	17	7	1
>60	1	1	0
Total	24	10	1

An air bone gap of more than 40dB correlated well with the presence of incus necrosis, as illustrated in Figure 13. Most cases of necrosis of the lenticular and long process of the incus were found to have an Air Bone Gap of 40-59dB.

Table 11
Extent of Mastoid pneumatization

X-ray Mastoids	N	Valid %
< 500mm^2	77	54.6
500-1000mm^2	52	36.9
>1000mm^2	12	8.5
Missing	9	

The X-rays of nine patients were not available for evaluation. Of the remainder, fifty-five percent of patients had mastoids of a size smaller than 500mm^2. There was a definite trend towards a smaller mastoid size, in keeping with the theories of diseased mastoids being poorly pneumatized.

Table 12

Middle ear and mastoid findings on intra-operative examination under the operating microscope

	N	%
IS joint discontinuity	23	15.3
Mastoid granulations	34	22.6
Tympanosclerosis	41	27.3
Status of Aditus		
Patent	71	47.3
Blocked	79	52.6

Incudostapedial joint discontinuity was seen in 23 ears, viz., all the cases in which the incus or the stapes was found to be necrosed. In one case, the lenticular process was necrosed, but the long process was found to be in contact with the head of the stapes, connected by a fibrous band, hence it was left undisturbed. Granulations and tympanosclerosis were found in the mastoid and middle ear in 22.6 % and 27.3% of patients respectively. The aditus was blocked in 52.6 percent of ears operated by us. Discontinuity of the incudo-stapedial joint was found to be more frequent in cases with a blocked aditus or the presence of granulation tissue.

COMPARISION OF THE 2 GROUPS : Categorized bivariate analysis

Table 13
Analysis of the symptoms related to ear discharge in normals versus incus necrosis

	Normal ossicles	**Incus Necrosis**	***p value***
Discharge			
Unilateral	62	8	0.114
Bilateral	64	16	
Duration of discharge			
<1yr	8	1	
1-5 yrs	28	5	0.849
5-10yrs	28	4	
>10yrs	62	14	
Amount of discharge			
Copious	77	17	0.253
Scanty	49	7	
Blood staining of discharge			
Always	0	0	
Occasionally	23	3	0.695
Never	101	21	
Most recent episode of ear discharge			
Presently discharging	18	6	
<6wks	56	10	
>6wks to <3m	21	5	0.505
3m to 1yr	26	3	
>1yr	5	0	

Patients who had necrosis of the ossicles were more likely to have long standing (more than 10 years) bilateral ear disease, with episodes of copious ear discharge. Most of them had discharge within 6 weeks of

surgery. Three patients gave history of occasional blood staining of the ear discharge. They also gave history of frequent cleaning of the ears. In our study, however, there was no statistically significant difference in frequency of symptoms of ear discharge in patients with normal ossicles and those with ossicular necrosis.

Table 14

Analysis of the symptoms related to hearing loss, normals versus incus necrosis

	Normal ossicles	**Incus Necrosis**	***p value***
Hearing loss			
Unilateral	58	7	0.085
Bilateral	66	17	
Duration of hearing loss			
<1yr			
1-5 yrs	8	2	
5-10yrs	31	4	0.716
>10yrs	27	4	
	60	14	
Degree of Hearing loss			
Inability to hear conversational speech	22	8	0.071
Inability to hear even shouted words	4	4	0.023

Patients with incudal necrosis were more likely to have bilateral, long standing hearing loss, but the difference was not statistically significant in

our study. There was, however, a significant difference in patients who reported that they were unable to hear even words shouted in their ears.

Table 15
Analysis of the Otoscopic findings in normals versus incus necrosis

	Normal ossicles	**Incus Necrosis**	***p value***
Status of ear			
Discharging	24	11	
Moist	86	12	0.014
Dry	16	1	
Size of perforation			
1 quadrant	17	4	
2 quadrants	46	5	0.076
3 quadrants	35	4	
4 quadrants	28	11	
Site of perforation			
Antero-inferior	118	21	0.248
Antero-superior	53	16	0.032
Postero-inferior	107	20	0.524
Postero-superior	46	13	0.083
Blood stained discharge	2	0	0.705
Round Window exposure	87	16	0.496
TM edge adhesion	17	7	0.059
IS joint area exposure	42	13	0.045
Middle Ear Mucosa			
Normal	59	7	
Oedematous	65	17	0.048
Polypoidal	2	0	
Granulations	0	3	0.004
Handle of Malleus			
Bare	73	12	0.309
Foreshortened	28	10	0.044
Tip eroded	11	5	0.087

Patients who had incus necrosis were more likely to have actively discharging or moist ears with oedematous middle ear mucosa and exposure of the incudo-stapedial joint area through the perforation. These differences were found to be statistically significant. There was also a significant association between a foreshortened handle of malleus and incus necrosis. The presence of middle ear granulations was significantly associated with incus necrosis. The adherence of the tympanic membrane edges to the middle ear mucosa was also strongly associated with the presence of incus necrosis.

Table 16

Distribution of degree of hearing loss according to the Goodman Scale

(Pure tone average of 500, 1000, 2000Hz)

Hearing loss (dB)		**Normal ossicles**	**Incus Necrosis**	***p value***
0-25	Normal	09	01	0.000
26-40	Mild	56	01	
41-55	Moderate	33	9	
56-70	Moderately-severe	21	07	
71-90	Severe	03	05	
>91dB	Profound	04	01	
Total		126	24	

Patients with necrosis of the incus were found to have moderate to moderately-severe hearing loss. Five patients also had severe hearing loss. This difference was found to be statistically very significant.

Table 17
Distribution of Air-Bone Gap

Air-Bone Gap (dB)	500Hz	1000Hz	2000Hz	3000Hz	4000Hz
<20 dB					
Normal	11	15	35	30	30
Incus Necrosis	01	01	07	16	03
20-39dB					
Normal	62	69	77	84	75
Incus Necrosis	05	06	12	12	14
40-59dB					
Normal	48	37	14	12	21
Incus Necrosis	17	14	05	06	07
≥60dB					
Normal	05	05	0	0	0
Incus Necrosis	01	03	0	0	0
p value	0.027	0.006	0.380	0.086	0.239

An air-bone gap of 40-59dB especially in the lower frequencies of 500 and 1000Hz was significantly associated with necrosis of the incus and incudo-stapedial joint discontinuity.

Table 18
Correlation of large Air Bone Gap with presence of incudal necrosis

	Normal ossicles	**Incus Necrosis**	***p value***
≤ 40dB	108	13	0.001
> 40dB	18	11	

Eleven patients with incus necrosis had an air- bone gap greater than 40dB. There was a statistically significant correlation between an air- bone Gap greater than 40dB and presence of incudal necrosis.

Table 19
Correlation of Sensorineural element of hearing loss with incus necrosis

Average bone conduction across frequencies	**Normal ossicles**	**Incus Necrosis**	***p value***
<20dB	101	14	0.024
≥20dB	25	10	

Out of the 24 patients who had incus necrosis, 10 patients had an average bone conduction across the frequencies 500 to 4000Hz worse than 20dB. This correlation with a mixed hearing loss was statistically significant.

Table 20
Extent of Mastoid pneumatization in normals versus incus necrosis

X-ray Mastoids	Normal ossicles	Incus Necrosis	*p value*
< 500mm^2	61	16	0.152
500-1000mm^2	45	7	
>1000mm^2	12	0	

Sixteen out of the 23 patients whose X-ray were available for study had an area less than 500mm^2. The remaining 7 had an area of less than 1000mm^2. Though there was a definite trend towards a smaller mastoid area, the difference was not statistically significant. No patient in our study with a large, well pneumatized mastoid with an area more than 1000mm^2 had a necrosed ossicle.

