Dissertation On

CLINICAL AND MICROBIOLOGICAL PROFILE OF ACTIVE TUBO TYMPANIC DISEASE

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CERTIFICATE

This is to certify that this Dissertation titled clinical and microbiological profile of active tubotympanic disease is a bonafide work done by Dr.Annie Johnny MS (ENT) Post graduate of upgraded institute of otorhinolaryngology, Madras Medical College, Chennai, during academic year 2004-2007.

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Introduction

INTRODUCTION

Chronic suppurative otitis media and its complications are among one of the most common conditions seen by otorhinolaryngologist, pediatrician and general practitioner. It is disease of multiple etiology and is well known for its persistence and recurrence inspite of treatment.

Chronic suppurative otitis media is a long standing inflammation of the middle ear cleft. From early days of otology, it is divided into two clinical types. Tubotympanic and atticoantral disease. Tubotympanic disease is characterised by perforation in pars tensa. As it follows a more benign clinical course, term `safe' is applied to it, though it is not always true. It is called active when in addition to tympanic membrane defect, middle ear mucosa is inflammed and edematous with production of excess of mucus or mucopus.

Even with newer antibiotics being licenced for use almost every year, chronic suppurative otitis media largely remains unconquered and continues to be one of the major causes of otologic morbidity. Its clinical significance is particularly related to its propensity to cause infectious complications as acute and chronic mastoiditis, petrositis, intracranial infections, and non-infectious sequel as chronic perforation of tympanic membrane, ossicular erosion, labyrinthine erosion,

tympanosclerosis, which are the major causes of hearing loss through out the world.

Management of CSOM begins with accurate documentation of tympanic membrane defect, preferably with operating microscope. Assessment of hearing loss by tuning fork test and pure tone audiometry is necessary as most of patients have associated conductive hearing loss. Appropriate therapy for otorrhea involves identification of offending organism by means of culture and sensitivity of middle ear discharge.

Almost all aspects of disease have been well studied over past few decades, but exhaustive review of available literature shows many authors focussed their attention primarily on bacterial flora, with comparatively fewer reports on mycological aspects, importance of which has been increasing in recent years because of excessive use of broad spectrum antibiotics, corticosteriods, and immune deficiency states. Number of Indian reports on this aspect have been meagre.

Causative bacterial flora and their sensitivity patterns are also subjected to change from time to time with emergence of multiple drug resistant strains. As for selection of first line therapy, it must be made by individual physician based on regional susceptibility data of bacterial pathogens.

By this study an attempt is made to reevaluate the role of bacterial and fungal pathogens in CSOM, with regional antimicrobial susceptibility data, so as to suggest management guidelines based on these observations.

Review of the Literature

REVIEW OF LITERATURE

Chronic suppurative otitis media is defined as the chronic or intermittent otorrhea through a persistent non-intact tympanic membrane.[2]

Anatomy

Tympanic membrane – Correct form and structure of tympanic membrane was first described by ***Henry Johes Shrapnell*** in 1830.[3] If forms partition between external auditory canal and middle ear. Shaped like an irregular cone, with apex being formed by umbo at tip of handle of malleus, a bright cone of light can be seen radiating from umbo to periphery in antero inferior quadrant. It is obliquely set, as a result, postero-superior part is more lateral than its antero inferior part. Adult tympanic membrane is 10mm tall, 8-9mm wide, and 0.1mm in thickness. Fibrous annulus anchors it in tympanic sulcus which is deficient superiorly at notch of Rivinus.

By anterior and posterior malleolar folds running from lateral process of malleus, it is divided into superior pars flaccida (Shrapnell's membrane) and inferior pars tensa. Shrapnell's membrane serve as lateral wall of Prussak's space (Superior recess of tympanic membrane).

Tympanic membrane is a trilaminar structure, Lateral surface is lined by squamous epithelium, medial layer is continuous with mucosal layer of middle ear, between these two layer is the fibrous layer or pars propia. It is thin and not orderly organized in pars flaccida.

Middle ear

Middle ear together with eustachian tube, aditus, antrum and mastoid air cells is called middle ear cleft. It is a sagitally oriented slit traversed by ossicular chain and lined by mucosal epithelium. Planes extending from tympanic annulus subdivide it into mesotympanum (lying opposite to pars tensa), epitympanum or attic (lying above pars tensa but medial to Shrapnell's membrane and bony lateral attic wall) hypotympanum (below level of pars tensa). The portion of middle ear around tympanic orifice of eustachian tube is called protympanum.

Middle ear is likened to a six sided box with roof, floor, medial, lateral, anterior and posterior walls.

Anterior wall – Here, thin plate of bone separate cavity from internal carotid artery. Anterior wall is dominated by bulge of semicanal of tensor tympani muscle. Tympanic orifice of eustachian tube is immediately interior to this bulge.

Posterior wall – Key feature is the pyramidal eminence and lateral to it is the chordal eminence. Aditus lies above the pyramid. Facial nerve runds in posterior wall just behind pyramid. Facial recess is the depression in the posterior wall lateral to pyramid.

Medical wall or surgical floor of middle ear- Presents a bulge called promontory due to basal coil of cochlea and 3 depressions; Sinus tympani, oval window niche and round window niche. Sinus tympani is defined by ponticulus superiorly, subiculum, inferiorly, mastoid segment of facial nerve laterally, and posterior semicircular canal medially. Oval window niche is anterior superior to ponticulus and is occupied by stapes foot plate. Above oval window is the canal for facial nerve and above it is the prominence of lateral semicircular canal. Round window niche found posteroinferior to promontory, being covered by secondary tympanic membrane.

Roof is formed by tegmen tympani separating tympani cavity from middle crucial from middle cranial fosse.

Floor is formed by thin plate of bone which separate tympanic cavity from jugular bulb.

Ossicles- They conduct round energy from tympanic membrane to oval window. They are,

Malleus – Has head, neck, handle, anterior and lateral processes. Head and neck lies in attic. Axis of rotation is along anterior ligament of malleus.

Incus – Has body and three processes Long, short and lenticular. Body articulates with head of malleus. Short process is anchored to incudal fossa. Long process extends inferiorly terminating at lenticular process which aritulates with head of stapes. It is prone to osteitic resorption owning to tenuous blood supply in face of chronic suppurative otitis media.

Stapes – It has head, neck, anterior, posterior crura and foot plate. Foot plate sits in the oval window.

Intra tympanic muscles – Tensor tympani and stapedius. They help to dampen very loud sounds thus preventing noise trauma.

Histology – Cilliated pseudostratified columar epithelium of respiratory tract extend up the eustachian tube as far as anterior part of middle car cavity. They are capable of producing mucus. More posteriorly mucosa changes to simple cubodial or stratified epithelium with no secretory elements. Medial aspect of tympanic membrane and mastoid air cells are lined by single layer of cells ranging from cuboidal to flat in shape.

Physiology of hearing

The purpose of auditory apparatus is to convert air borne vibrations to vibrations in inner ear fluids and then to nerve impulses to be transmitted to higher centers of hearing.

To over come impedance offered by inner ear fluids, sound energy of greater amplitude and less force need to get converted to lesser amplitude and greater force. It is achieved by impedance matching or transformer action of middle ear, accomplished by hydraulic action of tympanic membrane, lever action of ossicles and curved membrane effect., together giving total transformer ratio of 18:1. Phase differential between oval window and round window need to be maintained for normal hearing. It's is by preferential conduction to oval window by intact tympanic membrane and ossicles. Eustachian tube maintains equal pressures on either side of tympanic membrane for its efficient functioning. Transduction of mechanical energy to electrical impulses is at the level of organ of corti. Conduction of these electrical impulses to brain is via cochlear nerve to auditory nuclei in brain stem and from there to auditory cortex where these impulses are perceived as sound.

Epidemiology

Prevalence of CSOM varies between racial and socio economic groups. It is extremely common in native American Indian, Canadian and Alaskan inuits, Australian aborigines and New Zealand Maoris.[2] In U.K. prevalence is 0.6% of adult population. (***Browing*** – 1982).[4] In India, overall prevalence rate is 46 and 16 persons per thousand in rural and urban population respectively.[5] High prevalence in developing countries is due to adverse socioeconomic status, poor housing with damp and over crowded living quarters and limited access to medical care.[2]

Classification

A. Anatomical

a. Tubotympanic b. Atticoantral

Tubotympanic disease is characterised by perforation in pars tensa. They are not generally considered to be at risk of developing serious complications. So term `safe' ear used. But ***Browing***[6] (1984) pointed out that mucosal disease unassociated with cholesteatoma can have significant morbidity including mastoiditis, otitic hydrocephalus, lateral sinus thrombosis, extradural abscess, meningitis, brain abscess, petrositis, labyrinthitis and facial paralysis.

Atticoantral disease commonly involves pars flaccida and is characterized by formation of a retraction pocket in which keratin accumulate to produce cholesteatoma. Marginal perforations are also considered sinister because they may go in for cholesteatoma.

b. Pathological Classification[7]

a. Inactive (Mucosal) chronic otitis media.

There is a permanent defect of pars tensa, but no current evidence of inflammation either in middle ear mucosa or tympanic membrane. Ossicular chain may be eroded or fixed. The natural history of such an ear is to become active or remain inactive.

b. Active (Mucosal) Chronic Otitis Media

In addition to tympanic membrane defect, the middle ear mucosa is inflamed and edematous with production of excess mucus or mucopus. Such activity may be continuous or intermittent. In some cases, granulations or polyp may develop.

c. Active Squamous epithelial Chronic Otitis Media Cholesteatoma

Here, along with active mucosal otitis, there is squamous epithelial lined pocket full of squamous epithelial and

inflammatory debries. This most frequently occurs in pars flaccida but can occur from pars tensa retraction pocket also.

d. Inactive squamous epithelial chronic otitis media Retraction Pocket

It is the pars flaccid or pars tensa retraction, a part of which is out of vision to otoscopist which is considered abnormal because of potential to retain squamous debris, which may lead to cholesteatoma.

c. Clinical Classification

Thorburn[8] (1965) described tubotympanic disease into two types.

1. Permanent perforation syndrome.. LILLIE type I.

This consists of persistent perforation of tympanic membrane involving pars tensa. Margins of perforation are covered with healed epithelium. The ear may be completely dry for a long period or it may discharge intermittently. Such discharge may be caused by infected water passing through external canal or may spread up from Eustachian tube from nose blowing or sneezing., facilitated by air readily passing outward through perforation. In the dry state, tympanic membrane is pink and granulations or debris are not present in the tympanum. When

such an ear discharges secretions are mucoid or mucopurulent and may be very profuse. There is no odour to the discharge The mucosa in the tympanum becomes red and oedematous.

d. Chronic Tubotympanic Mucositis. LILLIE type II.

As the term implies, the ear presents a long standing infection characterized by an odourless mucoid or mucopurulent discharge which becomes very profuse and is associated with upper respiratory tract infection A large and often near total defect of tympanic membrane is usually present Only the limbus may persist. A shortened malleus may be present The exposed mucosa on the promontory is markedly thickened and red. The exposed ossicles are buried in this thick exuberant and edematous mucosa. Polyps may be present as a result of marked swelling of tympanic mucosa and these polyps may be associated with necrosis of ossicles or underlying portion of temporal bone.

In some of these cases, after a long period of suppuration, there is in growth of epithelium around perforation margins and into posterior tympanum or attic producing a secondary cholesteatoma. Such a case is classified as Lillie type III.

Types of Perforation

Described according to their anatomical location.[2]

Central perforations – located in par tensa and are surrounded by some residual tympanic membrane.

According to their location in relation to handle of malleus, they can be anterior, posterior or inferior

A subtotal perforation is a large defect surrounded by a completely intact annulus.

Marginal perforation is usually in the posterior part of tympanic membrane with pathological loss of annulus allowing direct exposure of the bony canal wall.

Attic perforation is the defect in pars flaccida.

Ethiopathogenesis

1. Environmental

As with any other medical illness – CSOM correlates with socioeconomic status, with lower groups having higher incidence. It is related to general health, diet and overcrowding at home.

2. Genetic

Mastoid air cell system is smaller in individuals with otitis media. The degree of initial mastoid aeration may be a predisposing factor, but once the condition developed cell system will decrease in size.

3. Previous otitis media

In majority of cases, CSOM occurs as a consequence of an episode of acute suppurative otitis media with perforation with subsequent failure of perforation to heal. Also it can be a sequel of otitis media with effusion as here, chronic retraction of tympanic membrane causes loss of fibrous layer. (***Smyth*** 1980)[9] which will not heal if there is a subsequent perforation[9]

4. Infective

Bacteria are almost invariably isolated, but they may be secondary invaders of a mucosa which is inflammed because of other factor., but they can perpetuate the process as they can produce substances that affect cilliary function (***Wilson & Colle*** 1988)[10]

5. Upper respiratory tract infection

A reasonable postulate is that if one area of respiratory mucosa is affected, there is an increased likelihood that another part will also be

affected, as many patients state that their ear start to discharge after an upper respiratory tract infection.

