

**PREVALENCE AND AETIOLOGY OF OTOMYCOSIS
IN SREE MOOKAMBIKA INSTITUTE
OF MEDICAL SCIENCES**



Dissertation

Submitted to

THE TAMILNADU Dr. M.G.R MEDICAL UNIVERSITY

**In partial fulfilment of the requirements for
the award of the degree of**

M.S. ENT

BRANCH IV

MAY 2020

CERTIFICATE - I

This is to certify that this dissertation entitled “**Prevalence and Aetiology of Otomycosis in Sree Mookambika Institute of Medical Sciences**” is a bonafide record of the work done by **Dr. ADHAVAN E.** under the supervision and guidance of **Dr. K.P Gopakumar, M.S.,** Professor, Department of ENT, Sree Mookambika Institute of Medical Sciences, Kulasekharam. This is submitted in partial fulfilment of the requirement of The Tamilnadu Dr. M.G.R. Medical University, Chennai for the award of M.S. Degree in ENT.

Dr. K.P. Gopakumar, MS.,
[Guide]

Professor and HOD
Department of ENT
Sree Mookambika Institute of
Medical Sciences [SMIMS]
Kulasekharam [K.K District]
Tamil Nadu -629161

Dr. Nepolean MD.,
[Co-guide]

Professor
Department of Microbiology,
Sree Mookambika Institute of
Medical Sciences [SMIMS]
Kulasekharam, K.K District,
Tamil Nadu -629161

Dr. Rema. V. Nair, M.D., D.G.O.,

Director
Sree Mookambika Institute of
Medical Sciences
Kulasekharam,
Kanyakumari District
Tamil Nadu - 629161

Dr. Padmakumar M.S, Mch

Principal
Sree Mookambika Institute
of Medical Sciences
Kulasekharam,
Kanyakumari District
Tamil Nadu - 629161

Urkund Analysis Result

Analysed Document: adhavan rough.pdf (D56684947)
Submitted: 10/8/2019 6:38:00 PM
Submitted By: addyuths@gmail.com
Significance: 3 %

Sources included in the report:

DR. ROBIN RICHARDS M - ENT DISSERTATION - PLAGIARISM.doc (D31630935)
otomycosis dissertation.docx (D31075568)
Dr purva thesis.docx (D41777422)
<https://www.jrmds.in/abstract/mycology-of-otomycosis-in-a-tertiary-care-teaching-hospital-1362.html>
<https://www.ijmhs.net/journals-aid-84.html>
https://www.researchgate.net/publication/51057736_Otomycosis_due_to_Filamentous_Fungi
bd0ac248-46d4-46fd-a16d-c1e292a11848
3facea34-ea13-4363-8dca-6c9b8789c905
7616a98a-c19f-468d-9781-f290d7fdd6a8

Instances where selected sources appear:

24

CERTIFICATE II

This is to certify that this dissertation work titled “**Prevalence and Aetiology of Otomycosis in Sree Mookambika Institute of Medical Sciences**” of the candidate **Dr. ADHAVAN .E** with registration Number 221714451 for the award of **M.S.** in the branch of **ENT [Branch-IV]**. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **3%** percentage of plagiarism in the dissertation.

Guide & Supervisor sign with Seal.

[Dr. K.P Gopakumar, M.S.]

DECLARATION

In the following pages is presented a consolidated report of the study **“Prevalence and Aetiology of Otomycosis in Sree Mookambika Institute of Medical Sciences”** on cases studied and followed up by me at Sree Mookambika Institute of Medical Sciences, Kulasekharam from 2017-2020. This thesis is submitted to the Dr. M.G.R. Medical University, Chennai in partial fulfilment of the rules and regulations for the award of MS Degree examination in ENT.

Dr. ADHAVAN E.,
Post Graduate
Department of ENT,
Sree Mookambika Institute of Medical
Sciences,
Kulasekharam, Kanyakumari District.
Tamil Nadu 629161.

ACKNOWLEDGEMENT

I am ever so indebted to God Almighty for showering me with blessings at this time which guided me in taking the right path and helped to complete this study.

*I owe my sincere gratitude to the Chairman **Dr. C.K. Velayuthan Nair** and Director **Dr. Rema V. Nair, SMIMS**, for allowing me to undertake this dissertation and accomplish with much avidity.*

*I would like to express my deep and sincere gratitude to **Prof. Dr. K.P. Gopakumar, HOD of ENT, SMIMS**, and **Prof. Dr Napoleon, HOD, Dept of Microbiology** for guiding me from the very beginning of the study. Their constructive comments and personal guidance have provided a good basis for the present thesis. I am much honored to have had the privilege of working under their supervision. I would be truly blessed to be guided and taught by an eminent professor, guide and clinician and only hope to live to these standards paved by him.*

*I am sincerely grateful to our institutions Academic Coordinator, **Dr. Mookambika** and the Deputy Medical Superintendent, **Dr. Vinu Gopinath** for their aid and support in academics and hospital allocations.*

*I am immensely thankful to my Associate Professor, **Dr. Chethan Kumar**, and my assistant professors, **Dr. Fakhruddin Niaz and Dr. Kiren T.**, for their constant support extended to me. They gave suggestions and more ideas for better understanding.*

*I am greatly indebted to the **ENT Residents of SMIMS** who undertook in the upbringing of this study, **Dr. Noufal Mon, Dr. R. Praseeda Prasad**,*

Dr. Geogin George Thottan., Dr. S. Kingsly for their constant support, understanding and willingness to help at times of need and doubt.

I thank the Audiologist, Ms. Arathy of our department for her help in performing this study, bringing light on audiology and acoustics and giving a clear comprehension of the study course.

I express my sincere thanks to Dr. Padmakumar, Principal, Sree Mookambika Institute of Medical Sciences for his constant support and approval.

I am grateful for the support the staff especially Sr. Chitra, Mrs. Meena and interns who gave their time and support to the Department of ENT, SMIMS and for their thoughtfulness throughout the work of this study.

I am sincerely grateful to my patients who willingly cooperated by providing details necessary as per the study requirements, thereby helping me complete the study in due time.

Professional life is never complete with personal life support, which makes me greatly indebted and expressing my eternal gratitude to my parents Dr. S. Elumalai, Mrs K Poongothai (pillar of my life) Er. P. Uthandaraman, Mrs K. Chandramala. Then my everloving and always prays for my success Dr. Arun Chandar my brother and Dr Sangeetha Ranganathan my sister in law and Master Sreekoushal , friends, fellow postgraduates, other staffs of SMIMS for their everlasting love and support as it is the least form of gratification possible from my side.

Least but not forgotten, everyone other person who contributed unequivocally their time, support, knowledge and any other form assistance is immensely appreciated.

TABLE OF CONTENTS

Sl. No.	Contents	Page no.
1	Introduction	1
2	Aims and Objectives	5
3	Review of Literature	6
4	Materials and Methods	57
5	Results & Interpretation	60
6	Discussion	75
7	Conclusion	83
8	Summary	84
9	Bibliography	i - vii
10	Appendix	

LIST OF TABLES

Sl. No.	Tables	Page No.
1	Symptoms	43
2	Signs	45
3	Isolates 01	49
4	Isolates 02	50
5	According to Gender and Age	60
6	Laterality of the Ear	61
7	According To Socio Economic Status	62
8	Variation of fungi and Month	63
9	According to presenting Symptoms and Number of patients	65
10	According To Pre Disposing Factors	66
11	Wax Presence	68
12	According To Specimens Sent For Culture	69
13	Organism Growth Distribution	70
14	Pre Disposing factor and growth	72
15	Associated Growth	74

LIST OF FIGURES

Sl. No.	Figures	Page No.
1	Aspergillus Niger – spores and Microscopic structure	11
2	Microscopic structure of Aspergillus fumigatus	12
3	SDA dish of Flavus and Microscopic structure	13
4	Candida species in dish	14
5	Filaments in microscope	15
6	Penicillium in SDA Dish and Microscopic structure	16
7	Pseudallescheria boydii life cycle	17
8	Malassezia in SDA- dish and Microscope structure	18
9	Six Hillocks and Formation of Adult Ear	20
10	Gross anatomy of pinna & parts	21
11	Blood supply of the pinna	24
12	The parts of the ear the outer middle and inner ear	25
13	Anatomy of middle ear	28
14	Otoscopic finding dry white fungal plug	44
15	Otoscopic finding shows white and black heads	44
16	Otoscopic finding shows black heads most probably Aspergillus	44
17	Otomycotic debris with tympanic perforation	44
18	According to Gender vs. Age	60
19	Laterality of the Ear	61
20	According to the Socio Economic Status	62

21	Variation of fungi vs. Month	64
22	According to presenting symptoms and Number of patients	65
23	According To Pre Disposing Factors	67
24	Wax Presence	68
25	According To Specimens Sent For Culture	69
26	Organism Growth Distribution	71
27	Pre Disposing factor and growth	73
28	Associated Growth	74

LIST OF ABBREVIATIONS

+FE	Positive for Fungal elements
ANIT-BIO	Antibiotic drops`
ANTI-BS	Antibiotics + steroid combination
Black-Myco Plug	Black mycological plug
CONGE-TM	Congested Tympanic Membrane
CSOM	Chronic suppurative otitis media
Dry MM	Dry mycelial mat
DNS	Deviated nasal septum
FA	Farmer
GR	Good response
HIV	Human immunodeficiency virus disease
HW	Housewife
PENICI-N	Penicillium Notatum
MIW -	Miscellaneous indoor worker
ASP -	Aspergillus
C albicans-	Candida albicans
P. Mirabilis	Proteus mirabilis
PR	Poor response/Same response
R mastoid cavity	Right mastoid cavity
SDA	Sabouraud's dextrose agar
WMM	Wet Mycelial mat
YELLOW SOFT D	Yellow Soft Debris

ABSTRACT

Introduction

Fungi constitute a large diverse group of about 120,000 heterotrophic organisms that differ characteristically from plants, bacteria and other protists in that they contain chitin in their cell walls. Most of the fungi are found as saprophytes in the soil and in decaying plant material, of which nearly 50,000 species have clinical importance¹. The virulence of the fungi is attributed to the fact that the fungus can grow at human body temperature (37°C) and the ability to produce various toxins and enzymes within the host and thus makes it pathological. Otitis externa can be a chronic or sub-acute, non-contagious, recurrent superficial mycotic infection of the external ear (pinna and external auditory canal or both). It can also infect the middle ear in cases of post-mastoidectomy or fenestration patients. However, deep fungal infections and systemic mycosis involving the ear are extremely uncommon.

Aims Methodology

The study was a hospital-based cross-sectional study conducted in the department of ENT, Sree Mookambika Institute of Medical Sciences Hospital, Kanyakumari from December 2017 to October 2019 (approximately 18 months). A total of 50 patients with the suspected otoscopic evidence of fungal debris were selected through clinical history, the predisposing factors and examination under 10% KOH study. Microscopy was done for the etiology of common isolates; the same was sent for fungal cultures.

Risks and Benefits of the Study:

- Benefits: Appropriate early diagnosis of causative factors for SNHL in diabetics to ensure prompt and effective management and to avoid or minimize the occurrence of complications.
- No risks so far have been detected following the study.

Conclusion

The incidence of Otomycosis is more during monsoon period. Females are more affected. Unilateral incidence of disease is common. CSOM is the most common predisposing factor. *Candida albicans* is the most common fungal isolate. *Aspergillus flavus* is the most common isolate in agriculturists and labourers (outdoor workers). *Aspergillus niger* is the common fungal isolate in females. Systemic illness play an important role in the recurrence of otomycosis. Inadequate aural toilet plays a role in recurrence.



Introduction

INTRODUCTION

The word fungus is directly derived from *Latin* which means ‘mushroom’ which was first used by Horace and Pliny¹. The word sponge was derived from Greek word ‘sphongos’ which refers to the microscopic structures. Fungi have worldwide distribution and grow in extreme environments from deserts to high salt concentration areas. Some can survive UV rays and cosmic radiation. Fungi constitute a large diverse group of about 120,000 heterotrophic organisms that differs characteristically from plants, bacteria and other protists in that it contains chitin in their cell walls. Most of the fungi are found as saprophytes in the soil and in decaying plant material, of which nearly 50,000 species have clinical importance¹. Fungi have fundamental roles in nutrient cycling and exchange in environment. Many species produce metabolites that are major sources of pharmacologically active drugs. Most important are the antibiotics such as penicillin G and other groups of penicillins and ciclosporin which is an antimetabolite. Fungi can also be used in day to day activities such as bread yeast which is essential food. Fungi are used extensively to produce industrial chemicals like citric, gluconic, lactic, and malic acids and industrial enzymes, such as lipases used in biological detergents and cellulases.

The virulence of the fungi is attributed to the fact that the fungus can grow in human body temperature (37°C) and the ability to produce various toxins and enzymes within the host and thus makes it pathological.

Otomycosis can be a chronic or sub-acute, non-contagious, recurrent superficial mycotic infection of the external ear (pinna and external Auditory canal

or both). It can also infect the middle ear in case of post mastoidectomy or fenestration surgery patient ². However deep fungal infections and systemic mycosis involving the ear are extremely uncommon.

Otomycosis is the fungal infections of external auditory canal which has worldwide distribution. However it is usually seen in the places of high humidity and in rainy places. Not only external environment but also various host conditions such as local or systemic may influence the occurrence of otomycosis. In this study we aimed to understand the various predisposing factors and the common isolates which are seen in this area². Along with symptoms, the clinical features along with otoscopic findings of the external canal may suggest otomycosis, but a proper identification of causative agents can be obtained by Fungal Culture which is beneficial in preventing recurrences and complications³.

Otomycosis is worldwide in distribution and it can occur from deserts to tropics and subtropical region. It is common as it contributes to about 5% to 20% of the all cases of infective otitis externa. It is one of the commonest manifestations in India, mostly in humid areas, especially during monsoons and rainy seasons. The fungal infection may resemble the desquamative form of diffuse otitis externa.

It is also called by several names-Singapore ear, hot weather ear, mildew ear, Otitis externa mycotica, Swimmer's ear, Tropical otitis, Panama ear, Abode ear, Mermaids ear.

Fungi can be broadly classified as moulds (filamentous fungi), yeasts or yeast like (dimorphic fungi).

We performed mycological analysis on 50 patients who attended our otorhinolaryngology outpatient department with suspected fungal debris from external auditory canal. In suspected case of otomycosis, sterile swab from the affected ear were sent for fungal cultures and studied. The objectives were to study the commonest modes of presentation, various types of isolates of fungi in this area, the predisposing factors, prevalence and etiology of the same. About 88% showed positive fungal cultures. Of these, a single isolate was found in 80%, mixed isolates were found in 8%, and 12% had no growth. The most common fungal pathogen found was *Candida albicans*, followed by *Aspergillus*. The causative factors for otomycosis were examined⁴. The prevalence of otomycosis has been reported to be as low as 9% of cases of otitis externa, and as high as 30.4% in patients presenting with symptoms of otitis externa. Prevalence is also influenced by the geographical area, as otomycosis is most commonly present in tropical and subtropical humid climates. It was more common in rainy season in the study which substantiates that it is common in humid conditions⁵. Otomycosis occurs more commonly in females than in males. Moreover it usually occurs most frequently in adults in the age group of 31-46 years and is less prevalent in children.

The most common fungal agents causing otomycosis are *Candida albicans*, *Aspergillus Niger*, *Aspergillus fumigatus*, *Candida tropicalis*, *Actinomyces*, and *Trichophyton*.⁴

Several factors can predispose to otomycosis which include super added bacterial infections usage of antibiotic and steroid combination ear drops , use of hearing aid, self-inflicted trauma like using a match stick or feather of birds to clean the ear, swimming in contaminated ponds, broad spectrum antibiotic therapy,

steroids and cytostatic medication, neoplasia and immune disorder. It is seen more frequently and more severely in immunocompromised patients compared to immunocompetent persons.⁵

The infection is usually unilateral and characterised by inflammation, itching, pain and suppuration with severe discomfort. Treatment options for otomycosis include elimination of predisposing factor such as foreign body, thorough canal cleaning and antifungal agents. Topical antifungals are specific (clotrimazole, miconazole, econazole, nystatin, tolnaftate, potassium sorbate), and non-specific (acetic acid, alcohol, boric acid, m-cresyl acetate, and gentian violet) and can be used for treatment of Otomycosis.



Aims & Objectives

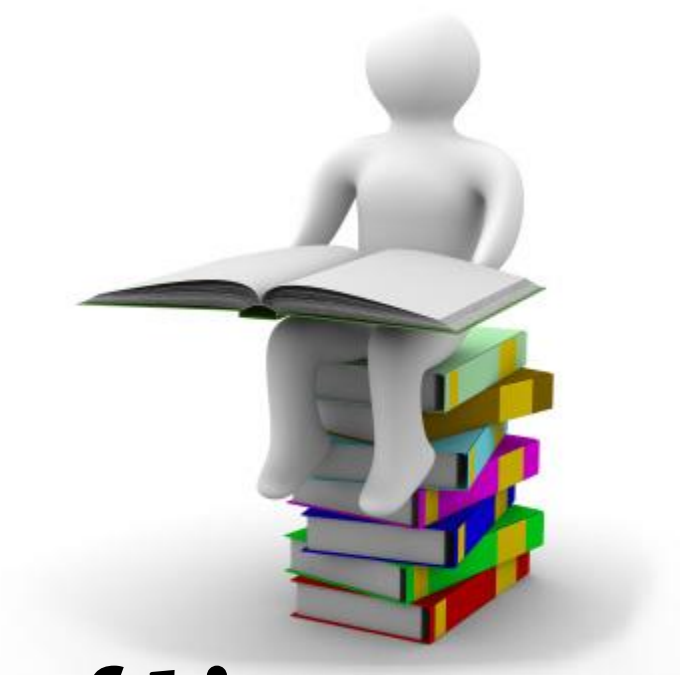
AIMS & OBJECTIVES

AIMS

- To study the prevalence of Otomycosis in patients suspected of otomycosis attending in our institution (Sree Mookambika Institute of Medical Sciences.)
- To identify the pre disposing factors involved in the cases of otomycosis.
- To evaluate the various presenting symptoms in patients with otomycosis.
- To identify common isolates which is prevalent in our institution and various etiology for the same.

OBJECTIVES:

- To study the prevalence of Otomycosis and its aetiology in patients attending in our institution (Sree Mookambika Institute of Medical Sciences.)
- To analyse the common causative organisms.



Review of Literature

REVIEW OF LITERATURE

Fungi have worldwide distribution and grow in extreme environments from deserts to high salt concentration areas. Some can survive UV rays and cosmic radiation. Fungi constitute a large diverse group of about 120,000 heterotrophic organisms that differ characteristically from plants, bacteria and other protists in that it contains chitin in their cell walls. Most of the fungi are found as saprophytes in the soil and in decaying plant material, of which nearly 50,000 species have clinical importance¹.

Fungi have fundamental roles in nutrient cycling and exchange in environment. Many species produce metabolites that are major sources of pharmacologically active drugs. Particularly important are the antibiotics such as Penicillin G and other groups of Penicillins and Cyclosporin which is an antimetabolite are also derived from fungi. Fungi can also be used in day to day activities such as bread yeast which is essential food. Fungi are used extensively to produce industrial chemicals like citric, gluconic, lactic, and malic acids, and industrial enzymes, such as lipases used in biological detergents and cellulases.

The virulence of the fungi is attributed to the fact that the fungus can grow in human body temperature (37°C) and the ability to produce various toxins and enzymes within the host and thus makes it pathological.

Definition:

It can be defined as a chronic or sub-acute, non-contagious, recurrent superficial mycotic infection of the external ear (pinna and external Auditory canal or both). It can also infect the middle ear in cases of post mastoidectomy or

fenestration surgery². However deep fungal infections and systemic mycosis involving the ear are extremely uncommon.

Synonyms:

- Singapore ear
- Otitis externa mycotica
- Hot weather ear
- Mildews ear
- Swimmer's ear
- Tropical otitis
- Panama ear
- Abode ear
- Mermaid's ear.

