STUDY OF BACTERIAL FLORA IN EXTERNAL AUDITORY CANAL AND MIDDLE EAR IN CHRONIC OTITIS MEDIA, TUBOTYMPANIC TYPE PATIENTS

Submitted by

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IN

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Under the guidance of

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Date: **Dr. SAI SUSHMA GADI.**

Place: Vijayapura.

LIST OF ABBREVATIONS

CSOM	Chronic Suppurative Otitis Media
TM	Tympanic Membrane
ASOM	Acute Suppurative Otitis Media
COM	Chronic Otitis Media
AOM	Acute Otitis Media
EAC	External Auditory Canal
HRCT	High Resolution Computed Tomography
PTA	Pure Tone Audiometry
MRSA	Methicillin-resistant Staphylococcus aureus
MSSA	Methicillin-sensitive Staphylococcus aureus
NP	Nasopharynx
MEE	Middle ear effusion
MDR	Multidrug resistant

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ABSTRACT

BACKGROUND:

Chronic suppurative otitis media (CSOM) is typified by persistent inflammation of the mastoid and middle ear mucosa, which results in ear drainage and TM perforation[1].

Geographical variation and patient demographic variables have been influencing the antibiotic resistance pattern of CSOM. Both bacterial isolate resistance and the excessive and illogical usage of broad spectrum antibiotics have grown widespread[1]. Staphylococcus aureus (both methicillin-resistant [MRSA] and methicillin-sensitive [MSSA]), Pseudomonas, Proteus, Coagulase-negative staphylococci, Enterococcus, and Anaerobes are among the frequently isolated bacteria[1].

AIMS AND OBJECTIVES:

1. To ascertain the aerobic bacterial isolates' microbial diversity in individuals with tubotympanic-type chronic otitis media.
2. To determine the antimicrobial susceptibility and resistance to antibiotics thereby helping in earlier management of the disease.

METHODOLOGY:

SOURCE OF DATA

This prospective study is being undertaken in the Department of Otorhinolaryngology at SHRI B M PATIL MEDICAL COLLEGE AND RESEARCH CENTRE AND HOSPITAL [BLDE (DU) UNIVERSITY] ,Vijayapura from April 2023 to January 2025.

METHOD OF COLLECTION OF DATA

- Preoperative examination of the patient including complete clinical history.
- Characteristics of patients including age, gender, residence.
- The patient is thoroughly examined, with a focus on otoscopic findings to

assess the condition of the tympanic membrane and to examine the oropharynx, nose, and throat.

- Patient will be subjected to investigations such as urine routine, blood routine examinations and aerobic culture of the discharge from both the external auditory canal and middle ear (under microscope).
- Sample from the external auditory canal is taken out with the help of Cotton Swab.
- Then, external auditory canal is cleaned using alcohol and normal saline and allowed to dry.
- Mucus extractor used to collect sample from middle ear.
- Both the samples are sent for aerobic culture and sensitivity.

RESULT:

Out of the 71 total swabs collected from external ear in our study, Staphylococcus aureus showed 100% sensitivity to Gentamycin and Linezolid, 95.5% to Clindamycin, 7.7% to Ciprofloxacin, and an overall sensitivity of 49.3% to the antibiotics used in this hospital. Pseudomonas aeruginosa showed 66.7% Amikacin and Colistin, 57.1% sensitivity to Meropenem, 50% to Cefepime, 38.5% to Ciprofloxacin and an overall sensitivity of 25.4% to the antibiotics used in this hospital.

Out of the total swabs collected from middle ear in our study, Staphylococcus aureus showed 100% sensitivity to Linezolid and Clindamycin and an overall sensitivity of 19.7% to the antibiotics used in this hospital. Pseudomonas aeruginosa showed 100% Amikacin and Colistin, 86.2% to Ciprofloxacin, 52.6% to Meropenem, 50% to Cefepime and an overall sensitivity of 59.2% to the antibiotics used in this hospital.

CONCLUSION:

Knowing the local susceptibility pattern of the causative agents is essential for treating the infection effectively and for developing antibiotic policy, as the susceptibility pattern of the pathogenic microorganisms is changing since antibiotics are widely used and readily available.

INTRODUCTION

Chronic inflammation of the middle ear, eustachian tube, and mastoid cavity is known as chronic otitis media (COM), and it can manifest as decreased hearing and recurrent ear discharge.

Based on whether the illness process affects the pars tensa or pars flaccida of the tympanic membrane (TM), chronic otitis media is typically divided into two types: tubotympanic and attico-antral.

Since there are no significant complications, tubotympanic is referred to as a safe or benign kind, whereas attico-antral is referred to as an unsafe or dangerous type due to related complications.

Out of all the side effects, hearing loss linked to persistent ear discharge is almost always noticeable. It is typically more severe than those linked to other forms of otitis media.

Antibiotics provided physicians with a tool to use even in the absence of a proper etiological diagnosis, and its irrational usage resulted in the rise of bacterial strains that were resistant to several drugs and the development of illness complications.

AIMS AND OBJECTIVES

1. To ascertain the aerobic bacterial isolates' microbial diversity in individuals with tubotympanic-type chronic otitis media.
2. To determine the antimicrobial susceptibility and resistance to antibiotics thereby helping in earlier management of the disease.

REVIEW OF LITERATURE

In 2013, Prakash R *et.al* conducted a study in Uttarakhand which showed Pseudomonas aeruginosa (19.89%) and Staphylococcus aureus (48.69%) were the most frequently isolated causal organisms out of the 191 aerobic isolates[2]. They came to the conclusion that efficient treatment, preventing complications and the emergence of antibiotic resistance, and ultimately lowering treatment costs all depend on understanding the etiological agents of CSOM and their susceptibility to antibiotics[2]

In 2022, Surendar Kumar concluded that "CSOM have a major impact on the quality of life of patients with the condition. Klebsiella pneumoniae (24.16%) was the commonest isolate followed by Pseudomonas aeruginosa (18.33%). Pus culture is a good and simple diagnostic tool to study the aetiology due to bacteria in CSOM"[3]

In 2009, in a study conducted by Lars Jonsson et al "Specimens from the external auditory canal (EAC) and the nasopharynx (NP) were obtained in order to correlate the bacterial findings with those of the MEE. Aerobic bacteria were invariably present in the MEE, the predominant aerobic species being *Pseudomonas*, found in 32%. Anaerobic bacteria were found in 45%: most anaerobes (55%) were cocci, 33% were of the *Bacteroides* species. The flora of the MEE correlated well with the bacteriological findings in the EAC cultures. In contrast, most organisms isolated from the NP represented the normal skin flora or were common respiratory pathogens."[4]

In 2013, a study was conducted by Shamweel Ahmad , which showed "major organisms isolated were Methicillin sensitive *Staphylococcus aureus* [MSSA] (45.1%) followed by Pseudomonas aeruginosa (19.5%). The sensitivity of S. aureus (MSSA) was 79.7% to ciprofloxacin, 69% to cotrimoxazole, and 82.5% to gentamicin whereas the sensitivity of *P. aeruginosa* was 100% to ceftazidime,

84.4% to ciprofloxacin, 90.6% to gentamicin, and 78.1% to Piperacillin. The study of microbial pattern and their antibiotic sensitivity determines the prevalent bacterial organisms causing CSOM in the local area to start empirical treatment of otitis media and its complications for a successful outcome, and thus to prevent the emergence of resistant strains."[5]

In 2002, A H C Loy in his study found that the "most common causal organisms isolated were Pseudomonas aeruginosa (33.3%) and Staphylococcus aureus (33.3%) followed by coagulase negative Staphylococcus (21.1%). Fungi accounted for 8.8% of isolates while 6.6% were anaerobes. Of the three antibiotics commonly available as topical eardrops, gentamicin has the highest susceptibility rate (82.6%), followed by neomycin (67.8%) and chloramphenicol (62.8%)"[6].

In 2021, study conducted by Wan Nur Anis Wan Draman MD etal showed "Microbial growth in 85 (93.4%) samples, but 6 (6.6%) samples had no growth. Among the samples with growth, 63 (69.2%) were monomicrobial, 13 (14.3%) were polymicrobial, and 9 (9.9%) were of mixed growth with more than three microorganisms. The most common bacteria isolated was *Pseudomonas aeruginosa* (32.6%) followed by *Staphylococcus aureus* (16.9%) and *Klebsiella* spp. (5.6%). The most sensitive antibiotics against *P aeruginosa* were ceftazidime, meropenem, piperacillin-tazobactam, and cefepime. *S aureus* showed the highest sensitivity toward rifampin, cefoxitin, and fusidic acid"[7].

ANATOMY OF EAR

External ear

Sound is collected, amplified, and sent to the middle ear by the external ear, which is made up of the auricle and external acoustic meatus. (fig.1)

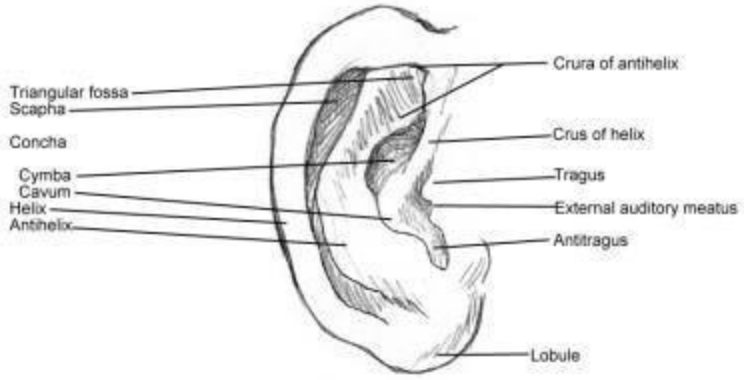

Fig.1 External ear (source Medscape)

Middle ear

Sound travels from the external ear to the fluid of the inner ear through the middle ear. It is an air-filled hollow that resides in the petrous portion of the temporal bone and connects to the nasopharynx via the Eustachian tube. (fig.2)

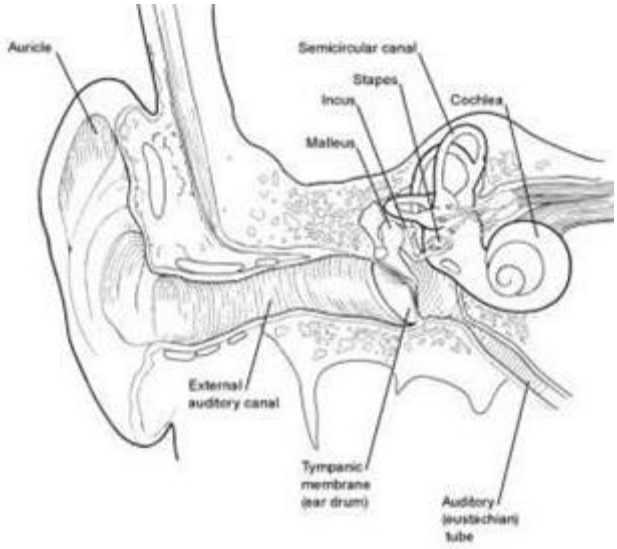

Fig.2 middle ear (Source: Medscape)

The walls of the tympanic cavity are:

- Tympanic membrane forms the lateral wall
- Posteriorly it is connected with mastoid antrum via aditus
- The round window covered by the secondary tympanic membrane is located posteriorly and separated by the promontory, whereas the oval window on the medial wall/labyrinthine wall is covered by the stapes footplate.
- The anterior wall, also known as the carotid wall, is the thin bony plate that divides the carotid canal from the tympanic cavity.
- Tegmen is the roof of tympanic cavity; it separates middle cranial fossa from the epitympanic recess.
- The floor is formed by the thin bony plate that separates internal jugular vein and middle ear.

Tympanic membrane

The external ear is separated from middle ear by a semi-transparent, oval tympanic membrane. It has 2 parts pars tensa where malleus handle is firmly attached to the membrane; where it forms concavity the umbo. Pars flaccida is part of the membrane which lies above lateral process of malleus(fig.3)

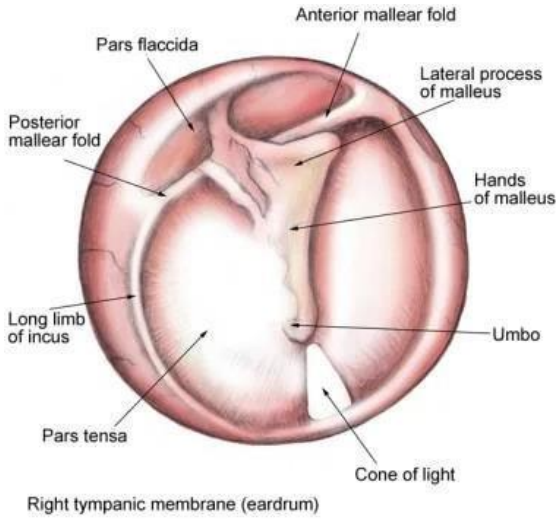

Fig.3 tympanic membrane (Source: Medscape)

The pinna gathers sound waves, which are then conveyed to the movable tympanic membrane and ultimately to the ear ossicles.