6. Auto immunity

It is likely that individuals with established auto immune disease will have higher incidence of CSOM, but only rheumatoid arthritis been proved till date. (***Camilleri et al*** 1992).[11]

7. Allergy

Allergic individuals have higher incidence of CSOM. Also allergy to topical antibiotics, or bacteria and their toxins may contribute to CSOM.

8. Eustachian Tube Malfunction

Abnormal Eustachian tube function associated with cleft palate, Down's syndrome, patulous eustachian tube predispose to CSOM.

9. Trauma

Iatrogenic (following grommet insertion) & Traumatic perforation which is large and infected may fail to heal causing CSOM.

Pathology

Inactive chronic otitis media - consistent finding is the loss of fibrous layer. So even if there is healing membrane bridging the defect is only an outer layer of squamous epithelium and inner mucosal layer. When perforation ins present, outer squamous epithelium meets middle ear mucosa at variable points.

In active mucosal disease, non secretory middle ear mucosa is replaced by respiratory type mucus secreting mucosa with goblet cells. Mucosa is hyperemic with underlying inflammatory response. Mastoid mucosa seldom undergoes metaplasia to secretory lining. Granulation tissue is more common here.

Clinical features

Two classic symptoms of CSOM are aural discharge and hearing loss.

Aural discharge of tubo tympanic disease in typically profuse, character varies form serous to mucous to frankly purulent. It is frequently intermittent, with increase in discharge associated with upper respiratory tract infection or contamination from external canal after bathing or Swimming, but can be continuous also May be blood stained

if there is associated aural polyps or granulations. Very rarely only it is foul smelling.

Hearing loss is usually conductive type, degree of which is influenced by,

a. Size and position of perforation Larger perforation – More hearing loss Posterior perforation – More severe loss because of reduction of round window baffle effect. Small anterior defect, offen produce no impairment of hearing at all.

b. Impairment of ossicular chain mobility either by ossicular necrosis or fixation.

c. Presence of middle ear pathology as edema and granulation tissue.

Paparella et al (1984)[12] pointed out a definite increase in incidence of sensory neural hearing loss, mainly in high frequencies, owning to diffusion of bacterial toxins across round window membrane to cochlea.

Pain is not a usual feature. Its presence should alert physician to possibility of more invasive pathology as with development of headache, vertigo and facial palsy. Features of secondary otitis externa

as itching, tragal tenderness, excoriation of external canal can be present with long standing profuse otorrhea.

Examination

1. Scars of previous ear surgery should be looked for on either side of pinna.

2. Otomicroscopic examination need to be done in all cases and findings documented. Critical screening of the following areas need to be done.

    a. External auditory canal, ear drum Carefully cleaned off wax, debris which prevent complete visualization of tympanic membrane.

    b. All four quadrants of pars tensa observed carefully and size and location of perforation noted.

    c. Presence of squamous epithelium in middle ear is documented.

    d. Status of middle ear mucosa is assessed through perforation. Other middle ear structures that may be visualized are Eustachian tube orifice, Promontory, round window and oval window niche.

e. Presence and location of granulation or polyps documented.

f. Integrity of ossicular chain as seen through perforation recorded. ie. any disruption of incudostapedial joint, necrosis of long process of incus, or foreshortening of handle of malleus.

3. An assessment of hearing loss done by standard Rinnie and weber tuning fork tests.

4. Nose and throat examined for focal sepsis if any.

5. General physical examination done to rule out systemic causes leading to persistence of CSOM.

Predisposing factors which lead to chronicity and recurrence of CSOM.

A. LOCAL

1. In ear

a. Persistent perforation of tympanic membrane leading to bacterial contamination directly from external ear. Also loss of middle ear "gas cushion" which helps to prevent reflux of nasopharyngeal

secretions into middle ear via Eustachian tube causes increased exposure of middle ear to pathogenic baceria from nasopharynx.[13]

b. Involvement of middle ear with squmous metaplasia and other irreversible pathologies.

c. Persistent obstruction to aeration of middle ear and mastoid air spaces caused by scarring, thickening of mucosa, polyps, granulations and tympanosclerosis.

d. Areas of sequestration or persistent osteomyelitis in mastoid.

2. In nose and throat

a. Eustachian tube dysfunction

b. Chronic rhinitis / allergic rhinitis

c. Atropic rhinitis.

d. Sinusitis

e. Adenoditis, chronic tonsillitis, pharyngitis

f. Nasal Polyposis.

g. chronic granulomatous disease of nose and paranasal sinuses.

B. SYSTEMIC

Diabetes mellitus, tuberculosis, allergic disorder, renal diseases, adrenal cortical insufficiency, chronic leukemia, chronic liver diseases, wegners granulomatosis, histocytosis X, dietary deficiencies, debility, agammaglobulinemia AIDS.

These conditions need to be adequately managed to obtain satisfactory results for medical and surgical treatment of CSOM.

Management

Swab from discharge should be sent for culture and sensitivity preferably prior to beginning of antimicrobial therapy. Audiologic evaluation is necessary as majority of patients will have conductive hearing loss Radiological examination is not necessary in uncomplicated cases of chronic suppurative otitis media with out cholesteatoma[2].

Microbiology

In normal individuals with intact tympanic membrane, culture swabs taken from middle ear mucosa will not grow any bacteria. In some, however upper respiratory tract flora as streptococci and pneumococci species may be isolated.

When tympanic membrane defect is present, and if the mucosa is already inflammed secondary bacterial colonisation occurs.[14] A wide range of organisms, both aerobic and anaerobic may be isolated, proportion of different organisms vary from study to study.

Morgagni was the first to describe suppuration in ear as a primary lesion. ***Andina*** and ***Aliman*** in 1950 stated in their work on importance of testing the resistance of pathogenic bacteria and their sensitivity.[15]

Collection of culture specimen

Special techniques, and procedures for improving bacterial isolation described. Materials available for culture are otorrhea, middle ear mucosa and mastoid portion of temporal bone.

4. Otorrhea

Conventional method is by inserting sterile cotton tip in an applicator in external canal. This technique is inadequate as tip of the applicator cannot reach focus of infection in the middle ear. So sample get contaminated with discharge collected in external canal.[16] But ***Raju K.G. et al.*** (1990)[17] got identical results with culture obtained from middle ear and conventional external ear swab.

A nichrome wire loop has been used as another technique.[16] But it has disadvantages, as specimen collected should be immediately inoculated into medium and is not suitable for transport and anaerobic culture as it dries up.

Aspiration through perforation of tympanic membrane with 2ml disposable syringe fitted with 24 G needle or 18 G needle with plastic cannula also described[16] but it is dangerous as it may injure middle ear. Further discharge may sometimes be inadequate to be aspirated to syringe.

If perforation is blocked by dry secretions or blood or granulations, or if tympanic membrane appears to be intact, culture of middle ear fluid can be obtained by tympanocentesis.

For higher yield of anaerobes, ***Induharan et al.*** (1996)[16] recommended pre reduced 28 G nichrome wire swab which they claim to be safe, simple and effective alternative to commercially available transport media.

2. Middle ear mucosa

Biopsy of middle ear mucosa may be desirable especially if there is large perforation of tympanic membrane or if patient undergoing exploratory tympanotomy or mastoidectomy. It provides a pathology

specimen and actual piece of infected tissue which may provide accurate microbiologic diagnosis than possibly contaminated secretion obtained from perforation.[18]

3. Mastoid portion of temporal bone

Since CSOM involves mucosal lining of mastoid and possibly periosteum as well, biopsy of mastoid bone performed during mastoidectomy provides diseased tissue for culture and histopathological examination. In certain cases, it may be appropriate to obtain needle aspiration of mastoid rather than open biopsy.[18]

Although several different organisms cultured, role of bacteria is often questioned as ***Picozzi et al*** (1982)[19] pointed out nearly 50% of ears with inactive CSOM, identical bacterial flora as that of active ears is cultured. But it is proven that bacteria can produce substances which affect cilliary function. There is also evidence that poly microbial infection is more damaging than monomicrobial infections. (***Brook*** 1987).[20]

Most common aerobic organisms isolated are, pseudomonas aeruginosa, staphylococcus aureus, proteus and Klebsiella, but their proportions vary from study to study. Staphylococcus aureus predominance was seen in various studies.[21,22] In study by ***B.N.Rao et al*** (1994)[23] Staphylococcus aureus was the predominant species (42.5%)

followed by pseudomonas aeruginosa. (21.6%) Pseudomonas was most common isolate in studies by ***R.D. Kulkarni et al*** (1993)[24], ***Fliss D.M. et al*** (1992)[25], ***Khanna et al*** (2000)[26], ***Attallah M.S.*** (2000)[27], ***Induharau R*** (1999)[28], ***Aslam MA.. et al*** (2004)[29].

Saini S et al (2005)[30] concluded in their study that staphylococcus aureus was the commonest isolate in paediatric age group and pseudomonas aeruginosa in adults. A study on bacterial culture from mastoid granulations by ***Albert R.R et al*** (2005)[31] found these granulations harbour polymicrobial pathogens, predominant aerobic isolates being coagulase negative staphylococci, pseudomonas aeruginosa, staphylococcus aureus and non-fermenting gram negative bacteria suggesting these organisms may be responsible for granulation.

A review of aerobic isolates in different studies

Year	Author	Pseudomonas	Staphylococcus	Proteus	Klebsiella
1992	Fliss DM[25]	84%	20%	15%	8%
1994	M. Rao[23]	21.6%	42.5%	18.3%	-
1995	Obi CL [22]	19.3%	33.6%	17%	4.3%
1997	Ashok Mittal[32]	48.7%	22%	3%	2%
1999	R. Induharan [28]	27.2%	23.6%	12%	-
2000	Attallah M.S. [27]	51%	31%	17%	-
2004	Aslam M.A.[29]	50.5%	23.6%	12%	-

Contrary to earlier opinion that type of flora are no different if cholesteatoma is present (***Sweeny, Picozzi*** and ***Browning*** 1982).[14] ***Vartiainen E*** (1996)[33] observed staphylococcus aureus was isolated in cholesteatoma ears more frequently than pseudomonas aeruginosa and in chronic ears without cholesteatoma, situation is reversed. He also observed bacteriological findings had no significant effect on incidence of complications caused by disease. Failure after surgical treatment were more common in pseudomonas ears.

Perhaps most exciting development in field of microbial flora of CSOM in recent years is the discovery of presence of non-sporing anaerobes, but its actual role in disease causation is a subject of speculation. Bacteroids melaninogenicus, bacteroids fragilis and other speces were isolated from 30-50% cases.[7] According to ***Sugita et al*** (1981)[34] they were most frequently isolated in ears with extensive cholesteatoma and granulation tissue formation. ***Kelly*** (1978)[35] demonstrated combination of aerobic and anaerobic organisms produce more marked inflammatory response than same organisms alone in animal model. ***Heinemann*** (1963)[36] a reported high incidence of anaerobes in otogenic intracranial infections. Indian literature shows 30% ***R.D. Kulkarni et al*** (1993)[24] 51.6% ***Beena Antony et al*** (1996)[37] of anaerobic isolates.

Fungal infections in CSOM was noted as early as in 1967 by ***Larine et al***.[38] Some cases of tubotympanic disease keep on discharging inspite of topical antibiotic ear drops or systemic antibiotics. ***Sen Gupta et al*** (1978)[39] proposed this intractable otorrhea to superimposed fungal infections over CSOM.

Fungal spores ubiquitously found in atmosphere readily establish themselves in warm and moist environment of middle ear, their growth being favoured by presence of epithelial debries along with long term use of topical antibiotics. Humidity in ears with CSOM found is significantly higher than normal ears[40] which aggravate the condition.

Prevalence of fungal infection also varies between studies.

Year	**Author**	**% of fungi**
1978	Sen Gupta[39]	25%
1992	Dincer A.D.[21]	28.6%
1994	B.N. Rao[23]	7.5%
1997	Ashok Mittal[32]	40.8%
1997	Ibekwe A.O[41]	25%
1998	Urmil Mohan[42]	13.7%
2000	Khanna .V[26]	9%

Most common fungal species implicated was aspergillus flavus.[26,32,39] But ***B.N.Rao,***[23] and ***Urmil Mohan***[42] reported candida as most common species. ***Tiwari.S*** (1995)[43] reported a case of pure fungal bilateral chronic suppurative otitis media caused by Aspergillus terreus which responded well to topical ketoconazole therapy. ***Albertt R*** (2005)[31] in their study on mastoid granulation reported single isolate of Aspergillus grown from granulation among total 79 cases. ***Martin T.J*** (2005)[44] in a retrospective review on fungal causes of tympanostomy tube otorrhoea observed an increase in incidence of fungal infections after period of widespread use of fluroquinolone antibiotics.