Table 21

Middle ear and mastoid findings on intra-operative examination under the operating microscope

	Normal ossicles	**Incus Necrosis**	***p value***
IS joint discontinuity	0	23	0.000
Mastoid granulations	23	11	0.005
Presence of tympanosclerosis	37	4	0.151
Status of Aditus Patent Blocked	 67 59	 4 20	 0.001

Granulations in the middle ear and mastoid were significantly associated with necrosis of the ossicles. Blockage of the aditus was also significantly associated with ossicular necrosis. Tympanosclerosis in the mastoid did not seem to affect the integrity of ossicles.

Table 22

Comparison of site of granulations as seen on intra-operative examination in normals versus incus necrosis

	IS Joint	Attic	Aditus	Mastoid	Others
Normal	3	0	6	16	4
Necrosis	2	2	7	8	1
p value	0.181	0.025	0.001	0.018	0.587

Of the 34 patients who were intra-operatively found to have granulations, 24 patients had granulations in the mastoid. Incus necrosis was found to correlate significantly with presence of granulations in the attic, the antrum or the aditus.

Table 23

Comparison of site of tympanosclerosis as seen on intra-operative examination in normals versus incus necrosis

	TM	Promontory	Ossicles	Mastoid	Others(ME)
Normal	27	4	4	4	8
Necrosis	2	0	1	2	2
p value	0.109	0.494	0.587	0.246	0.497

Forty-one patients were found to have tympanosclerosis on examination under the operating microscope. Twenty-nine had tympanosclerotic patches on the tympanic membrane, which was the most common site. Of the five who had tympanosclerosis around the incudo-stapedial joint, only one had ossicular necrosis.

Table 24
Factors affecting Malleus Necrosis :

	Normal Malleus	**Malleus Necrosis**	***p value***
Size of perforation			
1 quadrant	20	1	
2 quadrants	43	8	
3 quadrants	33	6	0.328
4 quadrants	30	9	
Status of ear			
Discharging	28	7	
Moist	84	14	0.717
Dry	14	3	
Presence of Incus necrosis	17	7	0.059
Status of malleus			
Tip bare	70	15	0.345
Foreshortened	33	5	0.394
Tip eroded	10	6	0.024
MEM			
Normal	57	9	
Oedematous	67	14	0.516
Polypoidal	1	1	

Necrosis of the malleus was significantly associated with presence of incus necrosis, implying that the same factors seemed to be causing both. The size of the perforation, presence of ear discharge, status of the middle ear mucosa did not seem to contribute to the presence of malleus erosion.

MULTIVARIATE LOGISTIC REGRESSION ANALYSIS

Table 25
Correlation of pre-operative factors with Incus necrosis

Risk Factor	Significance	Odds Ratio	95% Confidence Intervals	
			Lower	Upper
Postero-superior perforation	0.449	0.433	0.050	3.782
IS Joint exposure	0.165	4.532	0.537	38.229
Foreshortened HOM	0.050	2.992	0.984	9.098
Average ABG >40dB	0.289	1.933	0.571	6.539
Average AC with mod to mod-severe HL	0.029	1.713	1.055	2.781
Middle ear granulations	0.048	2.986	1.012	8.809
HOM tip erosion	0.758	1.280	0.266	6.168
Active ear discharge	0.044	0.371	0.141	0.975

A moderate to moderately-severe conductive hearing loss, an actively discharging ear, the presence of granulations in the middle ear and a foreshortened handle of malleus, turned out to be highly significant risk factors on multivariate analysis. The presence of granulations in the middle ear increased the risk of having ossicular necrosis by 3 times. Similarly, a patient with foreshortening of the handle of malleus had a 3 times higher risk of having incus necrosis. An actively discharging ear implied a 0.4 times

risk and a moderate to moderately-severe hearing loss gave a 1.7 times increased chance of having a co-existing ossicular necrosis.

Table 26
Correlation of intra-operative findings with Incus necrosis

Risk Factor	Significance	Odds Ratio	95% Confidence Intervals	
			Lower	Upper
Mastoid granulations	0.036	2.843	1.068	7.569
Aditus block	0.012	4.525	1.399	14.639
Malleus necrosis	0.599	1.351	0.440	4.149

There was a highly significant correlation between the presence of a blocked aditus and incus necrosis. Presence of granulations in the middle ear and the mastoid also contributed significantly to incudal necrosis. Necrosis of the handle of malleus was not always associated with incus necrosis.

DISCUSSION

Chronic suppurative otitis media (CSOM) refers to chronic infection (more than 6 weeks) of the middle ear cleft in the presence of a persistent tympanic membrane perforation. Defects of the ossicular chain may occur in all chronic middle ear diseases, with or without cholesteatoma. Discontinuity of the ossicular chain is confirmed only during an operation. The ability to predict the presence of ossicular discontinuity in a patient using certain pre-operative factors, would be of benefit in allowing the surgeon to plan ahead with regard to the need for an ossiculoplasty and also to give the patient a realistic explanation of the expected outcome.

Prevalence of incudal necrosis

Ossicular necrosis, according to all the current studies on the subject, predominantly affects the long process of incus. This has been the finding in patients with and without cholesteatomatous ear disease. Mirko Tos[11], studying 674 ears of CSOM without cholesteatoma found that 56 (8.31%) cases had necrosis of the long process of the incus. The body of the incus was necrosed in only 2 cases. Six cases had partial erosion of the stapes suprastructure and 15 had total loss of the suprastructure. The incus was the

most common ossicle to undergo necrosis, irrespective of the type of disease. Similarly, Jeng et al[13] found that of the 153 ears without cholesteatoma studied, 17 (11.1%) had ossicular necrosis, all of which occurred in the region of the incudo-stapedial joint. In the present study, we found that 24 (16%) out of the 150 studied had incus necrosis. The prevalence of incus necrosis in our study is a little higher than in the other studies of patients with non-cholesteatomatous ear disease. The reason for this could be long-standing duration of ear disease in most of our patients, leading to progressive bone erosion with repeated episodes of infection.

Significant risk factors for incudal necrosis by univariate analysis

1. Ear discharge :

Patients with chronic suppurative otitis media present to the surgeon at various stages in the disease. More often they present to the surgeon when there is active ear infection producing ear discharge. The presence of ear discharge is bothersome not merely because of the need to frequently clean the ear, but also because of the reduction in hearing levels produced by ear discharge. Some patients with moist ears have active infection even though there is very little discharge in the canal. In our study we found that the presence of active discharge or moistness strongly correlated with the

presence of incudal necrosis (p = 0.014). Having active ear discharge increased the risk of having ossicular necrosis by 0.4 times. Our findings are in concordance with those of Jeng et al[13] who found that the majority of their patients with ossicular discontinuity had either active ear discharge or moist ears.

The importance of ear discharge as a risk factor is that it signifies an on-going disease process. It may be due to an underlying focus of infection in the nasopharynx, Eustachian tube dysfunction, allergic rhinitis, chronic sinusitis or non- response to treatment. Whatever the cause, the presence of discharge is a poor prognostic factor for ossicular integrity, particularly that of the incus.

2. The handle of the malleus :

The status of the handle of malleus (whether foreshortened or not) and its association with incus necrosis has not been extensively studied. We decided to include this factor as the malleus handle is the only constantly visible part of the ossicular chain on otoscopy. We found 10 out of 24 cases (41.6%) with incus necrosis to have a foreshortened handle of malleus, and this was statistically significant (p = 0.044). A foreshortened handle of malleus increased the risk of incus necrosis 3 times (p = 0.050). We also

found erosion of the tip of the handle of malleus in 5 cases, but this was not statistically significant (p = 0.087). Foreshortening of the handle of malleus may be a result of adhesions and may be a hallmark of chronic ear disease and the processes that favour of bone resorption.