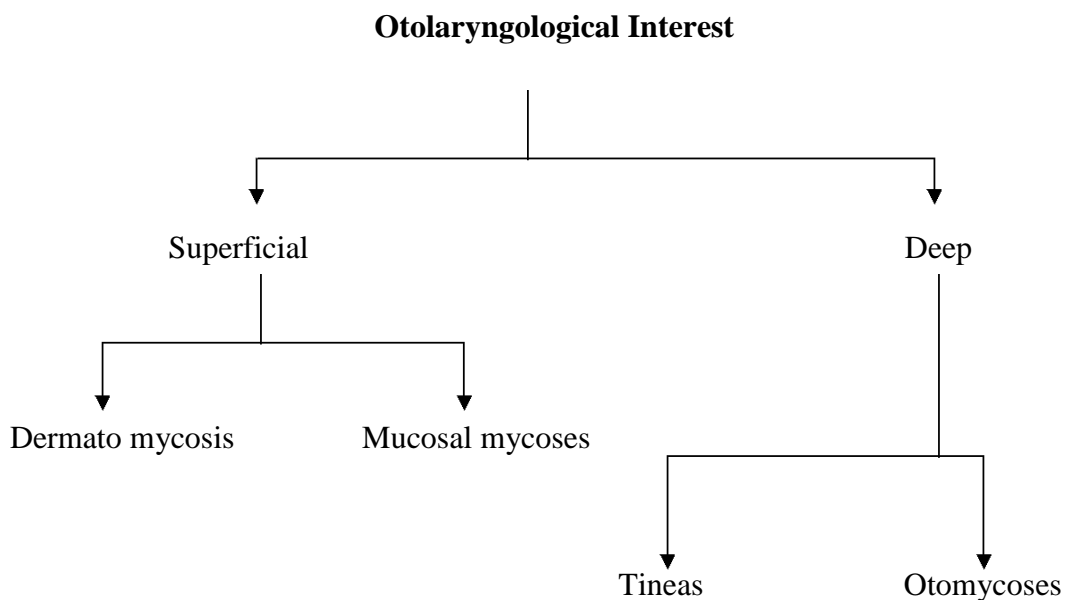
Classification of mycosis

Mycology is the branch of biology concerned with the systematic study of fungi, including their genetic and biochemical properties, their taxonomy, and their use to humans as a source of medicine, food, and psychotropic substances.

Fungal infections are more common and some of them are very serious and even fatal in immunocompromised individuals⁴. Further, the fungi that are able to cause disease in hosts is peculiar and unique to the species as it seems to have a peculiar trait of their metabolism which is not shared by taxonomically related species. Thus, the survival and growth of fungi at the elevated temperature of the body, the reduced oxidation-reduction environment of the time and the ability to overcome the host's defense mechanisms makes it more virulent and distinct from the virus and bacteria.

The best example is the dimorphic fungus which has transient adaptation to invasion and growth within tissue of the host. In nature they grow as soil saprophytes usually in a restricted environment producing mycelium and spores similar to other fungi. However, when their spores are inhaled or gain entrance to host by inoculation, by breach in the skin or trauma in to human or other animals, the organisms are able to adapt and grow in this different environment , causing a change in their morphology, metabolism, cell wall content, structure, enzyme systems and methods of reproduction.

The second major group of fungal diseases is the dermatophytoses. This is a closely related group of organisms with the ability to utilise keratin and to establish a kind of equilibrium albeit transitory with the host. Fungi are particularly remarkable for their ability to adapt and propagate in a wide variety of environmental situations.



Fungi can be broadly classified as moulds, yeast and yeast like (dimorphic having both mould and yeast form).

Organisms causing otomycosis

Hyaline moulds	Yeast like fungi
Aspergillus niger	Candida albicans
Aspergillus fumigates	Candida tropicalis
Aspergillus flavus	Candida krusei
Aspergillus terreus	Malassezia sympodalis
Aspergillus glaucus	
Penicillium species	
Pseudallescheria boydii	

ASPERGILLOSIS

Aspergillosis is broadly defined as group of disease caused by the members of the genus *Aspergillus*. Aspergillosis is one of the commonest etiologies of otomycosis. There are about 300 species of the genus *Aspergillus*.

They are abundant and most common on any environment. They are not only common in the soil and decaying vegetation throughout the world, but are also found on all types of organic waste from food left overs to catheter bags.

Aspergillus spores are air borne and constantly inhaled. After exposure to a cloud of *Aspergillus fumigatus* spores, the fungus can be recovered from the sputum for many days afterwards. *Aspergillus* organisms have been recovered from sulphuric acid, copper sulphate plating bath sand, formalinized pathology museum specimens⁶.

Although *Aspergilli* are constant in human environment, only about eight species have been consistently and authentically involved in human infectious diseases. They are *Aspergillus Niger*, *Aspergillus Flavus*, *Aspergillus Nidulans*,

Aspergillus Fumigatus, *Aspergillus Terreus*, *Aspergillus Niveus*, *Aspergillus Restrictus*, *Aspergillus Clavatus* and *Aspergillus Amesteloidami*. *Aspergillus Niger* is frequently seen in otomycosis.

Aspergillus Fumigatus accounts for almost all diseases both allergic and invasive fungal infestations.

Aspergillus species are thermotolerant. *Aspergilli* are remarkably adaptable and transformed themselves within the host or any environment to become pathogenic. *Aspergilli* have a marked tendency to invade blood vessels forming Thrombosis.

ASPERGILLUS NIGER (VAN TIEGHEM 1867)

Colony Morphology: culture on Sabouraud's Dextrose Agar (SDA) or Czapek's Solution Agar at 25°C: It grows to form a restricted colony with a diameter of 2.5-3.0 cm in ten days. The compact basal mycelium is white to yellow and soon bears abundant conidial structures which are black. The conidial spore heads are large, black and glabrous at first becoming radiate or splitting to loosely columnar. There is a distinct mouldy odour produced by the fungus.

Microscopic Morphology-

The conidiophores are 1.5-3.0 mm by 15-20 microns, smooth, colourless or turning dark towards the vesicles. The vesicles are globose about 60 microns in diameter and bears terigmata all over the surface.

Aspergillus Niger is more common and easily identifiable. They appear white to yellow mat like and the tips bear black conidia. It causes Aspergilloma, and is the most frequently encountered agent in otomycosis.

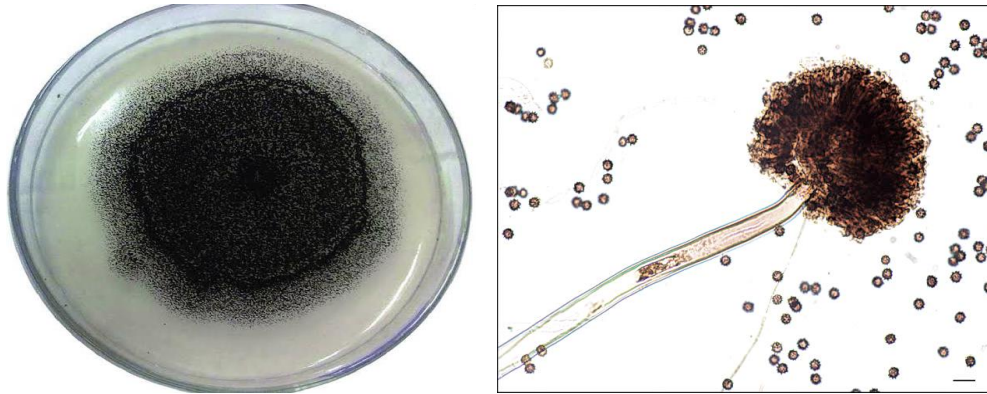


Fig 1: Aspergillus Niger – spores and Microscopic structure

ASPERGILLUS FUMIGATUS (FRESENIUS1850)

Colony Morphology: The organism grows rapidly on Sabouraud's Dextrose Agar (SDA) or Czepak's Solution Agar at 25 - 37 °C. They produce flat white colony that quickly becomes grey green with the production of conidia. The texture may vary from strictly velvety to deep felt, floccose or somewhat folded. The reverse of the culture tube or plate is generally colourless. The spore mass of the conidial heads are columnar, compact and often crowded. They range in size from 200-400 microns by 50-70 microns.

Microscopic Morphology: The conidiophores are short, smooth and up to 300 microns in length and 5-8 microns in diameter. It may have slightly green or brownish coloration, especially towards the upper part near the vesicle. The conidiophore gradually enlarges passing imperceptibly to form the expanded flask shaped vesicle.

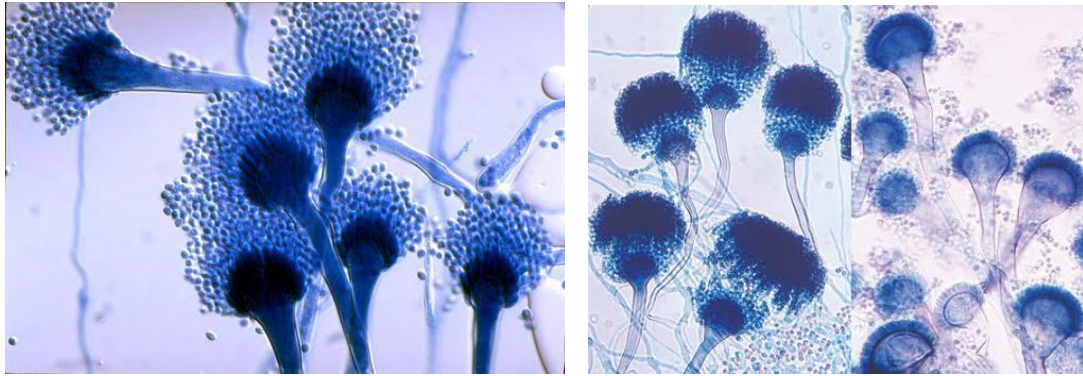


Fig 2: Microscopic structure of *Aspergillus fumigatus*

ASPERGILLUS FLAVUS (LINK 1809)

COLONY MORPHOLOGY: The organism grows on Sabouraud's Dextrose Agar (SDA) or Czapek's Solution Agar at 25°C. The growth is rapid (6-7cm in ten days) or slow (4 cm) and consists of a closed textured basal mycelium which is flat or radically furrowed or wrangled. Conidial heads are abundant and of intense yellow to yellow-green in colour. The reverse of the culture tube or plate is either pinkish or darker. Spore heads are radiate, splitting to form loose columns. They have an average diameter of 300-400 microns.

Microscopic Morphology: The conidiophores are thick walled, un-pigmented, and coarsely roughened up to 1mm length or more and 10-20 microns in diameter below the vesicles. Vesicles are globose to sub- globose. 10-15 microns in diameter and fertile (produces terigmata) over almost the entire area. The conidia are elliptical at first but later they are mostly globose (3.5-4.5 microns in diameter) and conspicuously echinulate.

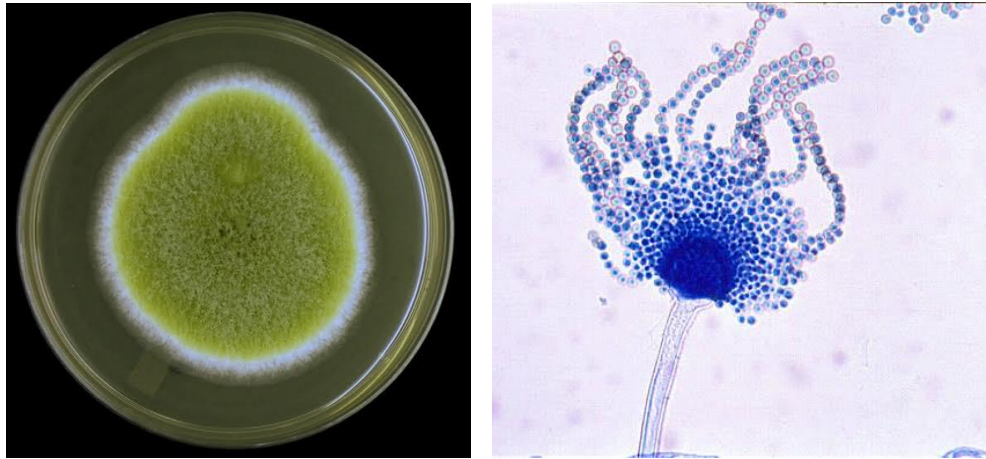


Fig 3: SDA dish of Flavus and Microscopic structure

ASPERGILLUS TERRUS (THOM 1918)

COLONY MORPHOLOGY:

The fungi grow on Sabouraud's Dextrose Agar (SDA) or Czepak's Solution Agar at 25°C. The growth of the colony is rapid reaching a diameter of 3-5 cm in ten days. The consistency is floccose to velvety. It may be furrowed or tufted sometimes and the colony can sporulate profusely. It grows in various colours from cinnamon to wood brown. The heads are 30-50 microns in diameter and 150-500 microns or more in length.

Microscopic Morphology: conidiophores are long and slender about 100-200 microns by 5-6 microns, smooth and uniform diameter throughout. The vesicles are hemispherical or dome like, about 10- 16 microns in diameter and merge imperceptibly with the conidiophores. Conidia are elliptical smooth and 2-2.5 microns in diameter. *Aspergillus Terreus* is another thermotolerant species that has been isolated from several cases of invasive Aspergillosis particularly in meningitis.

CANDIDA ALBICANS:

This is the only fungal species of importance to us in the genus *Candida*. These grow partly as spherical or as oval yeast cells measuring 2.5 X 4.0µm in diameter. These produce by budding and partly as a pseudo mycelium of non-branching filamentous cells which divide by constriction and budding from the division sites giving rise to yeast cells. Both these forms are thin walled, Gram positive and are not capsulated¹³.

Candida species are a common commensal of man and animals in the mouth, nose and throat. Infection is usually endogenous in origin, occasionally it can be exogenous. It may spread by contact or by air borne through infected dust.



Fig. 4: Candida species in dish

Laboratory diagnosis includes examination of the fungal plug on slide with 10% KOH. As these species may occur as commensal, demonstration of the same is not a final diagnosis.

Naked eye examination of a 48 hour SDA slope or plate reveals the presence of raised white moist and creamy colonies.



Fig. 5: Filaments in microscope

PENICILLIUM SPECIES⁷:

There are many species in the genus *Penicillium* found in the environment which are taken as common types of laboratory contaminants. Their main role is found in the production of antibiotics such as *Penicillium* group which is the most commonly used antibiotics in the world, some of the species are notorious and can cause mycotoxicosis also.

The growth of *Penicillium* species is rapid to develop colony and conidiophores arise in various forms producing phialides singly or in groups or from branched metulae giving brush like appearance. The conidia are unicellular and in chain with youngest at the base.

Penicillium marneffe is an exceptional species of this genus being as it is a dimorphic fungi. It is a primary pathogen in the immunocompromised patients in Southeast Asia and rest of the species are not that clinical significance. They have also been isolated in immunocompromised patients.

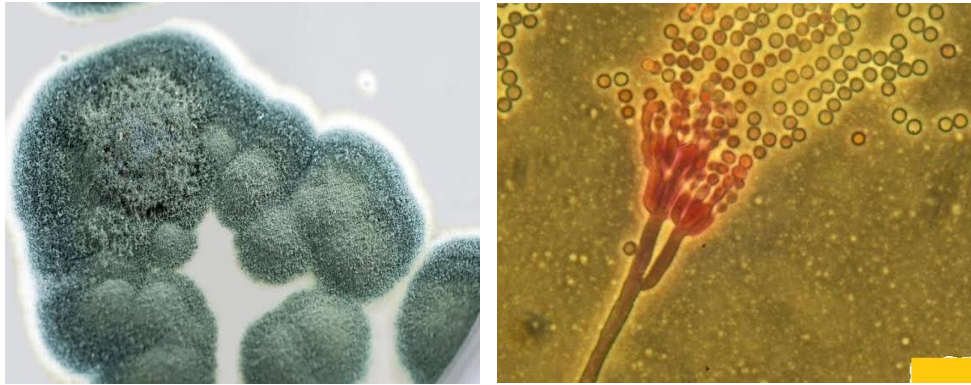


Fig. 6: Penicillium in SDA Dish and Microscopic structure

PSEUDOALLESCHERIA SPECIES⁷:

Pseudallescheria species *P. boydii* is a nascomycete and belongs to family microascaceae of the order microascales of phylum scomycota.

P. boydii is a soil saprophyte and in many other natural substrates. Its clinical importance is that now a days it has a tendency to produce opportunistic severe systemic fungal infection in immunocompromised patients. Its pattern of infection is very similar to that of *Aspergillus* species with invasion of blood vessels causing thrombus, respiratory tract producing *Aspergillomas* like fungi ball, with variable virulence in healthy individuals. It can also cause otitis externa and otomycosis and keratomycosis. It sometimes resembles and mistaken for *Aspergillosis*, particularly in the ear. It causes fungal ball in paranasal sinuses and can penetrate the meninges. It can cause white grain eumycetoma and endophthalmitis.

Laboratory diagnosis of *Pseudallescheria boydii* is a cottony fluffy white mold that rapidly turns brownish grey (mousy) with age, but the colony is black.

Microscopically septate hyphae with oval or club shape are seen. It produces elliptical, sperm shaped, single- celled conidia borne singly from the tips of long or short conidiophores.

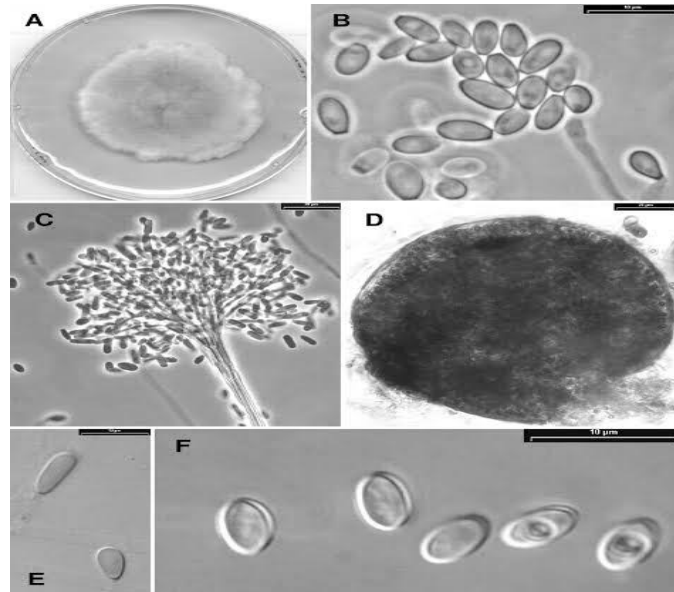


Fig. 7: Pseudallescheria boydii life cycle

Treatment of pseudoallescheriasis is similar to that of aspergillosis. However *P.boydii* may be found to be resistant to amphoterecin B and flucytosine. It is susceptible to the triazoles, itraconazole is the drug of choice.

MALASSEZIA SPECIES⁷:

Malassezia sympodalis:

Malassezia is a very important in causing different types of superficial cutaneous as well systemic fungal infections.

Malassezia species are lipophilic unipolar yeasts commonly recognized as commensals of normal skin in the areas rich in sebaceous glands in mammals and other animals that may be the source of infection under suitable conditions, usually causing skin conditions like pityriasis versicolor and otomycosis and otitis externa.

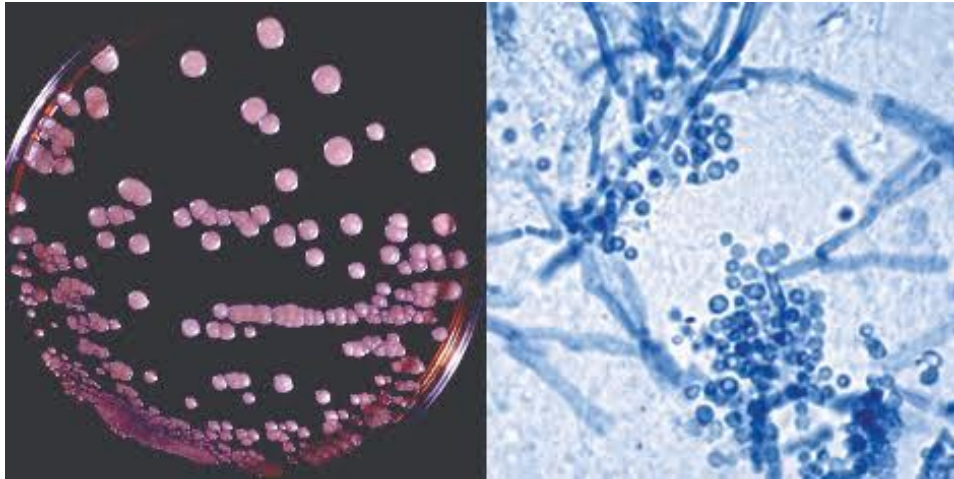


Fig. 8: Malassezia in SDA- dish and Microscope structure

The genus malassezia has undergone a wide variety of change since the recent past and its taxonomical status and understanding of pathogenesis has been highlighted. The species grow readily on skin surface rich in sebum consisting of cholesterol, squalene, triglycerides and free fatty acids.

Clinically, macular, erythematous, hyper pigmented (chronic) or hypo pigmented (achromic) lesion with fine scaling are present in this disease, but pathogenesis of the pigment variation has not been clearly elucidated.

The diagnosis of malassezia infection is based on typical clinical picture, positive direct microscopy and woods lamp examination in which scaly lesion usually show golden yellow fluorescence.

Malassezia species are usually present in large quantity and clusters of round yeast cells, 2 to 7 μ m in size with occasional budding, are observed, in KOH mounts, banana and grapes or spaghetti and meat ball forms are seen characteristically in Wet KOH mounts.

Malassezia is a lipophilic fungus is grown in SDA agar with oleic acid, olive oil and glycerol monosterate.

They form 3-6 mm small cream coloured yellowish colonies, slightly raised with irregular edges.

Treatment is by Ketaconazole (2%), selenium sulfide, zinc pyrithione (1%).

Pre disposing factors⁸:

1. External factors such as environment can play an important role- climate; it is more common during rainy season and areas of high humidity.
2. Better self-hygiene and usage of ear buds and self-probing can lead to trauma.
3. Swimming in infected water or still water which is mostly contaminated with fungi
4. Bacterial or viral or self-trauma to the external ear or tympanic membrane which will lead to infection.
5. Immunocompromised individuals, such as HIV infection, uncontrolled Diabetes mellitus, chemotherapy or steroid therapy.
6. Prolonged usage of antibiotic and steroid combination eardrops or systemic usage of steroids in conditions of respiratory failure, emphysema and severe bronchial asthma.