A 16 years retrospective analysis (from 1978 to 1991) on changing pattern of bacterial isolates and their sensitivities (***Nakagawa T. et al,*** 1994)[45] revealed a gradual rise in incidence of staphylococcus aureus infections while proteus infections gradually declined. Glucose non-fermenting gram negative rods, fungi and anaerobic bacteria increased during same period.

Latest development in field of microbiology of CSOM is demonstration of biofilms. Biofilms are "aggregated bacteria usually adherent to a surface, surrounded by an extra cellular matrix". They are recalcitrant to antibiotic treatment even though isolated bacteria are susceptible to antibiotics. Best studied pathogen for its propensity to form biofilm in pseudomonas aeruginosa. Biofilm associated

microorganisms are implicated in causation of, native valve endocarditis, infections associated with indwelling medical devices, chronic prostatitis as well as chronic otitis media. ***Dohar J.E. et al*** (2005)[46] demonstrated pseudomonas aeruginosa biofilm in middle ear in CSOM by scanning electron microscopy in monkeys. Further research regarding role of biofilms in antimicrobial resistance, chronic infections and as reservoir to pathogenic organisms is in progress.

Treatment

Treatment goals of uncomplicated CSOM are to eliminate infection, prevent further infection, and to restore normal function of middle ear. Both medical and surgical interventions play their role in achieving these aims.

Medical management

Three components of medical management are aural toileting, topical antibiotics and systemic antibiotics.

Aural toilet

It is important for successful treatment of CSOM, particularly when topical medications are used. Cleaning the discharge from external auditory canal will allow the topical agent to reach the middle ear in

adequate concentration. In a controlled trial on medical management of active CSOM ***G.G.Browning et al***[47] reported efficiency of treating actively discharging tubotympanic disease with aural suctioning alone is almost same as using aural toileting along with gentamicin . However, sample size of this study was very small and hence results were not statistically significant.

Methods

1. Cotton buds – self cleaning of ear by dry mopping with self made cotton buds is a convenient method for dealing with active discharge.

2. Syringing – Clearing debries and inflammatory exudate by syringing using normal saline at body temperature was recommended by ***Chui*** in 1982 and John ***Abramson*** 1984. Syringing of infected ear is not widely practiced nowadays.[48]

3. Suction, aspiration and debridement of inflammatory exudates under operating microscope is probably the most popular method Magnification offered by microscope aids in assessing size, site of tympanic membrane defect, extent and type of pathological changes, evidences of destructive disease in ossicular chain and presence of sequel of chronic

ear disease. It can be carried out at outpatient clinic either at weekly intervals[103] or daily as preoperative regimen. Preoperative conservative treatment has been shown to decrease significantly the number of culture positive ears prior to surgery.[48]

2. Topical antimicrobial therapy

Ototopical agents commonly used in treatment of CSOM are gentamicin, Neomycin, polymyxin B, and ciprofloxacin with or without steroids.

Although ototoxicity with aminoglycoside antibiotics has been demonstrated in animal model, there is no evidence that they cause sensory neural hearing loss in patients with CSOM (***Fairbanks*** 1981).[49] This may be due to combination of factors, as relatively low concentration of aminoglycosides reaching middle ear because of oedema of mucosa, which prevents direct absorption and also as human round window niche is deep and protected by pseudomembrane, less is the chance of ototoxicity with aminoglycosides in CSOM patients.[13]

Recent trials demonstrate increased efficacy and safety of fluroquinolone topical preparations like ciprofloxacin and ofloxacin over conventional polymyxin B, neomycin with or without hydrocortisone[50,51] and topical gentamicin.[52] A prospective observational

study on optimum duration of topical ofloxacin treatment[53] advise two weeks course from aspect of bacteriologic efficacy, although patients showing insufficient symptomatic improvement after 2 wk may benefit from another one to two weeks of therapy. Administration upto 4 weeks can increase clinical efficacy without causing safety problems. Drug concentration analysis[54]. With 0.3% ofloxacin shows high concentration in otorrhea, very low or not detected in serum, and highly variable middle ear mucosal concentrations. It shows invariably drug reaches infection site. An article of concern is by ***Jang CH*** (2004)[55] with report of ciprofloxacin resistant pseudomonas aeruginosa increasing recently. A meta analysis on effectiveness of 0.3% ofloxacin otic solution (***Abes G et al*** 2003)[56] conclude that ofloxacin otic ear drops is better than other antibiotic ear drops and other oral antibiotics in terms of overall cure rate.

It is conventional to use antibiotic steroid combination ear drops, but advantage of steroid combination has not been substantiated. ***Induharan et al*** (2005)[57] in their comparative study with gentamicin or gentamicin steroid combination ear drops for three weeks, found no difference in clinical and bacteriological improvement or ototoxicity with or without steroids.

Other preparation used as ototopical agents are,

- 1.5% acetic acid irrigation (one part of white vinegar in two parts water) at body temperature three times daily. Acetic acid irrigation removes accumulated debries and acidifies external canal, discouraging growth of pseudomonas and other bacteria.[58]

- Burrow's solution (13% aluminum acetate) has been used since late 19 century. It is effective in vitro in inhibition of growth of commonly occurring organism in CSOM.[59]

- Topical povidone iodine was compared to topical ciprofloxacin by ***Jaya et al*** (2003).[60] They reported equal results with superior advantage of no invitro drug resistance and reduced cost of therapy for povidone.

Systemic antibiotics

Success of antibiotic therapy is based upon conveyance of antibiotic to the site of bacterial activity in adequate concentration for adequate length of time. Unfortunately, fibrotic changes in chronic infection tend to isolate pockets of infection from effective blood supply so that systemic antibiotic therapy has been found disappointing in CSOM.

Because primary pathogen responsible is pseudomonas aeruginosa, choice of oral antibiotics are limited. Both ciprofloxacin and

Ofloxacin has good antipseudomonal activity. As per study by ***Gehauno P*** 1997.[61] Oral ciprofloxacin is an effective and well tolerated treatment for CSOM in adults. But these are not recommended in children due to possibility of causing arthropathies. So for children, choice is limited to broad spectrum pencillins and they need to be administered parentally.

Another topic of controversy is treatment for anaerobic bacteria. ***Browing et. al*** (1983)[62] pointed out that elimination of anaerobic bacteria from active ears does not render them inactive.

But superiority of therapy against anaerobes with clindamycin is illustrated by ***Brook I*** (1994).[63]

Following factors account for disappointing results of antimicrobial therapy in CSOM, particularly in diffuse mucosal disease involving mastoid bowl and middle ear cavity.

1. Poor drainage of inflammatory exudates.

2. Presence of destructive disease associated with osteitis, granulations and polyp.

3. Lack of information on efficacy of antimicrobial therapy in CSOM based on large scale controlled trials.

4. Presence of keratinizing squamous epithelium in middle ear.

5. Presence of mixed aerobic, anaerobic flora and their pathological synergy.

6. Possibility of infection with different strain of same species.

7. Possibility that certain strains have particular virulence in relation to chronically diseased ear.

8. Mucosal changes in active CSOM particularly in patients with long history of disease, characterized by subepithelial scarring and devasularisation both of which predispose to poor mucosal concentration of antimicrobial agent.

9. Presence of debries and inflammatory exudates in middle ear prevents topical antibiotic from acting on organism.

10. Resistant organisms.

11. Local and systemic predisposing factors.

Factors associated with susceptibility to complications were determined prospectively by ***Panda N.K. et al*** 1996.[64] As per them, complications are more likely in patients younger than 15 years, with short history of ear discharge, in patients whom anaerobic organisms

isolated in culture and in patients with granulation tissue found at surgery.

Surgical Treatment

If otorrhea recur or persist despite medical treatment, or if the patient feel handicapped by the residual conductive hearing loss, surgical therapy should be considered.

1. Tympanoplasty

Ideally, surgery should be carried out when infection has been adequately treated and middle ear mucosa is healthy, as chance of successful outcome is increased. In this situation, a tympanoplasty with repair of tympamnic membrane and ossicular chain is recommended.

2. Tympanomastoid surgery

In cases refractory to medical treatment, tympanoplasty combined with cortical mastiodectomy is indicated. Aims of this procedure are to aerate middle ear and mastoid, remove chronically inflamed tissue, repair the tympanic membrane defect, and reconstruct ossicular chain. The achievement of all there goals often require more than are procedure in different sittings.

Aims and Objectives

AIMS AND OBJECTIVES

This study was conducted with following objectives:

1. To study clinical profile of active tubotympanic chronic otitis media cases at upgraded Institute of otorhinolaryngology, Chennai.

2. To asses prevalence and distribution of bacterial and fungal organisms in CSOM.

3. To analyze antibiotic sensitivity and resistance pattern of bacterial isolates causing Chronic Suppurative Otitis Media (CSOM).

4. To suggest practical recommendations based on the observations.

Materials and Methods

MATERIALS AND METHODS

Study design

Hospital based clinical observational study.

Setting

ENT out patient division of Upgraded Institute of otorhinolaryngology, Madras Medical College and Government General Hospital, Chennai.

Duration

Specimen collection and analysis 1st January 2006 to 30th June 2006.

Evaluation and data interpretation 1st July 2006 to 30th September 2006.

Sample case selection

100 cases of active Tubotympanic Chronic Suppurative Otitis Media by systemic random sampling method.

Inclusion criteria

1. Adult patients with active tubotympanic type of CSOM i.e. Chronic (more than 3 months) continous or intermittent otorrhea through permanent defect in parstensa, with inflammed and edematous middle ear mucosa producing excess mucus or mucopus.

2. Perforation should be moderate / large sized.

3. History of partial or no response to prior treatment with commonly used ototopical agents like gentamicin, Neomycin, Polymyxin, Ciprofloxacin, before consultation.

Exclusion criteria

1. Pediatric age group – less than 15 yrs.

2. Clinically unsafe ears – i.e. with cholesteatoma granulations or aural polyps.

3. Overt clinical evidence of otitis externa with CSOM i.e external canal congested, inflammed, with otomycotic debries, Tragal tenderness.

4. Discharging ear through a pin hole perforation.

5. Cases undergone previous ear surgeries.

6. Suspected complications of CSOM.

7. Patients with clinical evidence of chronic sinusitis chronic tonsillitis.

8. Known or treated cases of pulmonary tuberculosis.

9. Known immune deficiency states – Diabetes mellitus, AIDS, renal diseases, bronchectasis.

All patients evaluated by detailed history and examination. Initially, collected discharge in external canal was cleaned by dry mopping method using sterile cotton wool tipped applicators. Otoscopic examination done, findings were documented. Audiologic evaluation done by Tuning fork tests and pure tone audiometry. Pure tone average and bone conduction at 4 KHZ recorded.

Method of Taking Ear Swab for Pus Culture and Sensitivity

Under aseptic precautions, ear discharge over tympanic membrane issuing from perforation was collected through sterile aural speculum thus minimizing external canal contamination by three separate sterile cotton wool tipped swab sticks which were then kept in

sterile culture tubes. They were immediately transported to microbiology laboratory.

Laboratory Methods

Swabs were processed for smear preparation and culture inoculation in a laminar flow biosafety hood.

A. Direct smear examination

Include gram stained smear and wet mount with 10% KOH.

Gram stained smears examined for presence of pus cells, epithelial cells, gram positive cocci, gram positive bacilli, and gram negative bacilli.

KOH smear examined for yeast cells, hyphae, pseydohyphae, and spores.

B. Culture for aerobic bacteria and their sensitivity patterns.

Material from aural swab was inoculated on

a. Blood agar

b. Chocolate Agar

c. Mac Conkey Agar

They were incubated at 37° C for 24 – 48 hrs. Any bacterial growth which occured was looked for after 24 hrs, and was identified by colony characters, microscopic morphology, and biochemical characters according to standard protocols.[1]

Bacterial sensitivity to various antibiotics as Cefoperazone - Sulbactam, Cefotaxime, Amikacin, gentamicin, ofloxacin, ciprofloxacin, were assessed by inoculating into Muller- Hinton agar by Kirby – Bauer Disk diffusion method.[1]

C. Culture for fungus

Aural swabs were inoculated into 2 tubes of sabouraud's dextrose agar one with antibiotic and second with antibiotic and cycloheximide. They were incubated at room temperature, Examined thrice weekly for first week, and once weekly for next 3 weeks. They were declared negative only if no growth occurred after one month. Isolates were identified based on colonial, microscopic morphology and biochemical characters according to standard protocols.[1]

Fungal infection was diagnosed by presence of fungal elements in smear examination and growth in culture. Cases which were negative by smear examination were nevertheless considered positive for fungus if

culture showed heavy confluent growth on both tubes. Anaerobic culture was not done due to technical and financial constraints.