The sensory supply of the tympanic membrane is as follows: Auricular branch of vagus nerve (Arnold nerve); tympanic branch of glossopharyngeal nerve (Jacobson nerve); and auriculotemporal nerve (mandibular branch of trigeminal nerve)

The maxillary artery's deep auricular, anterior tympanic, and stylomastoid branches send blood to the posterior auricle. The transverse sinus, dural veins, and external jugular vein are the sources of venous drainage.

The middle ear cavity contains the auditory tube, muscles, and nerves.

Ossicles

The ossicles are 3 in number (fig.4)

- Malleus
- Incus
- Stapes

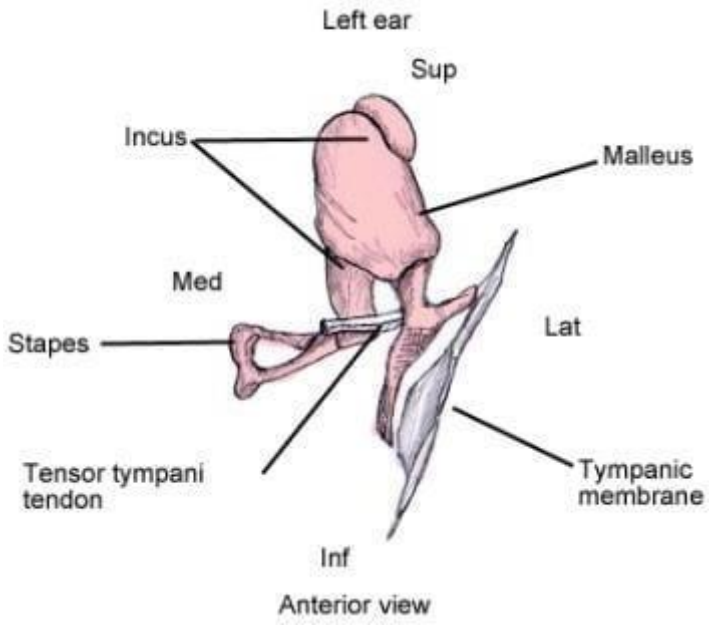

Fig.4 Ossicles (middle ear). (Source: Medscape)

Sound waves are amplified from air by ear ossicles to internal ear(perilymph). Sound waves striking the tympanic membrane cause pressure wave in the inner ear fluid.

Eustachian tube

Middle ear and the nasopharynx are communicating through the Eustachian tube. It equalizes pressure across the tympanic membrane.

Muscles

One of the middle ear's muscles is the stapedius muscle, which connects the posterior tympanum to the stapes neck. The malleus (as well as the tympanic membrane) is displaced medially by the tensor tympani tendon, which is attached to the handle of malleus. This dampens vibrations of sound.

Innervation

The horizontal part of the facial nerve traverses on the medial wall in the facial canal superior to stapes footplate.

The facial nerve branch, chorda tympani supplies the tongue in its anterior 2/3rd part (carries taste sensation) and also sublingual and submandibular salivary glands. The promontory of the medial wall containing tympanic plexus, is contributed by:

- Jacobson nerve (tympanic branch of glossopharyngeal nerve).
- Caroticotympanic nerves (superior and inferior): They are the sympathetic branches of carotid plexus that joins the glossopharyngeal nerve by it’s tympanic branch
- Communication with a branch of greater petrosal nerve

Vascular supply

The arterial supply of middle ear is by tympanic branch of the maxillary artery supplies tympanic membrane by its tympanic branch, the posterior auricular artery supplies mastoid and posterior cavity by its stylomastoid branch, middle meningeal artery (petrosal branch), ascending pharyngeal artery branch, internal carotid artery, and artery of the pterygoid canal and its branch. Venous drainage is formed by Superior petrosal sinus and the pterygoid plexus.

Inner ear

The inner ear, conducts sound to central nervous system, and also assists in balance. Auditory transduction, the conversion of acoustic energy to electrochemical energy, takes place within the inner ear. (fig.5)

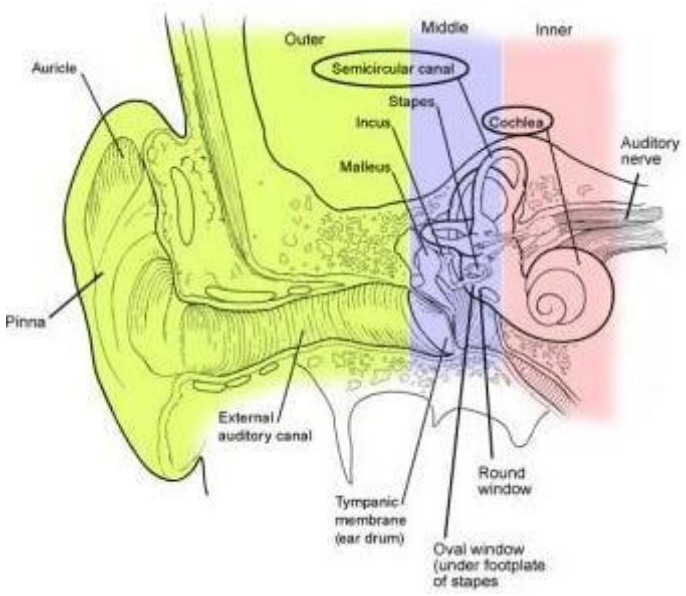

Fig.5 Inner ear in relation to middle and external ear. (Source: Medscape)

Inner ear consists of membranous labyrinth and bony labyrinth.

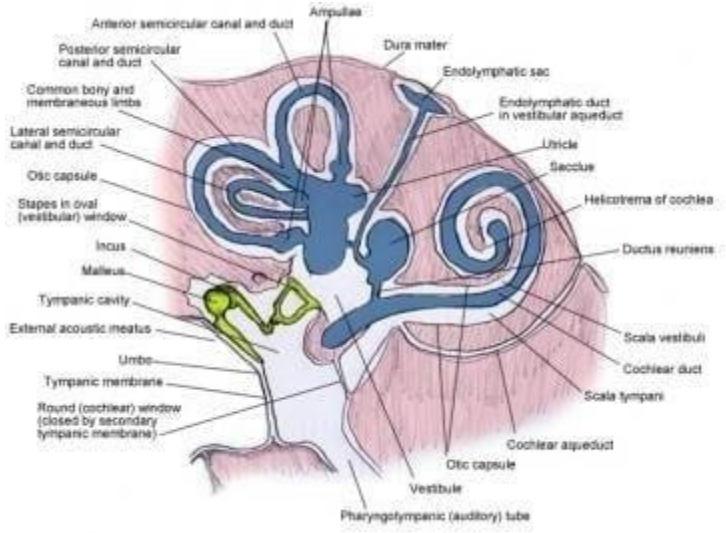

Fig.6 Inner ear: bony and membranous labyrinths. (Source: Medscape)

The bony labyrinth is within petrous part of temporal bone formed of bony cavities.

Membranous labyrinth.

Perilymph surrounds the membranous labyrinth and within it is endolymph. The membranous labyrinth also has cochlear, vestibular, and semicircular components. (fig.6)

Vestibule

The vestibule forms central part of the bony labyrinth (fig.7) The "otolithic organs" sense linear acceleration in the horizontal and vertical planes and play an important role in sensing the direction.

Semicircular and membranous canals

Semicircular canals form bony component of labyrinth and are 3 in number placed at right angles to two other canals. These canals lie above and behind the vestibule (lateral, superior and posterior)

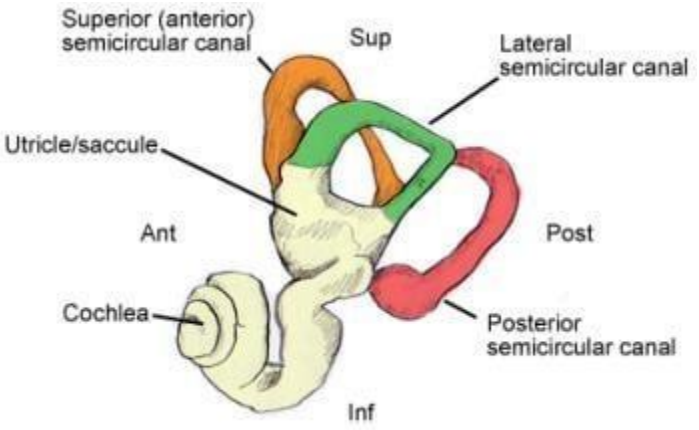

Fig.7 Inner ear. (Source: Medscape)

They play role in balance and angular acceleration detection. Cochlea The inner ear harbors cochlea. The transmission of electrical energy (within endolymph) occurs to the CNS via the cochlear nerve. (fig.8)

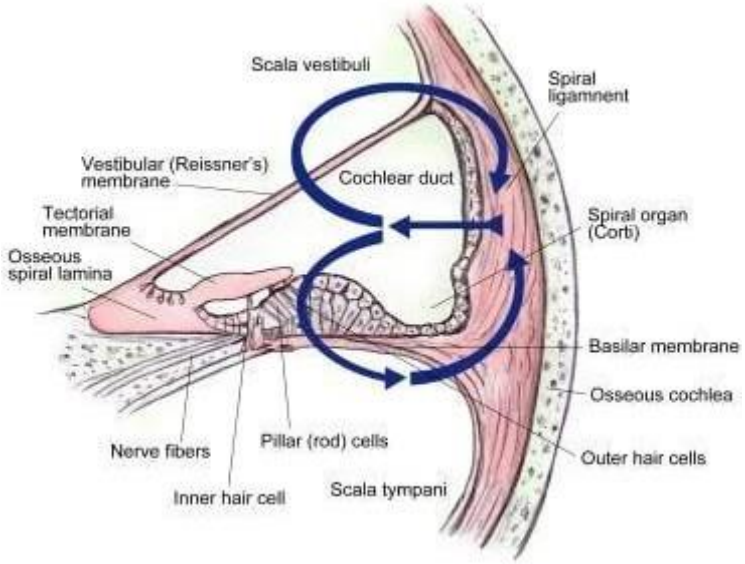

Fig.8 Cross-section of cochlea. (Source: Medscape)

The base of the cochlea is stimulated by higher frequencies and the apex is stimulated by lower frequencies.

CHRONIC SUPPURATIVE OTITIS MEDIA

CSOM has 2 types types:

a) Mucosal type, which is usually characterized by central perforation and affects “anterior part of middle ear cleft” and patients usually are not prone to major complications.

b) Squamosal type and mainly affects posterosuperior part of tympanic cavity and mastoid causing life threatening complications

In developing countries, CSOM is a significant health problem, resulting in severe local damage and life-threatening complications like lateral sinus thrombophlebitis, intracranial abscess, labyrinthitis etc.

Recently, antibiotic overuse has led to critical changes in bacterial strains and their antibiotic response. This has led to complexities in combating the situation.

Thus, updated information on antibiograms and the prevalence of microorganisms in CSOM patients is required to help in the treatment modalities.

Pathways and factors affecting otitis media.

Eustachian Tube Anatomy

By keeping pressure equalization, allowing the drainage of secretions from the middle ear, and impeding the entry of respiratory viruses and germs, a functioning Eustachian tube is crucial for the protection of the middle ear.

The epithelium that lines it serves as the primary barrier against organism invasion and colonisation.

The middle ear infections and inflammation that persist are caused by a combination of variables that are part of the pathogenesis of CSOM.

One of the main factors contributing to CSOM is impaired Eustachian tube function.

Microbiology

The microbiology associated with CSOM involves the presence of various microorganisms, often including fungi, in the middle ear.

Because of its ability to build biofilms and resistance to antibiotics, Pseudomonas aeruginosa is the most prevalent of these bacteria and is considered clinically important. Because it can produce toxic substances that cause tissue damage, Staphylococcus aureus coexists with it in an inflammatory pattern. Furthermore, a prevalent cause that may also result in cases of CSOM is Streptococcus pneumoniae.