Portal of Entry:

Directly through the skin, or self-trauma by a match stick or hair pins which patient commonly use, wherein the integrity of the skin is lost. The natural defense mechanism is lost causing the infection, the spores from air or from finger tips or nails may enter causing the disease.

ANATOMY OF PINNA AND EXTERNAL AUDITORY CANAL

The ear is constantly challenged by a variety of microorganisms such as fungi, virus, and bacteria, which is present in the external environment, it is contaminated by direct exposure or by break in the mucosa by injury through finger nail or match stick. Through evolution the organ has adopted many ways of preventing itself from disease. The accumulation of fungal debris in conducive environment allows colonization and infection of the ear. Understanding the principle of otomycosis and the diagnosis and treatment is important for understanding the microscopic anatomy and physiology of the external ear and factors that prevent the disease formation. The pathogenesis and the exclusive factor of fungi that affect the external ear are essential for the understanding of the disease.

A) DEVELOPMENTAL ANATOMY

The external auditory canal develops from the upper portion of first pharyngeal cleft and from the hillocks of HIS⁹. The groove extends as a funnel shaped tube to come in contact with the endoderm and intervening mesoderm. The endoderm is an extension of the pharyngeal pouch.

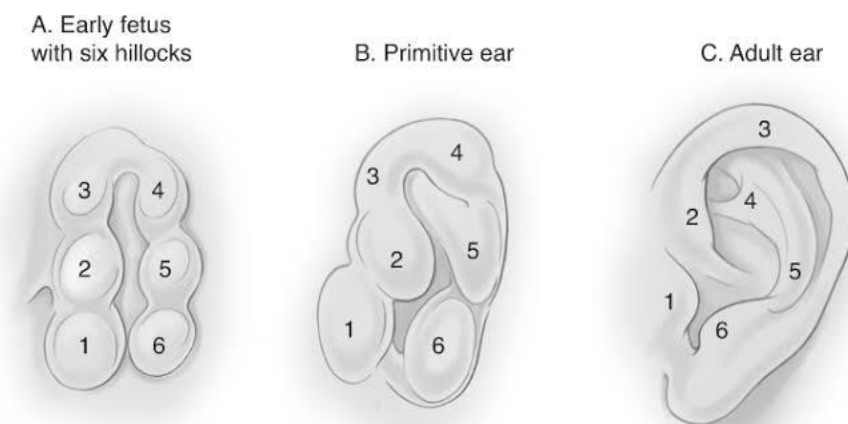


Fig. 9: Six Hillocks and Formation of Adult Ear

The intervening mesoderm later forms the middle fibrous layer of the tympanic membrane. The ectoderm in meatus proliferates to form an epithelial plug. The epithelial plug later on gives way and narrow slit forms between the future walls of external auditory canal and tympanic membrane⁹.

Within the mesoderm surrounding the deeper portion of the primitive meatus, four small centers of ossification develop. These later on fuse to form the tympanic bone. The cartilaginous part of meatus is formed by the extension of the developing auricular cartilage in to the future external meatus. The auricular cartilage develops from the fusion of six hillocks around the first pharyngeal cleft.

B) GROSS ANATOMY:

The Auricle is basically composed of fibro elastic yellow cartilage to which the skin and a small portion of subcutaneous tissue are closely attached. The cartilage nutrition is supplied by the perichondrium itself. The development of the cartilage is from inside to out which forms the outer part of the external canal. The length of the canal is around 24 -28 mm of which the outer 8mm is cartilaginous which is the area of our concern and the rest 16mm is bony, which extends up to the tympanic membrane.

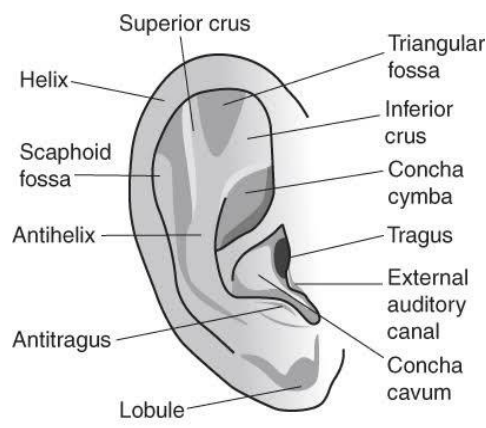


Fig. 10: Gross anatomy of pinna & parts

The epithelial lining of the canal is continuous with the epithelial covering of the auricle and outer layer of the tympanic membrane. There is a very thin layer of subcutaneous tissue between the skin and the cartilage in the lateral portion of the canal, but almost no subcutaneous tissue between the skin and bone of the medial part of the canal. The canal is obliquely placed with the anterior wall projecting medially about 4mm beyond the posterior wall. This end is marked, except at its upper part, by a narrow groove formed by the tympanic sulcus to which the circumference of the tympanic membrane is attached. Its lateral end is dilated and rough in the greater part of its circumference for the attachment of the cartilaginous meatus¹⁰.

The anterior, inferior and most of the posterior parts of the osseous meatus are formed by the tympanic element of the temporal bone. The posterosuperior region of the osseous part is formed by the squamous portion of the temporal bone.

The skin which envelops the auricle is continued into the external acoustic meatus and covers the outer surface of the tympanic membrane⁹. It is thin, shows no dermal papillae on sections and is closely adherent to the cartilaginous and osseous parts of the canal, Hence inflammatory conditions are extremely painful owing to the increased tension in these tissues.

The anterior wall of the cartilaginous part of the canal may have a dehiscence's known as the fissures of Santorini; these may allow spread of infection from canal wall into preauricular, parotid or temporomandibular joint. Medial to the bony cartilaginous junction is called isthmus, it is a narrow passage where foreign body or wax can get trapped.

Medial to the isthmus the canal converges and a space lateral to the tympanic membrane is the inferior tympanic recess. This space is important in the pathogenesis of otomycosis, because it is a region where the debris is difficult for cleaning or suctioning so the debris gets accumulated and aids in the pathogenesis.

ANATOMICAL RELATION OF THE CANAL WALL:

1. Superiorly and medially it is separated from the middle cranial fossa by a thick plate of bone
2. Posteriorly separated from mastoid cavity by the posterior portion of the tympanic bone.
3. Anteriorly is related to the glenoid fossa of the Temporal Mandibular joint and parotid gland ⁹.
4. Inferiorly is related to the infra-temporal fossa

NERVE SUPPLY OF EXTERNAL EAR:

SENSORY SUPPLY OF AURICLE:

- Medial surface :- Greater auricular (C2-C3) and Lesser Occipital nerve
- Anti- Helix and Concha -Auricular branch of Vagus.(Arnold's Nerve)
- Tragus, Crus of Helix and adjacent antihelix - Auriculo temporal nerve
- Small area in the root of Concha -Facial nerve.

SENSORY INNERVATION OF EXTERNAL AUDITORY CANAL:

- Anterior and superior quadrant of the canal-branch from the Auriculo temporal nerve.
- Posterior quadrant of the canal and floor: ARNOLDS NERVE OR ALDERMANS NERVE (Auricular branch of Vagus).

BLOOD SUPPLY OF EXTERNAL AUDITORY CANAL:

The arterial supply: the external meatus is derived from branches of the external carotid.-

- The auricular branches of the superficial temporal artery supply thereof and the anterior portion of the canal.
- The Anterior meatal wall, skin and the epithelium of the outer surface of the tympanic membrane is supplied by the deep auricular branch of the first part of the maxillary artery arises in the parotid gland behind the temporomandibular joint, pierces the cartilage or bone of the external meatus.
- Auricular branches of the posterior auricular artery pierce the cartilage of the auricle and supply the posterior portions of the canal⁹.

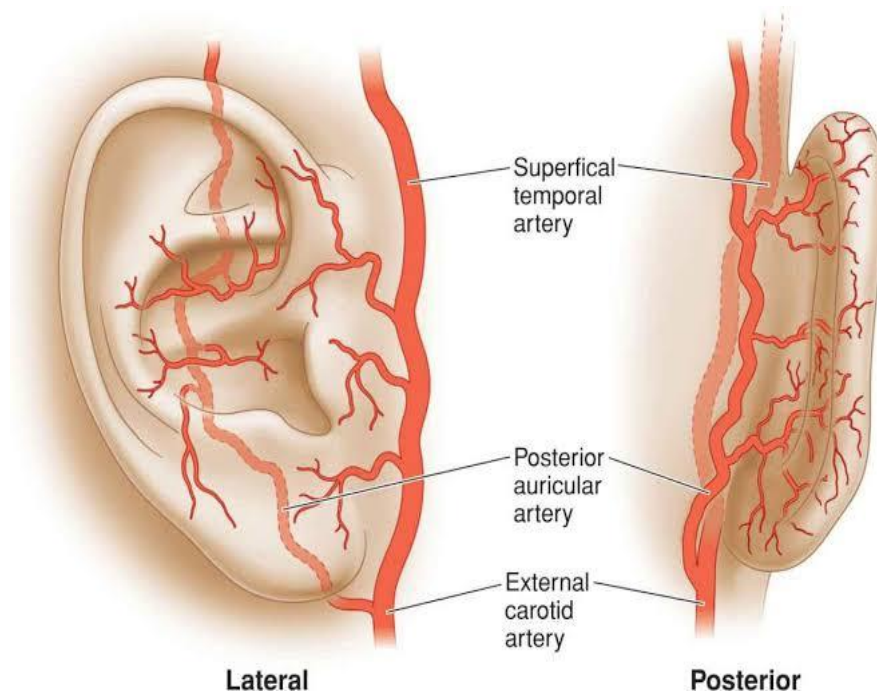


Fig. 11: Blood supply of the pinna

VENOUS DRAINAGE:

The veins drain into the external jugular vein, the maxillary vein and the pterygoid plexus of veins¹⁰. The veins accompany the arteries in course and name until they leave the region of the ear.

The superficial temporal vein combines with maxillary vein in the substance of the parotid to form the retromandibular vein. The retromandibular vein divides into two branches 1) an anterior which joins the facial vein to drain into the internal jugular and 2) a posterior which is joined by the posterior auricular vein just below the parotid gland to form the external jugular vein.

LYMPHATIC DRAINAGE⁹:

- Anterior and superior part of the canal drain into preauricular lymph nodes.
- Inferior part of the canal drains into infra auricular lymph nodes
- Posterior part of the canal drains into postauricular lymph nodes.
- Antihelix and concha drain into nodes at the apex of the mastoid process
- Superior aspect of the auricle drains into post auricular nodes.

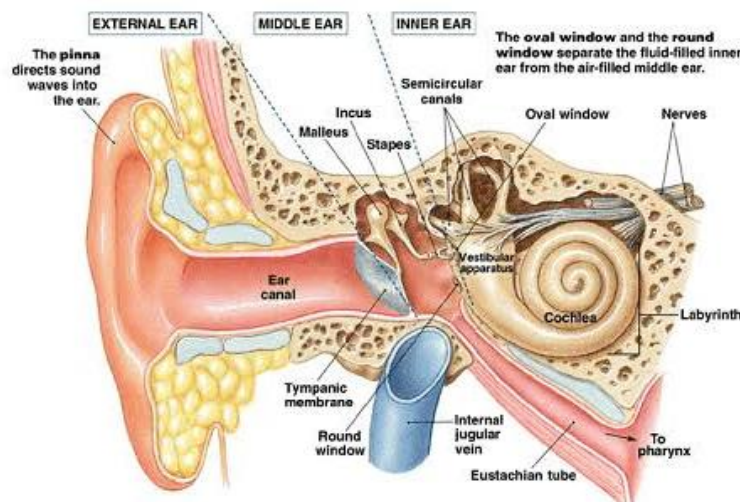


Fig. 12: The parts of the ear the outer middle and inner ear

E) MICROSCOPIC ANATOMY OF THE CANAL WALL:

The external auditory canal is made of the cartilaginous and the bony part. The cartilaginous part consists of the skin. The skin is made up of the hair, hair follicles and skin appendages. The hair follicles secrete sebum and cerumen via the sebaceous glands and the apocrine glands which is located in the epithelial layer. All the three appendages together called the apopilosebaceous unit. This produces water repellent waxy substance that protects the canal from the external invaders.

FUNCTIONS OF CERUMEN

Cerumen (ear wax) is a combination of secretions produced by sebaceous and apocrine glands admixed with desquamated epithelial cells. It forms a hydrophobic layer over canal skin, its acidic pH prevents the growth of microbes¹⁴. The presence of lysozymes & polyunsaturated fatty acids take over the microbicidal function.

Cerumen genetics and composition have attracted interest from otolaryngologists. The Native Americans have predominantly dry wax, a feature that supports their proposed migration from Asia. The concentration of DDT and other pesticides in cerumen has been used as a marker for occupational exposure. The biochemical analysis of cerumen by Nakashima in 1933 showed the presence of lipids as the major composition (46 -73 %), the remaining composition was formed by amino acids proteins and trace mineral ions. The fatty acids have been shown to have antibacterial activity in-vitro¹⁴. Driscoll and colleagues¹⁵ found that cerumen of diabetic patients was less acidic compared to that of non-diabetic controls, thus potentially favoring bacterial growth. However there was no significant difference in lysozyme or fatty acid concentration between the two groups.

In normal individuals, the ear and the EAC is self-cleansing and self-protective structure but various reasons can be subjective to the infection.

Absence of cerumen can predispose due to the following.

1. The self-removal of the wax using methods which can lead to injury to the skin and break in the natural defense mechanism causing the host be susceptible.
2. Cerumen naturally contains lysozyme which is antibacterial in nature so act of removing it can also predispose to the infection.
3. Cerumen also acts in the maintenance of the pH within the canal. So removal can raise the pH of the canal thus making it vulnerable for the infection.

ANATOMY OF THE MIDDLE EAR:

The middle ear cleft is made the following: 1.The tympanic cavity 2. Eustachian tube and 3.the mastoid air cell system.

Tympanic cavity:

The middle ear is a vertical air containing cleft in the temporal bone lying between the petrous portion which contains the cochlea and vestibule medially, and the tympanic membrane, with part of the squamous and tympanic portions laterally. Vertical, anterior and posterior diameter is about 15 mm each. The transverse diameter is 2mm mesotympanum, 4mm hypotympanum, and 6 mm epitympanum. The cleft communicates with the Nasopharynx through the auditory tube anteriorly and with the mastoid antrum through the aditus posteriorly. In clinical practice the middle ear cleft is taken to include the mastoid antrum and air cells. In section the cleft is hour glass shaped, being constricted, by the prominence of the first turn of the cochlea. At this point the distance between the medial and lateral walls of the

cleft is but 2mm, but the space widens above to 6 mm (epitympanic recess) and below to 4 mm (hypotympanum)¹¹.

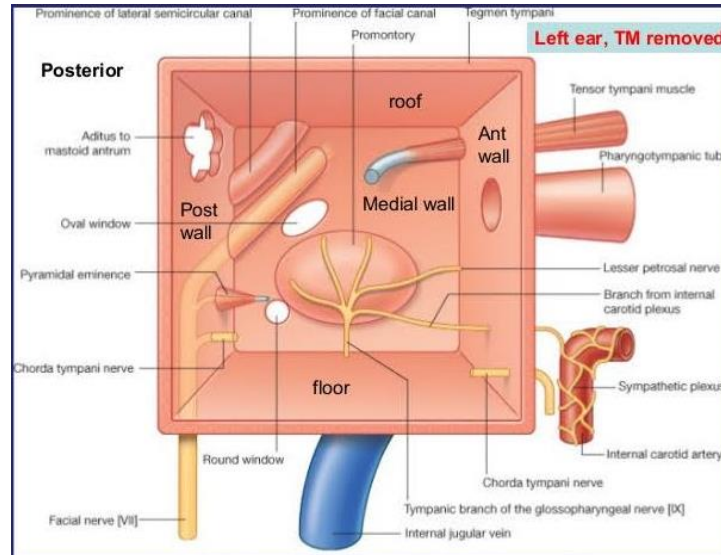


Fig 13: Anatomy of middle ear

Tympanic cavity:

The tympanic cavity is that part of the middle ear cleft situated between the tympanic membrane and the Cochlea. It is however, considerably bigger than the dimensions suggested by the tympanic membrane, having an upward extension behind the root of the external auditory meatus (epitympanic recess, attic) that accommodates the main body of the ossicles, and a variable extension downwards, below the level of the floor of the external acoustic meatus (hypotympanum) that usually contains air cells.

The cavity is irregularly quadrilateral with the shortest length anteriorly and allowing for variation, may be compared in size with a trimmed forefinger nail it is lined by a mucoperiosteal membrane, and filled with air, maintained from the nasopharynx via the auditory tube. Three small movable bones link the tympanic

membrane with the vestibule and together with their ligaments, muscles, and tendons and with chorda tympani nerve constitute the furniture of the cavity¹¹.

The Roof:

The anterior surface of the petrous portion of the temporal bone becomes thin where it joins the squamous portion, and at this point form the roof or tegmen, not only of the tympanic cavity but also of the mastoid antrum posteriorly and of the canal for the tensor tympani muscle anteriorly. The level and slope of the tegmen is approximately represented on the skull surface by the line of the lower border of the temporalis muscle and fascia where it is attached to the supramastoid crest and posterior root of the zygomatic process. Incomplete ossification of the petro squamous suture, as in early childhood, may allow passage of infection from the tympanic cavity or mastoid antrum to the middle cranial fossa, similarly venous channels that pass through this fissure may allow infection to reach the superior petrosal sinus. Persistent dehiscence in the tegmen have also been demonstrated¹¹.

The Floor:

Immediately below the level of the lower margin of the tympanic membrane is small thin bony plate, and beneath this only a thin convex plate separates the tympanic cavity from the superior bulb of internal jugular vein. The bone may be deficient in some cases, and the uncovered vein may come up to level of the membrane dangerously exposed position of middle ear disease and surgery. Antero medial to the vein the tympanic branch of the glossopharyngeal nerve enters the ear through the floor and middle ear from its origin¹³.

Anterior wall:

The floor of the middle ear slopes downwards and forwards parallel with the tegmen, medial and lateral walls coverage. The anterior wall which is vertical therefore angulates acutely with the floor and forms a hypotympanic recess where inflammatory secretions may accumulate. The position of the recess is indicated by the cone of light on the membrane, whose base points towards it. The anterior wall is extremely thin slat of bone which separates the cavity from the internal carotid artery. This slat of bone is perforated by the superior and inferior caroticotympanic nerves derived from the sympathetic plexus around the internal carotid artery sheath and by the Tympanic branch of internal carotid artery.

It consists of four openings

1. Canal of Huguier through which chorda tympani escapes from the middle ear.
2. The ascent of the artery in a medial and forward direction brings it to the medial side of the auditory tube and canal for the tensor tympani muscle, the openings of which constitute the upper features of the anterior wall.
3. The tympanic orifice of the Eustachian tube.
4. The Glasserian fissure containing the tympanic artery and the anterior ligament of the malleus, where a lateral angulation (processus cochleariformis) allows the tendon to pass laterally across the cavity to its insertion in the malleus neck¹¹.

Posterior wall:

The posterior wall is wider above than the anterior wall and resembles it in that the lower part is bony and the upper part open. The open approach (aditus) from the epitympanic recess to the tympanic antrum occupies the upper half of the posterior wall and the same line of division between pars flaccida and pars tensa of

the Tympani membrane, which when projected forward would enter the auditory tube, when projected backwards would pass into the aditus, below the aditus is conical projection called pyramid, which transmits the stapedial tendon to its insertion into the neck of stapes. At the pyramidal process the vertical portion of the facial nerve passes deep to the posterior canal wall. Lateral to the pyramid in the opening for the chorda tympani¹¹.

Lateral wall:

Lateral wall is mainly composed of the tympanic membrane. Posteriorly and anteriorly the membrane is close to the margins of the wall, but superiorly there is always an area of bone, called the scutum or outer attic wall. Inferiorly there is a small area of the down turned portion of the tympanic bone which separates the tympanic cavity from the medial part of temporomandibular joint¹³, the tympanic cavity is separated from external auditory meatus by the tympanic membrane, which forms the mesotympanum lateral wall and minor portion of epitympanum.

The fibrocartilagenous circumference of the pars tensa of the tympanic membrane is fixed into the tympanic sulcus. The upper limits of the sulcus are marked behind by the posterior canaliculus and in front by the anterior canaliculus of the chorda tympani nerve. The anterior canaliculus is placed at the medial end of the petrotympanic fissure. The visible aperture of which lodges the anterior ligament of the malleus and admits the anterior tympanic branch of maxillary artery¹¹.

Medial wall:

Medial wall is the lateral wall of the inner ear, it has two openings and several prominent features. Its central feature is the promontory formed by the first

turn of the cochlea. The surface of the promontory is grooved for the filaments of the tympanic plexus and below it the medial wall of the tympanic cavity is recessed and leads forwards, via communicating air cells, beneath the cochlea and behind and medial to the internal carotid artery, to the petrous apex.