Analysis

Results were analyzed for age, sex, unilateral or bilateral clinical presentation, otoscopic findings, hearing loss, prevalence of bacterial and fungal isolates, and their sensitivity and resistance patterns according to standard protocols.

Observations

OBSERVATIONS

Table 1 Age and Sex distribution

Age in Years	Male		Female		Total	
	Number	%	Number	%	Number	%
15-30	28	28%	30	30%	58	58%
31-45	18	18%	16	16%	34	34%
36-60	2	2%	4	4%	6	6%
Above 60	Nil	-	2	2%	2	2%
Total	48		52		100	

Table 2 Table showing duration of symptoms

Duration	No.	%
3-6 Months	2	2%
6 Months – 2Years	21	21%
More than 2 years	77	77%

Table 3 Symptomatology

Items	Discharge	Hard of Hearing	Tinnitus	Itching	Pain
Number	100	28	17	16	4
%	100%	28%	17%	16%	4%

Table 4 Table showing audiogram findings

Age in Year	Pure Tone Average	Bone Conduction at 4 KHz
15-30	36	21
31-45	40	21
45-60	40	20
Above 60	43	22

Table 5: Culture results

Items	Single infection	Mixed infection	Sterile
Number	52	38	10
%	52%	38%	10%

Monomicrobial growth	Pure bacterial	50	50%
	Pure fungal	2	2%
Polymicrobial growth	Mixed bacterial	10	10%
	Bacteria and fungi	28	28%
Culture sterile	Bacteria and fungi negative	10	10%

Table 6 Bacterial isolates

No.	Organisms	No.	%
1.	Pseudomonas aeruginosa	38	38%
2.	Staphylococcus aureus	22	22%
3.	Klebsiella	13	13%
4.	Aceinetobacter	11	11%
5.	Coagulase negative staph	7	7%
6.	Proteus species	5	5%
7.	Escherchia coli	2	2%

Table 6 Fungal isolates

No.	Organisms	No.	%
1.	Aspergillus flavus	12	40%
2.	Aspergillus niger	6	20%
3.	Candida species	6	20%
4.	Aspergillus fumigatus	3	10%
5.	Aspergillus terreus	2	6.6%
6.	Cladosporium species	1	3.3%

Table 7 Antibiotic sensitivity pattern of isolates

Antibiotic Tested		**Psudomonas 38**	**Staphylococci 22**	**Klebsiella 13**	**Aceinetobacter 11**	**Cons 7**	**Proteus 5**
Gentamicin	No.	25	13	8	2	7	5
	%	65.7%	59%	61.5%	18.1%	100%	100%
Amikacin	No.	35	21	13	8	7	5
	%	92.1%	95.4%	100%	72.7%	100%	100%
Ciprofloxacin	No.	30	17	12	8	6	5
	%	78.9%	77.2%	92.3%	72.7%	85.7%	100%
Ofloxacin	No.	37	20	12	10	7	5
	%	97.4%	90.9%	92.3%	90.9%	100%	100%
Cefotaxime	No.	12	12	11	2	5	2
	%	31.5%	54.5%	84.6%	18.1%	71.4%	40%
Cefoperazone Sulbactam	No.	37	22	13	11	7	5
	%	97.4%	100%	100%	100%	100%	100%