3. Middle ear granulations:

The presence of granulation tissue on the tympanic membrane was seen in 16 out of 17 patients (94.1%) with ossicular discontinuity in the study by Jeng et al[13] and was found to be statistically significant. In our study, 3 out of 24 ears (12.5%) with incus necrosis had middle ear granulations, which were visible through the perforation, on otoscopy. None of the patients without incus necrosis has middle ear granulations. The association between middle ear granulations and incus necrosis was found to be statistically significant (p = 0.004). The presence of granulations increased the risk of incus necrosis 3 times as compared to those ears without granulations (p = 0.048). Granulation tissue is a sign of overt middle ear infection with possible osteitis. It stands to reason, therefore, that granulations would predispose to osteitis of the incus and subsequent necrosis.

4. Incudo-stapedial joint area exposure :

In our study, 54% of patients with incus necrosis had exposure of the incudo-stapedial joint area through the perforation. This factor was found to have a statistically significant correlation with the presence of incus necrosis ($p = 0.045$). This has not been specifically studied in either of the previous studies, though Jeng et al[13] mention that subtotal perforations were associated with incus necrosis and Tos[11] found that a posterior or a total perforation was associated with incus necrosis.

Middle ear mucosa exposed to the outside atmosphere, the effects of water entry into the middle ear and the trauma of chronic vigorous self-cleaning is more likely to produce active ear discharge and granulations. Exposure of the incudo-stapedial joint, the smallest joint in the human body, may lead to further compromise of the already delicate blood supply of this region and promote ossicular necrosis. Exposure of this area may also predispose to microscopic migration of squamous epithelium into the middle ear, which would further promote necrosis of the incus.

5. Polypoid middle ear mucosa :

Oedematous middle ear mucosa is more likely to cause aditus block, decrease the ventilation of the mastoid, and to cause active discharge from

the ear, all factors which would lead to incus necrosis. Oedematous mucosa encasing the incus could compromise its blood supply or cause persistence of infection around the vulnerable long process of incus. In our study 70.8% of patients with incus necrosis had oedematous middle ear mucosa on otoscopy. This was found to be statistically significant (p = 0.048). In the study by Jeng et al[13], 76% of patients with ossicular discontinuity had polypoidal middle ear mucosa. Here, too, this risk factor was found to be a significant one.

6. Adhesion of perforation edges to middle ear mucosa:

Adhesion of the edges of a perforation to the middle ear mucosa is an occasional finding in many patients with non-cholesteatomatous CSOM. In our study, 7 out of 24 cases (29.2%) had adhesion of the perforation edges with middle ear mucosa, and this was found to have a statistically significant association with incus necrosis (p = 0.059). Jeng et al[13] found adhesion of the perforation edges to the promontory in 12 out of the 17 cases (70.5%) of ossicular discontinuity. Their finding was statistically highly significant (p=0.004).

Adhesions along a perforation edge, implies walling off of a portion of the middle ear, blocking further drainage. This would certainly lead to

persistence of infection around the incus region, particularly if such adherence is posteriorly situated. There may also be associated blockage of the aditus, which could further predispose to incus necrosis. Adhesion of the edges of the perforation to the middle ear mucosa may also be a precursor of migration of squamous epithelium into the middle ear, which in turn, would lead to ossicular necrosis.

7. Degree of hearing loss by symptoms:

Fifty-six percent of patients in our study had bilateral hearing loss. The majority of them had long standing hearing loss; with 40 patients reporting hearing loss for 20 years or more. A statistically significant correlation with ossicular discontinuity was found in patients who reported that they were unable to hear even words shouted in their ears ($p = 0.023$).

8. Degree of hearing loss by pure tone audiometry

In the study by Jeng et al[13], pure tone average hearing loss was found to correlate significantly with ossicular discontinuity. In our study, 7 out of 24 patients with necrosis of the incus were found to have moderate to moderately-severe hearing loss. Five patients also had severe hearing loss. This difference was found to be statistically very significant. Ten out of the

24 patients with incus necrosis in our study had an average bone conduction across the speech frequencies of more than 20dB, and this was statistically very significant. A patient with a greater hearing loss of the range of 40-70dB, as seen on average air conduction levels across the speech frequencies, was at a 1.7 times greater risk of having incus necrosis.
($p = 0.029$)

9. Air-Bone Gap:

Carrillo et al[14] in 2007 concluded that a 20dB or less ABG at 500Hz predicts an absence of ossicular discontinuity and an ABG of 30dB or greater at 2000Hz predicts the presence of ossicular discontinuity. Jeng's[13] study also found a significant correlation between a larger ABG and ossicular discontinuity.

In our study, an air-bone Gap of greater than 40dB, especially in the lower frequencies of 500 and 1000Hz was highly significant in association with necrosis of the incus and incudo-stapedial joint discontinuity($p= 0.001$).

10. Aditus Block

The presence of a blocked aditus is an extremely significant factor contributing to incus necrosis ($p < 0.001$). Aditus block, either with granulations or with thickened mucosa, increased the risk of incus necrosis 4.5 times ($p = 0.012$).

An aditus block would lead to inadequate ventilation and drainage of the mastoid, leading to negative pressure, exudation of secretions, stasis, infection, granulations and ossicular necrosis.

The studies by Tos[11] and Jeng et al[13], have not described the correlation of status of the aditus with incus necrosis. We feel this is an important finding, as preparations for ossiculoplasty are often made at this point, particularly when otoscopic appearances do not suggest the presence of ossicular discontinuity.

11. Granulations in the mastoid, as seen intra-operatively:

Granulations were present in 46% of ears with ossicular necrosis in our study. This association was found to be highly significant ($p = 0.005$). A total number of 34 patients were intra-operatively found to have granulations, 24 patients had granulations in the mastoid, of which 8 had

necrosis of the incus. Incus necrosis was found to correlate very significantly with presence of granulations in the attic, the antrum or the aditus. The presence of mastoid granulations increased the risk of incus necrosis 2.8 times as compared to those ears without granulations ($p = 0.036$)

Jeng et al[13] also found granulations in 16 out of 17 ears (94.1%) with ossicular discontinuity in their study. Schachern[16] has reported that granulation tissue may be trapped in non-draining spaces of the middle ear cleft. Chole[17] observed that granulation tissue can lead to bone erosion. Further, they noted that it is the presence of infection and inflammation rather than just the presence of cholesteatoma which leads to bone resorption.

Risk factors which were not significantly associated with incudal necrosis

1. Ear discharge and its duration:

The studies by Tos[11] and Jeng et al[13] do not take into account the duration of ear disease, as they felt that patients are poor historians. Patients usually knew that they had long standing ear disease, but were not able to remember more specific details about the onset of symptoms in their childhood.

In our study, the duration of ear discharge ranged from 3 months to 40 years, with a mean of 13.3 years. Forty patients reported having the disease for 20 years or more. Seven elderly patients reported intermittent ear discharge for 40 years. Most patients did not seem to be worried about their discharging ears, or to seek early treatment unless the disease was severe. In a large number of cases, there were financial constraints to seeking early treatment. However, there was no statistically significant difference in duration of symptoms of ear discharge in patients with normal ossicles and those with ossicular necrosis.

2. Laterality of disease :

Fifty-three percent of patients studied by us had history of bilateral ear discharge. Patients who had necrosis of the ossicles were more likely to have long standing (more than 10 years) bilateral ear disease, with episodes of copious ear discharge. Bilateral long standing ear disease may point towards a common aetiological factor like adenoid hypertrophy in childhood, allergic rhinitis or chronic sinusitis. However, there was no statistically significant correlation between bilateral ear disease and incus necrosis in our study. In the study by Jeng et al[13], only 4 out of the 17 with ossicular discontinuity had bilateral disease.