In the posterior portion of this overhung recess the air cells give way to a hollow niche which is broadly tented towards the tympanic cavity so that its upper third looks downwards and its lower two thirds look backwards.

The hard bone of the otic capsule which is considered as hardest bone in the body which hangs round the niche of the fenestra cochleae somewhat like a horse collar and, at the top of the collar, the bone. The upper margin of the collar thus determines the uniform shape of the fenestra vestibule which is convex upwards with its long axis horizontal. The fenestra leads into the vestibule of the inner ear and is closed by the footplate of the stapes. Immediately above, the prominence of the horizontal portion of the facial nerve canal (canal of fallopius) demarcates the convex upper margins of the fenestra and turns vertically downwards at its posterior extremity. From this point, as previously noted, the nerve has a slight lateral and backward inclination that carries it into the posterior wall of the tympanic cavity behind the pyramid and below the fossa incudis. There is thus a recessed triangular area posterior to the round window whose apex is the posterior end of the oval window, and whose posterolateral leg is the diverging descending portion of the facial nerve. This pyramidal retractor sinus tympanic again, may have cellular communication with the mastoid process and is of significance in these pathology and surgery of tympanic cavity¹³. Immediately above the fenestra vestibule and immediately anterior to the point where facial nerve turns medially to penetrate the

inner ear may be seen the processes cochleariformis where, opposite the neck of malleus, the canal for the tensor tympani turns laterally and allows the tendon of muscle to change direction accordingly¹¹.

Blood supply¹¹:

There are six arteries of the tympanic cavity; **two** large and **four** small

- Anterior tympanic branch of maxillary artery supplies tympanic membrane.
- Stylomastoid branch of posterior auricular artery supplies **posterior** part of cavity and mastoid air cells.
- Superficial petrosal branch of middle meningeal artery, which enters through the hiatus for the greater petrosal nerve.
- Superior tympanic branch of middle meningeal artery enters via canal for tensortympani.
- Inferior tympanic branch from ascending pharyngeal artery accompanying the tympanic branch of the glossopharyngeal nerve.
- Carotico tympanic branch of internal carotid artery, passes directly into cavity through the anterior wall.

Venous drainage¹¹

The veins drain into the pterygoid plexus and superior petrosal sinus, the latter by leaving the region of the mastoid antrum and passing through the concavity of the superior semicircular canal to gain the subarcuate fossa.

Nerves¹³

The nerves are contributory to or derivative from a plexus which ramifies over the promontory. This forms the tympanic plexus consists of tympanic branch

of glossopharyngeal nerve and superior and inferior cartico tympanic branches of sympathetic plexus of the internal carotid artery.

PHYSIOLOGY OF EXTERNAL AUDITORY CANAL

a) Auditory function:

External auditory canal is essential for effective sound transmission. It conducts the sound from external area and transmits to the middle ear. The canal is equivalent to a resonating tube with one open end approximately 2.5 cm long & 0.7 cm in diameter, closed on other end by the tympanic membrane. In the external canal sound pressures are greater for certain frequencies, those whose wave lengths are multiples of the length of external canal. Hence for the external auditory canal this increase in intensity of sound would correspond to resonant frequency between 2600 Hz to 3000 Hz. These effects which have been investigated in animals, humans, and models by Stinson¹¹, vary markedly, with changes in size and contour of the canal and with frequency and angle of incidence of the arriving sound wave.

b) Non-auditory functions:

The hair follicles in the cartilaginous portion of the external auditory canal represent the secondary sexual characters. These prevent foreign bodies from entering the external canal. Hair follicles of external auditory canal are somewhat unusual, in that they do not have erector pili muscles⁸.

The cerumen gives a water proofing effect on the canal due to the hydrophobic action of lipids. The stagnation of water following swimming or use of topical oils or the effect of active CSOM macerates the canal wall skin and the further use of match sticks or hairpins to clean the canal wall hastens the entry of

pathogenic organisms. Cerumen maintains a low pH & contains anti-microbial compound such as lysozyme.

PATHOPHYSIOLOGY OF OTITIS EXTERNA

The microscopic anatomy of the external auditory canal plays a pivotal role in the prevention of any bacterial and fungal infections. The hair follicles, apocrine and the sebaceous unit helps in maintaining the homeostasis of prevention of the disease so any obstruction or inflammation of the pilosebaceous unit can lead to the infection. This is a main factor in the pathogenesis of otitis externa.

This obstruction occurs primarily from intracellular oedema, which is part of the inflammatory response to increased environmental temperature and humidity. The patient experiences the obstruction as a sense of fullness and itching. The usual response to itching is to scratch and this act disrupts surface epithelium allowing contamination by surface microbes.

The above causes the disruption in the host defence mechanism thus gives the opportunity for acute diffuse external otitis which is usually bacterial in nature, the fungal infections may be superimposed on previous bacterial infections or due to their treatment in form of antibiotic and steroid combination.

Senturia and associates¹⁸ have described several contributing factors in addition to the well recognised effects of heat, humidity and trauma, they noted that absence of the protective coating of cerumen particularly due to the removal of the lipid surface layer by repeated washing, cleaning the canal or swimming subjects the canal to greater risk of infection. In addition they noted that the pH in the external auditory canal is usually acidic, a fact that reduces susceptibility to

infection. Neutralisation or alkalisation of the canal impairs the natural ability to ward off bacterial or fungal contamination of the canal.

Senturia and associates¹⁸ divided the clinical manifestations of diffuse otitis-externa into three stages:

- a) Pre-inflammatory
- b) Acute-inflammatory
- c) Chronic stage

a) PRE-INFLAMMATORY STAGE:

The heat and humidity of the environment causes some inflammation and increases the permeability which in turn there is an increase in the aqueous content of the stratum corneum and produce intracellular oedema, perceived as fullness in the ear.

b) ACUTE-INFLAMMATORY STAGE:

Trauma to the epithelium, causes break in the continuity of the intact epithelium and subsequent microbial contamination and multiplication can produce pain in the auricular region. The auricle and tragus may be tender to manipulation. Examination of the canal frequently shows slight narrowing of the lumen due to edema of the external canal and the presence of clear odourless secretion with some accumulated debris. As the pain increases the canal skin is subjected to further trauma, the oedema, pain and tenderness increases and the discharge becomes purulent. In the most severe cases the lumen may be totally occluded by the oedema of the skin and sub-cutaneous tissue. Extension of infection beyond the canal lining may lead to periauricular cellulites, lymphadenitis and a marked increase in pain.

c) CHRONIC STAGE :

In the absence of totally adequate treatment a chronic condition may occur in which the skin of the canal is markedly thickened and feels indurated to palpation. Histologically, the skin demonstrates hyperkeratosis and acanthosis. The auricle and concha frequently show secondary changes.

Such as accumulated lichenification and superficial ulceration. This causes further edema and accumulation of fluids.

The treatment of diffuse bacterial external otitis is based on the stage of presentation. Generally the treatment is successful .In some cases the treatment may set a stage for the production of otomycosis. However in these conditions the fungi become the dominant microbial agents.

CLASSIFICATION OF OTITIS EXTERNA:

Mawson¹⁰ (1967) gave a comprehensive classification of otitis externa (Aetiological classification)

I) Infective:

a) Bacterial

- Impetigo contagiosa
- Retroauricular intertrigo
- Furunculosis
- Erysipelas of auricle(cellulitis)
- Diffuse otitis externa
- Lupus vulgaris(tuberculosis)
- Malignant otitis externa

b) Fungal:

- otomycosis

c) Viral:

- Herpes simplex
- Herpes zoster
- Otitis externa haemorrhagica.

II) Reactive:

- Eczematous dermatitis
 - a) Infective
 - b) Non-infective
- Seborrhoeic dermatitis
- Keratosis obturans
- Neurodermatitis
 - a) Pruritis
 - b) Neurotic excoriation (Purigo)
 - c) Dermatitis artifacta
 - d) Phobias
- Lupus erythematosus
- Psoriasis

Another classification of otitis externa [Scott and Brown]⁹ based on clinical factors is as follows.

CLASSIFICATION SUB-CLASSIFICATION

- i) Localised otitis externa. (furunculosis)
- ii) Diffuse otitis externa
 - a) Idiopathic
 - b) Traumatic
 - c) Irritant
 - d) Allergic
 - e) Bacterial
 - f) Fungal

Climatic/Environmental

- iii) Part of generalised skin conditions
- iv) Invasive otitis externa

Seborrhoeic dermatitis

- a. Allergic dermatitis
- b. Atopic dermatitis
- c. Psoriasis
 - Granulomatous, necrotizing or malignant otitis externa.
- v) Others: Keratosis obturans.

In 1942, William Gill divided otitis externa based on pathophysiology into three varieties.

1. Nonpathogenic otitis externa.
2. Pathogenic otitis externa
3. Mycotic otitis externa.

In 1945 dark classified otitis externa as:

- Edematous otitis externa
- Purulent otitis externa
- Granulomatous otitis externa
- Membranous otitis externa
- Haemorrhagic otitis externa.

Walter. B. Shelly and E. T. Peny ²¹ stated that otitis externa can be classified into:

1. Maceration dermatitis
2. Primary dermatitis
3. Allergic dermatitis.

PREDISPOSING FACTORS FOR OTOMYCOSIS

In normal individuals, the EAC is a self-cleansing and self-protective structure but various factors can predispose.

Absence of cerumen can predispose to infection due to the following.

1. The self-removal of the wax using by methods like using a match stick or hair pin or finger nail can lead to injury to the skin and break in the natural defense mechanism causing the host to be susceptible for fungal infection.
2. Cerumen naturally contains lyzosome which is antibacterial in nature so act of removing it can also predispose to the infection.
3. Cerumen also acts in the maintenance of the pH within the canal. So removal can raise the pH of the canal thus making it vulnerable for the infection.

Morrison & Mackay (1976)²², found a high incidence of excessive negative middle ear pressure in patients suffering from recurrent otitis externa. The

dysfunction of eustachian tube function and the negative pressure that persists, causes the patient to scratch his auditory canal.

Robert. C. Bruner and Leiland House²³ in the study of thermodynamics stated that the skin of the external canal is a dynamic structure within a certain limit of ambient temperature and humidity, it maintains an optimum moisture content and temperature. Dermatomycosis were observed in 34% of total cases in a study by K.Murat Ozcan²⁴ and same pathogenic fungi were isolated from ear. Vaginal mycosis was observed by Paulose et al in 3% of cases²⁵.

Fungal infection in otorhinolaryngology are often associated with decrease in immune function and are often regarded as colonisation rather than invasion and therefore not requiring treatment. This assertion is far from truth Gregson and LaTouche²⁶ suggested that active fungal infection, predominantly by species of aspergillus and candida, may be more common than is recognized and may explain failure to obtain dry ears in many cases of otorrhea. In CSOM, the development of otalgia, itching and the presence of hyphae indicate the presence of fungi. Recent reports of aspergillus involving certain areas in otorhinolaryngology, eg. Paranasal sinuses with intracranial complications, suggest that fungi can be pathogenic in ear infections as well fungi become pathogenic in an already inflamed mucosa unless they are treated when isolated in CSOM.

Then the environmental temperature exceeds 100° F and relative humidity exceeds 70%, the skin tends to become macerated predisposing this structure to infection.

According to Ediey Jones and Tom Norman³³ the known influence of temperature and exposure add support to the theory that the clinical picture of this type of acute diffuse external otitis may be varied in different geographical cases and climates.

CLINICAL PRESENTATION

CLINICAL FEATURES

Symptoms:

1. Nocturnal itching and pain worsens in night.
2. In case of aspergillus niger infection there is a lot of irritation. There is a sense of discomfort which is localised to deeper part of canal. Itching is more common in Candida.
3. Discharge may be present from metabolic product of the fungus. It may be persistent, Scanty, Colourless from the ear. In case of mixed infection there will be excessive discharge.
4. Ear discomfort is one of significant symptoms. It may associated with pain when combined with a superadded bacterial infection.
5. Headache may be associated with ear pain.
6. Fullness of ear due to edema and debris in the ear causing varying degree of conductive deafness.
7. There may be Vertigo and Tinnitus due to EAC pressure changes.

Signs:

- i) External examination of the ear is almost normal except it can cause ulcerations in severe cases on the lateral surface.

- ii) The external auditory canal may contain a mass formed of mixture of fungus, mucous, epithelial debris, exudates and cerumen.

Meyer was the first to describe the fungal infection in the ear in 1844 and this created a doubt that the fungal infections are secondary to the bacterial toxins or a primary infection on its own. Although some may disagree, the overwhelming evidence today confirms the notion that fungi can be primary pathogens.

Table 1: SYMPTOMS

Symptoms	Murat Ozcan et al ²⁴ (2003) (%)	Paulose et al ²⁵ (%)	Pradhan et al ²⁸ (2003) (%)	M. M. Yehia et al ⁴¹ (1990) (%)
Itching	95.4	88	93	78
Earache	54	70	86	51.4
Ear discharge	33.3	58	9.2	62.6
Hearing loss	47.1	33	96	38
Tinnitus	37.9	11.4	-	48

The study by Paulose & ET al²⁵ from Bahrain reported a series of 193 patients in whom the presenting symptoms noted was as follows;

- Itching 88% (170 patients)
- Blocked sensation of the ear 87% (168 patients) (Otagia)
- Discharge 30% (58 patients)
- Tinnitus & Deafness 11.4% (22 patients)

The same symptoms can occur in other inflammatory conditions of the external auditory canal. So a careful ear examination and cultures of the fungi are beneficial to make a definitive diagnosis.

The most frequent complaint in otomycosis is itching and tragal tenderness pain in canal^{8, 18, 19}; Patients have an irresistible urge to itch the canal with fingertip, match stick or any other instruments. As discussed earlier this causes sub epidermal invasion of the fungi. The itching and scratching in process can lead to dull and deep seated pain in the canal. This can be associated with scanty and foul smelling ear discharge. Accumulation of fungal debris in the canal and the edematous and congested canal can lead to conductive hearing loss.

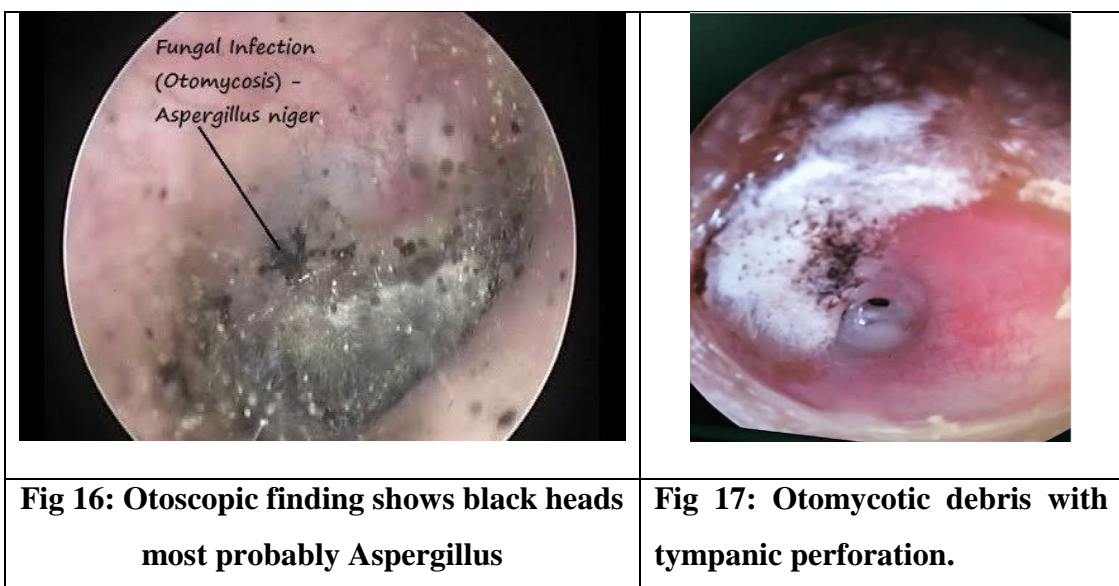
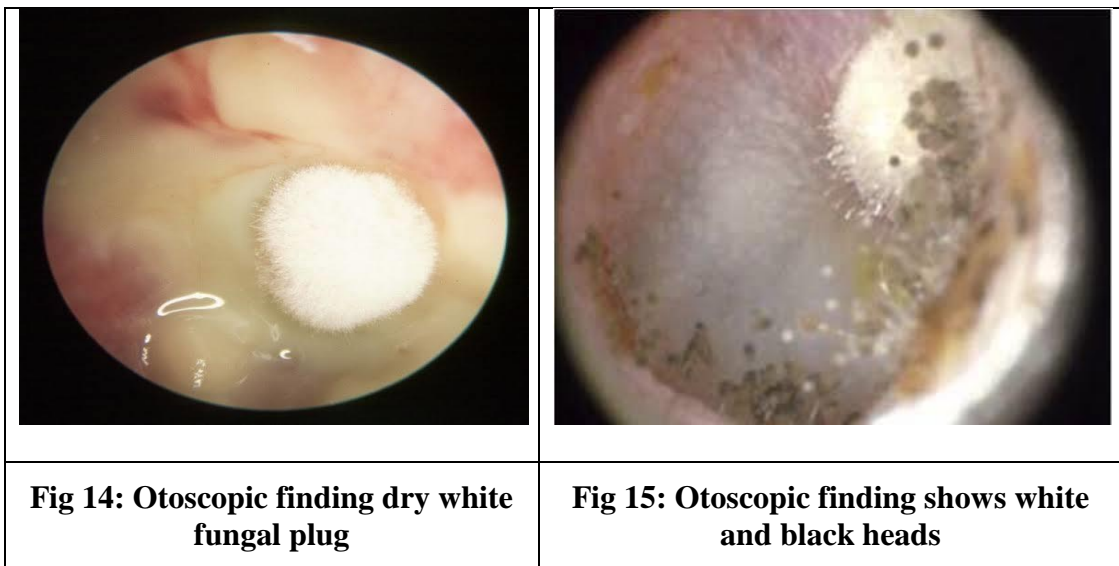


Table 2: SIGNS

Sl. No	SIGNS	Youseff and Abdou Cairo ³⁴	P. Kurnatowski and A. Filipiak ³¹
1	Pinna – Swelling and redness - Perichondritis		28.4%, 33.6%
2	Mycological presentation in external canal		
	a) Mycologic plug	65%	14.7%
	b) Wet mycelia mat	25%	
	c) Soft debris	10%	8.62%
	d) Dry mycelia mat	10%	
	e) Absence of wax	Absent	Absent
3	Tympanic membrane	16%	23.3%

INCIDENCE OF OTOMYCOSIS:

Many fungi have been implicated in the clinical disease process and as many as 61 different species have been identified in selected studies but the most common organisms are *Candida* species and *Aspergillus* species. In one large study of more than 12000 ears affected by external otitis, fungi were identified in over 10%.

Pathogens vary from temperate to tropical climates but the above two common species are found to affect in both climates. The wide spread incidence of otomycosis is not surprising because fungi especially *Aspergillus Niger* can be cultured from air and house dust. The External auditory canal maintains a perfect environment for the growth of fungi due mainly to the existing warm, humid environment. The distribution of fungal species in otomycosis depends on the geographic location. It was found that

Candida species predominated in the temperate climates whereas Aspergillus species dominated in the tropical climate. Many cases of otomycosis in the study were mixed infection of fungal and bacterial, the most common bacterial isolates being Pseudomonas species, staphylococcus aureus and Proteus species.

In a study of Beaney and Broughton³² have said that the greater frequency of infections are seen in the tropical countries due to changes in the composition of cerumen induced by sweating.

In the study conducted by Ferguson and Associates³³ had shown that cerumen can aid in the growth of fungi in-vitro. Initial symptoms of otomycosis and bacterial otitis externa are often indistinguishable although pain tends to be the dominant complaint in bacterial infections. The sensation of itching deep in the canal induces the patient to scratch ear canal with the fingertip or an instrument and this facilitates sub-epidermal invasion of the fungi.

Acute otomycosis tend to become chronic if the condition is misdiagnosed or unrecognized or inadequately treated. Secondary bacterial invasion can also compound the problem.

In a study of Oliveri³⁵ and Associates in Sicilian patients with otomycosis, found that apart from external environment, probing of the external auditory canal was the important risk factor for the development of the disease.

The use of topical antibiotic-steroid ear drops have found to be another risk factor for the development of otomycosis, but the experience neither of Oliveri and Associates nor of Muglistan and Donoghue³⁶ who noted that there was no significant rise in incidence of otomycosis by the use of topical steroid preparations but they

may disguise any allergic reaction to fungal toxins and associates³⁷ found in their study that 70% of patients had itching, discomfort in 54%, tinnitus in 50%, hearing impairment in 35% and ear discharge in 35% of patients.

PHYSICAL EXAMINATION AND DIAGNOSIS

On Examination in cases of otomycosis generally demonstrates canal congestion and edema with the presence of fungal debris within the canal. In initial stage of the disease the fungal debris in the canal wall may be bare minimum and then with progression of the disease the fungal debris become the mucous plug in form of cotton wooly mass or a wet newspaper like debris.