Figure - 1
Bar Diagram showing age and sex distribution

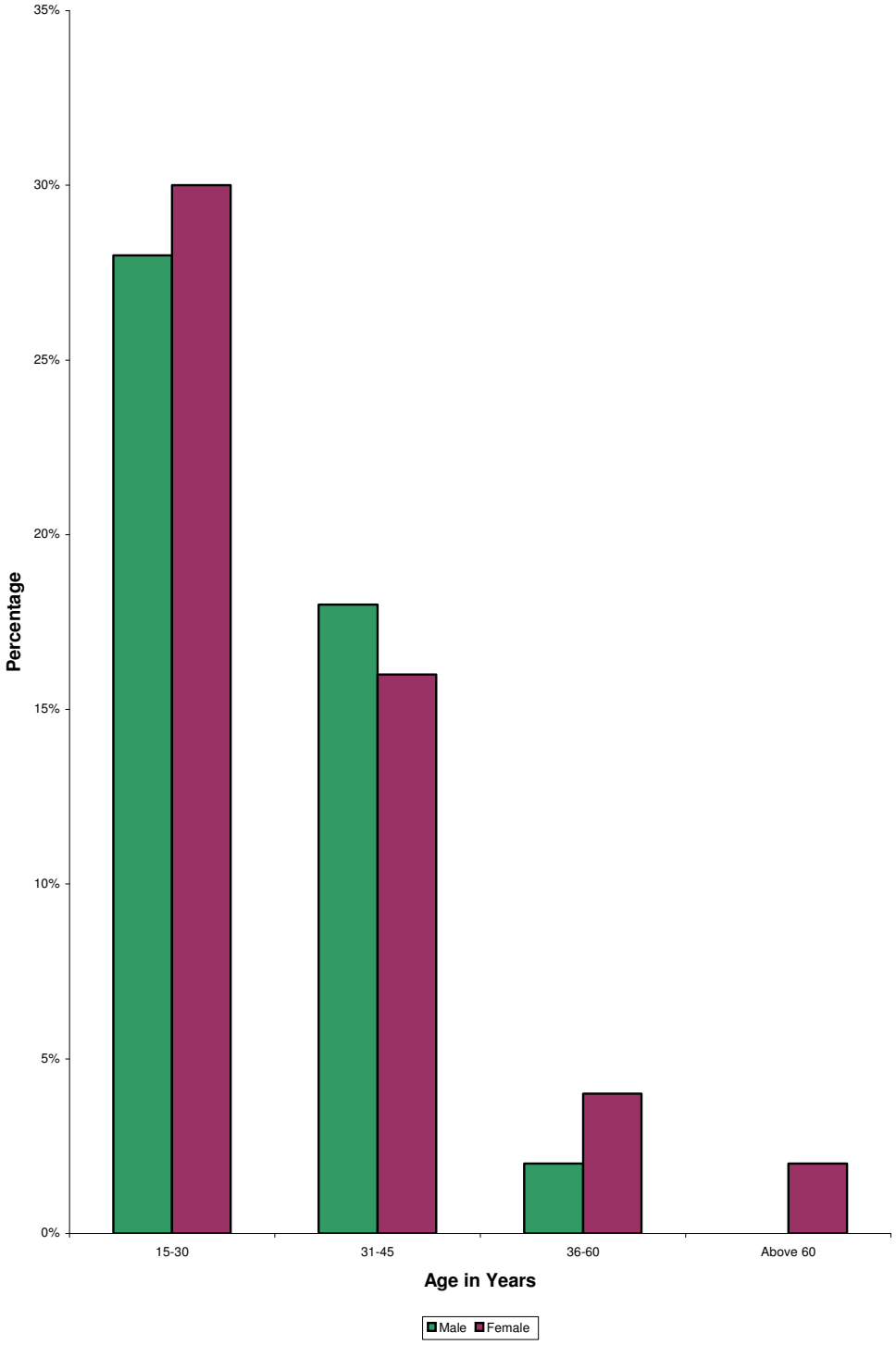


Figure - 2
Pie Chart showing unilateral or bilateral disease

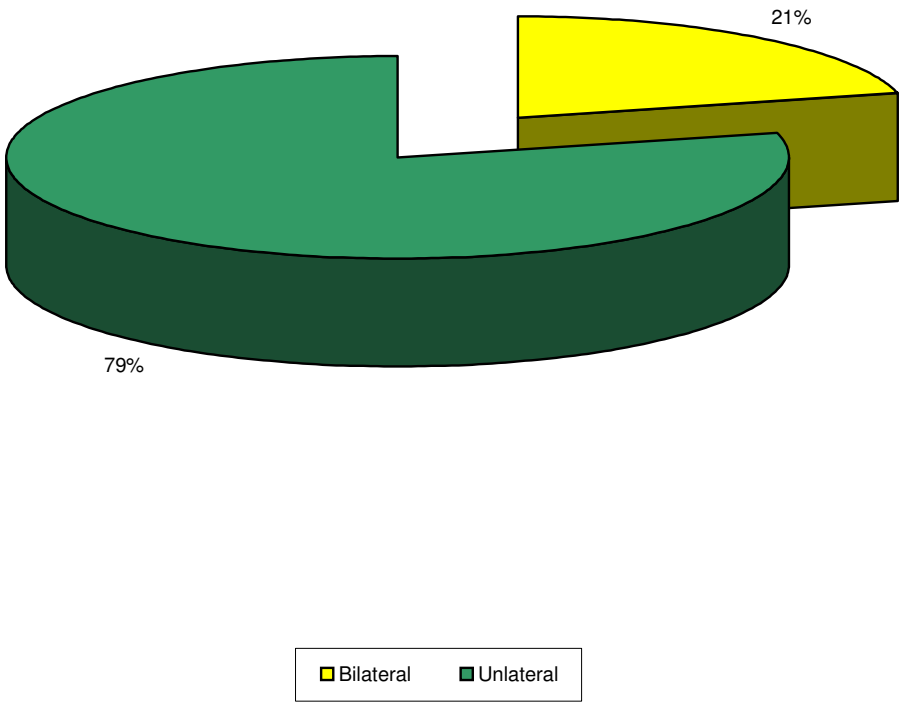


Figure - 3
Pie Chart showing duration of illness

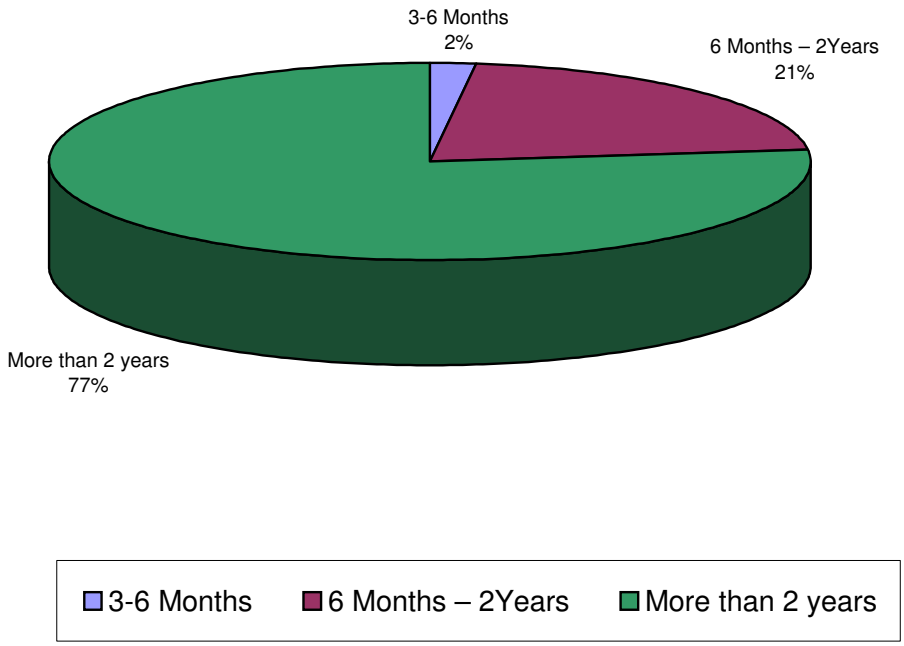


Figure - 4
Bar Diagram showing symptomatology

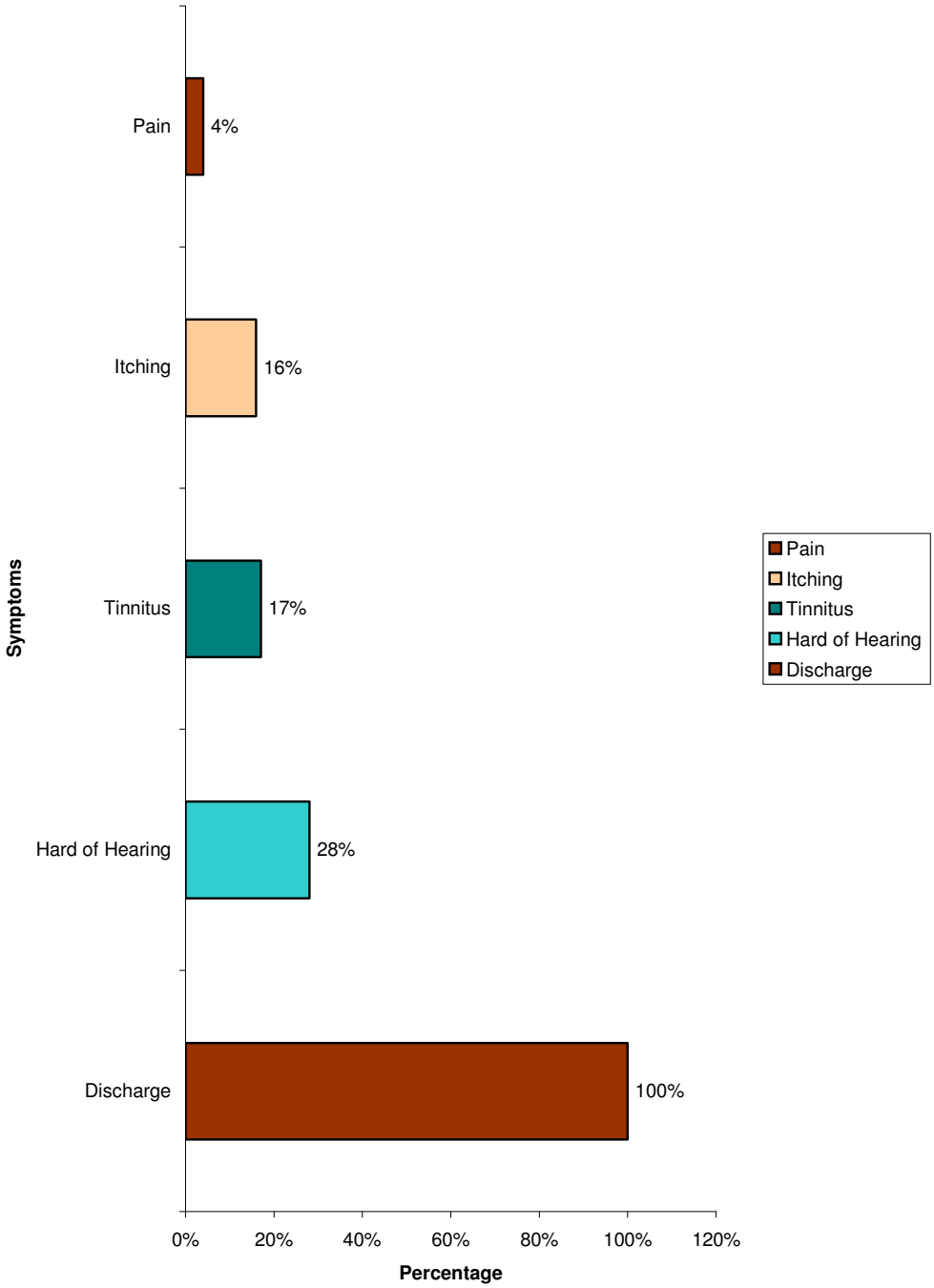


Figure - 5
Pie chart showing condition of middle ear mucosa on otoscopy

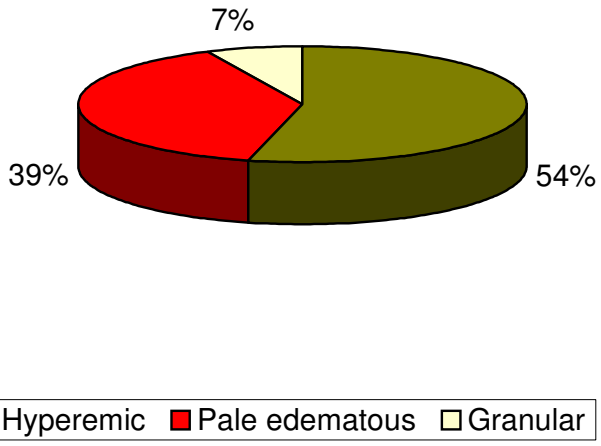


Figure - 6
Bar diagram showing pure tone average and bone conduction of 4 KH3

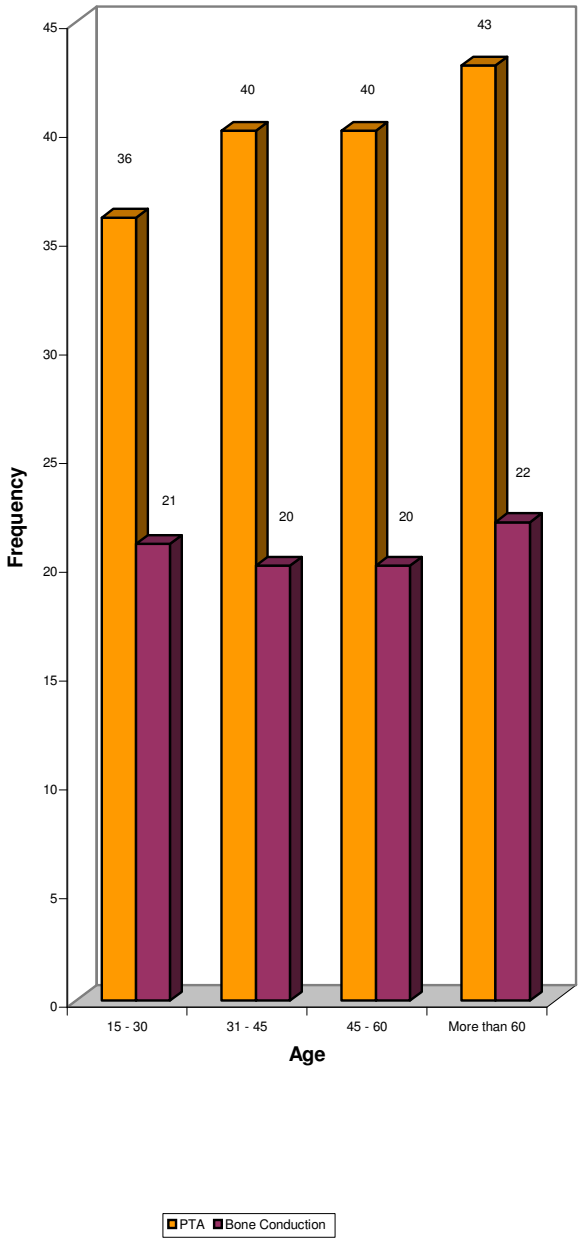


Figure- 7
Pie chart showing breakup bacteriology

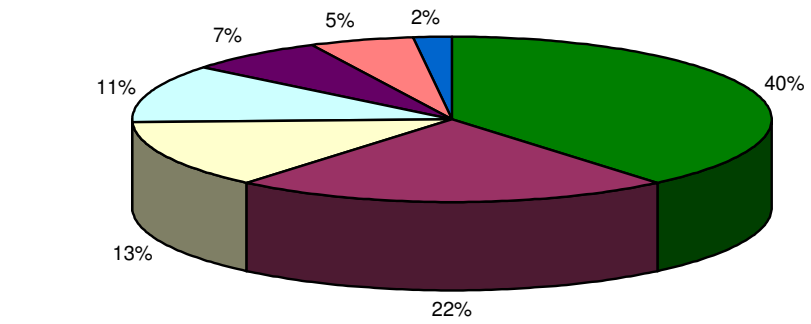
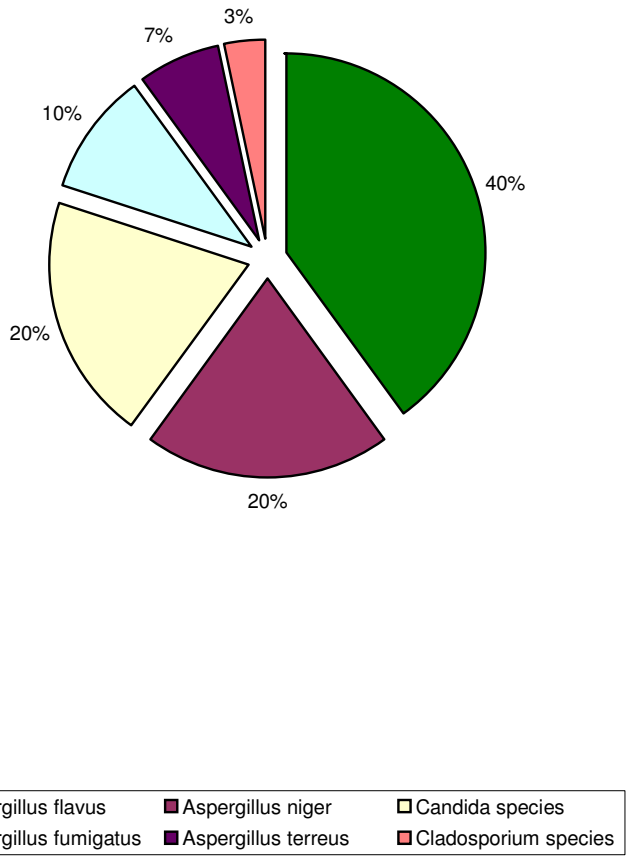