The advancement of the illness can also be caused by fungal infections like Aspergillus and Candida, as well as other bacteria like Haemophilus influenzae, Moraxella catarrhalis, Proteus spp., and anaerobic germs like Peptostreptococcus spp. and Prevotella spp. Moreover, anaerobic etiological organisms are the agents of causality.

Genetics and immunology

Genetics and the immune system have a major role in the management of CSOM for protecting against mucosal infections such as CSOM, immunoglobulins IgG, IgA, and secretory IgA are the most efficient. The mucosa of the middle ear cavity produces sIgA locally, which aids in preventing bacterial colonization and attachment.

Complications and sequelae

Serious and sometimes fatal side effects of CSOM include conductive or sensorineural hearing loss, extracranial problems (such as facial paralysis, Subperiosteal abscess, mastoiditis), and intracranial problems (such as meningitis, cerebral abscess). Meningitis and brain abscess due to CSOM can even cause death.

Hearing loss (both conductive and sensorineural) is regarded as the third condition that affects older individuals' that hinders physical and mental well-being in developed countries, following arthropathy and hypertension.

On the other hand, information about these health issues is still scarce for the populations of less developed nations.

In order to lessen health problems and social economic adversity by acting at the appropriate moment, it is necessary to have an accurate understanding of the incidence rate of AOM across populations

ANTIBIOTIC RESISTANCE

Any therapeutic drug's effectiveness is jeopardized by the possibility of tolerance or resistance developing right after administration. It is important to treat viral, bacterial, fungal, and parasitic infections as well as chronic illnesses like diabetes and cancer; it also applies to conditions that affect or are caused by any living thing, including people, animals, fish, plants, insects, and so on. Resistance may be caused by a variety of physiological and biochemical processes. It is impossible to overstate the complexity of the mechanisms underlying the emergence and spread of resistance in the infective agents, and causes paucity of noteworthy progress in the effective prevention and control of resistance development is the lack of fundamental understanding of these subjects.

The late 19th century discovery of these antimicrobial organisms sparked a hunt for suitable prophylactic and therapeutic measures, but it wasn't until the discovery and introduction of antibiotics fifty years later that effective therapy was achieved. The discovery of antibiotics marked a watershed in human history and has transformed medicine in many ways, saving countless lives. Unfortunately, the fast emergence of resistance strains has coincided with the usage of these miracle

medications.

Although the antibiotic penicillin was discovered in 1928, it wasn't until 1949 that Dorothy Crowfoot Hodgkin's X-ray crystallographic research showed the whole structure of this very simple molecule. In 1959, comprehensive synthesis validated the structure. Research on interactions led to the development of the field of chemical biology/genetics.

The history of antibiotics, like any biological study, is full of false assumptions, misreading, incorrect forecasts, and other errors that have occasionally resulted in the truth. The goal of this account is to emphasize the truth. In addition to its influence on the management of infectious diseases, the antibiotic discovery is rightfully important in present era. Numerous other therapeutic uses of "antibiotics" as antiviral, antitumor, or anticancer drugs have resulted from studies involving these compounds, which frequently reveal unanticipated nonantibiotic effects that point to a range of other biological activities. Alternative uses, such as the treatment of cardiovascular illness or the use of immunosuppressive drugs, have occasionally outweighed the significance of antibacterial activity.

Unfortunately, there is a serious environmental cost associated with the enormous demand for these priceless medications. Production advancements have made chemicals more affordable, which promotes off-label and nonprescription use. The packaging of the oldest and most often used antibiotics is (likely) the largest source of cost. Naturally, the abundance of these harmful substances on Earth has greatly aided in the selection of resistant breeds. Generations of antibiotic-resistant bacteria have resulted from years of continuous selection pressure from human antibiotic usage, including overuse, misuse, and underuse, and they have spread throughout the biosphere's microbial populations. There may not be a finer illustration

of Darwinian concepts of selection and survival than this—a man-made circumstance placed on nature rather than a natural process.

Alexander Fleming developed penicillin in 1928, and two members of the penicillin discovery team identified a bacterial penicillinase in 1940, several years before penicillin was first used as a medication. Widespread usage of the antibiotic led to the emergence of resistant strains that might render the medication inactive, prompting research into chemically altering penicillin to stop cleavage by penicillinases (β-lactamases).

Class	Examples	Target	Resistance mechanisms
B-lactams	Ampicillin (AMP) Amoxicillin (AMX)	Targets: Penicillin binding proteins (PBPs). Inhibition of cell wall synthesis. Bactericidal.	Beta-lactamases, modification of penicillin binding proteins (PBPs), efflux pumps and membrane impermeability.
B-lactamase resistant penicillin	Dicloxacillin (DCX)	Targets: Penicillin binding proteins (PBPs). Inhibition of cell wall synthesis. Dicloxacillin is stable against hydrolysis by a variety of beta-lactamases. Bactericidal.	Modification of penicillin binding proteins (PBPs), efflux pumps and membrane impermeability.
Aminoglycosides	Kanamycin (KM) Streptomycin (STP)	Targets: Binding of 30 ribosomal subunit.Disrupts translation, inhibits protein synthesis. Bactericidal.	Modification of antibiotic by acylation, phosphorylation or adenylation.
Tetracycline's	Tetracycline (TET)	Target: Binding of 30 ribosomal subunit. Inhibits protein synthesis. Bacteriostatic.	Efflux, ribosomal protection and enzymatic inactivation.
Fluoroquinolones	Ciprofloxacin (CIP)	Targets the GyrA subunit of DNA gyrase, and topoisomerase IV. Inhibition of DNA synthesis. Bacteriicidal.	Target modification, efflux pumps.
Macrolides	Erythromycin (ERI)	Targets: 50S ribosomal subunit. Inhibits protein synthesis. Bacteriostatic.	Target modification, mutations in 23S rRNA, efflux pumps and enzymatic inactivation. Phosphotransferases: phosphorylation of hydroxyl group. Glycotransferases: glycosylation of hydroxyl group. Esterases: hydrolyzation of lactone ring.
Trimethoprim	Trimethoprim (TMT)	Targets: dihydrofolate reductase (DHFR). Inhibits folate synthesis. Bacteriostatic.	**Resistant** forms of the DHFR enzyme. **Mutations** in gene promoter and upstream genetic elements lead to overexpression of intrinsic DHFR enzyme.
Sulfonamides	Sulfamethoxazole (SMX)	Targets: dihydropteroate synthase (DHPS). Inhibits folate synthesis. Bacteriostatic.	Resistant forms of DHPS enzymes, mutations in *dhp* gene.

Table 1: Most widely used antibiotics along with their mechanisms of action and resistance mechanisms (source: ResearchGate)

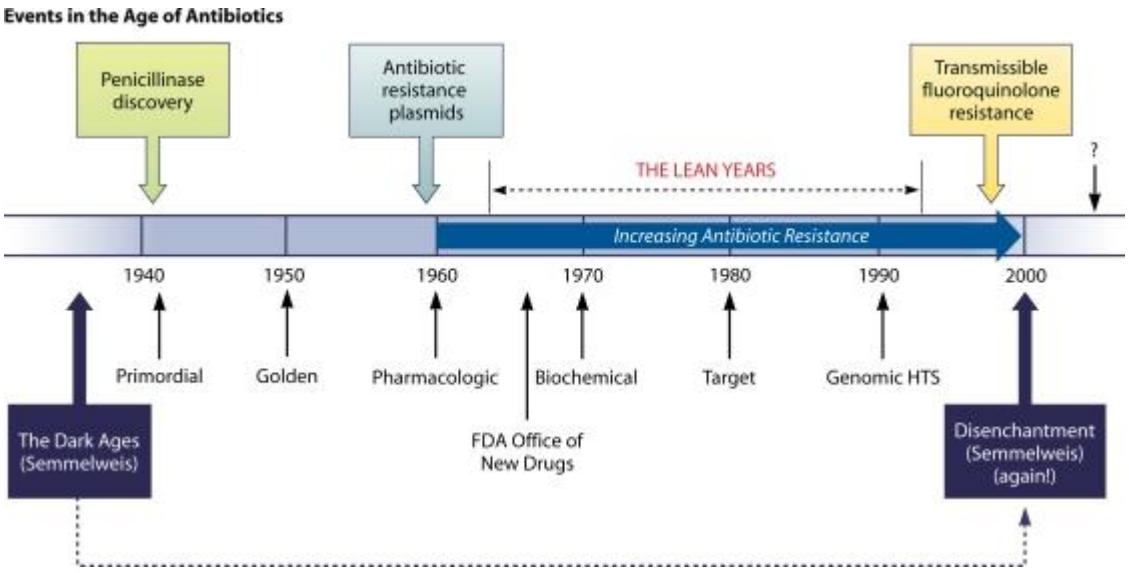

Fig 9: Progression of antibiotic discovery and resistance development

(source: ASM journals)

THE HISTORY OF THE DISCOVERY OF ANTIBIOTICS AND THE EMERGENCE OF ANTIBIOTIC RESISTANCE

The preantibiotic era, known as the "dark ages," the "primordial" (the introduction of chemotherapy through the use of sulfonamides), "lean" , the "biochemical" , the "target," the "genomic/HTS" and the "disillusion" . Other significant events in this history include the FDA Office of New Drugs' establishment following the thalidomide scandal, which resulted in more stringent regulations for drug safety, including the use of antibiotics. Novel compound registration was slowed as a result. Semmelweis promoted hand washing as a means of preventing infection prior to the discovery of antibiotics; nowadays, this practice is highly advised as a means of preventing transmission.

The fact that gene exchange is a ubiquitous characteristic of bacteria that has taken place during generations of microbial evolution has only recently come to light. The significance of horizontal gene transfer (HGT) in genome evolution has been brought to light by the identification of probable bacterial gene sequences in

eukaryotic genomes. The discovery and dispersion of genomic islands including pathogenicity genes and other functional gene clusters in many bacterial genera have since disclosed additional facets of gene transfer. Plasmid-mediated transmission of antibiotic resistance has also been shown recently.

SUPERRESISTANCE AND SUPERBUGS

After the administration of antibiotics, numerous bacterial pathogens associated with human illness outbreaks have evolved into multidrug-resistant (MDR) strains. For instance, MDR M. A common disease in both industrialized and underdeveloped countries, TB is the 20th century's equivalent of an ancient one. Escherichia coli, Haemophilus influenzae, Klebsiella pneumoniae, Proteus mirabilis, Pseudomonas aeruginosa, Staphylococcus aureus, Staphylococcus epidermidis, and Streptococcus pneumoniae are among the other serious infections that are hospital-associated. The term "superbugs" describes microorganisms that have increased morbidity and mortality as a result of numerous mutations that have high levels of resistance to the antibiotics which are used for their treatment; these microbes have fewer therapeutic options, and hospital stays are longer and more expensive. Super resistant strains have occasionally additionally developed improved transmissibility and pathogenicity. In actuality, one may classify antibiotic resistance as a pathogenicity factor.

The most common Gram-negative organisms, including Escherichia coli, and Klebsiella pneumoniae, cause a wide range of illnesses.

Pseudomonas aeruginosa is now a potent hospital-acquired illness. In this instance, the most effective medicines (such β-lactams and aminoglycosides) were compromised because antibiotic resistance mechanisms developed concurrently with the release of new antibiotic derivatives. Because P. aeruginosa is extremely

persistent and evades the human immune system, it poses a serious risk to people with cystic fibrosis. Long-term antibiotic use in people with cystic fibrosis is linked to the development of resistance.

Another more recent Gram-negative pathogen that is mostly nosocomial is Acinetobacter baumannii. It has the same set of r genes and pathogenicity determinants as the pseudomonads, which leads to higher rates of morbidity and mortality. The strong survival and biodegradation skills of Acinetobacter organisms in the environment are assumed to be the source of their infectious qualities; many strains also have high rates of spontaneous transformation and are naturally competent for DNA absorption.

Nowadays, Staphylococcus aureus, a Gram-positive bacterium, is the most infamous superbug. It is debatable whether it is the most dangerous superbug because it is unclear how much of its negative image stems from the widespread media attention. Because it is present in 30% of people as a nasal commensal and has long been connected to common skin illnesses like boils, aureus is closely associated with humans. After penicillin was discovered, it appeared that S. aureus infections were manageable. Methicillin, the first designed antiresistance antibiotic, was discovered and introduced in 1959, and it was believed to be a effective against penicillinases. However, the emergence of MRSA, which today stands for multidrug-resistant S. aureus, inexorably gave rise to other multiantibiotic-resistant strains in just three years. With improved virulence and transmission traits, MRSA has recently spread outside of hospitals and emerged as a significant community-acquired (CA) pathogen.