In the early stages of disease the fungal debris may be minimal and difficult to detect and remove from the canal. In long standing cases the debris may accumulate and it can totally occlude the canal depicting cotton like mass. Usually this fungal debris were identified to be adherent to the postero-superior osseous part of the external auditory canal wall.

The skin appears edematous and ulcerated. Aural toileting like suctioning or syringing of the ear reveals the congestion of the canal wall and bleeding epithelium.

The tympanic membrane is often not affected. The debris may have yellowish tinge when it is mixed with the cerumen or a bluish discoloration due to mixture of blood during instrumentation by an ear buds or match stick or trauma to canal wall. Sometimes otomycosis may be associated with bacterial infection which can further complicate the clinical diagnosis. The canal should be cleaned gently with suction, and complete cleaning of the canal can facilitate for the proper treatment of the condition.

Even adequate cleaning of the otomycotic ear may be enough for the treatment. Care must be given when the fungal debris must be removed from the anterior recess. During cleaning of the canal wall care should be given and moisture must be completely dried since moisture can elevate the growth of the fungi.

The various differential diagnoses should be kept in mind and a brief note of it is mentioned as follows

1. **Contact dermatitis**⁴⁴ is a type inflammatory skin disorder recognized as eczematous inflammation allergic or irritant skin reaction can occur in the external canal due to contact with an external agent like creams , soaps, chromium or nickel jewelry or antibiotics like Neomycin.
2. **Eczematous otitis externa**⁴⁵: a non-contagious inflammatory crusting disease of the skin in response to endogenous or exogenous stimuli, characterised by erythema, oedema, vesiculation of the epithelium.
3. **Seborrhic dermatitis**⁴⁶ is seen usually behind and below the ear lobes. It is caused due to dandruff. It forms crusts.
4. **Impetigo contagiosa**⁴⁷ is a chronic skin infection caused by streptococcus and staphylococcal infection. It enters into the superficial layers of the skin by minor cuts, and it is seen as reddish sores on skins and it progresses to a blister which is filled with serum. Later the blisters burst to exude serum which dries to form yellow crusts.
5. **Furunculosis**⁴⁸ is caused by staphylococcal infection of the hair follicle. It can be local or diffused. If local it is confined to the single site. In case of the diffuse the pain is whole ear. The pain is aggravated chewing and moving the jaw .As the

condition progresses the pain becomes more severe and the meatus may become occluded by the swelling causing deafness. In severe cases the oedema may spread to the post auricular sulcus producing forward displacement of the pinna. Examination of the ear reveals the tender swelling in the cartilaginous portion of the meatus with a normal deep canal and tympanic membrane beyond it. Tragal tenderness is present and there may be peri-auricular lymphadenopathy.

STUDIES ON TREATMENT:

In a study by Bavaja and P.L. Dhingra³⁸, aspergillus fumigatus, aspergillus Niger, Candida albicans, penicillium species and fusarium species were various isolates from external auditory canal. It is evident from observations that aspergillus fumigatus (52%) was the commonest Isolate followed by aspergillus Niger (28%). Candida albicans (12%), penicillin species (4%) and fusarium species (4%).

Table 3: ISOLATES 01

Isolates	Baveja et al³⁸ No. of isolates	Murat Ozcan et al²⁴ No. of Isolates
Aspergillus fumigatus	52%	17.9%
Aspergillus niger	28%	44.8%
Candida albicans	8%	11.9%
Penicilium species	4%	-
Fusarium species	-	-

Murat Ozcan and co-workers²⁴ in their study found that the most common pathogenic fungus was aspergillus Niger (44.8%) and concluded that administration

of 4% boric acid solution in alcohol and frequent suction cleaning of the ear canal might be a cost effective treatment for otomycosis since 77% of patients were treated effectively this way.80% of the resistant cases had mixed fungal bacterial infection and 50% of them vein had dermatomycosis. These resistant cases were treated by administration of tioconazole ointment.

Table 4: ISOLATES 02

Isolates	Joy et al 40 (1980)	Yassin et al 39 (1978)	Yehia et al 41 (1990)	Jaiswal (1990) 43	Pahwa et al 42 (1983)	Paulose et al 25 (1989)
A.niger	45.9	51.2	70.9	34.0	56.3	54.4
Mucor spp.	6.5	2.3	-	-	6.3	-
Candida spp.	8.7	4.6	7.3	46.0	6.3	17.0
A fumigatus	15.7	-	5.6	-	15.6	25.1
A flavus	24.3	18.3	15.6	-	4.7	-
Other Aspergillus	-	13.7	-	-	3.1	-
Rhizopus spp.	-	-	0.6	12.0	-	-
Penicillium spp	1.1	5.3	-	-	4.7	3.5
Other fungi	-	4.6	-	8.0	3.0	-

As noted in the chart above studies by different authors show those aspergillus groups are the most common fungi causing otomycosis.

No particular drug regimen has been proposed for the disease. Only systemic and complete cleaning of the external auditory meatus may be enough for the treatment of otomycosis.

Administration of mildly acidic drops, such as boric acid and alcohol or modified Burow's solution may be part of the initial therapy. Topical antifungals may be administered in resistant cases. When combined with suction cleaning of the ear canal. 4% boric acid solution appears to be a cost effective initial therapy.

Suction cleaning of the ear canal is very important and must be a part of therapy. There are no reports of ototoxicity with antifungals in the literature.

MANAGEMENT

1. Predisposing factors should be eliminated such as control of diabetes and to stop the instillation of antibiotic and steroid combination ear drops.
2. Removal of the fungal debris must be done. It is seen that any amount of debris can predispose for the formation of otomycosis. So aural toileting in form of gentle suctioning or dry mopping must be done with utmost care. If in case of furuncle or any other inflammatory condition of the ear it may cause then a course of systemic antibiotics and analgesics can be given and removal of the debris can be done.
3. A combination of Iodine ointment (2%) with Tannic acid (2%) can also give since its effective against Candida.
4. Instillation of topical antifungal combination ear drops must be given, which are
 - Clotrimazole: it is most common anti-fungal agent being used in day to day practice. It acts against Candida; aspergillus and dermatophytes .it is available as ear drops and lotions.
 - Nystatin: it is obtained from strptomyes. noursei it is available as ointment, cream, powder and tablet form.

- **MOA:** it acts on ergosterol in fungal cell membrane and forms pores in cell membrane. This causes the death of the fungi. It is more effective against Candida but not Aspergillus.

- Amphotericin B :

It is derived from *Streptomyces nodosus*

- **MOA:** it acts on ergosterol in fungal cell membrane and forms pores in cell membrane. This causes the death of the fungi.

Spectrum: aspergillum, blastomyces, Candida, cryptococcus. Coocidioides.

Available as 0.15% eardrops and as 3% solution cream for topical application.

- Econazole: it is used as solution and as 1% cream

Its spectrum is diverse it acts on Aspergillus, Candida and effective in immunocompromised patients.

- Miconazole: it has a same mechanism of action and same spectrum of action.

Other antifungal agents can be used are: Gentian violet drops can be used.

Natamycin ,ketoconazole, Whit field ointment, Tolciclat, Tioconazole, Fenticonzole, Oxiconazole, ciclopiroxolomine, Tolnafate, Flucytosine, Acetic acid, selenium sulfide, undecylenic acid, triacetin etc.

Prevention:

1. Keep ear dry: almost care to be given for water should be prevented from entering the ear
2. Restriction in usage of ear buds and other self-probing.
3. Usage of ear stoppers when entering to a swimming pool or any ponds so that the water doesn't enter the ear
4. To avoid over the counter usage of antibiotic or antibiotic and steroid combination topical drops for prolonged duration.

The breakthrough in fungal infection was first made by Lagenback in 1839. Among the numerous fungal diseases in man and animals Aspergillosis was the first to be discovered. Florentine botanist Michelli coined the term aspergilla in his “Nova Plantarium Genera” of 1729. Fungal infections of the external ear was initially discovered by Meyer in 1844. Meyer was the first person to provide strong evidence that fungal infections can be the primary pathogens in the ear rather than secondary pathogens.⁴

Meyer first described fungal infections of the external ear in 1844. He also sparked a long lasting debate about whether fungi are the primary infectious agents or are secondary pathogens that affect the skin of the external auditory canal after it has been exposed to bacterial toxins. There is overwhelming evidence to confirm the notion that fungi can be primary pathogens.⁴

Intractable otorrhoea which occurred either due to otitis externa or post radical mastectomy was a great challenge for cure until Gregson and La Touche suspected otomycosis as the cause for the resistant otorrhoea. Out of 180 patients they found 80 patients to have fungal infection, and temperate climates being a predisposing factor.

The prevalence rate of fungal otitis was studied by Ahmad Yegane Mogadam et al in 2009. The study included patients suffering from external otitis. Samples were taken using sterile swabs and identified using direct smear and cultivation techniques. Out of the 910 patients included in the study suffering 52 patients (5.7%) were diagnosed with otomycosis, the sex ratio was distributed as 16 patients (30.8%) males and 36 (69.2%) females. The most common causative organism being *Aspergillus Niger* (32, 61.5%) while only one case (1.2%) was

diagnosed to be Scopulariopsis. The study done in Kasahn which has a hot and dry climate. Also, result shows a high prevalence rate of *A. Niger* in otomycosis patients.⁵¹

H.S. Satish et al 2013, done a prospective study with 200 cases of clinically diagnosed otomycosis, of which 60 patients were immunocompromised. After a detailed history and clinical examination, required investigations were carried out to confirm the diagnosis. All patients were treated with topical Clotrimazole and 20 of them who did not respond to it were treated with Fluconazole. In this study, otomycosis was found to be more common among males (53%) and majority in the age-group 21-30 years (42%). The disease was predominantly unilateral (89%), but bilateral involvement was seen more in the immunocompromised group. *Aspergillus* species (77%) was the most commonly isolated fungus in the immunocompetent group while *Candida* (53.4%) was commonly isolated in the immunocompromised group. All the patients were treated with Clotrimazole eardrops. Twenty of the immunocompromised patients who did not respond to it were successfully treated with Fluconazole. Six of the immuno-compromised patients had TM perforation due to otomycosis. They concluded as Otomycosis is fungal infection of the external ear and infrequently affects the middle ear. It presents with symptoms of itching, ear discharge, blocking sensation, and earache. Important predisposing factors for otomycosis include trauma to the EAC, use of antibiotic/antibiotic-steroid eardrops, and immuno-compromised status. The disease is predominantly unilateral with bilateral involvement more in the immuno-compromised patients. *Aspergillus* species and *Candida* are the most commonly isolated fungi among the immuno-competent and immuno-compromised patients respectively. Complications occur in the immunocompromised patients.⁵²

In 2013 a study was undertaken by Gokale SK et al to determine the prevalence of various fungal agents and also the numerous predisposing factors involved in otomycosis. A total of 130 patients were suspected to have otomycosis and were included in the study and mycological examination was done [direct microscopic examination and culture]. 85% of the patients were diagnosed with otomycosis. The important and common predisposing factors that were identified from the study include Swimming, usage of oils and eardrop solutions. The most common complaints the patients presented with were Otagia and pruritus. The fungal agents that were isolated include *Aspergillus Niger* being the most common followed by *A. fumigatus*, *A. flavus*, *C. albicans*, *candida non albicans*, *Mucor* and *penicillium*. Hence it was concluded that otomycosis is very common in a temperate country like India clinical and mycological examination are very effective in diagnosis and treatment for otomycosis.⁵³

Metwally Abdelazeem et al 2015 in his study described Otomycosis as an external auditory canal fungal infection, with middle ear complications occurring infrequently. In the study they assessed mycological analysis of fungal debris from external auditory canals of 110 clinically diagnosed otomycosis patients. Fungal growth was identified for in the inoculated culture plates. The study showed that the most common fungal agent of otomycosis being *Aspergillus Niger*. Young males of age group 21-40 being the common candidates for otomycosis. The incidence of new cases being more in the summer and spring season. The common victims affected based upon occupation were manual workers and students. Pruritus was the commonest presenting chief complaint with trauma playing the most common predisposing factor. Different factors in the study population caused variations in the epidemiological profile of otomycosis infection.⁵⁴

The prevalence, predisposing factors and the presentation were all analysed by Surinder Singh et al in their study conducted in 2017. The study included 150 clinically suspicious patients of otomycosis and samples from their external ear were analysed for mycological infection. Mycological diagnosis confirmed otomycosis in 69.3% of the clinically suspected patients. The season of fall had the highest incidence, and again young males of 21-40yrs were the common candidates similar to the other studies. Occupation in a very dry and dusty environment was a major predisposing factor. Itching was the most common symptom among most patients. The commonest organism was found to be *Aspergillus flavus*(46.2%) followed by *A. niger*(38.4%), *Candida albicans* (7.7%), *A. fumigatus*(4.9%), *A. nidulans*(1.9%) and *C. parapsilosis*(0.9%). Hence they concluded that a high index of clinical suspicion of otomycosis is important to prevent unnecessary use of antibiotics. The fungal pathogens in dry dusty regions were found to be different from the ones in hot humid areas and this requires attention in future susceptibility tests which can improve the management of patients with otomycosis.⁵⁶



Materials & Methods

MATERIALS AND METHODS

METHODOLOGY

Study Design: Hospital based cross sectional study.

Inclusion Criteria:

- New Patients with otitis externa and otoscopic evidence of debris in external auditory canal.
- New Patients with CSOM and with otoscopic evidence of debris in external auditory canal
- Post mastoidectomy patients with new otoscopic evidence of debris in external auditory canal.

Exclusion Criteria:

- Patients with otitis external and without otoscopic evidence of debris in external auditory canal.
- Patients not available for follow-up study
- non cooperative patients

SAMPLING

Sampling Population: Patients with clinically suspected otomycosis.

Sample Size Calculation: It is calculated by the formula $4pq/d^2$

p=available local prevalence rate in target population (obtained from comparable studies)

q=100-p,d=relative precision i.e. 20% of previous prevalence

In this study,

$$N = \frac{4pq}{(d^2)}$$

p - 85% (according to Gokale SK et al 2013)⁵³

q - 15%

d - 15% of p = 12.75

Minimum sample size = 31.37

Sampling technique: Convenient Sampling.

INVESTIGATIONS

- History Taking
- Otoscopic Examination
- KOH study
- Fungal culture

STUDY PROCEDURE:

After acceptance of the study protocol by our Institutional Research & Human ethics committee, the patients that fulfill the inclusion criteria was included in the study. Written informed consent was taken and all the selected patients was explained in detail about the procedure. Proforma was filled for all the patients included in the study.

For patients presenting with suspected fungal debris in external auditory canal was selected. Under strict aseptic precautions, after swabbing the pinna and adjacent area of the ear with antiseptic, the debris in the external auditory canal was collected by moist sterile swabs or by sterile forceps. The collected specimen was promptly transported to the lab and processed on the same day. KOH mount was done and the various species of the swab was studied in this study. The KOH test for fungus is conducted on an outpatient basis and patients do not need to prepare in

advance. If fungal cultures are required, the test is performed by a technologist who specializes in microbiology.

Collection: a sterile swab is taken, the otomycotic debris is collected with utmost care and is being sent to a laboratory, the scrapings are placed in a sterile covered container. The scrapings are placed directly onto a microscope slide and are covered with 10% or 20% potassium hydroxide.

1. The slide is left to stand until clear, normally between five and fifteen minutes, in order to dissolve skin cells, hair, and debris.
2. To enhance clearing dimethyl sulfoxide was added to the slide. To make the fungi easier to see lacto phenol cotton blue stain was added.
3. The slide is gently heated to speed up the action of the KOH.
4. Adding calcofluor-white stain to the slide will cause the fungi to become fluorescent, making them easier to identify under a fluorescent microscope.
5. The slide was placed under a microscope to read, after that the data were collected and results were interpreted.

Sample size of each group: 50 (one group)

Total sample size of the study: 50

No risks so far have been reported in prior studies.



Results & Interpretation

RESULTS AND INTERPRETATION

Table 5: According to Gender and Age

S No	Age group	Male	Female	Total
1	<15	2(4%)	4(8%)	6(12%)
2	16-30	8(16%)	2(4%)	10(20%)
3	31-45	2(4%)	13(26%)	15(30%)
4	46-60	5(10%)	4(8%)	9(18%)
5	>60	4(8%)	6(12%)	10(20%)
Total		21	29	50

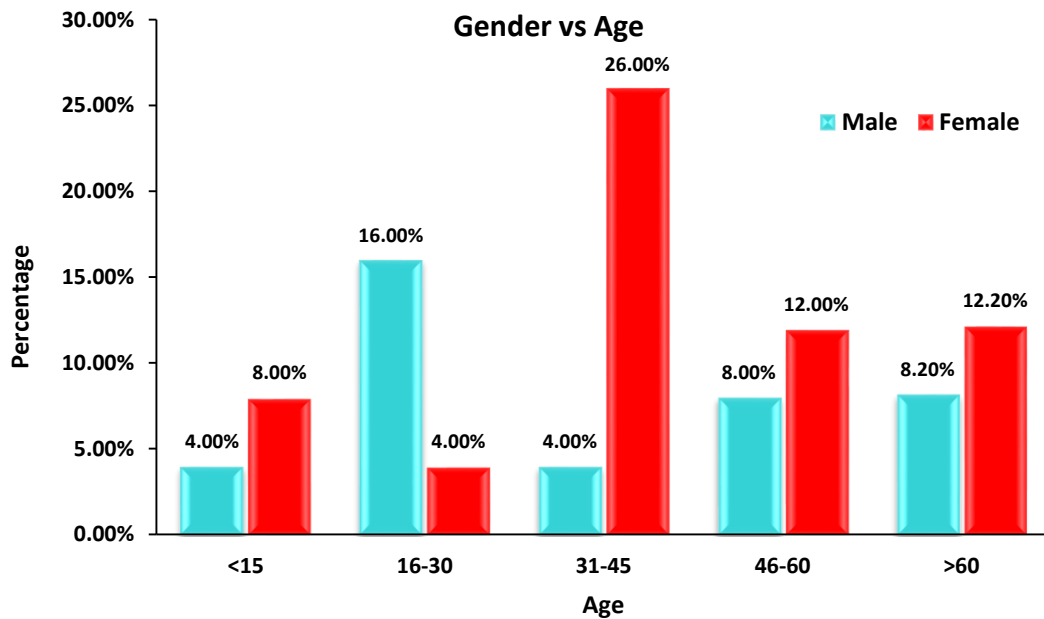
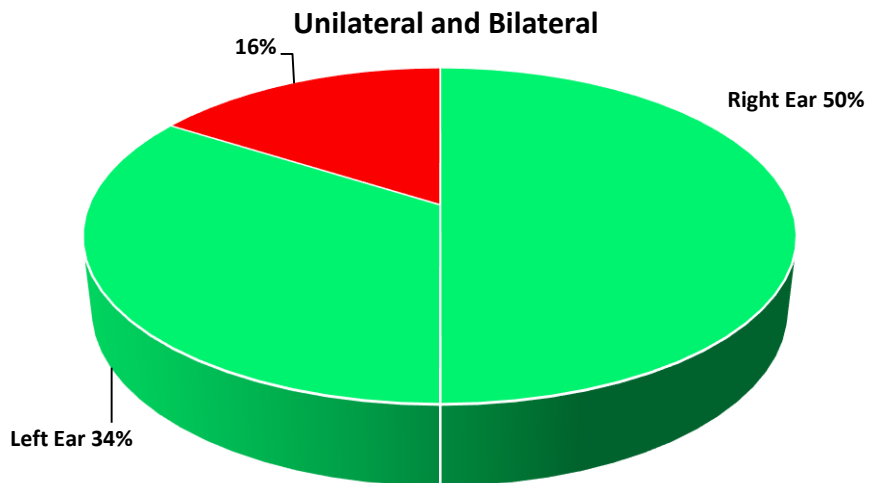


Fig 18: According to Gender vs. Age

In 50 cases of suspected with otomycosis studied in Sree Mookambika Institute Of Medical Sciences, in the age and gender were evaluated in which it was highest in the age group of 31-45 age and more prominent in females about 49 %.

Table 6: Laterality of the Ear

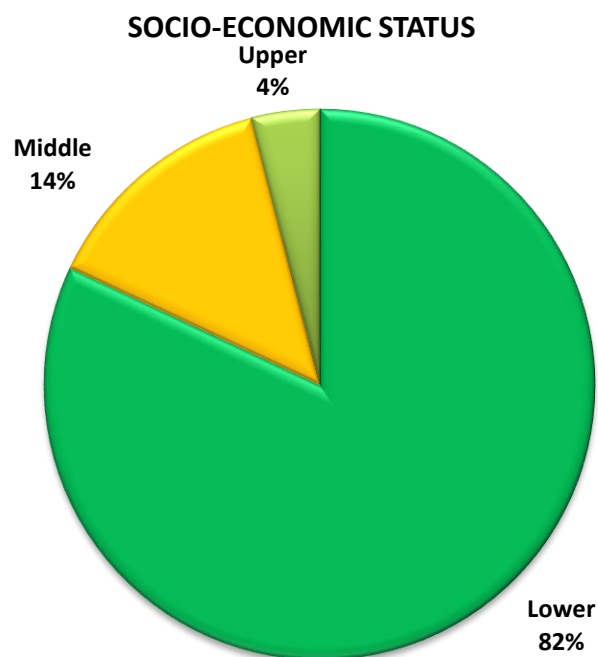
Laterality	Right ear	Left Ear	Total
Unilateral	25(50%)	17(34%)	42(84%)
Bilateral			8(16%)
Total			50

**Fig 19: Laterality of the Ear**

In cases of 50 suspected cases of otomycosis it was more a unilateral cases about (84%) in which it was more on right side which was (50%) and in left was 34%. Bilateral cases were seen in about 16%.