Figure - 8
Pie Chart showing distribution of fungal species



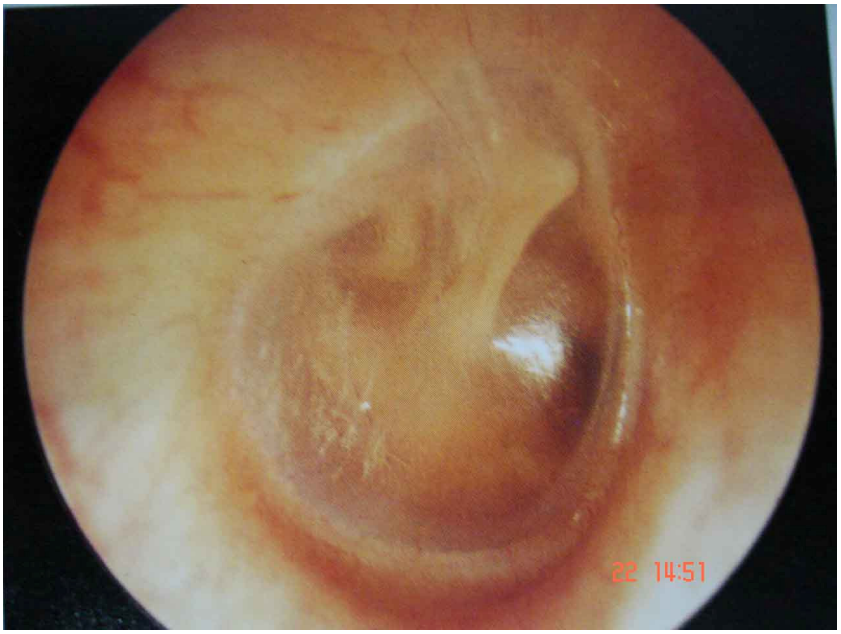


Plate No.1 : Normal Tympanic Membrane

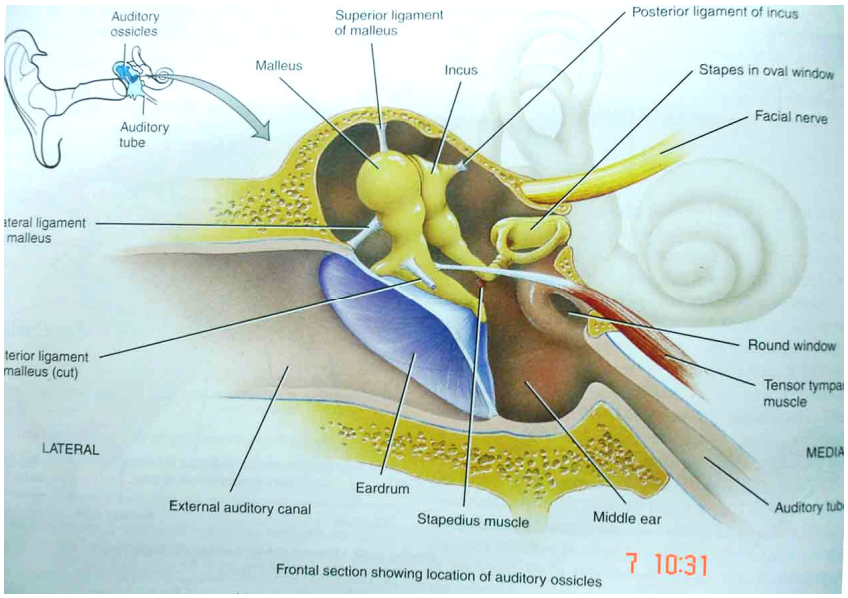


Plate No.2 : Frontal section showing contents of middle ear

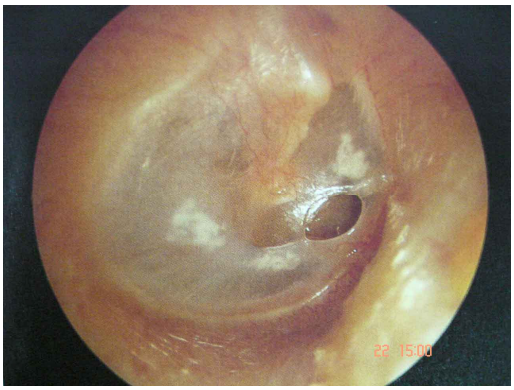


Plate No.3 : Right CSOM Anterior perforation

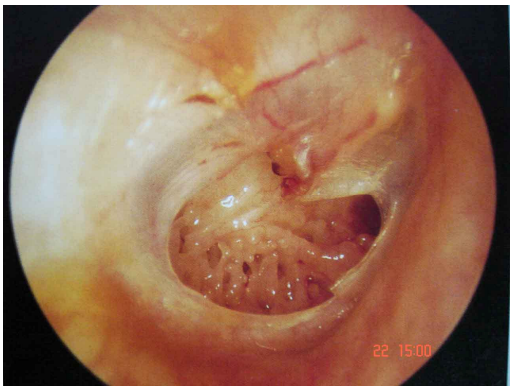


Plate No.4 : Right CSOM Inferior perforation

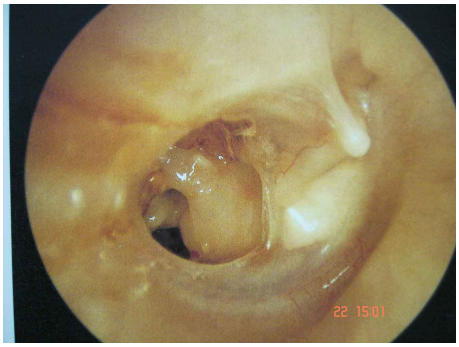


Plate No.5 : Right CSOM Posterior perforation

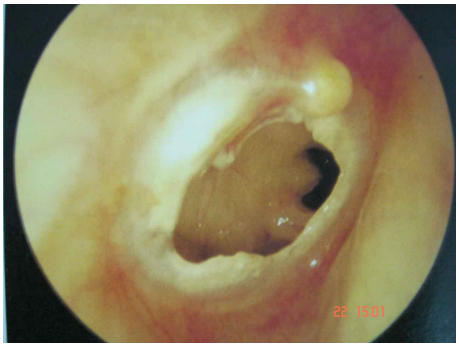


Plate No.6 : Right CSOM Antero- Inferior perforation

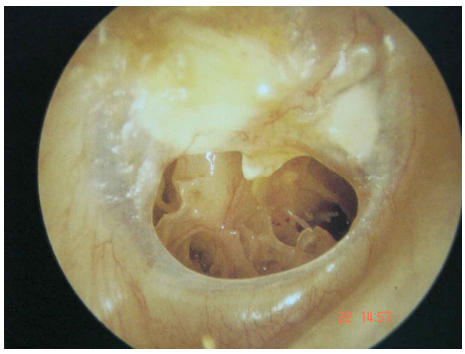


Plate No.7 : Inactive right CSOM. Permanent defect in pars tensa. Normal middle ear mucosa.

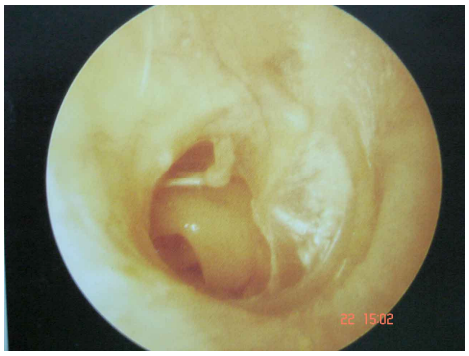


Plate No.8 : Inactive right CSOM. Intact long process of incus can be seen.

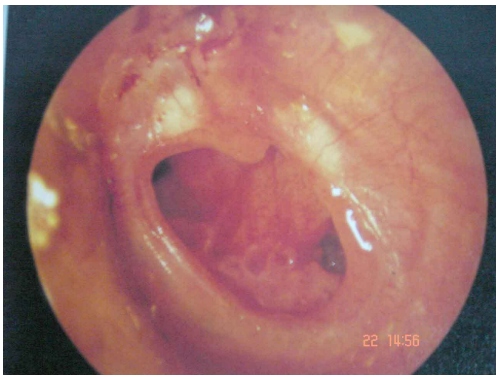


Plate No.9 : Active left CSOM Middle ear mucosa inflammed, edematons.

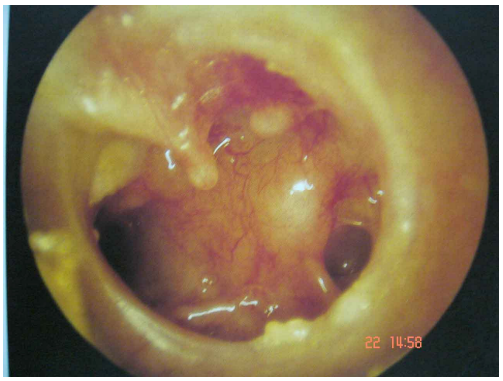


Plate No.10 : Active left CSOM Stapes can be seen through perforation. Long process of incus eroded.

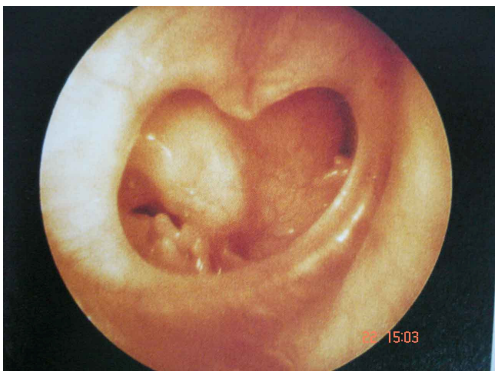


Plate No.11 : Right CSOM with eroded handle of malleus.

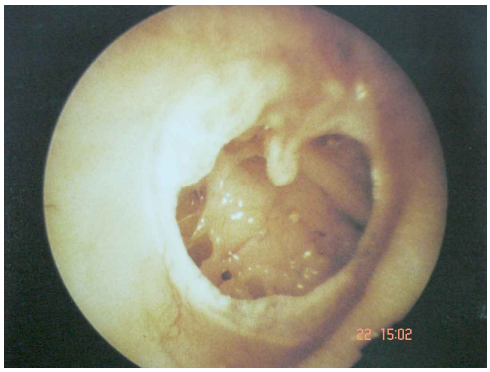


Plate No.12 : Right CSOM. Sub total perforation

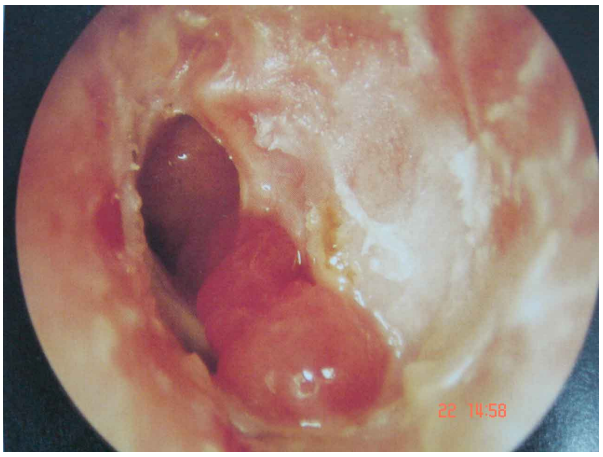


Plate No.13 : Active left CSOM. Anterior pars tensa defect with middle ear mucosa hypertropied forming polyps.

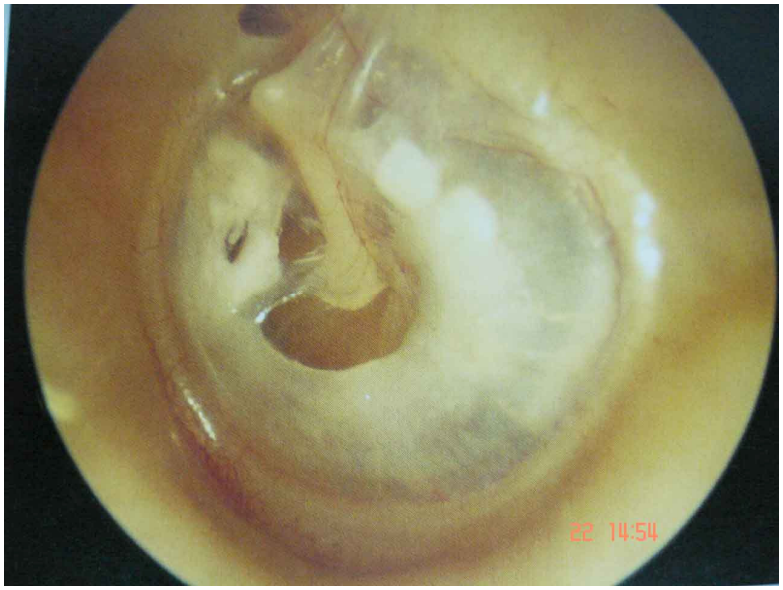


Plate No.14 : Scarred pars tensa associated with left healed otitis media.

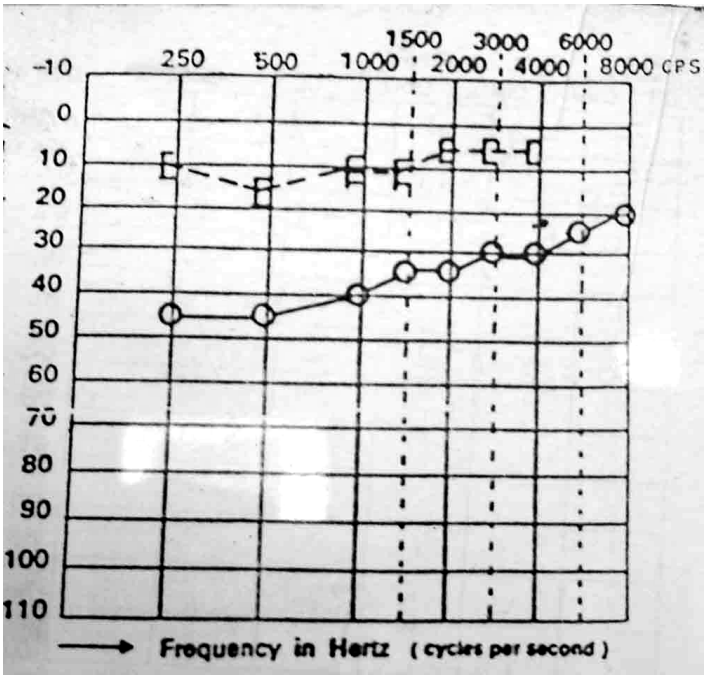


Plate No.15 : Pure tone audiogram - showing mild conductive hearing loss right ear.

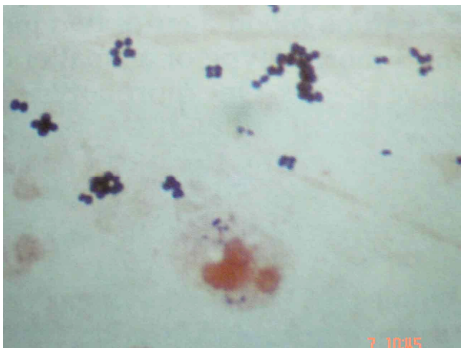


Plate No.16 : Gram stained smear showing gram positive cocci in pairs

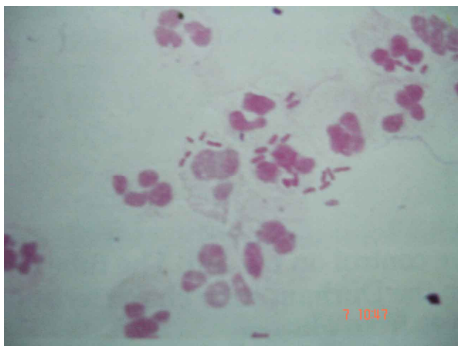


Plate No.17 : Gram stained smear showing gram negative bacilli amidst of pus cells and epithelial cells.

Plate No.18 : Pseudomonas aeruginosa colonies on Mac Conkey agar.

Plate No.19 : Aspergillus flavus colonies on sabouraud's agar

Discussion

DISCUSSION

Out of the 100 cases studied, age distribution was such that 58% of cases in 15-30 years, 34% in 31-45 years, 6% in 46-60 years, and 2 cases above 60 years. An apparent decrease in incidence as age advances can be noted. According to western literature population prevalence of adult otitis media does not vary with age.[7] A study from Benghazi, Libiya[23] report age distribution among as 0-15 year- 44.17%; 15-20 yrs-25%; 3-45 years- 19.17%; 46-60 years- 9.16%; 60 and above 2.5%. The difference from existing literature in this study may be attributed to selection criteria for sample cases, as pediatric age group, patients with diabetes mellitus, associated frank otitis externa were excluded from study. Also, present study being conducted at a tertiary care center, referral patterns may also influence outcome. Large scale population studies only can accurately delinate incidence and age distributions, particularly for widely prevalent condition as CSOM.