Resistance from Within

The presence of genes in bacterial genomes that have the potential to produce a resistant phenotype, such as proto- or quasi-resistance, is referred to as intrinsic

resistance. Different antibiotic response phenotypes are displayed by various genera, species, strains, etc. Gene amplification, is a frequent genetic pathway to increased antibiotic resistance, particularly for resistance to trimethoprim, and sulfonamides.

Through saturation mutagenesis of bacterial genomes, phenotypic investigations of partial or "complete" gene knockout libraries enable the discovery of particular mutants causing antibiotic hypersensitivity reactions. A resistance phenotype is thought to result from overexpression of the relevant wild-type gene. Numerous organisms have been the subject of such prognostic investigations, which have resulted in the prediction of novel resistance classes. A resistance phenotype may not result from many of the purported "susceptibility" genes that have been found, such as genetically recessive genes. However, these methods reveal information about the systems biology of resistance and uncover putative r genes. Similar predictive data has been obtained from RNA microarray analysis of antibiotic effects. In other words, titration can result in a decrease in the intracellular concentration of the inhibitor when the number of copies of the antibiotic's target genes increases.

Yassin and Mankin identified potential target locations for ribosomal function inhibitors using a mutant method. Numerous RNA regions that could be new targets for small-molecule translation inhibitors were described by rRNA studies. Despite claims to the contrary, our novel studies show that there are still a lot of promising therapeutic targets in antimicrobial discovery that need to be explored. A useful strategy for prolonging antibiotic lives would be to accurately predict resistance and take relevant action.

The Resistome

Placing environmental bacteria on antibiotic-containing media in a lab setting has long been recognized to isolate antibiotic-resistant bacterial strains. Given that the majority of actinomycetes that manufacture antibiotics have genes that confer resistance to the substances they produce, this is not surprising. Certain enzymatic changes of the antibiotics have been found to be the resistance mechanisms in a number of cases. It has long been recognized that streptomycetes produce a range of β-lactamases, which could potentially be the cause of various clinical forms of β-lactam resistance.

Many strains were innately multidrug resistant, meaning they could withstand an average of seven or eight antibiotics. The ambient antibiotic resistome is the term used to describe the population of r genes found in nature. The quantity and kind of resistances would obviously fluctuate depending on the surroundings.

The Subsistome

The ability of hundreds of strains to survive or proliferate on one or more distinct antibiotics as the only sources of carbon and nitrogen. A number of strains that developed well on common antimicrobials, such as aminoglycosides, fluoroquinolones, and other types, were discovered. Naturally occurring catabolic pathways that aid in the digestion of antibiotics offer a wealth of possible resistance determinants.

Resistance as a Result of Human Activity

Populations of resistant strains in all environments are constantly under selection and maintenance pressure. We must presume that commercial manufacturing supplies the great majority of the antibiotics present in the biosphere, as the only data suggests that naturally occurring antibiotic-producing strains

contribute very little in the form of antibiotics in their native settings. The following are some other applications for antimicrobial agents: (i) the promotion of animal growth and prevention; (ii) human treatment and prevention; (iii) aquaculture; (iv) domestic pets; (v) cloning and pest management for plants and agriculture; (vi) use as biocides in household cleaning products, toiletries, and hand care items; and (vii) cloning, selection, and culture sterility in industry and research.

RESISTANCE GENETICS

Numerous investigations into the genetic components of the various processes linked to resistance development, including gene pickup, heterologous expression, HGT, and mutation, have been prompted by the emergence and spread of antibiotic-resistant bacteria.

Transmission of Resistance Genes

Any element present in bacteria has the ability to acquire genes and facilitate their transmission. Although there are some similarities and distinct distinctions between Gram-positive and Gram-negative bacteria, the most prevalent means of horizontal gene transfer is plasmid-mediated transmission. On various vectors, they are commonly observed as phage "fingerprints" flanking genes that encode virulence or resistance. Such occurrences seem to be very typical in S. aureus. Conjugation-based gene transmission has been thoroughly investigated in lab settings and in microcosms that mimic natural environments, and the frequency of the transfer events frequently varies greatly.

Both virulence and pathogenicity genes are very promiscuous in the streptococci, meningococci, and allied genera; transformation seems to be the main route for DNA trafficking. Lastly, concerning direct environmental DNA uptake, Acinetobacter spp. are frequently subject to HGT and are inherently competent;

pathogenic strains usually possess sizable genomic islands. Throughout evolutionary history, horizontal gene transfer has taken place. Two distinct sets of events can be distinguished, primarily by the duration of each event and the degree of selection pressure.

Numerous genetic mechanisms linked to the evolution of antibiotic-resistant populations have been described in laboratory research; phages, transformation, and plasmids have well-established roles, but there may be more mechanisms at play. For instance, large mixed microbial communities, like those found in biofilms, may enhance bacterial cell-cell fusion. A prospective r gene's low-level expression in a novel host would offer some defense against an antagonist; greater expression would result from later gene customization through mutation and selection. Bacterial pathogens exposed to high concentrations of antibiotics for prolonged periods of time during therapeutic use experience strong selection pressure, which raises resistance levels. It is unknown how an ambient gene becomes a clinical gene, but it clearly happens with some facility. There are many steps from source to clinic. Physical methods that favor DNA exchange, such as physical contact by immobilization on a filter or agar surface, can boost HGT in the laboratory under a range of conditions. Antibiotics may promote the development of antibiotic resistance, particularly when used at subinhibitory concentrations. Antimicrobials, for instance, have been demonstrated to promote phage formation from lysogens and to improve gene transfer and recombination, partly by triggering the SOS system. These elements might be crucial in increasing the frequency of gene exchange in settings that offer the best conditions for gene acquisition, like farms, hospitals, and sewage systems.

Positively, it should be mentioned that research on the mechanisms of

antibiotic resistance and the gene transfer mechanisms that go along with them in pathogens has been crucial in the development of recombinant DNA techniques, which have served as the experimental basis for the contemporary biotechnology sector. Biology was revolutionized by the application of plasmid cloning techniques and restriction enzymes. The development of suitable bifunctional antibiotics and corresponding genes in pro- and eukaryotes were among the few modest technological changes needed to extend the bacterial recombinant DNA techniques to genetic manipulations in plants, animals, and humans. With benefits to every facet of pure and applied biology becoming more and more apparent, the applications are genuinely universal.

METHODS TO MANAGE OR DECREASE THE DEVELOPMENT OF ANTIBIOTIC RESISTANCE

The concurrent emergence of resistant strains is, by far, the biggest detrimental effect of antibiotic use, which has led to ongoing attempts to regulate antibiotic use. One early example was erythromycin, which was first used to treat S. pneumonia as an alternative to penicillin. Antibiotic resistance is evidently unavoidable. Strict regulations on human antibiotic use, accurate prescription requirements, prohibitions on antibiotic use without a prescription and controlled therapeutic use in agriculture and animal husbandry are some of the action items suggested.

The most prevalent type of resistance to the majority of antibiotic classes is the ability to pump antibiotics out of cells, which is a characteristic shared by the majority of environmental bacteria and their pathogenic cousins. An appealing approach for the development of modified or combination therapies is the creation of substances that obstruct the cell's ability to release active inhibitors.

The practice of "cycling" antibiotics, which entails periodically switching out front-line antibiotics in hospitals with different structural classes, has been discussed extensively over the years. The issue strains (or r genes) are rapidly reselected when similar drugs are reintroduced. It could be challenging to properly disinfect the "infected" intensive care units while switching between antibiotics in huge hospital complexes.

Combinations of inhibitory substances with distinct mechanisms of action are used as a related strategy.

There have been numerous attempts to prevent, suppress, or get around disease resistance mechanisms. The β-lactam antibiotics have proven the most successful in these attempts. Clavulanic acid and its derivatives are often used in conjunction with β-lactam antibiotics because they are strong inhibitors of β-lactamase enzymes. Although these combinations have proven to be quite successful, bacteria have outwitted us and developed a variety of β-lactamases that are resistant to clavulanate inhibition.

The key component of bacterial disease control. In an ideal society, antibiotic use would be greatly curtailed and, ideally, restricted to hospital surgical procedures under rigorous monitoring, as there would be effective vaccines against all infectious diseases. There aren't many commonly used antibacterial vaccinations, nevertheless, despite years of work. The pneumococcal vaccine's success serves as an example of what is possible.

MATERIALS AND METHODS

STUDY DESIGN: PROSPECTIVE STUDY SOURCE OF DATA:

This prospective study is conducted in the Department of Otorhinolaryngology in **SHRI B M PATIL MEDICAL COLLEGE AND RESEARCH CENTRE AND HOSPITAL [BLDE (DU) UNIVERSITY]**, Vijayapura from April 2023 to January 2025.

METHOD OF COLLECTION OF DATA:

- Preoperative examination of the patient including complete clinical history.
- Characteristics of patients including age, gender, residence.
- The patient is thoroughly examined, with a focus on otoscopic findings to assess the condition of the tympanic membrane and to examine the nose, throat, and oropharynx.
- Patient will be subjected to investigations such as urine routine, blood routine examinations and aerobic culture of the discharge from both the external auditory canal and middle ear (under microscope).
- Sample from the external auditory canal is taken out with the help of Cotton Swab.
- Then, external auditory canal is cleaned using 70% alcohol and normal saline and allowed to dry for 30-40 seconds to achieve sterile area.
- Mucus extractor is used to collect sample from middle ear.
- Both the samples are sent for aerobic culture and sensitivity.
- All isolates underwent an antimicrobial susceptibility test utilizing the Kirby Bauer disc diffusion method on a Mueller Hinton agar plate. Clinical Laboratory Standards Institute (CLSI) criteria were used to interpret the results.

INCLUSION CRITERIA:

1. Individuals with a diagnosis of chronic otitis media-tubotympanic type.
2. Patients who have had ear discharge for longer than three months and is currently active.
3. Patients who have not received topical or systemic antibiotic treatment in the previous seven days.

EXCLUSION CRITERIA:

1. Patients with chronic otitis media who have taken topical and systemic antibiotics within the seven days prior to presentation.
2. Atticoantral COM patients.

SAMPLE SIZE:

- With anticipated Proportion of Klebsiella CSOM patients 24%[3], the study would require a sample size of 71 patients with 95% level of confidence and 10 absolute precision
- Formula used $n=z^2p*q$

$$\frac{}{d^2}$$

- Where Z=Zstatisticatαlevelofsignificance
- d^2=Absolute error
- P=Proportion rate
- q= 100-p

STATISTICAL ANALYSIS:

- The data obtained will be entered in a Microsoft Excel sheet, and statistical analysis will be performed using statistical package for the social sciences (Verson 20).
- Results will be presented as Mean±SD, Median and interquartile range, frequency, percentages and diagrams**.**

OBSERVATIONS AND RESULTS

A hospital-based prospective study was conducted with 71 patients to study the bacterial flora in external auditory canal and middle ear in chronic otitis media, tubotympanic type patients.