3. Size and Site of perforation :

Fifty-two percent of patients in our study had large perforations, involving 3 or 4 quadrants of the pars tensa. Eleven out of 24 patients with ossicular necrosis had a perforation involving all four quadrants of the drum. This has not been found significant in either of the studies by Tos[11] and Jeng et al[13]. The size of the perforation is determined to some extent by the severity of the initial infection. However, it appears that the extent of damage to the tympanic membrane does not correlate with damage to the ossicles.

4. Exposure of the round window niche :

Exposure of the round window through the perforation was found in 16 out of the 24 cases of incus necrosis in our study, however this association was not found to be statistically significant. Exposure of the round window would contribute towards greater hearing loss in an ear with a perforation.

5. Radiology

Jeng et al[13] found that 12 out of the 17 cases in their study with ossicular discontinuity had poorly pneumatized mastoids, as judged by their appearance on mastoid X-rays or CT scans. A less pneumatized mastoid

would signify a long standing active ear disease and thus point towards chances of having ossicular necrosis.

In our study, 66% of cases with incus necrosis had mastoid area of less than 500 mm^2 as measured planimetrically on X-ray Law's view. Seven cases had an area between 500 and 1000 mm^2. There was a definite trend towards a smaller, less pneumatized mastoid in cases with incus necrosis, but this was not statistically significant in our study ($p = 0.152$).

Significant risk factors for incudal necrosis by multivariate analysis

While univariate analysis of all the various risk factors studied indicated that at least 11 risk factors for incudal necrosis existed, multivariate analysis showed that the only 6 variables that each remained independently and significantly related to the presence of incudal necrosis were an actively discharging ear, foreshortening of the handle of the malleus, middle ear granulations, a pure tone air conduction average hearing loss of the moderate to moderately-severe range, intra-operative findings of mastoid granulations and a blocked aditus. When compared to the results of Jeng et al[13] who also analyzed some of these risk factors using a logistic regression model, similar findings were noted. Additionally, the present study has shown that separation of preoperative and intraoperative risk

factors which may be predictive of incudal necrosis, enables the surgeon performing mastoidectomy with tympanoplasty for patients with chronic suppurative otitis media to be prepared in advance for possible simultaneous ossiculoplasty.

CONCLUSION

In conclusion, four pre-operative factors were found to be reliable, independent predictors of incus necrosis. These were, the presence of an actively discharging ear, foreshortening of the handle of the malleus, middle ear granulations and a pure tone air conduction average hearing loss of the moderate to moderately-severe range (40-70dB). Intra-operative findings of mastoid granulations and a blocked aditus were also highly significant, independent factors increasing the risk of incus necrosis.

These four pre-operative factors and two intra-operative factors could be considered reliable indicators of incus necrosis in chronic suppurative otitis media without cholesteatoma.

BIBLIOGRAPHY

1. Coates H. Chronic suppurative otitis media without cholesteatoma. In: *Advanced Therapy of Otitis Media,* Alper CM, Bluestone CD, Hamilton, Ontario. BC Decker, 2004: 299-305

2. Mills RP. Management of chronic suppurative otitis media. In: Kerr AG, *Scott-Brown's Otolaryngology,* 6th Edition, London. Butterworth-Heinemann,1997: 3: 3/10/1-11

3. Veldman JE, Braunis WW. Revision surgery for chronic otitis media: A learning experience. *Annals of Otorhinolaryngology* 1998; 107: 486-491

4. Vartianen E, Vartianen J. Hearing results of surgery for chronic otitis media without cholesteatoma. *ENT Journal* 1995; 74: 165-169

5. Vedantam Rupa. When to suspect complicated suppurative otitis media. In : *Advanced Therapy of Otitis Media,* Alper CM, Bluestone CD, Hamilton, Ontario. BC Decker, 2004: 312-317

6. Youngs R. Chronic suppurative otitis media – Mucosal disease. In : Ludman H, Wright T, *Diseases of the Ear*, 6th Edition, Arnold, Oxford University Press, New York, 1997; 27: 374-385

7. Youngs R. Chronic suppurative otitis media – cholesteatoma. In : Ludman H, Wright T, *Diseases of the Ear*, 6th Edition, Arnold, Oxford University Press, New York, 1997; 386 - 397

8. Raz Y. Conductive hearing loss. In: *Advanced Therapy of Otitis Media*, Alper CM, Bluestone CD, Hamilton, Ontario. BC Decker, 2004: 419-424

9. Merchant SN, Rosowski JJ. Auditory physiology. In: Glasscock ME, Gulya AJ, *Surgery of the ear*, 5th Edition, BC Decker, 2002; 3 : 59-82

10. Sismanis A. Tympanoplasty. In: Glasscock ME, Gulya AJ, *Surgery of the ear*, 5th Edition, BC Decker, 2002; 24: 463-485

11. Tos M. Pathology of the ossicular chain in various chronic middle ear diseases. *Journal of Laryngology and Otology* 1979; 93 : 769-780

12. Walshe P, McConn Walsh R, Brennan P, Walsh M. The role of computed tomography in the preoperative assessment of chronic suppurative otitis media. *Clinical Otolaryngology Allied Sciences* 2002; 27(2): 95-97

13. Jeng F, Tsai M et al. Relationship of preoperative findings and ossicular discontinuity in chronic otitis media. *Otology & Neurotology* 2003;24: 29-32

14. Carrillo RJC, Yang NW, Generoso TA. Probabilities of ossicular discontinuity in chronic suppurative otitis media using pure tone audiometry. *Otology & Neurotology* 2007; 28:1034-1037

15. Thomsen J, Bretlau P, Jorgensen MB. Bone resorption in chronic otitis media: the role of cholesteatoma: a must or an adjunct? *Clinical otolaryngology* 1981; 6:179-186

16. Schachern P, Paparella MM, Sano S, Lamey S, Guo Y. A histopathological study of the relationship between otitis media and mastoiditis. *Laryngoscope* 1991; 101: 1050-1055

17. Chole RA. Chronic otitis media, mastoiditis, petrositis. In : Cummings CW, *Otolaryngology Head & Neck Surgery*, 4th Edition. St. Louis : CV Mosby, 2004 : 4 : 2988-3012

18. Davis A. Epidemiology of hearing impairment. In : Ludman H, Wright T, *Diseases of the Ear*, 6th Edition, Arnold, Oxford University Press, New York, 1997; 7 : 130-137

19. Valvassori GE. Imaging of the temporal bone. In: Glasscock ME, Gulya AJ, *Surgery of the ear*, 5th Edition, BC Decker, 2002; 11: 227-259

20. Flisberg K, Zsigmond M. The size of the mastoid air cell system. *Acta Otolaryngologica,* 1965: 60: 23-29

21. Todd NW, Pitts RB, Braun IF, Heindel H. Mastoid size determined with lateral radiographs and computerized tomography. *Acta Otolaryngologica (Stockh)* 1987;103:226-231

22. McGrew MB, Jackson CG, Glasscock ME. Impact of mastoidectomy on simple tympanic membrane repair. *Laryngoscope* 2004:114: 506-511

23. Frootko NJ. Reconstruction of the middle ear. In: Kerr AG, *Scott-Brown's Otolaryngology,* 6th Edition, London. Butterworth-Heinemann, 1997: 3: 3/11/1- 30

Appendix A

Informed Consent

Patient's Name :

Hospital Number:

The procedures and investigations required for the study **"Preoperative predictors of ossicular necrosis in chronic suppurative otitis media, without cholesteatoma"** have been fully explained to me in a language I understand. I have no objection to enroll myself in this study, or for the use or publication of the data collected for any scientific or academic purpose.