Gender distribution shows 1:1 male/ female ratio (48% and 52% respectively). It was similar to earlier studies. Obviously CSOM has no sex preponderance.

Duration of symptoms was more than 2 years in majority of patients (77%). An Indian study[42] states 64.7% had history of symptoms more than one year duration. This typical long standing history of

otorrhea has to be seen in accordance with poor living conditions, over crowding, nutritional deficiencies and limited access to medical care existing in developing countries all contributing to perpetuation of disease process.

Occurrence of unilateral and bilateral disease was in the ratio of 3: 1(79% and 21% respectively). ***Saini S et al***[30] (2005) reported similar figures. (75% and 25% respectively). ***Urmil Mohan***[42] (1998) in his study at Amritsar observed 86% unilateral and 14% bilateral disease.

On analyzing symptomatology, obviously all patients presented with otorrhea. Hard of hearing was next predominant symptom (28%) Itching was present in 16% cases. Tinnitus in 17%,pain was the least mentioned complaint (7 and 4% respectively). ***B. N. Rao***[23] (1994) reported pain as next common symptom to otorrhea (15%) followed by itching, hearing loss and tinnitus 97.5%, 5.8 and 0.8% respectively.

Apparent difference may be due to different case selection criteria, as for present study small pin hole perforations, pediatric age group, associated frank otitis externa, and suspected complications of CSOM were excluded.

Condition of middle ear mucosa as seen on otoscopy was 54% pale and edematous, 39% hyperemic, and 7% granular mucosa. Similar findings were observed by ***Jaya C et al***[60]

On studying audiometry findings, pure tone average was around 40. Bone conduction at 4 KH_z as measure of sensory neural component involvement showed no significant sensory neural hearing loss. These levels did not vary among age groups. Most of the literature shows there is correlation between sensory neural hearing loss and chronic ear disease. [65,66,67].

Dumich and ***Harner***(1983)[68] failed to demonstrate any evidence of sensory neural hearing loss in their series of 200 patients.

Analysis of culture results showed monomicrobial infection in 52% (Pure bacterial 50%, pure fungal 2%) polymicrobial (Mixed bacteria 10%, Bacterial and Fungal 28%) 38% and Sterile culture on 10% ***R.D. Kulakerni et al*** (1993)[24] in their study reported 62.5%, 7.5% and 7.5% respectively. As per ***Urmil Mohan*** (1998)[42], it was 60.4%, 6.1%, 25.8%. ***Sen Gupta et al*** (1978)[39] observed 66.4%, 13.6% and 8.6%, while in ***B.N. Rao*** series[23] 68.5% were monomicrobial, 32.4% were polymicrobial and 10% were sterile cultures. The difference could be due to difference in patient population studied and geographic variations.

Most common organism isolated in present study was pseudomonas aeruginosa (38%) followed by staphylococcus aureus (22%), Klebsiella (13%), Aceinetobacter (11%), coagulase negative

staphylococci (7%), proteus species (5%) and escherechia coli (2%). Other studies which observed pseudomonas predomiancne was by ***Fliss DM***[25] (1992) (Israel), ***Ashok Mittal*** (1997-Chaudigarh)[32], ***Attalah MS*** (2001-Saudi Arabia)[27] and ***Aslam MA*** (2004-Rawalpindi)[29] Studies by ***B.N. Rao*** (1994-Libiya)[23] and ***Obi CL.*** (1995-Nigeria)[22] showed staphylococcal predominance. ***R. Induharan et al*** (Malyasia)[28] reported almost equal incidence of pseudomonas (27.2%) and staphylococcus aureus (23.6%).

A remarkable feature of this study in comparison to studies done else where is the increased isolation of Klebsiella (13%) and aceinetobacter (11%). Except for ***Fliss DM***[25] (8%) all others report only 2-5% isolation of Klebsiella. They all had higher occurrence of proteus upto 15%.

In literature, there is reported proven association between climate and bacteriology of CSOM. ***Yildirim A et al*** (2005)[69]. Observed that when weather is warmed, frequency of isolation of enteric bacteria were increased significantly. So, the higher incidence of Klebsiella in this study may be positively correlated with comparatively hot humid climate of the particular geographic area (Chennai) where the study was conducted.

Reported prevalence of aceinetobacter is 4.8% (***Ashok Mittal*** 1997)[32], 1.37% (***Beena Antony et al*** 1996)[37] to none. ***Dadswell J.U.***[70] studied on acenetobacter and similar organisms in ear infections. His observation was that although these organisms were considered less virulent forms few strains can be pathogenic especially once they acquire multiple drug resistance. They are ubiquitous in nature, found commonly in contaminated water sources. Higher isolation rates in this study may again be attributed to geographical variations and different line of antimicrobials routinely used in different parts.

Coagulase negative staphylococci was isolated in 7% cases. Even though they are generally considered non-pathogenic, their association in CSOM cases can be attributed to extreme lowering of resistance in middle ear due to invasion by other organisms. Under these circumstances, they assume pathogenic role either singly or more often in combination with other organism[32].

Sterile culture was observed in 10 swabs.

This can be explained in two ways[32]

1. Middle war exudates fail to yield positive results due to presence of strictly anaerobic bacteria.

2. Presence of viral agents, particularly when the infection was caused by respiratory syncitial virus, influenza virus or adenovirus.

Fungal isolates were obtained in 30% cases, 28%, along with bacteria and 2% fungi alone. Reported incidence of secondary fungal inspection in other studies are ***Sen Gupta et al***[39] 25%., ***Dincer A.D. et a***l[21]. 28.6%, ***B.N. Rao et al***[23] 7.5%. ***Ashok Mittal et al***[32] 40.8%; ***Ibeukwa A.O***[41] 25%; ***Urmil Mohan***[42] -13.7%; and ***Khanna V***[26]-9%. The availability and use of broad spectrum antibiotics with or with out steroids in period before consultation was probably responsible for apparently higher incidence of fungal isolates in the present study. A retrospective analysis of ***Nakagawa et al***[45] 1994 noted frequency of isolation of fungi, anaerobic bacteria, and glucose non-fermenting gram negative rods were gradually increasing while incidence of proteus infections gradually declined over 16 years period (1976-1991). Also ***Martin TJ*** (2005)[44] reports fungal infection are on increasing trend after the period of wide spread use of fluroquinolone antibiotics.

Distribution of fungal species showed Aspigillus flavus (40%), Aspergillus niger (20%) Candida (20%), Aspergillus terreus (6.6%) Cladosporium species (3.3%). Simialr pattern was observed in three other studies[26,32,39]. But ***B.N. Rao***[23] and ***Urmil Mohan***[42] reported Candida as most common species.

Analysis of antibiogram showed majority of pseudomonas, staphylococcus aureus, and Klebsiella strains are gaining resistance to commonly used antibiotics as gentamicin, cefotaxime and ciprofloxacin. They still have marked sensitivities (over 90% of isolates) to amikacin, Ofloxacin with around 100% sensitivity to cefoperazone sulbactam. Aceinetobacter strains showed multi drug resistant pattern of commonly used antibiotics as gentamicin, Amikacin, ciprofloxacin, cefotaxime. They were markedly sensitivie to ofloxacin.

Coagulase negative staphylocci and proteus strains were markedly sensitive to all antibiotics except cefotaxime.

This is in accordance with recent literature showing high efficacy of fluorquinolone antibiotics in treatment of CSOM, especially ofloxacin.[50,51,52,53,54,56]

Unfortunately for pediatric age group our choice is limited to either ototopic fluroquinolones or parentral broad spectrum pencillins or aminoglycosides. Pattern of increasing resistance to cefotaxime and gentamicin make situation complex. Cefoperazone sulbactam can be considered as a reserve drug in difficult clinical situations, as it attains almost 100% sensitivity to all isolates.

Summary and Conclusion

SUMMARY AND CONCLUSIONS

1. Gender distribution of CSOM shows no male : female preponderance.

2. Duration of symptoms in more than 77% patients were more than 2 years.

3. Majority of cases were unilateral (79%)

4. All patients with otorrhea, next common symptom being hard of hearing.

5. Mean pure tone average, was 40, No significant sensory neural hearing loss detected among the 100 cases.

6. Pseudomonas aeruginosa was the most common isolate. Klebsiella and aceineto bacter showed an increased incidence of 13% & 11% in the this study.

7. 30% cases showed positive results with fungal culture. i.e one in 3 patients may have superimposed fungal infections in active chronic suppurative otitis media.

8. Most common fungal isolate was Aspergillus flavus.

9. Antibiotic with maximum sensitivity to all isolates was cefoperazone –sulbactam. Ofloxain and amikacin also showed remarkable sensitivities . (more than 90%) to all isolates. Most of isolate found to be resistant to commonly used antibiotics as cefotaxime, gentamicin.

Limitation of Study

LIMITATIONS OF STUDY

Despite sincere efforts to minimise pitfalls and draw backs the present study has following has following handicaps.

1. Ideal recommendation for assessing tubotympanic disease and culture sample collection is examination under operating microscope, pus being collected by suction aspiration from middle ear. It could not be adhered because of obvious practical difficulties.

2. Ideally, culture swab should be taken before starting antibiotics. It was not possible, as study was conducted at a tertiary care referral center, where most of patients already had treatment with broad spectrum antibiotics before consultation, details of which was difficult to collect.

3. Anaerobic culture was not done due to technical and financial constraints.

4. Though sample size was 100, for assessing antibiotic sensitivity to each organism separately, number of cases in subclass was too small eg:- Total number of proteus isolates among hundred, cases was five. Percentage antibiotic sensitivity pattern of proteus was calculated among the five isolates which is statistically insignificant. A prospective study with large sample (eg. as around 100 isolates of proteus alone) has its own practical constraints with respect to duration.

Recommendations

Recommendations

RECOMMENDATIONS

1. As many bacterial strains implicated in chronic suppurative otitis media are gaining resistance to commonly used antibiotics as cefotaxime, gentamicin and even to ciprofloxacin, culture directed antibiotic therapy is superior and advisable to empirical broad spectrum antibiotic therapy.

2. In chronic suppurative otitis media 1 in 3 patients can have super added fungal infections. So, it is worth while to look for fungi in all cases of intractable prolonged otorrhea, not responding to antibiotics.

3. Importance of dry mopping in treatment of CSOM is stressed. Dry mopping along with culture directed antibiotic for optimum duration, with regular assessment of clinical response to treatment is advisable.

Indiscriminate use of broad spectrum over-the counter (OTC) ototopical preparations with or without steroids for prolonged period is to be avoided.

Annexures

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PROFORMA

1. Name 2. Age 3. Sex

4. Hospital No. 5. Study No.

6. Presenting symptoms

(a) Ear Discharge

- Duration (Total)
- Frequency
- Duration of present episode
- Nature of discharge
- Quantity

(b) Hard of hearing (Yes/ No)

(c) Tinnitus (Yes/ No)

(d) Pain (Yes/No)

(e) Itching (Yes/ No)

(f) Others

7. Past medical treatment

(a) History of Rx for present illness.

- Ototopical medications (Yes/ No) - Duration
  - Medication used
- Antibiotic (oral/ inj) (Yes/ No) - Duration
  - Medication used.

8. Past surgical history

9. Family history

10. Personal history Smoking (Yes/ No)

Over crowding (Yes/ No)

History of self cleaning ear (Yes/ No)

History of dip baths (Yes/ No)

11. Socioeconomic status

12. ENT examination - Ear

a. External canal – Normal/ inflamed/ excoriations

b. Discharge- Mucoid/ Mucopurulent / purulent.