1. SEX DISTRIBUTION:

Out of the 71 patients studied enrolled in this study 56.3% were females and 43.7% were males. Females were more compared to males in the study. (table.2) (fig 10)

Gender	No. of patients	Percentage
Female	40	56.3
Male	31	43.7
Total	71	100.0

Table 2. Distribution of patients according to sex

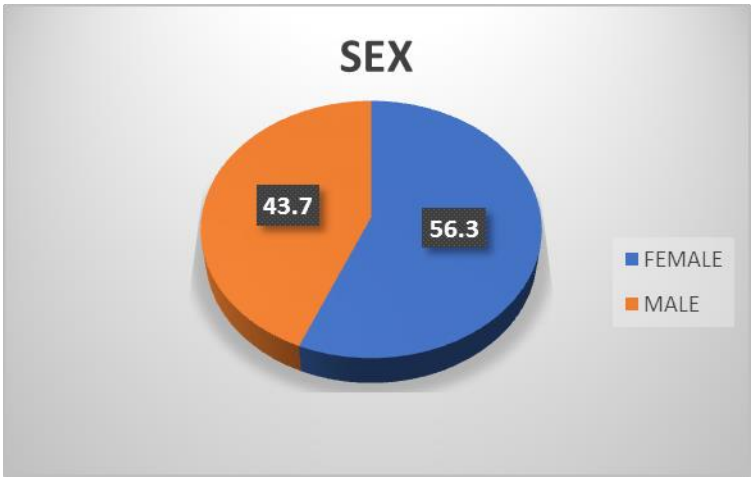

Fig.10: Gender distribution

2. AGE DISTRIBUTION

The present study included 71 patients, the most commonly affected age group was 10 to 19 years, which included 36(21.2%) patients. (table.3) (fig 11)

Age (Years)	No. of patients	Percentage
< 10	2	2.8
10 – 19	20	28.2
20 – 29	12	16.9
30 – 39	17	23.9
40 – 49	6	8.5
50 – 59	9	12.7
60+	5	7.0
Total	71	100

Table 3: Distribution of patients according to age (years)

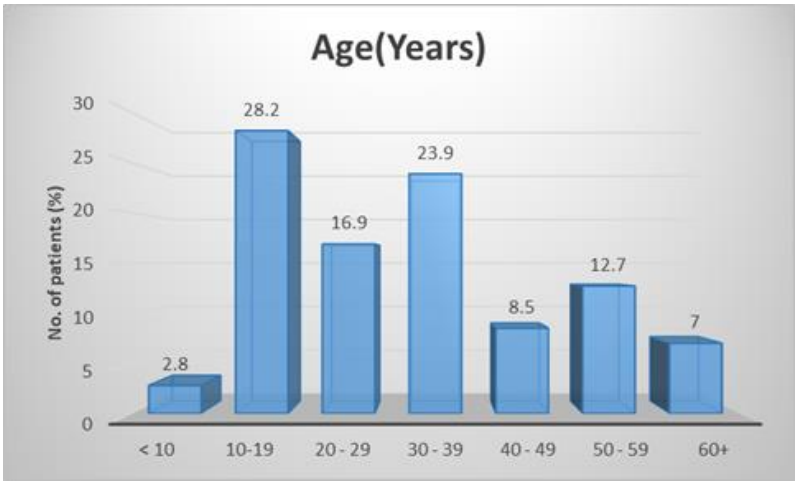

Fig. 11: Distribution of age

3. DISTRIBUTION OF SAMPLE TAKEN FROM DISEASED EAR

Out of 71 patients, sample from left ear discharge was taken from 54.9% of patients and right ear discharge was taken from 45.1% of patients. (table 4) (fig 12)

Diseased ear	No. of patients	Percentage
Left	39	54.9
Right	32	45.1
Total	71	100.0

Table 4: Distribution of sample taken from Diseased ear

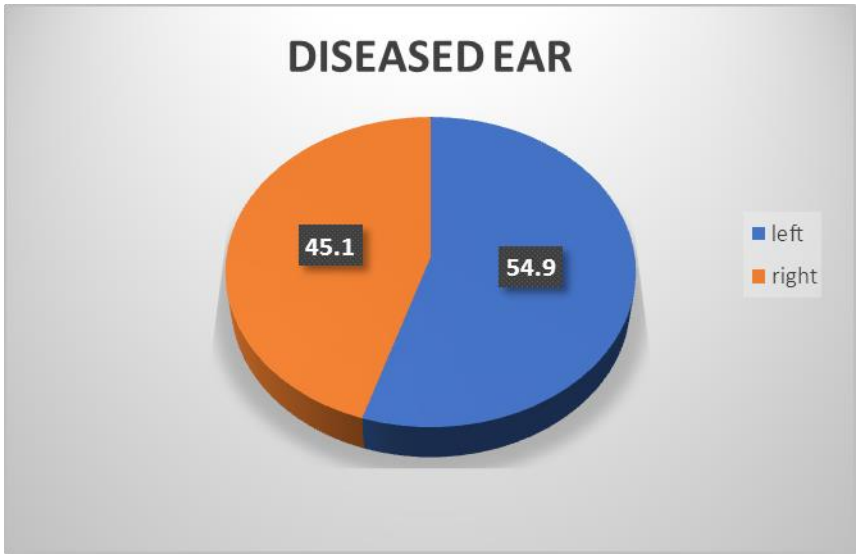

Fig. 12: Sample from diseased ear

4. DISTRIBUTION OF PATIENTS ACCORDING TO DURATION OF EAR DISCHARGE

Out of 71 patients, ear discharge from childhood was maximum, seen in 38% of patients, from 3 months to 1 year was seen in 23.9% , 2yrs to 4yrs was seen in 25.4%, 5 yrs to 7 yrs was seen in 8.5% of patients, 8yrs to 10 yrs was seen in 4.2% of patients. (table 5) (fig 13)

Duration of ear discharge (years)	No. of patients	Percentage (%)
<=1	17	23.9
2-4	18	25.4
5-7	6	8.5
8-10	3	4.2
childhood	27	38.0
Total	71	100.0

Table 5: Distribution according to duration of ear discharge

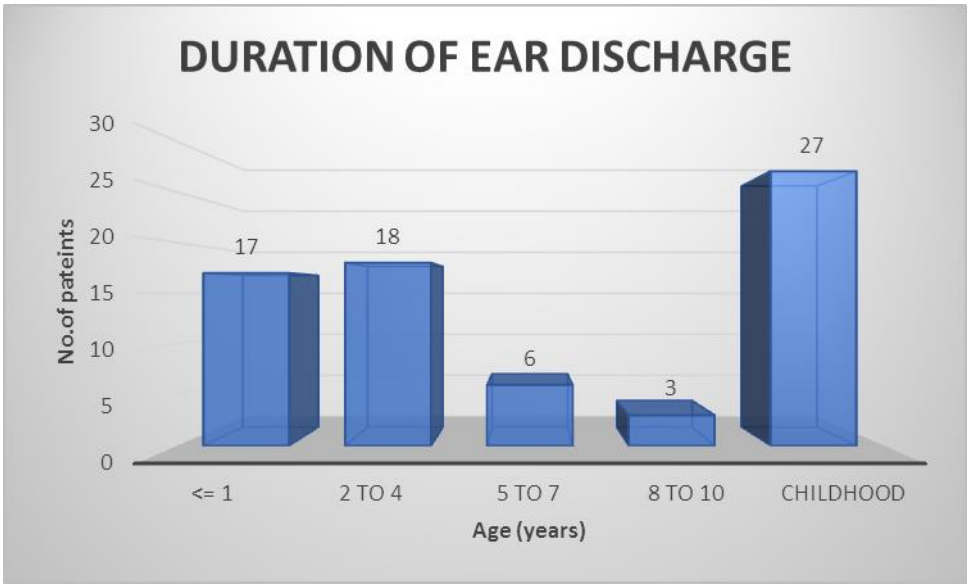

Fig 13: Duration of ear discharge

5. DISTRIBUTION ACCORDING TO SIZE OF PERFORATION

In present study, out of 71 samples collected, large central perforation was most commonly seen in 27 (38%) of patients. (table 6) (fig 14)

Size of perforation	No. of patients	Percentage (%)
Large central perforation	27	38.0
Medium central perforation	20	28.1
Small central perforation	11	15.5
Subtotal perforation	13	18.3
Total	71	100.0

TABLE 6: Distribution according to size of perforation

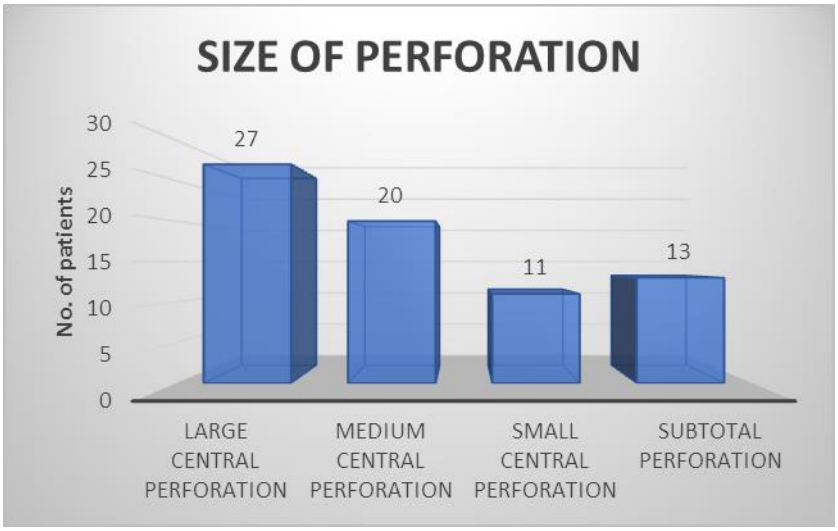

Fig.14: Sizes of perforation

6. DISTRIBUTION ACCORDING TO DEGREE OF HEARING LOSS

In our study no hearing loss was seen in 11.3% of patients, whereas very severe hearing loss was seen in 1.4% of patients. (table 7) fig 15).

Degree of hearing loss (db)	No. of patients	Percentage (%)
<20	8	11.3
20-40	26	36.6
41-60	31	43.7
61-80	5	7.0
>80	1	1.4
Total	71	100.0

Table 7: Distribution according to degree of hearing loss

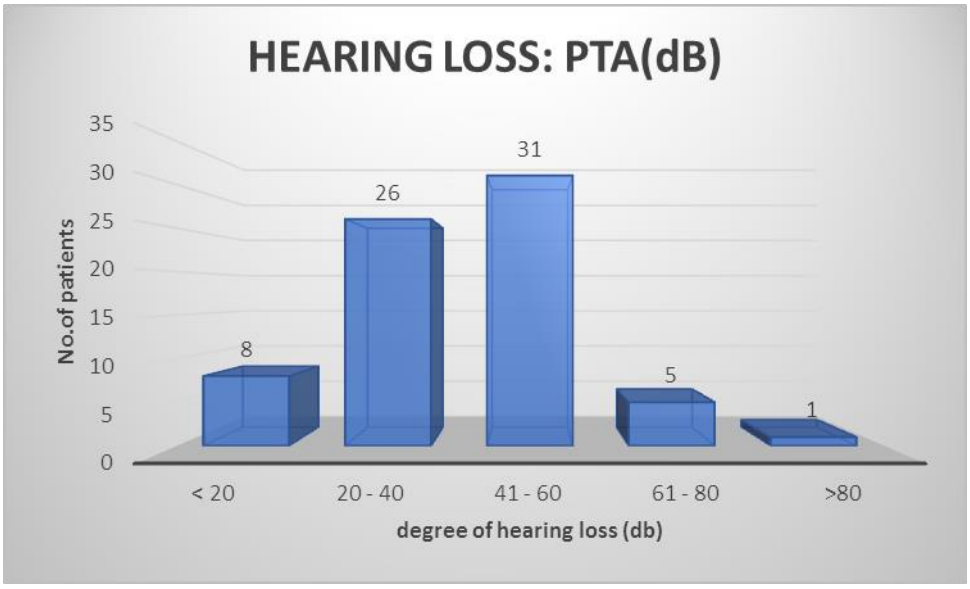

Fig 15: Degree of hearing loss (db.)