Signature of the patient / relative

Relationship to the patient

Date:

Address

Witness

Appendix B

PROFORMA

Name :
Age : **Sex :** **Hosp No:**
Address:

Tel :
email :

History :

Ear discharge :
Right / Left ear/ Both
Duration :(weeks)
Amount : copious / scanty Last discharge :
Blood stained : always / occasionally / never

Hearing loss :
Right / left / both
Duration : (weeks)
Degree : Ability to hear conversational speech Yes / No
Ability to hear words shouted in ear Yes / No

On Examination (Otomicroscopy) :

Actively discharging ear / moist ear / dry ear
Blood stained ear discharge: Yes / no

Size of Perforation : One / two / three / all four quadrants

Site of Perforation : Antero-inferior/ Antero-superior/ Postero-inferior/ Postero-superior

Round Window exposed : Yes / No
Presence of Granulations : yes / no Site :
Adherence of the tympanic membrane to promontory : yes / no
Handle of Malleus : bare / foreshortened
Incudo-stapedial joint exposed : Yes/ No
Middle ear mucosa : Polypoidal / normal / oedematous

Pure tone Audiogram : (at 500, 1000, 2000, 3000, 4000Hz) : Right : Left :
Bone Conduction :
Air Conduction :
Air bone gap :

X-ray mastoids :
Right : cellular / sclerosed
Left : cellular / sclerosed

Ossicular Status (intra op assessment) :

Normal / discontinuity present / fixity

Necrosis of Incus : Long process / body
Necrosis of Malleus : Handle / Head
Necrosis of Stapes : Head / Suprastructure
Incudo-stapedial joint discontinuity : yes /no

Granulations : On the promontory / Round window niche / Incudostapedial joint /Stapes footplate/ Attic / Aditus / Mastoid / others

Tympanosclerosis : Present/ Absent
Site : TM / Promontory / Ossicles / Mastoid / others

Aditus : patent / blocked

<u>If ossicular discontinuity present :</u>
Reconstruction done : Yes / No
Autograft / prosthesis used

Type of reconstruction :

Post op Hearing status : (3 months post op)

Pure tone Audiogram : at 500, 1000, 2000Hz
Right :
Left :

Air bone gap :

Appendix C

DATA SHEET

Figures

Figure 1: Incus and Malleus in various stages of necrosis (a) Incus with part of body and lenticular process eroded (b&c) Incus with erosion of long and lenticular processes (d) Malleus (e) Malleus with eroded handle (f) Malleus with eroded tip (g) Head of malleus