Table 7: According To Socio Economic Status

SES	Salary	Freq.	Percent	Valid Percent
Lower	<2101	41	82.0	82.0
Middle	2102-7007	7	14.0	14.0
Upper	>7008	2	4.0	4.0
		50	100.0	100.0

**Fig 20: According to the Socio Economic Status**

In cases studied according to the BJ Prasad salary score the salary⁴⁹ of <2101 was lower and 2101-7007 was middle class and more than 7008 is high class. So according to the salary was studied and was found to be it was common in lower class. This may be due to the fact the lack of awareness and self-hygiene of the society.

Table 8: Variation of fungi and Month

Month	Frequency	Percent	Valid Percent	Cumulative Percent
Jan	2	4	4	4
Feb	2	4	4	8
Apr	1	2	2	10
May	5	10	10	20
Jun	2	4	4	24
Jul	1	2	2	26
Aug	4	8	8	34
Sept	4	8	8	42
Oct	9	18	18	60
Nov	11	22	22	82
Dec	9	18	18	100
Total	50	100	100	

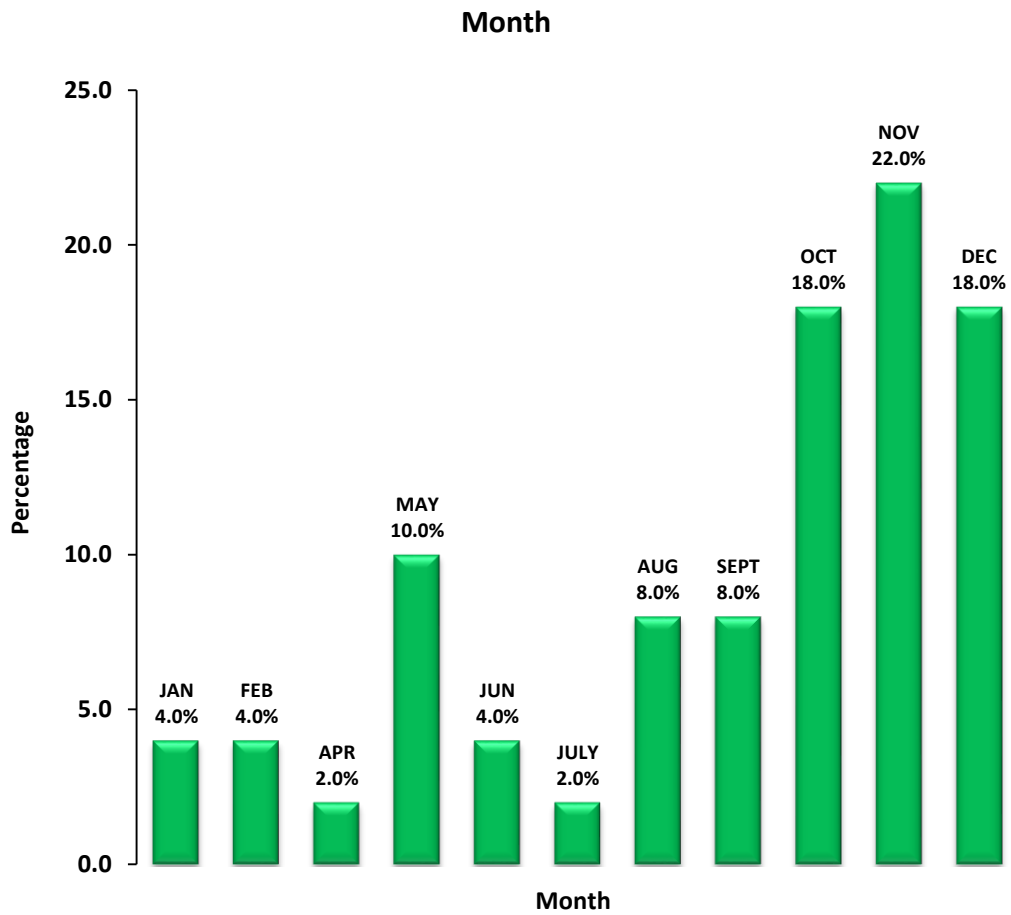
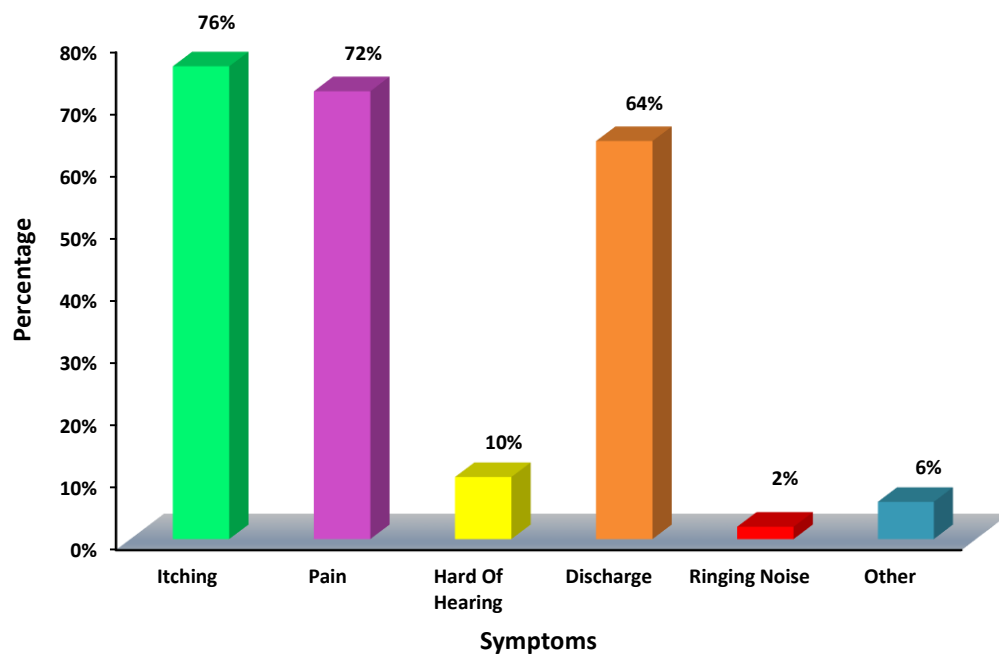


Fig 21: Variation of fungi vs. Month

According to the month variation was studied the number of cases. The rainy and winter season of October to December were the maximum cases about 58% of the cases, and in the summer of May were 10 percent.

Table 9: According to presenting Symptoms and Number of patients

S. No	Symptoms	Number
1	Itching	38 (76%)
2	Pain	36 (72%)
3	Hard Of Hearing	5 (10%)
4	Discharge	32(64%)
5	Ringing Noise	6 (12%)
6	Other	3 (6%)

**Fig 22: According to presenting symptoms and Number of patients**

In the cases studied the most important symptom was itching and it was constituted to 76% and followed by pain which was 72 percent and about 64% were associated with ear discharge. Ear ache mostly seen in bacterial otitis externa. The other symptoms were associated were ringing noise (12%) and hard of hearing (10%).

Table 10: According To Pre Disposing Factors

S. No	Pre Disposing Factors	Total [50(100%)]
1	Diabetes	12(24%)
2	TB	1(2%)
3	Irradiation	0
4	H/O Previous Ear Infection	10(20%)
5	H/O Ear Surgery	3(6%)
6	Rx with Steroids/Antibiotics	3(6%)
7	Local Antibiotics	5(10%)
8	Allergy to Dust/Drugs	4(8%)
9	Swimming in Ponds	8(16%)
10	Ear Probing	11(22%)
11	Usage of Ear Buds	11(22%)

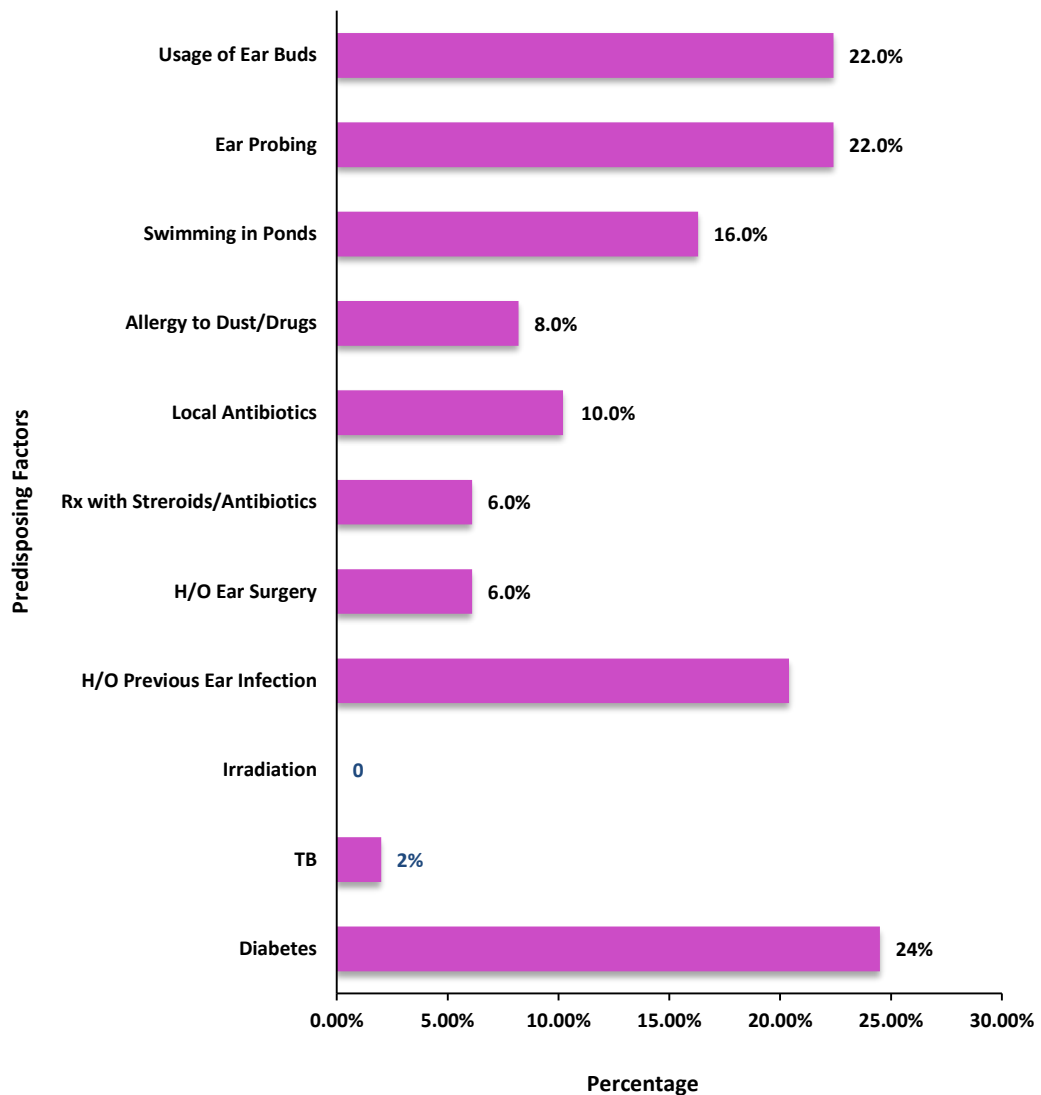
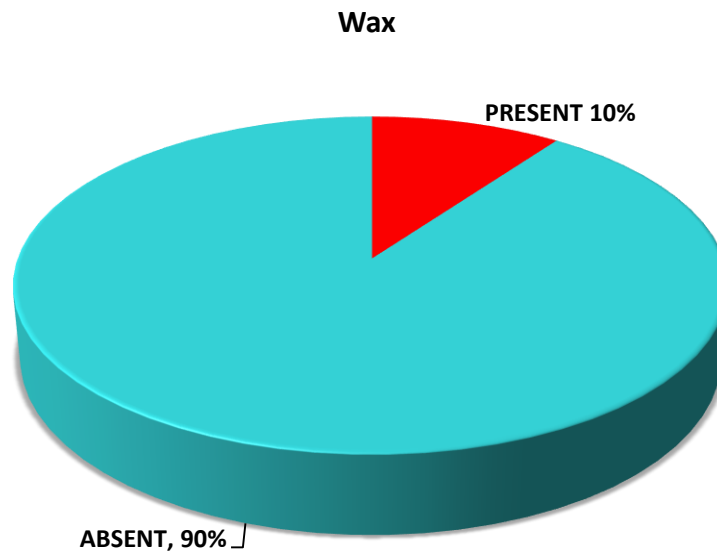


Fig 23: According To Pre Disposing Factors

The various predisposing factors was studied in this study and found that ear probing (44%) was most associated predisposing factor, followed by diabetes (24%), then was the history of previous ear infection (20%), the other predisposing factors were (swimming in ponds(16%) , local topical antibiotics (6%) and history of previous ear surgery(6%).

Table 11: Wax Presence

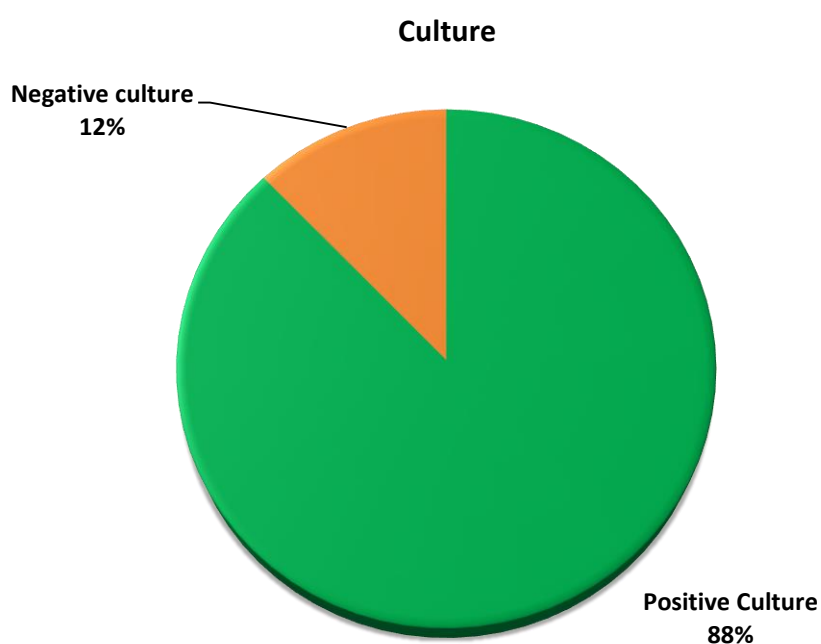
Wax	Frequency	Percent	Valid Percent
Present	5	10.0	10.0
Absent	45	90.0	90.0
Total	50	100.0	100.0

**Fig 24: Wax Presence**

The otoscopic findings of the cases studied the presence or absence of wax was seen. Since the presence of cerumen is found to be a protective factor in the protection of otomycotic growth. The studies shows that it was absent in 90% of the cases studied. And was present in 10 present in cases.

Table 12: According To Specimens Sent For Culture

S. No.	Total sample tested	Positive Culture	Negative Culture
1	50	44(88)	6(12%)

**Fig 25: According To Specimens Sent For Culture**

In cases of suspected cases of otomycosis the culture sent was positive in 44 cases of 50 cases (88%) in cases sent and negative in 6 cases (12%)

Table 13: Organism Growth Distribution

Organism Growth Distribution	Frequency	Percentage
Candida non Albicans	7	14
C. Albicans	15	30
Asp. fumigatus	10	20
Asp. niger	4	8
Asp. flavus	4	8
Penicillium	1	2
Mucormycosis	3	6
No Growth	6	12
Total	50	100.0

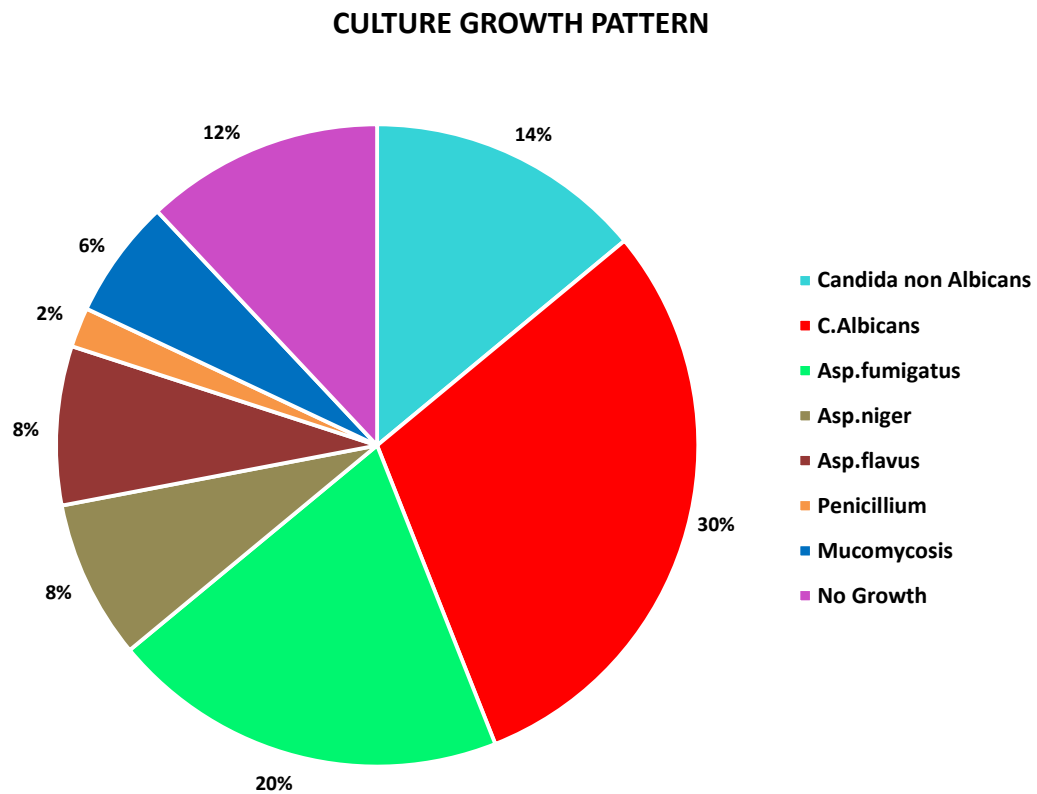


Fig 26: Organism Growth Distribution

The cases which was positive was 44 cases(88%) of 50 cases in which the most common isolate in my study was candida albicans(30%) followed by Aspergillus fugimatus (20%) then by candida non albicans(14%) then by Aspergillus niger and flavus each was 8 percent , and the least isolate was Mucormycosis(6%)

Table 14: Pre Disposing factor and growth

Pre Disposing Factor	Number	A. Flavus	A. Fumigatus	A. Niger	Candida	C. Albicans	Mucomy cosis
Diabetes	12	1	3	2	2	4	-
TB	1	-	-	-	-	1	-
Irradiation	0	-	-	-	-	-	-
Prev Ear Inf	10	1	5	-	1	3	-
H/O SX	3	-	1	-	1	1	-
Rx Steroids	3	-	1	-	-	2	-
Local AB	5	-	1	1	1	2	-
Allergy	4	-	2	-	1	1	
Swimming	8		3	-	2	2	1
Ear Probe	11	2	2	-	1	5	1
Ear Buds	11	1	2	-	2	5	-