7. DISTRIBUTION OF ORGANISMS ISOLATED FROM EXTERNAL EAR

Out of 71 samples collected from external ear in COM patients, most common organism was found to be Staphylococcus aureus seen in 35 (49.3%) patients, followed by Pseudomonas aeruginosa seen in 18 (25.4%) patients. (table 8) (fig 16)

Organism (external ear)	No. of patients	Percentage
Acinetobacter baumannii	3	4.2
E Coli	4	5.6
Klebsiella sps	11	15.5
Pseudomonas aeruginosa	18	25.4
Staphylococcus aureus	35	49.3
Total	71	100.0

TABLE 8: DISTRIBUTION OF ORGANISMS ISOLATED FROM EXTERNAL EAR

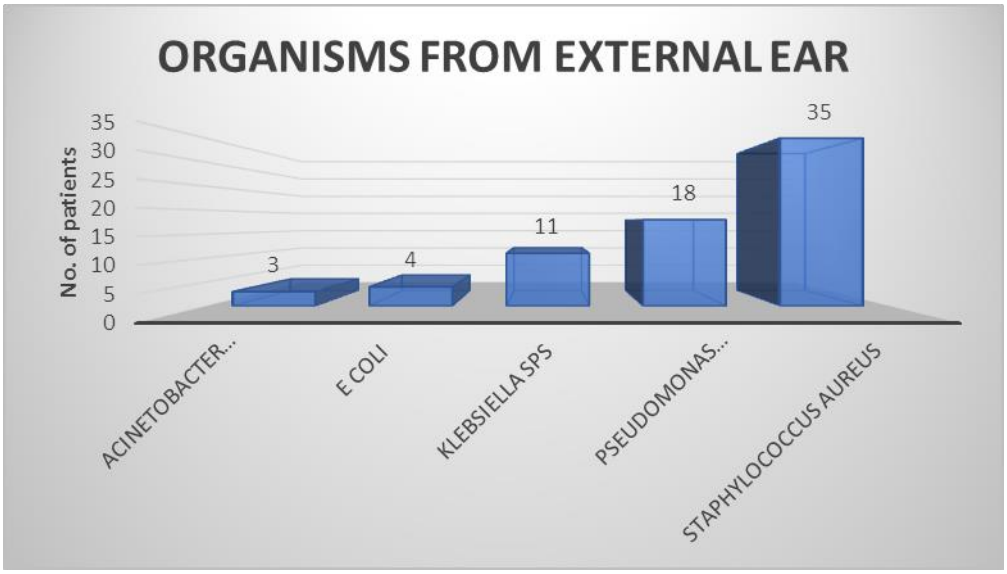

Fig.16: Organisms isolated from external ear

8. DISTRIBUTION OF PATIENTS BASED ON ANTIBIOTIC SENSITIVITY (EXTERNAL EAR)

Out of 71 samples collected from external ear, patients showed maximum sensitivity to Clindamycin (31%), followed by Meropenem (19.7%). (table 9) (fig 17)

Antibiotic Sensitivity (External Ear)	No. of patients	Percentage
Amikacin	3	4.2
Cefaperaxone/Sulbactum	1	1.4
Cefepime	2	2.8
Ciprofloxacin	13	18.3
Clindamycin	22	31.0
Colistin	3	4.2
Gentamycin	3	4.2
Linezolid	10	14.1
Meropenem	14	19.7
Total	71	100.0

TABLE 9: DISTRIBUTION OF PATIENTS BASED ON ANTIBIOTIC SENSITIVITY (EXTERNAL EAR)

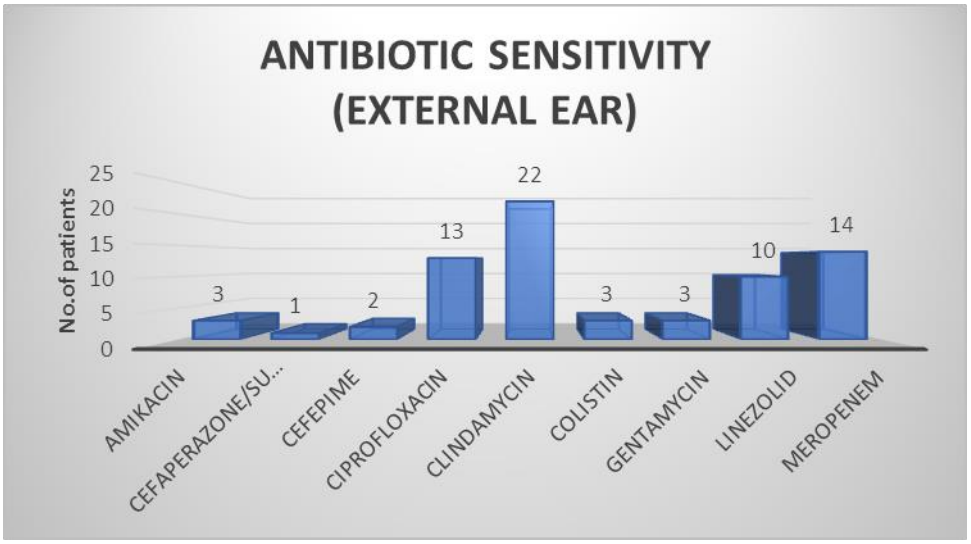

Fig.17: Antibiotic sensitivity distribution (external ear)

9. DISTRIBUTION OF ORGANISMS ISOLATED FROM MIDDLE EAR

Out of 71 samples collected from middle ear in COM patients, most common organism was found to be Pseudomonas aeruginosa seen in 42 (59.2%) patients, followed by Staphylococcus aureus seen in 14 (19.7%) patients. (table 10) (fig 18)

Organism (external ear)	No. of patients	Percentage
Acinetobacter baumannii	1	1.4
E Coli	2	2.8
Klebsiella sps	12	16.9
Pseudomonas aeruginosa	42	59.2
Staphylococcus aureus	14	19.7
Total	71	100.0

TABLE 10: DISTRIBUTION OF ORGANISMS ISOLATED FROM MIDDLE EAR

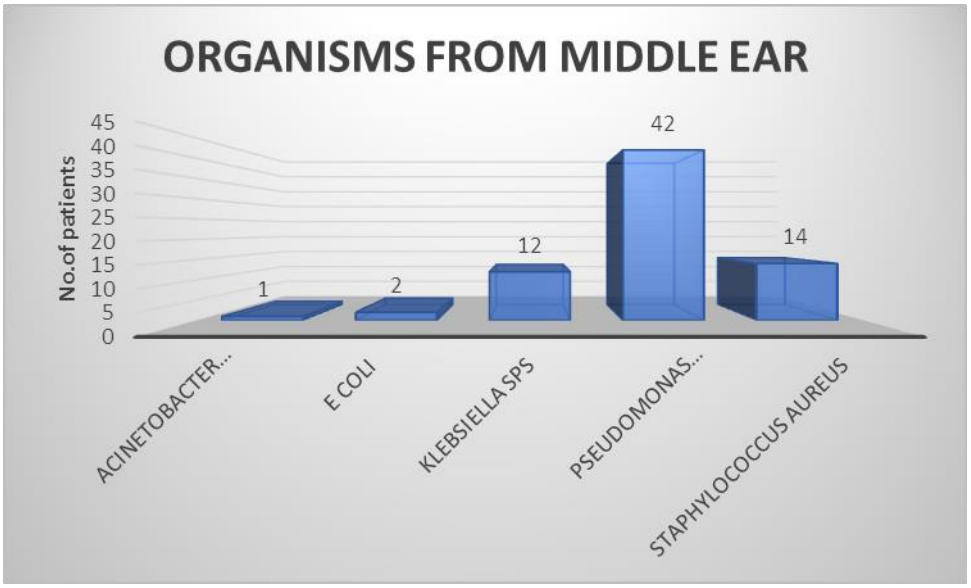

Fig.18: Organisms isolated from middle ear

10. DISTRIBUTION OF PATIENTS BASED ON ANTIBIOTIC SENSITIVITY (MIDDLE EAR)

Out of 71 samples collected from middle ear, patients showed maximum sensitivity to Ciprofloxacin (40.8%), followed by Meropenem (26.8%). (table 11) (fig 18)

Antibiotic Sensitivity (Middle Ear)	No. of patients	Percentage
Amikacin	4	5.6
Cefaperaxone/Sulbactum	1	1.4
Cefepime	2	2.8
Ciprofloxacin	29	40.8
Clindamycin	11	15.5
Colistin	2	2.8
Linezolid	3	4.2
Meropenem	19	26.8
Total	71	100.0

TABLE 11: DISTRIBUTION OF PATIENTS BASED ON ANTIBIOTIC SENSITIVITY (MIDDLE EAR)

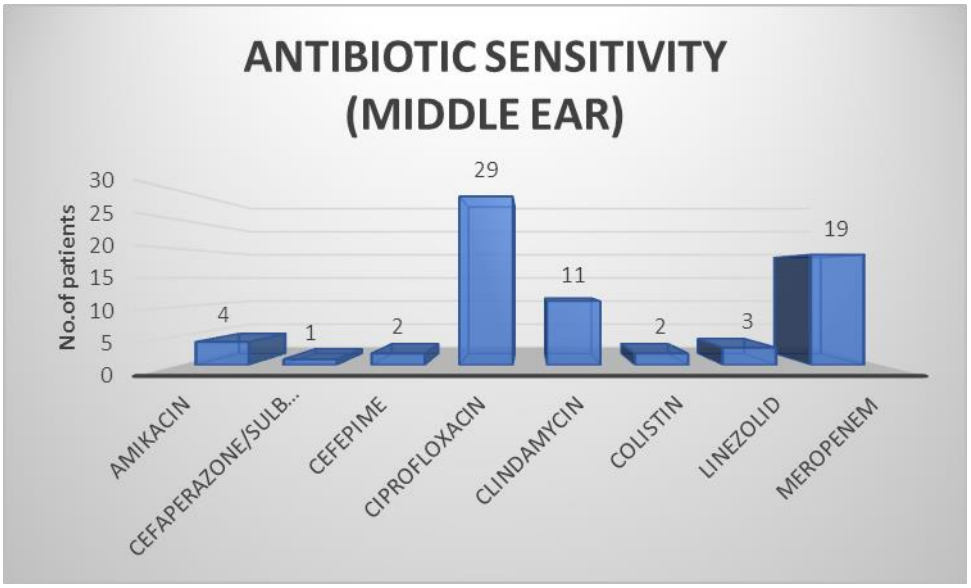

Fig.18: Antibiotic sensitivity distribution (middle ear)

11. ANTIBIOTIC SENSITIVITY PATTERN OF ORGANISMS ISOLATED FROM EXTERNAL EAR

Organism (external ear)	Amikacin	Cefaperazone/Sulbactam	Cefepime	Ciprofloxacin	Clindamycin	Colistin	Gentamycin	Linezolid	Meropenem	Total
Acinetobacter baumannii	0 0%	1 100.0%	0 0%	0 0%	1 4.5%	1 33.3%	0 0%	0 0%	0 0%	3 4.2%
E Coli	0 0%	0 0%	0 0%	1 7.7%	0 0%	0 0%	0 0%	0 0%	0 0%	3 21.4%
Klebsiella sps	1 33.3%	0 0%	1 50%	6 46.2%	0 0%	0 0%	0 0%	0 0%	3 21.4%	11 15.5%
Pseudomonas aeruginosa	2 66.7%	0 0%	1 50%	5 38.5%	0 0%	2 66.7%	0 0%	0 0%	8 57.1%	18 25.4%
Staphylococcus aureus	0 0%	0 0%	0 0%	1 7.7%	21 95.5%	0 0%	3 100%	10 100%	0 0%	35 49.3%
Total	3 100%	1 100%	2 100%	13 100%	22 100%	3 100%	3 100%	10 100%		71 100%
Chi square=107.109 p=0.000										

Table 12: Antibiotic sensitivity pattern (External ear)

12. ANTIBIOTIC SENSITIVITY PATTERN OF ORGANISMS ISOLATED FROM MIDDLE EAR

Organism (middle ear)	Amikacin	Cefaperazone/Sulbactam	Cefepime	Ciprofloxacin	Clindamycin	Colistin	Linezolid	Meropenem	Total
Acinetobacter baumannii	0 0%	1 100.0%	0 0%	0 0%	0 0%	0 0%	0 0%	0 0%	1 1.4%
E Coli	0 0%	0 0%	0 0%	0 0%	0 0%	0 0%	0 0%	2 10.5%	2 2.8%
Klebsiella sps	0 0%	1 50%	4 13.8%	0 0%	0 0%	0 0%	0 0%	7 36.8%	12 16.9%
Pseudomonas aeruginosa	4 100%	0 0%	1 50%	25 86.2%	0 0%	2 100%	0 0%	10 52.6%	42 59.2%
Staphylococcus aureus	0 0%	0 0%	0 0%	0 0%	11 100%	0 0%	3 100%	0 0%	14 19.7%
Total	4 100%	1 100%	2 100%	29 100%	11 100%	2 100%	3 100%	19 100%	71 100%
Chi square=156.273 p=0.000									

Table 12: Antibiotic sensitivity pattern (Middle ear)

DISCUSSION

SEX DISTRIBUTION:

We included 71 participants in our study. Of these patients, 31 (43.7%) were men and 40 (56.3%) were women. Females were more frequently affected than men in our study, which is consistent with a study by Shrestha et al. that revealed a ratio of 1.23:1 between 127 females and 103 males[8].

Whereas, in a study conducted by Kombade et al, males, 80 (52.3%), were predominantly affected as compared to females, 73 (47.7%) out of 153 cases[9]. The difference in results may be due to geographical reason.

AGE DISTRIBUTION:

The age group with the highest COM incidence in our study was 10–19 years old (28.2%), whereas the age group with the lowest prevalence was patients under 10 years old (2.8%).

The age range of 21–30 years had the highest number of patients in a study by Kombade et al.[9], followed by 11–20 years (21.6%).

According to a study by Metri Basavaraj[10] in India, the age range of 1–20 years old accounted for 52.8% of the most often affected CSOM patients, followed by 21–60 years old (45.9%).

AFFECTED SIDE:

Out of 71 patients, sample from left ear discharge was taken from 54.9% of patients and right ear discharge was taken from 45.1% of patients.