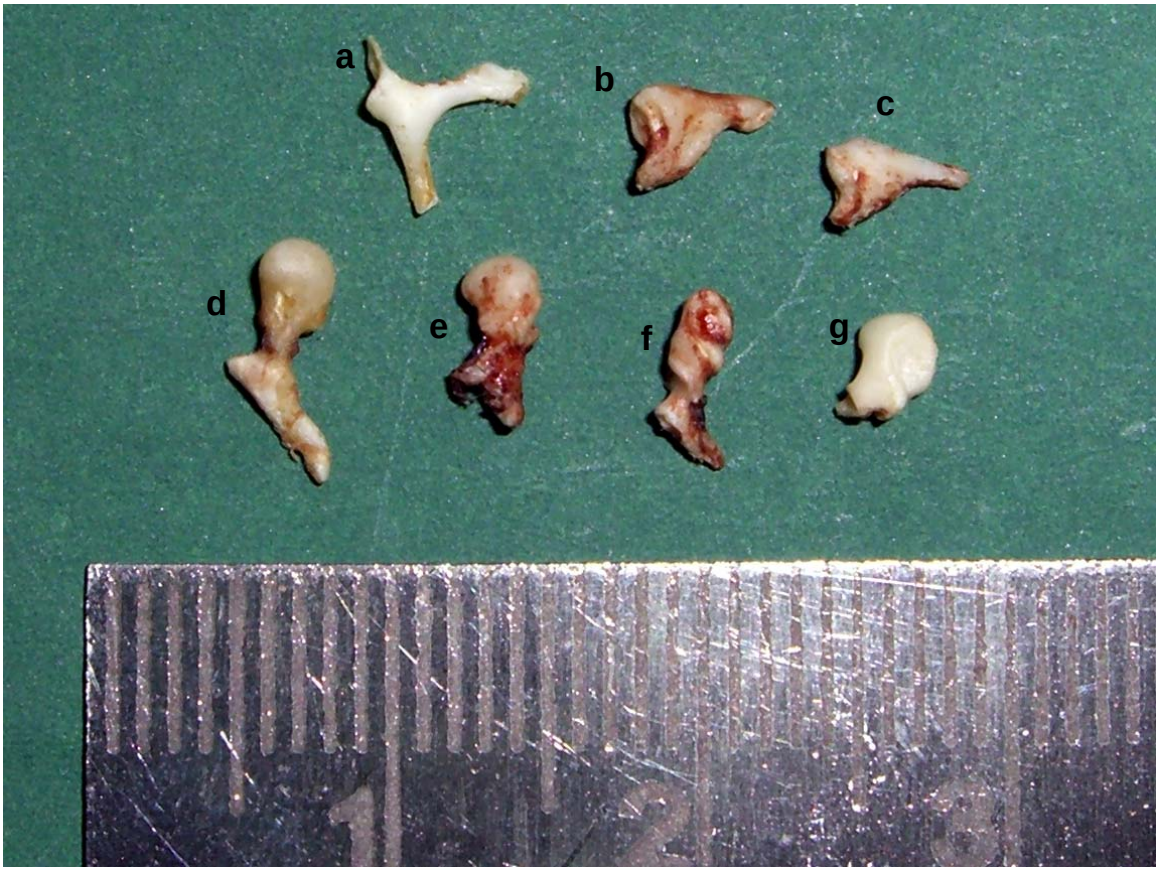


Figure 2: Intra-operative photograph of an incus being refashioned

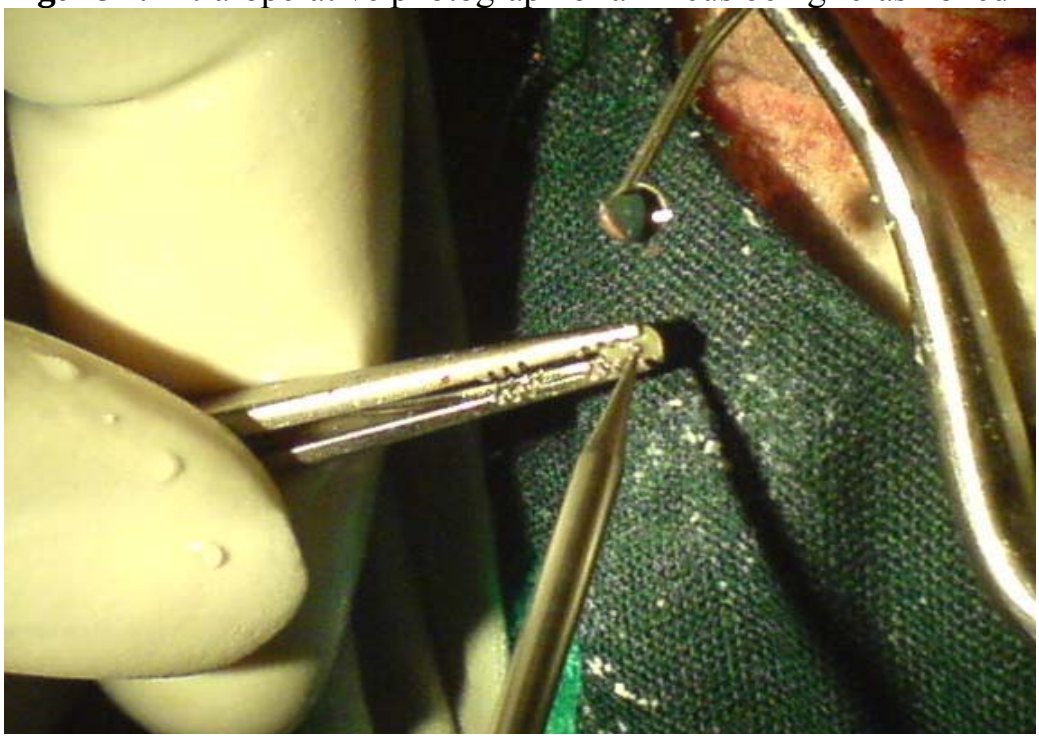


Figure 3: Refashioned incus placed on the stapes head

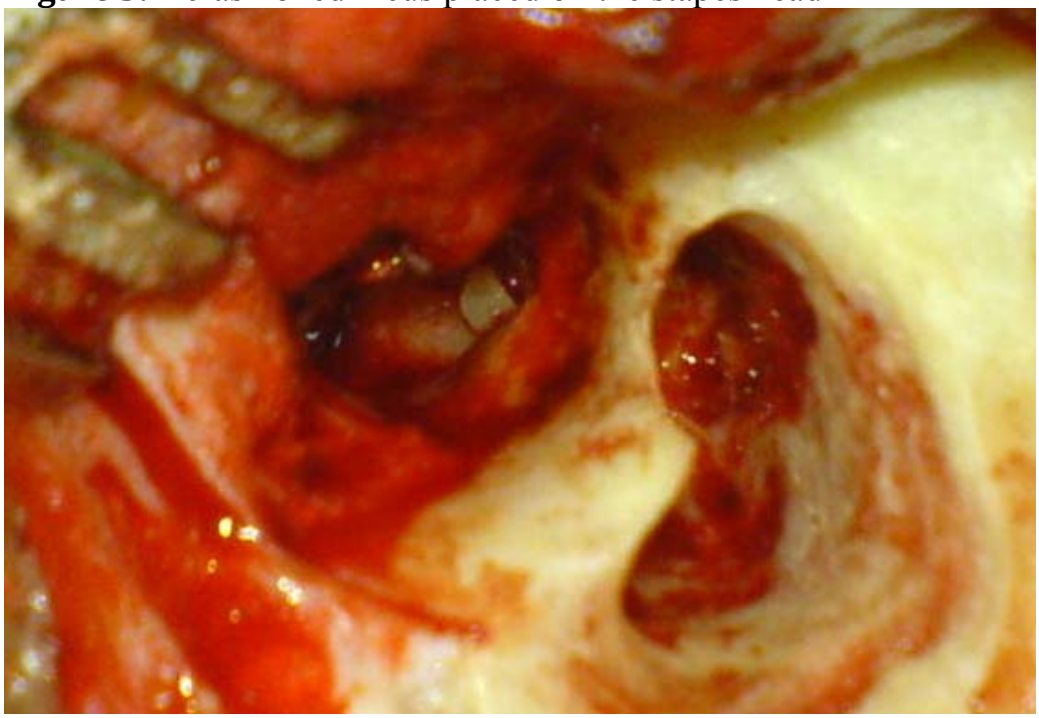


Figure 4: Audiogram of a patient with 40-50dB conductive hearing loss on the left, typical of ossicular necrosis

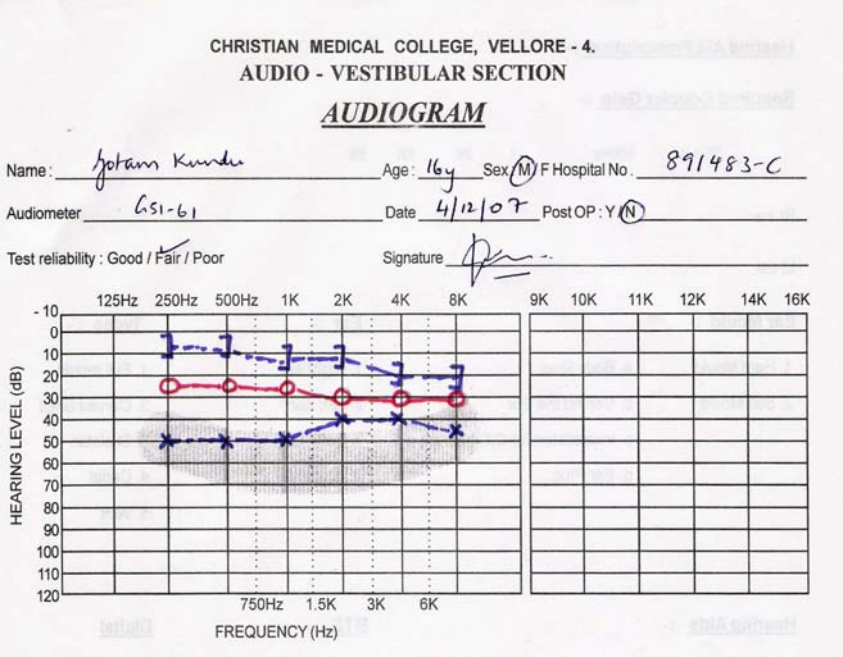


Figure 5: Audiogram of a patient with an ABG of 40-50dB on the right, typical of ossicular necrosis. Note the bone conduction dip at 4KHz