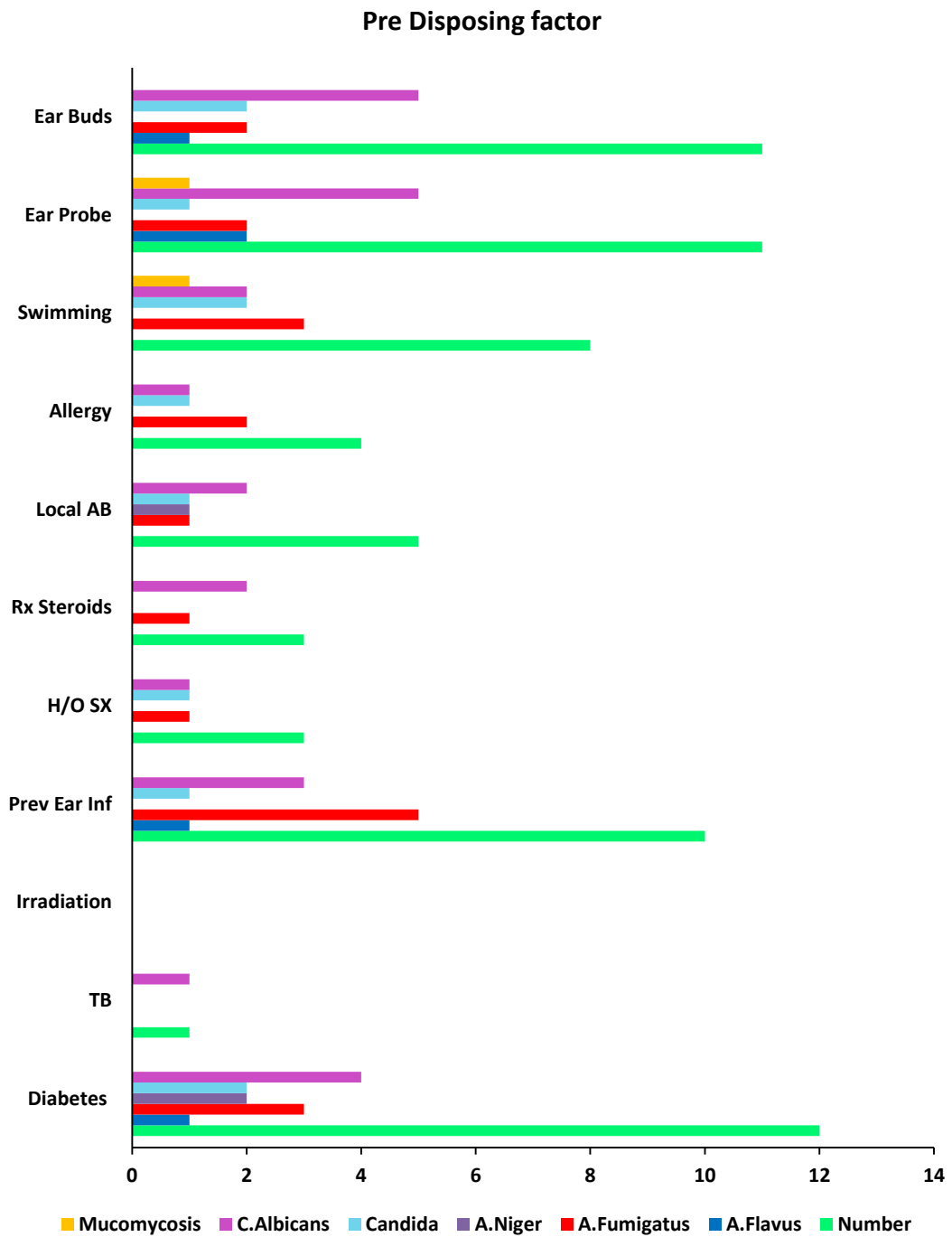
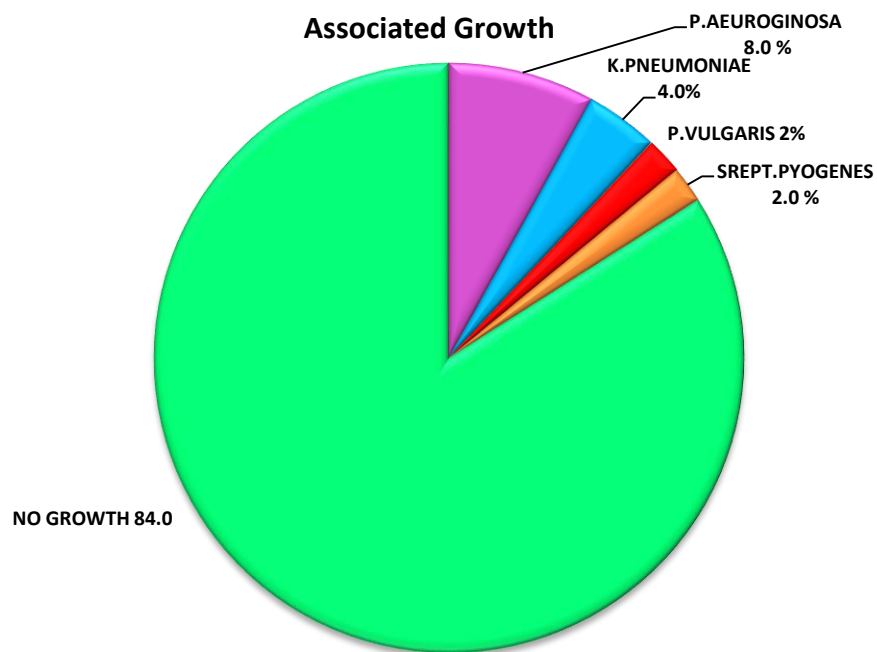


Fig 27: Pre Disposing factor and growth

Table 15: Associated Growth

Growth	Frequency	Percent	Valid Percent	Cumulative Percent
P. Aeuroginosa	4	8	8	8
K. Pneumonia	2	4	4	12
P. Vulgaris	1	2	2	14
Strep Pyogenes	1	2	2	16
No Growth	42	84	84	100
Total	50	100	100	

**Fig 28: Associated Growth**

The associated growth of the bacterial along with fungal culture was also studied it showed about 84 percent was no growth and the most common associated growth was Pseudomonas aeuroginosa followed by Klebisella Pneumonia and Streptococcoal Pyogens.



Discussion

DISCUSSION

Otomycosis is a common infection of the external auditory canal in this region and as such there are no specific cause attributed to its high prevalence in this region of the country. So an analysis as to the prevalence and aetiology will help in prevention of the disease, knowing the causative organisms and making guidelines to the effective management of the condition. So a study of 50 patients with suspected otomycosis attending the ENT OPD was conducted in Sree Mookambika Institute of Medical Sciences and the following was found. The main predisposing factor for otomycosis is the chronic infective conditions of the ear and the clinical diagnosis is a frustration for clinicians. Otomycosis remains an uncommon disease often overlooked and goes unnoticed and unattended until severe pain occurs. Several authors have drawn attention to various aspects of otomycosis.

Fungi could be mentioned as a normal micro-flora in the external auditory canal playing an important role in otomycosis. Various factors pave the way for such saprophytic organisms to gain a foot hold in the external canal. Fungal infections in the ear are increasing especially with indiscriminate use of topical antibiotic-steroid preparations and unhygienic habits. Treatment of otomycosis consists of adequate aural toilet, altering the pH of the external canal with acidifying agents and anti-fungal topical ear drops.

The above observations have been discussed as follows.

Age:

The incidence of otomycosis was found to occur in age groups 3- 80 years. The youngest case in this study was a 3 year old girl who had otomycosis. The

oldest patient in the study was a 70 year old male who was a diabetic. The study reveals that fungal infection was more common among adolescent and middle aged individuals. This age group from 16-30 and 31-45 years constitute 30% and 20 % of incidence respectively. The above age group 31-46 years includes predominantly housewives. Housewives are exposed to cold damp environment doing household chores inside as well as in the fields and students are exposed to outside environment.

The age incidence is in accordance with the studies of T. Mugliston and G.O'Donoghue³⁶ (1985) 21%, Yehia MM and Al Habib HM and ShehabNM⁴¹(1990) 48%, KO Paulose and S. All Khalifa²⁵ (1989) 41%, Baveja and Dhingra³⁸ (1993) 50%, Murat Ozcan²⁴ and colleagues in their study had 31-60 year age group as suffering the most and they attribute it probably to religious practice of head coverings as a predisposing factors which is mandatory in Turkey.

Sex:

In this study the incidence of otomycosis was found more among females (58%). This is in accordance with the study conducted by Yehia MM, and Al Habib HM. and NM Shehab⁴¹ (1990). The young and middle aged females are among the most common to be affected by the disease.

Housewives in India as a whole and in South India in particular have a lot of family burden to share as predominantly the males work in fields, working in damp, cold conditions lead to exposure to dust and deposition of fungal spores. The unhygienic practice of self-cleaning of the ear canal with dirty fingers, hair pins, match sticks hastens the deeper invasion of the fungus. However T. Mugliston and

G.O'Doughue³⁶ (1989) from London in their study found little difference in the sex incidence. On the contrary, Paulose, Al Khalifa Shenoy, Sharma²⁵ (1989) – 58% and CP Baveja Dhingra PL, Natarajan R.³⁸ (1993) 64% found that incidence was found more among males.

Laterality distribution:

Otomycosis is commonly a one sided disease this study is a proof to this fact. It is found in this study that 84% of cases had otomycosis in a single ear and only 16% of the patients had the disease in both ears. The above results are in accordance with studies by KO Paulose, Al Khalifa, P. Shenoy and RK Sharma²⁵ 87% and T. Mugliston and G.O. Donghue³⁶ (1985) 89% who also reported that otomycosis is predominantly a unilateral disease. In their study the incidence of otomycosis in both ears was 13% and 11% respectively. As far as the side of ear is concerned the above studies could not find any difference but in our study the incidence was more in the right ear. The unilateral nature of the disease confirms the phenomenon reported by Dewit (1974), that the disease is not highly infectious.

Seasonal incidence:

In this study majority of cases were reported between October to December (58%) that constitute rainy season in Kanyakumari District. Fungi abound in any soil or in sand which contains decomposing vegetable matter, droppings of cattle, goats and other domestic animals. This is desiccated rapidly in the tropical sun and is dispersed as wind-blown particles. The air borne fungal spores are carried on droplets of water vapour a fact which we believe correlates with the higher incidence during the above season. The above observations are in accordance with

the study by Muglistan and G.O Donaghue³⁶ in 1985 and Sood VP and Sinha A, Mohapatra LN⁵⁷ (1964) and Beg MH, HH Bughari AT⁵⁸ (1983).

The excessive sweating during summer in tropics dilutes the wax and reduces the protective sleeve property. Further the high humidity and the various factors in the canal give suitable conditions for many fungi and bacteria to be established there. The relative high humidity in the external auditory canal and the temperature in the canal which approximates that of the body are the prime factors in any germinating medium. It is likely that dormant spores of fungi lying in the canal start multiplying under these favourable conditions of optimum temperature and humidity. (Sood VP, and Sinha A, Mohapatra LN. 1964)⁵⁷.

Presence of high humidity shifts the pH of the external canal to alkaline side. The alkaline pH further helps the growth of fungi. In our study the increased incidence of otomycosis was noted between June and October (high humidity in these months).

Presenting complaints:

All the patients in our study presented with itching (76%) and other common complaints were ear ache (72%), and ear discharge (64%).

The above complaints and their incidence as mentioned earlier were in accordance with KO Paulose, Al Khalifa, P.Shenoy, RK Sharma²⁵ et al (88%), Yehia MM and Alhabib HM and Shehab NM.⁴¹ (78%).

Although pain tends to be the dominant complaint in bacterial infections, the most common complaint in otomycosis was severe itching sensation deep inside the canal, patients frequently report an irresistible urge to scratch the ear canal with the

finger tip, and of any sharp instrument like hair pins. Though such trauma is not necessary for the production and progression of the disease, it may facilitate sub epidermal invasion of the fungi. Itching generally progress to a dull and deep seated pain that may later on be associated with ear discharge. The accumulation of fungal debris in the inflamed and narrow external canal frequently produces hearing loss.

Predisposing factors:

In our study the predisposing factors taken into consideration were history of swimming or water entry into ear canal while bathing in ponds, use of local antibiotic or steroid drops, and trauma to external canal or any associated medical disease.

The most predominant predisposing factor was trauma to external canal and use of antibiotic drops. The trauma was in the form of inadvertent use of match sticks, bird's feather, and finger tips to clean the external canals, aural syringing or poorly maintained mastoid cavity. The percentage of patients in this group were 44% each. History of swimming in local ponds, poorly maintained swimming pools was present in 16% of case as predisposing factor, these finding are in accordance with the study of Sheikh et al 42% and Beany GPE and Broughton A³². The lipid mantle layer formed by the cerumen has long been considered as the key factor for the protection of the canal wall. Its removal by frequent irrigation of the external layer is thought to be the reason why frequent bathing in tropical climates is incriminated as the cause of recurrent otomycosis.

According to Beany GPE and Broughton A³², swimming in sea and fresh water is not necessarily the mode of entry of infection into the external ear canal but

the constant irrigation of the outer canal during swimming and diving repeatedly removes nature's waterproof lining of the external canal. Any factors which prevents the ready drainage of water from the ear canal will lead to maceration of the meatal skin and provide an excellent culture for the growth of bacterial and fungal organisms. In our study diabetes mellitus was a important predisposing factor. It is due to high level of glucose in the tissues which may favour the growth of fungi in external auditory canal this substantiates 25% of the otomycotic cases in our study being diabetic.

Liston and Siegel⁵⁹ (1986) report that many authors attribute the recent increase in incidence of otomycosis to increasing use of antibiotic and steroid ear drops, systemic and local steroid usage. However this was not the experience of Mugliston T, and O'Donoghue²¹ who noted that there has been no significant increase in the incidence of otomycosis with the widespread use of topical combination of antibiotic and steroid drops. According to Mogliston T, and O'Donoghue³⁶, the local steroid drops seem to suppress the allergic reaction of fungal toxins and this concept was kept in mind because of the disease recurrence after drug withdrawal. In our study it shows only 6% of cases using antibiotic- steroid combination ear drops.

Role of cerumen:

In our study about 90% of all the cases had no cerumen in the external canal. This is accordance with study of KO Paulose and Al Khalifa, P.Shenoy RK Sharma²⁵ (1989) and Youseff and Abdua MH³⁴, ear wax contains numerous amino acids, saturated and unsaturated fatty acids which have an inhibitory effect on fungi (Senturia¹⁸ 1957). However till date this remains controversial

cerumen may support luxurious growth of aspergillus species despite containing the above contents (Akobjanoff 1954)⁶⁰.

Otoscopic findings:

In our study the most common otoscopic finding was the presence of black mycologic plug in 52% of patients, 20% patients presented with white wet mycelial mat of fungal spores, 16% with dry mycelial mat, 8% as soft grayish white debris and 4% as cotton wooly mass.

These findings were in accordance with Youseff YA, and Abdou MH.³⁴ (1962) 65% Sheikh et al 72% (1993) who found that predominant mycological picture was of grayish white nature.

Associated ear disease:

After adequate aural toilet various underlying changes in the canal wall and the nature of the tympanic membrane was noted. 42% of cases had congested tympanic membrane with edema of canal wall. This is in accordance with K.O. Paulose et al²⁵ (1989) who found similar findings in their study.

Fungal distribution:

The fungal debris removed from the external auditory canal of all patients were subjected to 10% KOH slide preparation and examined for presence of fungal elements, those positive for fungal elements were also cultured on the sabourauds dextrose agar for fungal growth. In our study *Candida Albicans* was isolated in 30% of cases, *Aspergillus fumigatus* in 20% of cases, *Candida non-albicans* in 14% *Aspergillus flavus* in 8% and *Mucormycosis* in 6% of cases. The predominance of *Candida Albicans* in this study is in accordance with that conducted by Sinha and

Mohapatra⁵⁷ (1961) 60%, Paulose and Al Khalifa, P. Shenoy, RK Sharma²⁵ (1989) *A. niger* grows on cerumen, epithelial scales and debris deep in the external canal, the resulting plug of mycelium and debris causes irritation, itching pain and deafness.

Beany GPE and Broughton A³² found that aspergillus species were known to produce antibiotics which eliminate bacterial complicators. This substantiates the low association of bacterial growth in aspergillus cases in our study.

The difference in pattern of isolates obtained in our study may be due to the different geographical distribution of fungus. Not much epidemiological data were available pertaining to our region for comparison.



Conclusion

CONCLUSION

- The incidence of Otomycosis is more during monsoon period.
- Females are more affected.
- Unilateral incidence of disease is common.
- CSOM is the most common predisposing factor.
- Candida Albicans is the most common fungal isolate.
- Aspergillus flavus is the most common isolate in agriculturists and labourers. (Outdoor workers)
- Candida Albicans is the common fungal isolate in females.
- The frequency of recovery of certain fungal species such as A. flavus, A. niger, A. terreus and Candida albicans from Otomycosis reflects the great ubiquity of the distribution of their spores and hence exposure to them.
- Systemic illness play an important role in the recurrence of otomycosis
- Inadequate aural toilet plays a role in recurrence.



Summary

SUMMARY

The present study was aimed at finding out the common etiology of the patients attending with suspected case of otomycosis in our college and the predisposing factors for Otomycosis. This was done at Sree Mookambikai Institute of Medical Sciences And Hospital attached to MGR University for a period of one and half years from 2017 to 2019. 50 patients clinically diagnosed as Otomycosis were selected for this study and the different predisposing factors were analyzed.

- The incidence was more during the monsoon period (58%) and this was due to increased humidity in the atmosphere.
- Females were more affected (58%) than males. This may be due to the fact, that females are more exposed to fungal spores, as they bathed in ponds more than males. The outdoor air is an important vehicle for locally prevalent fungal flora.
- Most of the cases were unilateral (84%). It was more common in left ear (50%) and right ear (34%)
- The most common predisposing was diabetes (24%).
- Itching was the most common symptom (76%).
- Pain was the second commonest symptom (72%).
- Candida Albicans was the most common fungal isolate (30%).
- Aspergillus fungimatus was the commonest isolate in patients with CSOM as the predisposing factor (51.39%).
- Aspergillus flavus was the commonest isolate in patients with Otomycosis using ear drops (65.52%).

- *Aspergillus fumigatus* was the second common fungal isolate (24%).
- *Aspergillus Niger* was the commonest isolate in females (17.73%) and miscellaneous indoor workers as they spend more time indoor and are exposed to house dust. *Aspergillus nigers* can be cultured from house dust.
- *Aspergillus terreus*, *Aspergillus fumigatus*, *Aspergillus glaucus* were also isolated.
- Different species of *Aspergillus* may be the dominant organism in different climates.
- *Aspergillus* needs dead organic matter as a substrate for their growth.
- Mucormycosis was isolated in 3 cases (6%).
- *Pseudomonas aurogeniosa* was the most common bacterial isolate (8%).
- *Proteus*, *streptococcus pyogens* and *Klebsiella* were the other common isolates.
- Systemic diseases play a role in otomycosis since diabetes was a common systemic disease in our study.
- Inadequate aural-toileting played a role in recurrence.



Bibliography

BIBLIOGRAPHY

1. Bowman SM, Free SJ. The structure and synthesis of the fungal cell wall. *Bioessays*. 2006 Aug;28 (8):799-808.
2. Kaur R, Mittal N, Kakkar M, Aggarwal AK, Mathur MD. Otomycosis: a clinicomycologic study. *Ear, nose & throat journal*. 2000 Aug;79 (8):606-9.
3. Vennewald I, Klemm E. Otomycosis: diagnosis and treatment. *Clinics in dermatology*. 2010 Mar 1;28 (2):202-11.
4. Jawad Ahmed NA. A clinicopathological study of Otomycosis (Doctoral dissertation).
5. Prasad SC, Kotigadde S, Shekhar M, Thada ND, Prabhu P, D'Souza T, Prasad KC. Primary otomycosis in the Indian subcontinent: predisposing factors, microbiology, and classification. *International journal of microbiology*. 2014;2014.
6. Dr. Jagadish Chander. Text book of medical mycology. New Delhi:
 - a. Mehta Publishers, Apr.2002.
7. Kurnatowski P, Filipiak A. Otomycosis: prevalence, clinical symptoms, therapeutic procedure. *Mycoses*. 2001 Dec;44(11-12):472-9.
8. Anwar K, Gohar MS. Otomycosis; clinical features, predisposing factors and treatment implications. *Pakistan Journal of Medical Sciences*. 2014 May;30(3):564.
9. Right, David Arthur. Scott Brown's otolaryngology. 5th Edn. Vol.1, 8-9.
10. Susan (2008). Borley, Neil R. (ed.). *Gray's Anatomy: The Anatomical Basis of Clinical Practice* (40 ed.). Edinburgh: Churchill Livingstone/Elsevier. pp. Chapter 37. "Inner ear", 633–650.

11. Stinson MR. The spatial distribution of sound pressure within scaled replicas of the human ear canal. *J Acoust Soc Am*, 1985;78: 1596
12. Stern JC, Lucente FE. Otomycosis. *Ear, nose, & throat journal*. 1988 Nov;67(11):804.
13. R. Ananthanaryan and CKJ Paniker. Text book of microbiology. 5thEdn. Orientlongman, 562.
14. Petrakis NL, Doherty M, Lee RE, et al. Demonstration and implication of lysozyme and immunoglobulins in human ear wax *Nature*, 1971; 229: 119-120.
15. Kaspar A, Newton O, Kei J, Driscoll C, Swanepoel DW, Goulios H. Prevalence of otitis media and risk-factors for sensorineural hearing loss among infants attending Child Welfare Clinics in the Solomon Islands. *International journal of pediatric otorhinolaryngology*. 2018 Aug 1;111:21-5.
16. Sreepada GS, Kwartler JA. Skull base osteomyelitis secondary to malignant otitis externa. *Current opinion in otolaryngology & head and neck surgery*. 2003 Oct 1;11(5):316-23.
17. Strauss MB, Dierker RL. 15 Otitis externa associated with aquatic activities (swimmer's ear). *Clinics in dermatology*. 1987 Jul 1;5(3):103-11.
18. Senturia BH, Marcus MD and Lucente FE. Disease of external earan otologic – dermatologic manual. Gune and Stratton, New York, 1980: pp66-69
19. Russell JD, Donnelly M, McShane DP, Alun-Jones T, Walsh M. What causes acute otitis externa?. *The Journal of Laryngology & Otology*. 1993 Oct;107(10):898-901.