Pulsatile/ Non-pulsatile

Profuse/ Scanty

b. Perforation -Site

Size – Medium ( ) Large ( ) Subtotal ( )

c. Remnant TM – Normal/ Congested/ Tympanosclerotic patches.

d. Middle ear mucosa – Hyperemic ( ) pale edematous ( ) granular

e. Other findings

Nose: positive findings if any

Throat : positive findings if any

Final Diagnosis Audiogram PTA:

Bone conduction at 4 KH_z

Ear swab pus c/s Dated:

Results

Gram staining

Bacterial culture and sensitivity

KOH Smear:

Fungal Culture:

MASTER CHARTS

Sl. No.	Name	Age	Sex	Presenting Symptom	Duration	U/L or B/L	Middle Ear Mucosa	PTA	Bone con at 4KHz	Bacteria	Fungus	Antibiotic	
												Sensitive	Resistant
1.	Chellam	27	F	O,H	>2	B	P	36	15	St	-	G,A,O, Cef, CS	Cip
2.	Mariammal	52	F	O	>2	U	H	38	18	St	-	G,A,O,Cef, CS	Cip
3.	Rajagopal	41	M	O	>2	U	P	40	20	Cons	-	G,A,Cip,O,Cef, CS	
4.	Daniel	28	M	O,I	1	U	P	40	18	A,K	AFU	K-G, A, Cip, O, Cef, CS A - A,O,CS	A-G, Cip, Cef
5.	Gajapathy	37	M	O	>2	U	P	38	18	A,K	ANi	K - G,A,Cip, O,CS A - A.O,CS	A-G, Cip, Cef
6.	Bakya	32	F	O,H,T,I,P	>2	B	H	38	21	P	AFa	G,A,Cip,O,Cef, CS	Cef
7.	Arivudia Nambi	45	M	O,H,I	8/12	B	G	44	21	Cons	AFa	G,A, Cip, O, Cef, CS	
8.	Parvathi	40	F	O	1½	U	P	40	20	P	AFa	G,A,Cip, O, CS	Cef
9.	Thajuden	39	M	O	>2	U	H	42	20	P	AFa	G,A,Cip, Cef, CS	
10.	Venu	29	M	O.H.I	>2	U	H	30	20	P	Ca	A, Cip, O, Cs	G, Cef

Sl. No.	Name	Age	Sex	Presenting Symptom	Duration	U/L or B/L	Middle Ear Mucosa	PTA	Bone con at 4KHz	Bacteria	Fungus	Antibiotic	
												Sensitive	Resistant
11.	Bagaraman	55	M	O,H	>2	U	P	42	22	St	Ate	G,A,Cef, CS	Cip
12.	Saraswathy	40	F	O	>2	U	H	38	22	E	-	G,A,Cip, O, CS	Cef
13.	Jayamma	40	F	O	>2	U	H	40	18	E	-	C,S	G,A,O,Cip, Cef
14.	Kumar	27	M	O	1½	U	H	32	18	Cons	-	G,A,O,Cip, Cef, CS	
15.	Rajesh	25	M	O,I	>2	U	P	36	16	St	Ani	A,O,Cip, Cef, CS	G
16.	Resmi	30	F	O,H,I	1	U	P	39	20	P,K	-	G,A,O, Cip, Cef,CS	
17.	Shanmugam	20	M	O.I	>2	U	H	35	18	A	AFa	A,O,Cip, CS	G,Cef
18.	Thyagu	20	M	O,H,T	>2	U	P	40	18	Pro.	AFa	G, A,O, Cip,CS	Cef
19.	Shanthi	29	F	O	>2	U	H	36	16	St.	-	G,A,O,Cip, CS	Cef
20.	Philomina	29	F	O	>2	U	H	32	15	Cons	-	G,A,O,Cip, Cef, CS	
21.	Poovarasan	15	M	O	4/12	U	P	33	20	P	Clad	G,A,O, Cef, CS	Cip
22.	Divya	15	F	O,H,T	6/12	U	P	40	21	P	ANi	G,O, Cip,Cef, CS	A

Sl. No.	Name	Age	Sex	Presenting Symptom	Duration	U/L or B/L	Middle Ear Mucosa	PTA	Bone con at 4KHz	Bacteria	Fungus	Antibiotic	
												Sensitive	Resistant
23.	Vasu	40	M	O,H,T	1	B	P	42	20	St, K	AFa	Cip, O,Cef, CS	G
24.	Umapathy	24	M	O	>2	U	H	38	18	A	-	G,A,Cip, O, CS	G,A, Cef
25.	Devi	30	F	O,H	1½	U	G	39	19	St	-	G,A,Cip, O, CS	Cef
26.	Maheswari	25	F	O	>2	B	H	40	21	P,St	-	G,A,Cip, O, CS	Cef
27.	Dhanalakshmi	19	F	O	>2	U	H	32	15	P	-	G,A,Cip, O, CS	G,Cef
28.	Amudha	28	F	O	>2	U	H	30	15	St		G,A,Cip, O, CS	Cef
29.	Saravanan	20	M	O	>2	U	P	36	20	St		G,A, Cip, O, CS	Cef
30.	Antony	18	M	O	1	U	G	35	18	Cons.		G,A,Cip, O, CS	Cef
31.	Anushya	29	F	O	>2	U	P	34	17	Cons.	-	G,A,Cip, O, CS	Cef
32.	Veketaramani	38	F	O.H.T.I,P	>2	B	P	40	20	K	C	A,Cef, O,CS	G, Cip
33.	Vijayaraj	38	M	O,I,T	>2	U	H	36	22	-	C		
34.	Kantha	65	F	O	1½	U	H	44	22	P,K	C	A,Cip, O,CS	Cef, G

Sl. No.	Name	Age	Sex	Presenting Symptom	Duration	U/L or B/L	Middle Ear Mucosa	PTA	Bone con at 4KHz	Bacteria	Fungus	Antibiotic	
												Sensitive	Resistant
35.	Beegam Bee	56	F	O,H,T	>2	U	P	40	20	Pro.K	AFa	A, Cip, O, CS	Cef, G
36.	Pankaraj	27	M	O	1	U	P	38	28	A	-	Cip, O, CS	G,A, Cef
37.	Sujatha	26	F	O	>2	U	H	36	20	K	-	G,A, Cip, CS	
38.	Mallika	45	F	O	>2	U	H	44	18	A	-	Cip, O, Cef, CS	G,A, Cef
39.	Rajamuarugan	25	M	O	>2	U	H	36	16	Pro.	-	G,A, Cip, O, Cef, CS	
40.	Srinviasan	27	M	O.H,T	>2	B	H	36	15	P	-	-	G,Cip, O, Cef, CS
41.	Poomadevi	30	F	O	1	U	P	35	15	St.	-	Cip, O, Cef, CS	G,A
42.	Deepa	20	F	O,I	>2	U	H	32	18	P	C	G,O, Cef, CS	A,Cip
43.	Kumari	35	F	O	1	U	H	34	20	-	-		
44.	Lokanathan	36	M	O	>2	U	P	38	18	-	-	G,A,Cip, O, CS	Cef
45.	Sumathi	29	F	O	>2	U	P	40	20	St.	-	G,A,Cip, O, CS	Cef
46.	Athiya	35	F	O	>2	U	P	38	22	-	-	G,A,Cip, O, CS	Cef

Sl. No.	Name	Age	Sex	Presenting Symptom	Duration	U/L or B/L	Middle Ear Mucosa	PTA	Bone con at 4KHz	Bacteria	Fungus	Antibiotic	
												Sensitive	Resistant
47.	Rajamurugan	18	M	O,I,P	1½	U	P	30	20	K	C	A, Cip,O,Cef, CS	G
48.	Damodharan	45	M	O	>2	U	H	40	20	P	-	G,A,O,Cef,CS	Cip
49.	Antony	37	M	O,H,T	8/12	B	H	44	22	K	-	G,A,Cip,Cef,CS	O
50.	Kousalya	24	F	O	>2	B	H	34	21	St, Pro.	-	G,A,Cip,O,CS	Cef
51.	Rajaraman	45	M	0,H	1	U	H	44	18	Cons.	-	G,A,O,Cef,CS	Cip
52.	Manikandan	15	M	0	>2	B	P	34	18	P	-	G,A,Cip,O,Cef,CS	
53.	Ramalingam	50	M	0,T	>2	U	H	44	22	P	-	G,A,Cip,O,Cef,CS	
54.	Kasi	58	M	O	1½	U	P	46	24	-	-		
55.	Rajendran	43	M	O,T	>2	B	P	36	22	A	-	A,Cef,CS	G,Cip,O
56.	Lalitha	28	F	O	>2	U	H	38	21	P	AFa	G,A,Cip,O,Cef,CS	
57.	Shanthammal	62	F	O.H,T	1	U	P	42	22	P	-	G,A,Cip,O,Cef,CS	
58.	Ramesh	31	M	O	>2	U	P	36	20	P	AFa	G,A,Cip,O,Cef,CS	Cef

Sl. No.	Name	Age	Sex	Presenting Symptom	Duration	U/L or B/L	Middle Ear Mucosa	PTA	Bone con at 4KHz	Bacteria	Fungus	Antibiotic	
												Sensitive	Resistant
59.	Abirami	47	F	O.H,T	>2	B	P	40	18	-	-	G,A,Cip,O,Cef,CS	
60.	Rajendran	40	M	O	>2	U	H	40	22	-	-	A,Cip,O,CS	Cef,G
61.	Dharani	25	F	O	>2	U	H	35	20	P	-	A,Cip,O,G,Cef,CS	G
62.	Ravi	25	M	O	>2	U	P	34	20	St	-	A,O,Cip,CS	Cip
63.	Veketawaran	29	M	O	>2	U	H	40	18	P	-	G,A,O,Cip,CS	G,Cef
64.	Padma	28	F	O,H,T	1	U	G	42	18	A	-	A,Cip,O,Cef,CS	Cef
65.	Parvathi	39	F	O	>2	U	P	40	20	P	-	G,A,O,Cip,CS	G
66.	Arunkumar	40	M	O	>2	U	H	40	20	P	Afa	A,Cip,O,Cef,CS	G,Cef
67.	Sengiah	41	M	O,H,T,P	>2	B	H	44	18	St	-	A,Cip,O,CS	Cef
68.	Karthikeyan	17	M	O	1	U	G	30	15	K	-	G,A,Cip,O,CS	Cef
69.	Kavitha	18	F	O	>2	U	H	31	15	P	-	G,A,Cip,O,CS	Cef
70.	Kuberan	34	M	O,H,T	>2	B	H	42	22	-	-	G,A,Cip,O,CS	

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												Sensitive	Resistant
71.	Sekhar	33	M	O.H	>2	B	H	44	22	P,K	-	G,A,O,CS	Cef
72.	Vesanthi	18	F	O	>2	U	H	42	21	P	-	G,A,O,CS	Cip,Cef
73.	Singaram	34	M	O	>2	U	P	36	20	St	-	G,A,Cip,O,CS	Cef
74.	Veketeswaran	34	M	O	>2	U	P	38	20	St	-	A,Cip,O,Cef,CS	G
75.	Kuppan	25	M	O,T	>2	U	H	28	18	P	-	G,A,Cip,O,CS	Cef
76.	Saravanan	28	M	O,I	1	U	G	30	15	P	AFa	G,A,Cip,O,CS	Cef
77.	Lingam	33	M	O	>2	U	P	34	22	A	-	A,Cip,O,CS	G,Cef
78.	Suban	29	M	O,H	>2	B	H	36	20	P	-	G,A,Cip,O,CS	Cef
79.	Indira	30	F	O	>2	U	H	40	18	St	-	G,A,O,Cef,CS	Cip
80.	Jayasree	18	F	O	>2	U	H	36	18	St	-	G,A,Cip,O,CS	Cip
81.	Sasikala	30	F	O	>2	U	H	34	15	P	-	A,Cip,O,CS	Gen,Cef
82.	Prema	29	F	O,H,I	>2	B	H	40	20	-	AFu		

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												Sensitive	Resistant
83.	Muthumari	23	F	O	>2	U	P	32	20	P	-	A,Cip,O,CS	G,Cef
84.	Deepika	24	F	O	>2	U	P	38	18	-	-		
85.	Soundeswari	38	F	O	>2	U	H	40	20	St	ANi	A,Cip,O,Cef,CS	G
86.	Sasikumar	28	M	O	>2	U	H	40	20	P	-	A,O,CS	Cef,G,Cip
87.	Sivakumar	32	M	O,I	>2	B	H	42	22	St		A,Cip,O,Cef,CS	G
88.	Rasi	28	M	O,H	>2	U	P	32	21	St	Ate	A,Cip,O,Cef,CS	G
89.	Velankanio	30	F	O	>2	U	P	34	18	P	-	A,O,CS	Cef,G,Cip
90.	Buvaneswari	18	F	0,H	>2	B	P	36	18	K	-	G,A,Cip,O,CS	Cef
91.	Laksmi	30	F	O	>2	U	H	32	15	P	-	GA,Cip,O,CS	Cef
92.	Yamuna	18	F	O,H	>2	B	H	34	16	St	-	A,Cip,O,Cef,CS	G
93.	Bagya	17	F	O,I	>2	U	H	40	18	P	AFu	A,Cip,O,Cef,CS	G,Cef
94.	Mohammed	15	M	O.H	>2	B	H	42	20	-	-		

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												Sensitive	Resistant
95.	Shanthakumar	34	F	O	>2	U	H	40	20	P	A Ni	A,Cip,O,G,CS	G,Cef
96.	Priyadarshini	16	F	O	1	U	G	40	18	P,A	-	G,A,Cip,O,CS	A,Cef
97.	Kumari	30	F	O	>2	U	P	38	16	Pro	-	G,A,Cip,O,CS	Cef
98.	Kamala	28	F	O.H,I	>2	U	P	40	16	P	A Ni	A,O,CS	G,Cef,Cip
99.	Amudhavalli	33	F	O	>2	U	H	42	22	K,A	-	G,A,Cip,O,CS	Cef
100.	Thahira	32	F	O	>2	U	H	36	22	-	-		