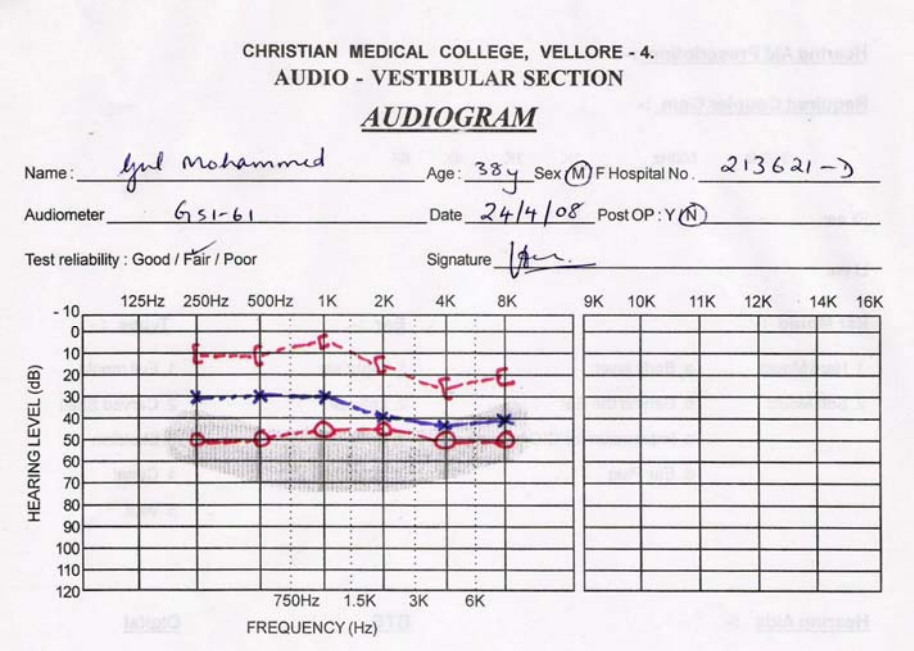


Figure 6: Xray of a sclerosed mastoid in a patient with incus necrosis

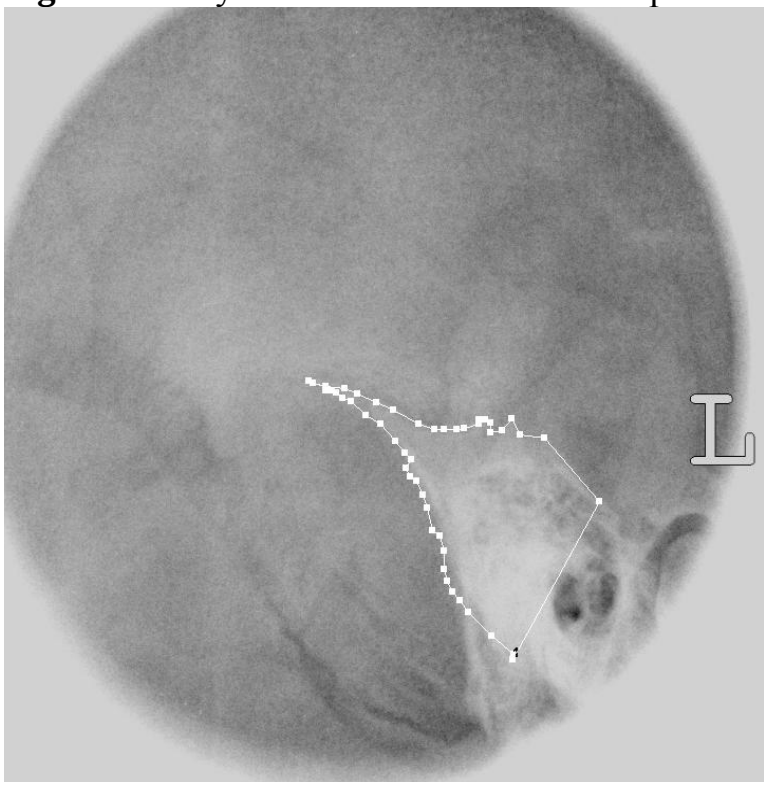


Figure 7: Xray of a well pneumatized mastoid of a normal ear

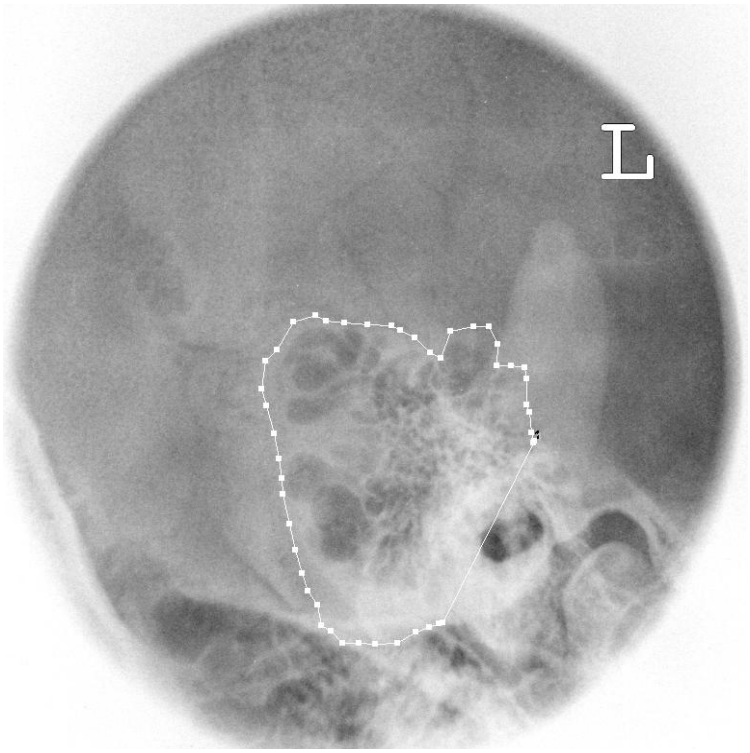


Figure 8 : Age and Sex Distribution

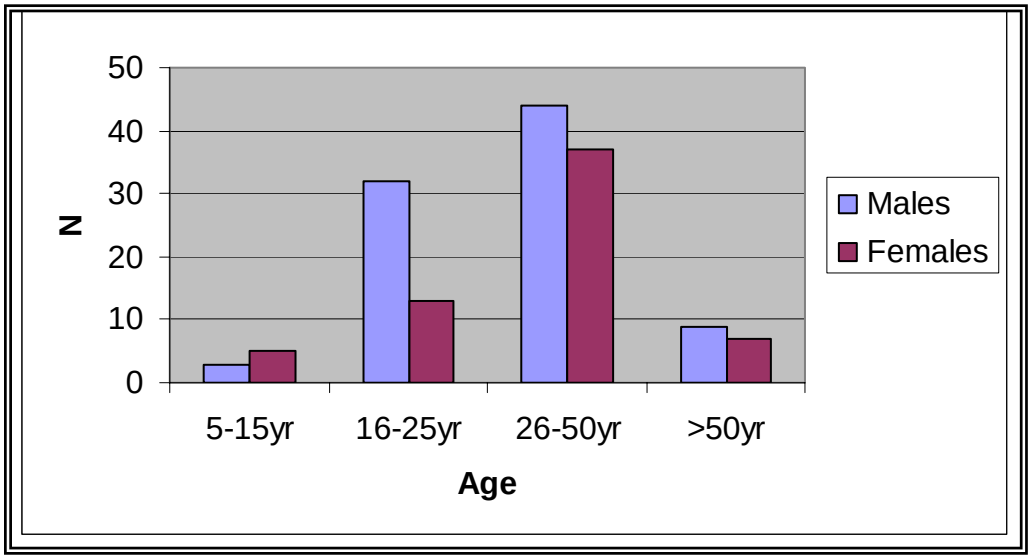


Figure 9 : Duration of ear discharge

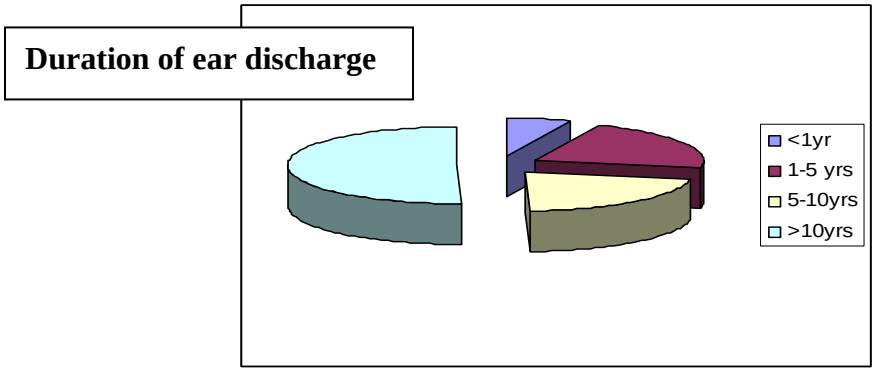


Figure 10 : Most recent episode of ear discharge

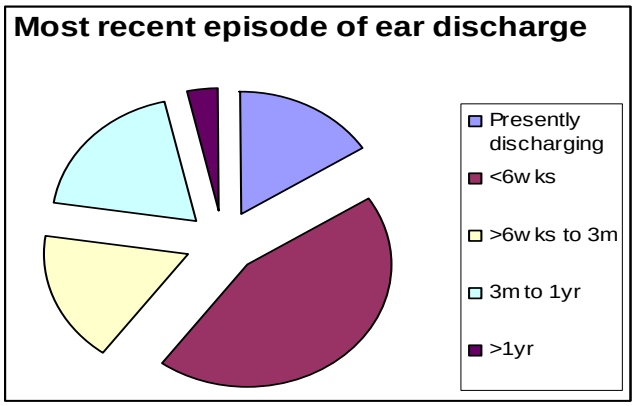