Study conducted by Wan Draman et al showed unilateral ear involvement in 96.6% of cases, where right ear was more commonly affected than left ear, and bilateral ear involvement in 3.4% of total cases[7].

ISOLATION OF ORGANISMS:

In our study, out of 71 samples collected from external ear, most common organism was found to be Staphylococcus aureus seen in 35 (49.3%) patients, followed by Pseudomonas aeruginosa seen in 18 (25.4%) patients whereas, from the samples collected from middle ear, most common organism was found to be Pseudomonas aeruginosa seen in 42 (59.2%) patients, followed by Staphylococcus aureus seen in 14 (19.7%) patients. Least common organism was Acinetobacter baumannii from both the swabs.

In a study conducted in Pakistan, Pseudomonas aeruginosa (38%) was the most common bacterial isolate, followed by staphylococcus (28%), Proteus mirabilis (21%), E coli, (3%), Klebsiella (3%) and Candida (2%)[11].

Study conducted by Basavaraj Hiremath et al reported the predominant organism to be Pseudomonas aeruginosa (38.79%) and Staphylococcus aureus (32.75%)[12].

SENSITIVITY TO ANTIBIOTICS:

Out of the total swabs collected from external ear in our study, Staphylococcus aureus showed 100% sensitivity to Gentamycin and Linezolid, 95.5% to Clindamycin, 7.7% to Ciprofloxacin, and an overall sensitivity of 49.3% to the antibiotics used in this hospital. Pseudomonas aeruginosa showed 66.7% Amikacin and Colistin, 57.1% sensitivity to Meropenem, 50% to Cefepime, 38.5% to Ciprofloxacin and an overall sensitivity of 25.4% to the antibiotics used in this hospital.

Out of the total swabs collected from middle ear in our study, Staphylococcus aureus showed 100% sensitivity to Linezolid and Clindamycin and an overall sensitivity of 19.7% to the antibiotics used in this hospital. Pseudomonas aeruginosa showed 100% Amikacin and Colistin, 86.2% to Ciprofloxacin, 52.6% to Meropenem,

50% to Cefepime and an overall sensitivity of 59.2% to the antibiotics used in this hospital.

Mansoor et al concluded that sensitivity pattern of Pseudomonas aeruginosa showed that “amikacin was active against 96% of isolates followed by ceftazidime 89%, ciprofloxacin 85%, gentamicin 81%, imipenem 76%, aztreonam 42% and ceftriaxone 21%”[13].

According to the study conducted by R.Indudharan et al “the sensitivity of *P aeruginosa* was 100% to ceftazidime, 98.9% to ciprofloxacin, 96.3% to gentamicin, and 95.4% to polymyxin B, whereas the sensitivity of *S aureus* was 98.6% to ciprofloxacin, 97.4% to cloxacillin sodium, 96.5% to cotrimoxazole, and 90.7% to gentamicin”[14].

CONCLUSION

The bacteriological profile of COM in our study showed a high prevalence of Staphylococcus aureus in external ear and Pseudomonas aeruginosa from middle ear with different distributions in different age groups and varying degree of antibiotic sensitivities. Linezolid was found to be the most suitable antibiotic for Staphylococcus aureus. Amikacin was found to be the most suitable antibiotic for Pseudomonas aeruginosa. Knowing the local susceptibility pattern of the causative agents is essential for treating the infection effectively and for developing antibiotic policy, as the susceptibility pattern of the pathogenic microorganisms is changing since antibiotics are widely used and readily available.

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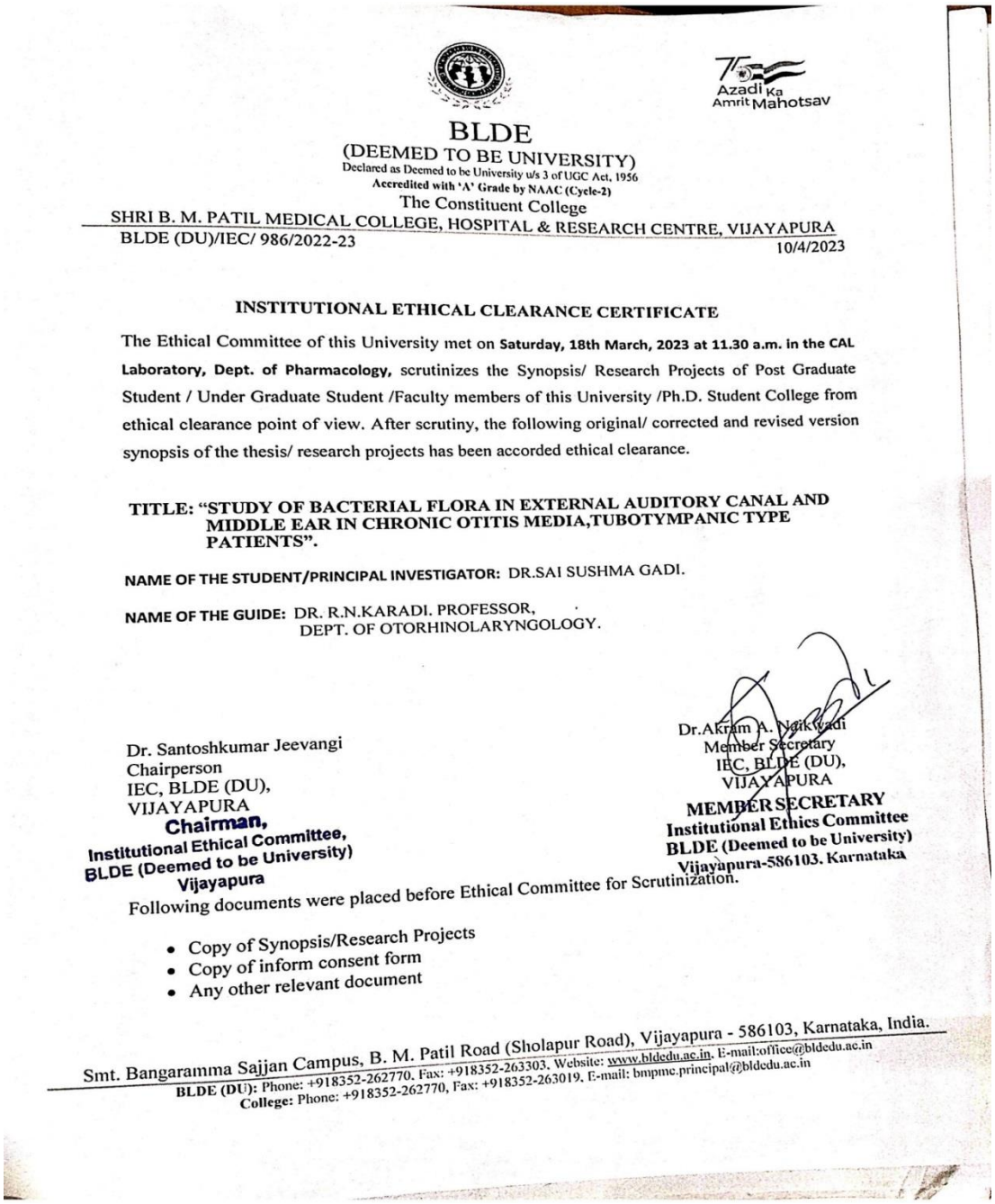
ANNEXURE I

ETHICAL CLEARANCE CERTIFICATE



BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 986/2022-23 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "STUDY OF BACTERIAL FLORA IN EXTERNAL AUDITORY CANAL AND MIDDLE EAR IN CHRONIC OTITIS MEDIA,TUBOTYMPANIC TYPE PATIENTS".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.SAI SUSHMA GADI.

NAME OF THE GUIDE: DR. R.N.KARADI. PROFESSOR,
DEPT. OF OTORHINOLARYNGOLOGY.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA
Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA
MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.
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ANNEXURE – II

PROFORMA

SCHEME OF CASE TAKING

DOA:	DOD:	DOS:	
1) NAME: 2) AGE: 3) SEX: 4) RELIGION: 5) OCCUPATION: 6) RESIDENCE:			CASE NO: IP NO:

7) CHIEF COMPLAINTS:

8) HISTORY OF PRESENTING ILLNESS:

9) PAST HISTORY:

10) FAMILY HISTORY:

11) GENERAL PHYSICAL EXAMINATION:

12) VITALS

PR:

BP:

RR:

Temp:

13) OTHER SYSTEMIC EXAMINATION:

14) LOCAL EXAMINATION

EAR Right Left

- Pinna
- Preauricular area
- Postauricular area
- External auditory canal
- Right Left
- Tympanic membrane

Pars Tensa

Pars flaccida

- Mastoid Tenderness
- Fistula sign
- Tragal Tenderness
- Facial nerve function
- Nystagmus

- Tuning Fork test Rinnes
- Webers

ABC

- NOSE
- ORALCAVITY
- OROPHARYNX
- EXAMINATION UNDER MICROSCOPE :
- WITH COLLECTION OF CULTURE SWAB

15) FINALDIAGNOSIS:

19) COMMENTS:

RESULTS OF AEROBIC CULTURE FROM EXTERNAL AUDITORY CANAL AND MIDDLE EAR:

ANNEXURE –III

INFORMED CONSENT FORM

BLDE (DEEMED TO BE UNIVERSITY)

SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA- 586103

TITLE OF THE PROJECT **"STUDY OF BACTERIAL FLORA IN EXTERNAL AUDITORY CANAL AND MIDDLE EAR IN CHRONIC OTITIS MEDIA, TUBOTYMPANIC TYPE PATIENTS"**

PG STUDENT - Dr. SAI SUSHMA GADI

DEPARTMENT OF OTORHINOLARYNGOLOGY

PG GUIDE - Dr. RN KARADI

DEPARTMENT OF OTORHINOLARYNGOLOGY

BLDE (Deemed To Be University)

SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE,

VIJAYAPURA – 586103

All aspects of this consent form are explained to the patient in the language understood by him/her.

PURPOSE OF RESEARCH:

I have been informed about this study. I have also been given a free choice of participation in this study.

PROCEDURE:

I am aware that in addition to routine care received, I will be asked a series of questions by the investigator. I have been asked to undergo the necessary investigations and treatment, which will help the investigator in this study.

RISK AND DISCOMFORTS:

I understand that I may experience some pain and discomfort during the examination or during my treatment. This is mainly the result of my condition, and the procedure of this study is not expected to exaggerate these feelings that are associated with the usual course of treatment.

BENEFITS:

I understand that my participation in this study will help to improve the survival of the patient and will bring about a better outcome.

CONFIDENTIALITY:

I understand that the medical information produced by this study will be a part of Hospital records and will be subject to confidentiality and privacy regulation. Information of a sensitive personal nature will not be a part of the medical records but will be stored in the investigator's research file and identified only by a code number. The code-key connecting name to numbers will be kept in a separate location. If the data are used for publication in the medical literature or for teaching purposes, no name will be used, and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand that I may see the photographs and videotapes and hear the audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time. **Dr. SAI SUSHMA GADI** is available to answer my questions or concerns. I

understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation.

If during the study or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that DR.SAI SUSHMA GADI may terminate my participation in the study after she has explained the reasons for doing so and has helped arrange for my continued care by my own physician or physical therapist if this is appropriate.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study if such injury were reported promptly, the appropriate treatment would be available to me, but no further compensation would be provided. I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained the purpose of the research, the procedures required and the possible risks and benefits to the best of my ability in the patient's own language.

Dr. SAI SUSHMA GADI Date

_____ _____

(Investigator)

STUDY SUBJECT CONSENT STATEMENT

I confirm that **DR. SAI SUSHMA GADI** has explained to me the purpose of the research, the study procedures that I will undergo, and the possible risks and discomforts as well as benefits that I may experience in my own language. I have read, and I understand, this consent form. Therefore, I agree to give consent to participate as a subject in this research project.

Participant / Guardian Date

_____ _____

_____ _____

Witness to signature Date

MASTER CHART

SL NO	NAME	AGE	SEX	UHID NO	DISEASED EAR	DURATION OF EAR DISCHARGE	SIZE OF PERFORATION	PTA (db)	ORGANISM FROM EXTERNAL EAR	ANTIBIOTIC SENSITIVITY (EXTERNAL EAR)	ORGANISM FROM MIDDLE EAR	ANTIBIOTIC SENSITIVITY (MIDDLE EAR)
1.	Shridevi Sharanbasappa Udachana	34	F	320103	left	1	LCP	52	E Coli	Meropenem	E Coli	Meropenem
2.	Akshaykumar Shivanand Rathod	15	M	76472	right	childhood	SCP	30	Staphylococcus aureus	Clindamycin	Staphylococcus aureus	Clindamycin
3.	Reshma Mustafa Mirajakar	28	F	111082	left	childhood	LCP	31.6	Staphylococcus aureus	Clindamycin	Pseudomonas aeruginosa	Ciprofloxacin
4.	Aishwarya Bindappa Goundi	17	F	105484	left	childhood	MCP	43.3	Staphylococcus aureus	Clindamycin	Staphylococcus aureus	Clindamycin
5.	Renuka Hugar	30	F	211087	left	childhood	MCP	30	Staphylococcus aureus	Clindamycin	Pseudomonas aeruginosa	Ciprofloxacin
6.	Shivanand Sharanappa Halli	58	M	203740	left	1	LCP	66	Staphylococcus aureus	Clindamycin	Staphylococcus aureus	Clindamycin
7.	Sunil Talawar	39	M	235925	left	childhood	subtotal perforation	53	Staphylococcus aureus	Clindamycin	Pseudomonas aeruginosa	Meropenem
8.	Veerpakshi Duragappa Piraga	52	M	231594	right	5	LCP	44.2	Pseudomonas aeruginosa	Meropenem	Pseudomonas aeruginosa	Meropenem
9.	Mahadevi Jyoteppa Pujari	15	F	227111	right	childhood	LCP	60	Klebsiella sps	Ciprofloxacin	Klebsiella sps	Ciprofloxacin
10.	Ajith Nivalakhed	22	M	242782	right	childhood	LCP	23.8	Klebsiella sps	Ciprofloxacin	Pseudomonas aeruginosa	Ciprofloxacin
11.	Rajashree Suresh Bajantri	42	F	213378	left	childhood	subtotal perforation	50	Staphylococcus aureus	Clindamycin	Staphylococcus aureus	Clindamycin
12.	Yamanappa Pundalikappa Goundi	70	M	140177	left	8	subtotal perforation	71.6	Pseudomonas aeruginosa	Meropenem	Pseudomonas aeruginosa	Meropenem
13.	Siddanna Laxman Kalate	55	M	228149	right	childhood	LCP	55	Staphylococcus aureus	Clindamycin	Staphylococcus aureus	Clindamycin
14.	Shivalila Srikanth Patil	52	F	252895	left	8	LCP	58	Acinetobacter baumannii	Colistin	Klebsiella sps	Meropenem
15.	Sadashiv Khandegol	25	M	295767	left	4	MCP	26.6	Staphylococcus aureus	Gentamycin	Acinetobacter baumanni	Cefaperazone/Sulbactum
16.	Rukmavva Naguad	70	F	267765	left	1	MCP	68.3	Klebsiella sps	Cefepime	Pseudomonas aeruginosa	Ciprofloxacin
17.	Santosh Ramchandra Kinagi	25	M	262182	left	childhood	SCP	22.5	Pseudomonas aeruginosa	Amikacin	Pseudomonas aeruginosa	Amikacin
18.	Taira Sayad Madar	8	F	221500	left	childhood	LCP	48.3	Staphylococcus aureus	Linezolid	Pseudomonas aeruginosa	Ciprofloxacin
19.	Siddamma Mallikarjun Ukkali	19	M	169930	right	childhood	subtotal perforation	48	Klebsiella sps	Meropenem	Klebsiella sps	Meropenem
20.	Kallappa Channappa Kumbar	44	M	37271	left	6	LCP	28.2	Staphylococcus aureus	Clindamycin	Staphylococcus aureus	Clindamycin
21.	Karan Vijaykumar Bagali	12	M	138891	right	1	SCP	16.6	Pseudomonas aeruginosa	Colistin	Pseudomonas aeruginosa	Colistin
22.	Kartik Narasapp Bajentri	10	M	146119	left	4	LCP	38	Pseudomonas aeruginosa	Ciprofloxacin	Pseudomonas aeruginosa	Ciprofloxacin
23.	Amruta Ramaji Misal	11	F	321113	left	1	MCP	26.6	Staphylococcus aureus	Clindamycin	Pseudomonas aeruginosa	Ciprofloxacin
24.	Mallamma Mallappa Hadapad	73	F	199430	left	1	MCP	53.3	Pseudomonas aeruginosa	Colistin	Pseudomonas aeruginosa	Colistin
25.	Rayanna Shrisail Walikar	16	F	184457	right	childhood	LCP	36.8	Pseudomonas aeruginosa	Ciprofloxacin	Pseudomonas aeruginosa	Ciprofloxacin
26.	Renuka Amogi Wadeyar	14	F	195756	right	5	LCP	48	Staphylococcus aureus	Clindamycin	Pseudomonas aeruginosa	Ciprofloxacin
27.	Malingaraya Shivappa Yattapur	13	M	156624	left	childhood	subtotal perforation	44	Staphylococcus aureus	Clindamycin	Pseudomonas aeruginosa	Ciprofloxacin
28.	Preetam Manohar Rathod	14	M	122113	left	1	LCP	35	Staphylococcus aureus	Clindamycin	Pseudomonas aeruginosa	Ciprofloxacin
29.	Iramma Shrishail Ganganalli	38	F	70939	right	childhood	SCP	55	Klebsiella sps	Ciprofloxacin	Klebsiella sps	Ciprofloxacin
30.	Ranjeeth Gurajalkar	22	M	221975	left	4	subtotal perforation	51.6	Pseudomonas aeruginosa	Ciprofloxacin	Pseudomonas aeruginosa	Ciprofloxacin
31.	Aishwarya Ashok Ammanni	18	F	206753	right	childhood	MCP	45	Pseudomonas aeruginosa	Meropenem	Pseudomonas aeruginosa	Meropenem
32.	Sarojini Kori	56	F	284677	left	2	SCP	20	Pseudomonas aeruginosa	Meropenem	Pseudomonas aeruginosa	Meropenem
33.	Davalappa Mallikarjun Bagali	13	M	334204	left	1	MCP	24.8	Staphylococcus aureus	Linezolid	Pseudomonas aeruginosa	Meropenem
34.	Mahadevi Appasaheb Mantur	55	F	135504	left	1	LCP	63.3	Staphylococcus aureus	Clindamycin	Staphylococcus aureus	Clindamycin
35.	Nagamma Dasappa Koudimatti	39	F	223267	left	childhood	LCP	22.3	Pseudomonas aeruginosa	Ciprofloxacin	Pseudomonas aeruginosa	Amikacin
36.	Sumalata Byalal	27	F	211084	right	4	MCP	60	Pseudomonas aeruginosa	Cefepime	Pseudomonas aeruginosa	Cefepime
37.	Lalsab	50	M	80565	left	childhood	LCP	80	Acinetobacter baumannii	Cefaperazone/Sulbactum	Klebsiella sps	Meropenem
38.	Jainabbi	65	F	90166	right	1	MCP	45	Staphylococcus aureus	Linezolid	Staphylococcus aureus	Linezolid
39.	Lalbee Inamdar	12	F	397320	left	childhood	subtotal perforation	36	Pseudomonas aeruginosa	Meropenem	Pseudomonas aeruginosa	Meropenem

40.	Savitri Kademani	35	F	90327	right	2	LCP	42	Staphylococcus aureus	Gentamycin	Pseudomonas aeruginosa	Ciprofloxacin
41.	Rajkumar	35	M	15016	left	2	SCP	23	Staphylococcus aureus	Clindamycin	Staphylococcus aureus	Clindamycin
42.	Sneha Manganavar	14	F	14711	right	childhood	LCP	53	Staphylococcus aureus	Linezolid	Staphylococcus aureus	Linezolid
43.	Bouramma	60	F	381584	left	1	MCP	53	Klebsiella sps	Ciprofloxacin	Klebsiella sps	Ciprofloxacin
44.	Vaishali Hattalli	15	F	360338	right	3	SCP	26.6	E Coli	Meropenem	Pseudomonas aeruginosa	Meropenem
45.	Sangamesh Shahapur	45	M	379675	left	childhood	subtotal perforation	48	Staphylococcus aureus	Gentamycin	Pseudomonas aeruginosa	Ciprofloxacin
46.	Deepa	17	F	13240	left	2	MCP	20	Staphylococcus aureus	Linezolid	Staphylococcus aureus	Linezolid
47.	Laxman Chambar	38	M	341320	right	6	LCP	33	Staphylococcus aureus	Clindamycin	Pseudomonas aeruginosa	Ciprofloxacin
48.	Asif Kaladagi	21	M	332886	left	4	MCP	26.6	Staphylococcus aureus	Linezolid	Pseudomonas aeruginosa	Ciprofloxacin
49.	Shreeshail	26	M	337722	right	8	LCP	38	Klebsiella sps	Meropenem	Klebsiella sps	Meropenem
50.	Laxmi Anand	30	F	9080	right	2	MCP	20	Acinetobacter baumannii	Clindamycin	Staphylococcus aureus	Clindamycin
51.	Kavita Iranna Hadappad	27	F	322581	left	5	SCP	22	Staphylococcus aureus	Linezolid	Klebsiella sps	Cefepime
52.	Devakki	35	F	284158	right	childhood	MCP	40	E Coli	Ciprofloxacin	Klebsiella sps	Ciprofloxacin
53.	Mohan Hiralal Rathod	34	M	8179	right	1	subtotal perforation	90	Klebsiella sps	Ciprofloxacin	Pseudomonas aeruginosa	Ciprofloxacin
54.	Bahuraj Vithal Madar	12	M	8631	right	2	MCP	20	Staphylococcus aureus	Linezolid	Klebsiella sps	Meropenem
55.	Basavva Bidari	43	F	56810	right	1	MCP	36.6	Pseudomonas aeruginosa	Meropenem	Pseudomonas aeruginosa	Ciprofloxacin
56.	Divya	14	F	313567	left	childhood	LCP	32	Staphylococcus aureus	Linezolid	Pseudomonas aeruginosa	Ciprofloxacin
57.	Nakush Kashling Salagar	26	F	279272	left	5	subtotal perforation	45	Klebsiella sps	Ciprofloxacin	Pseudomonas aeruginosa	Ciprofloxacin
58.	Sidaray Ningappa Rugi	40	M	80833	right	1	LCP	40	Klebsiella sps	Meropenem	Klebsiella sps	Meropenem
59.	Geetha Shankar	30	F	8274	right	3	LCP	53.3	Staphylococcus aureus	Clindamycin	Staphylococcus aureus	Clindamycin
60.	Sharada Chavan	48	F	7481	left	4	SCP	20	Pseudomonas aeruginosa	Ciprofloxacin	Pseudomonas aeruginosa	Ciprofloxacin
61.	Shubam Rajput	25	M	213075	left	1	LCP	45	Pseudomonas aeruginosa	Amikacin	Pseudomonas aeruginosa	Amikacin
62.	Vijayalaxmi Talageri	8	F	244918	right	3	MCP	50	Staphylococcus aureus	Clindamycin	Staphylococcus aureus	Clindamycin
63.	Boramma Biradar	30	F	134404	left	1	LCP	41.6	Staphylococcus aureus	Linezolid	Pseudomonas aeruginosa	Ciprofloxacin
64.	Maulasab Babusaheb Lingasur	37	M	4611	right	4	subtotal perforation	46.6	Staphylococcus aureus	Clindamycin	Klebsiella sps	Meropenem
65.	Boramma Hanchanal	50	F	135990	right	2	LCP	36.6	E Coli	Meropenem	E Coli	Meropenem
66.	Mallu Madar	30	M	136278	right	childhood	SCP	20	Staphylococcus aureus	Ciprofloxacin	Pseudomonas aeruginosa	Ciprofloxacin
67.	Guruputra Mahadev Dhane	28	M	4709	left	1	SCP	15	Staphylococcus aureus	Clindamycin	Pseudomonas aeruginosa	Ciprofloxacin
68.	Rajashree Talawar	33	F	930	left	childhood	subtotal perforation	50	Pseudomonas aeruginosa	Meropenem	Pseudomonas aeruginosa	Ciprofloxacin
69.	Samarth Vittal Madaraki	14	M	80764	right	childhood	subtotal perforation	56	Pseudomonas aeruginosa	Meropenem	Pseudomonas aeruginosa	Meropenem
70.	Laxmibai Siddappa Somanal	33	F	80377	right	childhood	MCP	43	Klebsiella sps	Amikacin	Pseudomonas aeruginosa	Amikacin
71.	Yellavva Navalagi	50	F	68707	right	4	MCP	38.6	Staphylococcus aureus	Clindamycin	Pseudomonas aeruginosa	Meropenem

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