Figure 11 : Distribution of Air Bone Gap across frequencies

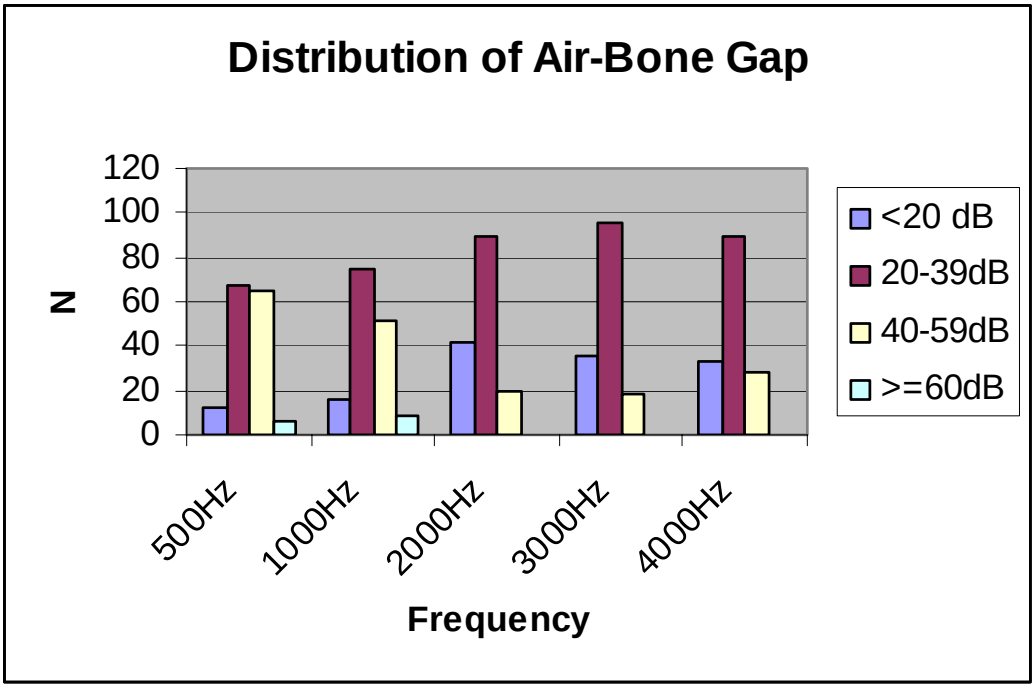


Figure 12 : Correlation between presence of incudal necrosis and degree of hearing loss

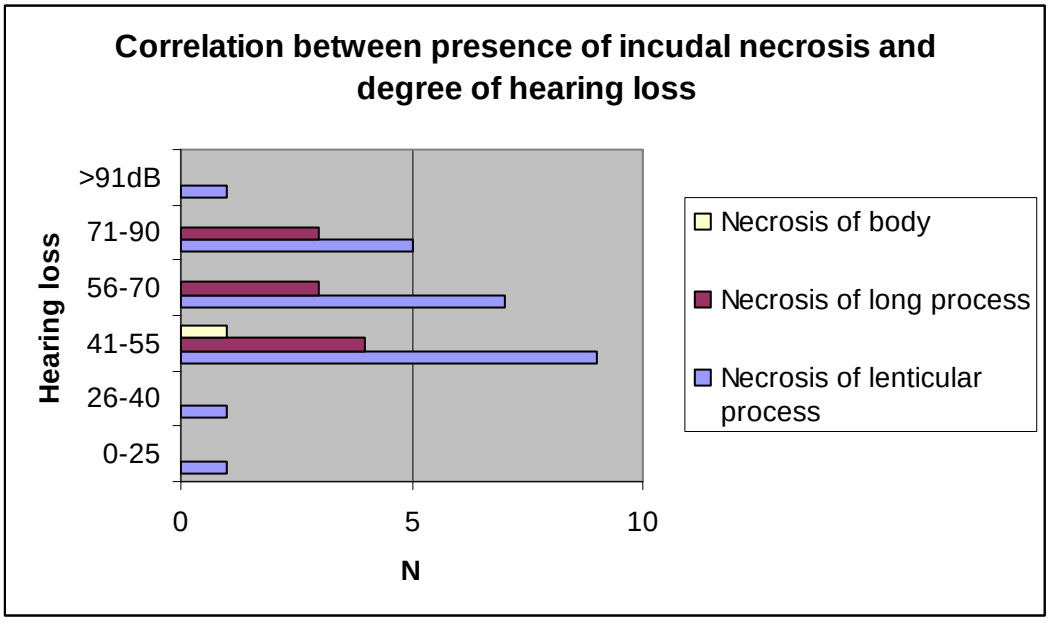
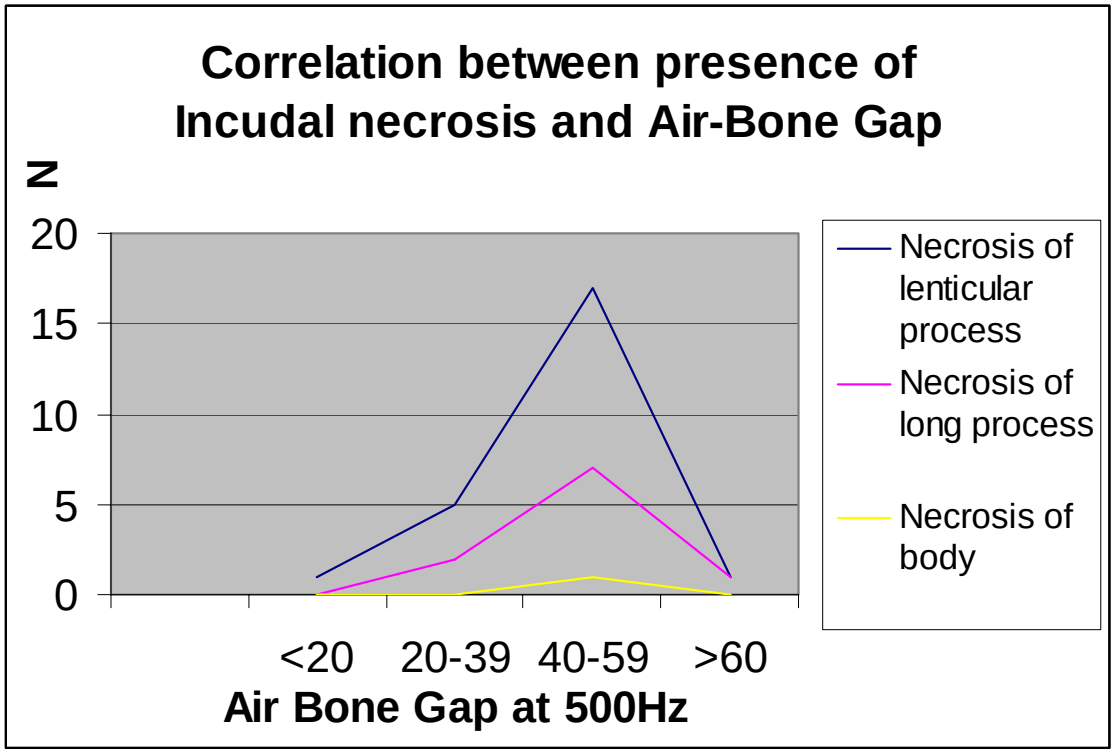


Figure 13 : Correlation between presence of incudal necrosis and Air-Bone Gap


Correlation between presence of Incudal necrosis and Air-Bone Gap
N
20
15
10
5
0
<20
20-39
40-59
>60
Air Bone Gap at 500Hz
Necrosis of lenticular process
Necrosis of long process
Necrosis of body