20. Gill WD. XXXIV Otitis Externa. *Annals of Otolology, Rhinology & Laryngology*. 1942 Jun;51(2):370-7.
21. Janardhan D. *Otomycosis In Relation To Chronic Suppurative Otitis Media- A Study (Doctoral dissertation)*.
22. Morrison AW, Mackay IS. The aetiology of otitis externa—a new concept. *The Journal of Laryngology & Otology*. 1976 May;90(5):495-7.
23. Bruner, Robert C., and Leland R. House. “XXXII Thermodynamics of the External Auditory Canal.” *Annals of Otolology, Rhinology & Laryngology* 76, no. 2 (June 1967): 409–13. doi:10.1177/000348946707600209.
24. K. Murat Ozcan, MugeOzcan, AydinKaraarslan, FilizKaraarslan. *Otomycosis in Turkey: predisposing factors, aetiology and therapy*. *The J Laryngol& Otology*, 2003; 117: 39-42.
25. Paulose KO, Al Khalifa S, Shenoy P, Sharma RK. Mycotic infection of the ear (otomycosis) : a prospective study. *J Laryngolotol*, 1989; 103: 30-5
26. Gregson AEW and La Touche. Significance of mycotic infection in the aetiology of otitis externa. *JLO*, 1961; 75: 165-170.
27. Yehia MM, Al; Habib HM, Shehab NM. *Otomycosis : A common problem in North Iraq*. *J LaryngolOtol*, 1990; 104: 380-389.
28. BibuPradhan, NhuchheRatnaTuladhar, Ramchaya Man Amatya. *Prevalence of otomycosis in outpatient department of otolaryngology in Tribhuvan University, Teaching Hospital, Kathmandu, Nepal*. *Ann OtolRhinolLaryngol*, 2003; 112: 384-387.
29. Hawke M, Wong J, Kraijden S. *Clinical and microbiological features of otitis externa*. *J Otolaryngol* 1984; 132: 289-295.

30. Linstrom CJ, Lucente FE. External otitis. In English GM (ed) Otolaryngology, BC Decker, Philadelphia; 1990.
31. P.Kurnatowski and A.Filipiak. Otomycosis: prevalence, clinical symptoms, therapeutic procedure. *Mycoses*. 2001; 44: 472-479
32. Beany GPE and Broughton A. Tropical otomycosis. *J Laryngol Otol*, 1967; 81: 987-997.
33. Ferguson BJ and Mitchell TT. Stimulation of *Aspergillus niger* growth on exposure to cerumen - presented at the annual meeting of the American Academy of Otolaryngology. *Head & Neck Surg*, 1987
34. Youssef YA, Abdou MH. Studies of fungus infection of the external ear. *J Laryngol & Otol*, 1967; 81: 401-412.
35. Oliveri S, Capello G, Napolitano MG. Et al. Otomycosis etiologia ed. Analisi di Alchui Faltori Predisponenti. *Boll Ist Sieroter, Milan*, 1984; 63(6): 537-542.
36. Mugliston T. and O'Donoghue G. Otomycosis - A continuing problem. *J Laryngol Otol*, 1985; 99: 327-333
37. Than KM, Naing KS. And Min M. Otomycosis in Burma and its treatment. *Am J Trop Med Hyg*, 1980; 29(4): 620-623. CP
38. Baveja, P.L. Dhingra, R. Natarajan. Mycological profile in otomycosis and its response to an antifungal agent. *Ind J Otolaryngol Head & Neck Surg*, 1993; 2(4)..
39. Yassin A, Maher A, Moawod MK. Otomycosis : A survey in the eastern province of Saudi Arabia. *J Laryngol Otol*, 1978; 92: 869-76.
40. Joy MJ, Agarwal MK, Samanth HC, et al. Mycological and bacteriological studies in otomycosis. *Ind J Otolaryngol*, 1980; 32:72-5.

41. Yehia MM, Al; Habib HM, Shehab NM. Otomycosis : A common problem in North Iraq. *J Laryngol Otol*, 1990; 104: 380-389
42. Pahwa VK, Chamiyal PC, Suri PN. Mycological study in otomycosis. *Ind J Med Res*, 1983; 77: 334-8
43. Jaiswal SK. Fungal infection of ear and its sensitivity pattern. *Ind J Otolaryngol*, 1990; 19-22.
44. Rietschel RL, Fowler JF, Fisher AA. Fisher's contact dermatitis. PMPH-USA; 2008.
45. Jacobsson S, Karlsson G, Rigner P, Sanner E, Schrewelius C. Clinical efficacy of budesonide in the treatment of eczematous external otitis. *European archives of oto-rhino-laryngology*. 1991 May 1;248(4):246-9.
46. Gupta AK, Bluhm R. Seborrheic dermatitis. *Journal of the European Academy of Dermatology and Venereology*. 2004 Jan;18(1):13-26.
47. Dillon HC. Impetigo contagiosa: suppurative and non-suppurative complications: I. Clinical, bacteriologic, and epidemiologic characteristics of impetigo. *American Journal of Diseases of Children*. 1968 May 1;115(5):530-41.
48. Siwicki AK, Anderson DP, Rumsey GL. Dietary intake of immunostimulants by rainbow trout affects non-specific immunity and protection against furunculosis. *Veterinary immunology and immunopathology*. 1994 May 1;41(1-2):125-39.
49. Georgopapadakou NH. Antifungals: mechanism of action and resistance, established and novel drugs. *Current opinion in microbiology*. 1998 Oct 1;1(5):547-57.

50. Borgers M. Mechanism of action of antifungal drugs, with special reference to the imidazole derivatives. *Reviews of infectious diseases*. 1980 Jul 1;2(4):520-34.
51. MogadamAY ,Asadi MA , Dehghani R , Hooshyar H. The prevalence of otomycosis in Kashan, Iran, during 2001-2003. *Jundishapur J Microbiol* 2009; 2(1): 18-21
52. Satish HS, Viswanatha B, Manjuladevi M. A Clinical Study of Otomycosis. *IOSR J Dent Med Sci* 2013;5(2):57-62.
53. GokaleSK ,Suligavi SS , Baragundi M, Anushka D, Manjula R. Otomycosis : A Clinico Mycological Study. *Int J Med Health Sci* 2013,2(2):218-23.
54. Abdelazeem M , Gamea A, Mubarak H, Elzawawy N. Epidemiology, causative agents, and risk factors affecting human otomycosis infections. *Turk J Med Sci* (2015); 45: 820-6
55. Mangal A, Kumar V, Panesar S, Talwar R, Raut D, Singh S. Updated BG Prasad socioeconomic classification, 2014: A commentary. *Indian Journal of public health*. 2015 Jan 1;59(1):42.
56. Singh S, Singh H, Kaur A. Otomycosis : A Clinical and Mycological Study. *Indian J Basic Appl Med Res* 2017; 6(2):271-5.
57. Sood VP, Sinha A, Mohapatra LN. Otomycosis : A clinical entity clinical and experimental study. *J Laryngol Otol* 1967; 81: 997-1004.
58. Beg MHH, Bughari AT. Otomycosis in Karachi, *Practitioner* 1983; 227: 1767-1770.
59. Liston L, Leighton Seigul. Tinactin in the treatment of fungal otitis externa. *Laryngoscope*, 1986; 96: 699.

60. Akobjanoff and Senturia BH. The chemistry of cerumen – A preliminary report. *J Invest Dermatol* 1954; 23: 43-49.
61. Mohamed Anwarullah, PA Jayakar. A microbiological study of otomycosis in Vishakapatnam. *J Indian Med Assoc*, 1987; 85(8).



Appendices

APPENDIX - I



**SREE MOOKAMBIKA INSTITUTE
OF MEDICAL SCIENCES
KULASEKHARAM**

RESEARCH COMMITTEE

CERTIFICATE

This is to certify that The Research Protocol Submitted
by Dr. ADHAVAN E
Faculty / Post Graduate from Department of OTDRHINO LARYNGOLOGY
..... Titled
PREVALENCE AND AETIOLOGY OF OTOMYCOSIS
IN SREE MOOKAMBIKA INSTITUTE
OF MEDICAL SCIENCES
is approved by the Research Committee.


Chair Person


Convenor
(Dr. P. S. KRISHNAN MURUGAN)

Prof. B. H. O. S.
Dept. of Microbiology
Sree Mookambika Institute of Medical Sciences
Kulasekharam 629 167

Date: 16/11/2017

APPENDIX - II



INSTITUTIONAL HUMAN ETHICS COMMITTEE

SREE MOOKAMBIKA INSTITUTE OF MEDICAL SCIENCES,
KULASEKHARAM, TAMILNADU

Communication of Decision of the Institutional Human Ethics Committee(IHEC)

SMIMS/IHEC No: 2 / Protocol no: 28 / 2017

Protocol title: PREVALENCE AND AETIOLOGY OF OTOMYCOSIS IN SREE MOOKAMBIKA INSTITUTE OF MEDICAL SCIENCES
Principal Investigator: Dr.Adhavan.E
Name& Address of Institution: Department of Otorhinolaryngology Sree Mookambika Institute of Medical Sciences
<input checked="" type="checkbox"/> New review <input type="checkbox"/> Revised review <input type="checkbox"/> Expedited review
Date of review (D/M/Y): 05-12-2017
Date of previous review , if revised application:
Decision of the IHEC:
<input checked="" type="checkbox"/> Recommended <input type="checkbox"/> Recommended with suggestions
<input type="checkbox"/> Revision <input type="checkbox"/> Rejected
Suggestions/ Reasons/ Remarks:
Recommended for a period of : Eighteen months

Please note*

- Inform IHEC immediately in case of any Adverse events and Serious adverse events.
- Inform IHEC in case of any change of study procedure, site and investigator
- This permission is only for period mentioned above. Annual report to be submitted to IHEC.
- Members of IHEC have right to monitor the trial with prior intimation.

Reneegayangadhas

Signature of Member Secretary (IHEC)



APPENDIX - III

PROFORMA

Title: Prevalence and aetiology of Otomycosis in Sree Mookambika Institute of Medical Sciences

Name: _____ **Date:** _____
Age: _____

Sex: _____

IP No.: _____ **Unit:** _____

Address: _____

Socio Economic status: Poor / Average / affluent

Chief Complaints: Unilateal / Bilateral if unilateral- Right ear / Left ear

- | | | |
|----------------------------|----------|-----------|
| 1. Itching | yes / no | Duration- |
| 2. Ear ache | yes / no | Duration- |
| 3. Ear discharge | yes / no | Duration- |
| 4. Hard of hearing | yes / no | Duration- |
| 5. Ringing noise | yes / no | Duration- |
| 6. Other complaints if any | | Duration- |

Past H/o

- | | | |
|--|----------|-----------|
| 1. Diabetes Mellitus- | yes / no | Duration- |
| whether on any antiglycemic drugs or insulin | | |
| 2. Tuberculosis | yes / no | Duration- |
| 3. Irradiation - | yes / no | |
| 4. Ear infection | yes / no | |
| 5. Surgery | yes / no | |
| 6. Treatment with steroids / antibiotics | yes / no | |
| 7. Local antibiotic drugs | yes / no | |
| 8. Allergy | yes / no | |

Habits

1. Swimming / taking bath in ponds - yes /no
2. Ear probing yes/no
3. Usage of ear buds yes/no

EXAMINATION:

Ear:	Right ear	Left ear
E.A.C. :		
Presence of Wax:		
Nature of Debris:		
Associated Ear disease:		

EXAMINATION OF NOSE:**EXAMINATION OF THROAT:****SPECIAL:**

10% KOH smear
Microscopic examination:
Mycological culture:

MYCOLOGICAL REPORT:

Culture Report:

Associated growth:

ANNEXURE-II : MASTER CHART

Case No	Name	Age	Sex	Occupation	OP No.	Presenting complaints							Side			Predisposing factors				Past h/o	
						Itching	Days	Pain	Days	Ear discharge	days	hearing loss	days	Left	Right	Both	Swimming	Use of ear drops	Injury to canal wall		Associated systemic disease
1	Jaismi	60	F	House wife	18119678	yes	1 mon	yes	3 wks	yes	3 wks	yes	2 wks	-	yes	-	no	no	yes	-	-
2	Joseph Jaya	78	F	House wife	18288055	yes	2 wks	no	-	no	-	no	-	-	yes	-	no	no	yes	DM- 18 yrs	DM
3	Purusothaman	59	M	laburer	17111503	yes	4 days	yes	4 days	no	-	yea	5 days	yes	-	-	no	no	Yes	DM- 6 yrs	DM
4	Srejith	26	M	student	17161001	Yes	3 days	yes	3 days	yes	5 days	no	-	-	yes	-	no	no	yes	-	-
5	Rani	54	F	House wife	18297681	yes	1 mon	no	-	yes	1 week	yes	1 ,mon	-	yes	-	no	no	yes	-	-
6	Jagadesh	35	M	Watch man	19111716	yes	1week	yes	1 week	yes	1 week	no	-	yes	-	-	yes	No	Yes	-	-
7	Kamala nair	65	F	House wife	17265585	no	-	yes	1week	yes	1week	no	-	yes	-	-	no	yes	no	DM- 5 yrs	DM
8	Rejin	25	M	student	19123323	yes	5 days	Yes	5 days	yes	5 days	no	-	-	yes	-	yes	yes	yes	-	-
9	Sajitha	40	F	House wife	16070394	no	-	yes	5 days	yes	4 days	no	-	yes	-	-	no	no	yes	DM- 5 yrs	DM
10	Aswini	40	F	House wife	19135611	yes	2 wks	yes	2 wks	no	-	no	-	-	yes	-	no	yes	yes	-	-
11	Sheriba	50	F	House wife	17245810	yes	2 wks	yes	2 week	yes	3 days	no	-	-	yes	-	no	yes	no	-	-
12	Venu kutheni	50	F	Hose wife	18044971	yes	1 mon	yes	1 ,mon	no	-	no	-	-	yes	-	NO	No	yes	-	-
13	Raja sekaran	57	M	labourer	19114891	yes	5 days	no	-	yes	5 days	yes	3 days	yes	-	-	no	no	no	-	-
14	Saraswathi	40	F	labourer	17232419	no	-	yers	1mon	no	-	yes	3 days	-	yes	-	no	no	no	-	-
15	Subha	36	F	House wife	19171671	yes	2 yrs	yes	2 days	yes	2 days	no	-	-	-	yes	no	no	no	-	-
16	Bala krishnan	60	M	Retiered officer	17157238	No	-	yes	2 days	no	-	NO	-	yes	-	-	no	no	no	-	-
17	Ajil	16	M	student	180210130	yes	10 days	yes	10 days	no	-	No	-	yes	-	-	no	yes	yes	-	-
18	saraswathy	44	F	House wife	18067536	yes	2 wks	yes	2 wks	yes	2 wks	no	-	yes	-	-	no	yes	yes	DM- 5 yrs	DM
19	Sreeja jainth	31	F	House wife	18038644	yes	1 mon	yes	1 mon	yes	1mon	no	-	yes	-	-	yes	no	yes	-	-
20	Subha	32	F	housewife	17123130	yes	7days	yes	7days	no	-	No	-	yes	-	-	no	no	no	-	-
21	Raja sundari	66	F	housewife	19132744	yes	4 days	yes	4 days	yes	4 days	no	-	yes	-	-	no	no	no	DM-5 yrs	DM
22	saraswathy	53	F	housewife	1811385	yes	6 days	yes	6 days	yes	6 days	no	-	-	yes	-	no	no	no	DM- 15 yrs	DM
23	Joseph vinu	72	M	watchman	1831833	yes	1 week	no	-	no	-	no	-	yes	-	-	no	no	no	-	-
24	Subi	15	F	student	15161719	yes	2 wks	yes	2 wks	yes	2 wks	No	-	yes	-	-	no	no	no	-	-
25	Jinesh	28	M	doctor	18010900	no	-	yes	15 days	no	-	no	-	-	yes	-	no	no	no	-	-
26	Ramu	35	F	teacher	1810171	yes	3 days	yes	3 days	yes	3 days	no	-	-	yes	-	no	no	no	-	-
27	Raghul	18	M	student	19117161	yes	3 wks	yes	3 wks	no	-	no	-	-	yes	-	no	no	no	-	-
28	Rajiv	42	M	labourer	17010010	yes	2 days	yes	2 days	yes	2 days	no	-	-	-	yes	no	no	no	-	-
29	Usha	34	F	Nurse	18173712	No	-	no	-	yes	5 days	yes	5days	-	yes	-	yes	no	no	-	-

30	Saju	30	M	conductor	18271973	yes	6 days	yes	6 days	yes	6 days	no	-	-	yes	-	No	No	No	-	-
31	Vijaya mary	27	F	hosewife	18037336	yes	20 days	yes	20 days	yes	20 days	no	-	yes	-	-	no	no	yes	-	-
32	Ponnaya	60	M	carpenter	18127959	Yes	5 days	No	-	no	-	no	-	-	yes	-	no	yes	No	-	-
33	Raja	26	M	teacher	18116135	yes	3 days	yes	3 days	yes	3 days	no	-	-	yes	-	no	no	No	-	-
34	Manoj	61	M	Driver	19171617	yes	1 week	yea	1 week	yes	1 week	No	-	-	yes	-	no	no	no	-	-
35	Sheeba	71	F	housewife	19161512	yes	6 days	yes	6 days	yes	6 days	no	-	-	-	yes	no	no	no	DM-15 yrs	DM
36	Mani	62	M	carpenter	16155375	Yes	4 days	yes	4 days	no	-	no	-	-	yes	-	no	no	no	DM-5 yrs	DM
37	Manonmani	62	F	House wife	1805380	yes	10 days	yes	10 days	no	-	no	-	-	yes	-	no	no	yes	-	-
38	Deeshma	29	F	teacher	16166677	yes	3 days	yes	3 days	yes	3days	no	-	yes	-	-	no	no	No	-	-
39	Jameela	36	F	doctor	17249495	yes	5days	no	-	no	-	no	-	yes	-	-	yes	no	yes	-	-
40	Nisha	27	F	receptionist	18270280	yes	3 days	yes	3 days	no	-	no	-	-	yes	-	no	no	no	-	-
41	Nandhini	3	F	-	19061716	yes	3 days	yes	3 days	no	-	no	-	yes	-	-	no	no	no	-	-
42	Santhi	39	F	housewife	17128356	Yes	3 days	yes	3 days	yes	3 days	no	-	-	-	yes	no	no	yes	-	-
43	Parvathi	70	F	House wife	1803105	yes	5 days	no	-	no	-	no	-	-	-	yes	no	no	NO	-	-
44	Latha kumari	35	F	officer	19024478	yes	5 days	yes	5 days	yes	5 days	No	-	yes	-	-	no	no	no	-	-
45	Tamil selvan	25	M	engineer	18179823	yes	5 days	no	-	no	-	yes	5 days	yes	-	-	no	no	No	-	-
46	Prasath	45	M	Shop keeper	1909919	yes	3 days	yes	4 days	yes	3 days	no	-	-	-	yes	yes	yes	yes	DM-10 yrs	DM
47	Sabarish	12	M	student	18178131	yes	5 days	yes	5 days	No	-	no	-	-	yes	-	no	no	no	-	-
48	Sree latha	43	F	housewife	18186133	no	-	yes	2 days	yea	2 days	no	-	-	yes	-	no	no	No	-	-
49	lakshmanan	68	M	cashier	17127704	yes	5 days	yes	5 days	yes	5 days	no	-	-	yes	-	no	no	no	DM- 6 yrs	DM
50	Tamil selvi	44	F	houswife	16220381	yes	4 days	no	-	no	-	no	-	-	yes	-	no	no	yes	-	-

Family h/o	Case NO	Examination			Associated ear disease	Examination of throat	Nose	10% KOH Microscopic examination	Culture report
		Ext.aud.canal	Presence of wax	Nature of debris					
-	1	Black D	-	Purp.bla	-	N	N	-	No growth
Diabetes	2	Black D	-	Yellowish brown	-	N	N	-	No growth
Diabetes	3	Green black D	-	wet newspaper like debris	-	N	N	+FE	C.Albicans
-	4	Whitish CWDE	-	whitish CWD	-	N	N	+FE	Candida non albicans
Systemic hypertension	5	Black D	-	black mycologic plug	-	N	N	+FE	Mucomycosis
-	6	Black D	-	black, WMM	CSOM	N	N	+FE	Asp.fumigatus
Diabetes	7	Yellowish white D	-	yellow soft D	-	N	N	+FE	C.Albicans
-	8	Black D	-	Black WMM	-	N	N	+FE	Asp.niger
-	9	Yellowish white D	-	Dry MM	-	N	N	+FE	C.Albicans
-	10	Black D	-	Wstm M	-	N	N	+FE	Asp.flavus
Diabetes	11	Yellow brown D	-	Black myco plug	CSOM	N	N	+FE	Asp.fumigatus
-	12	Brownish Black D	-	Black myco plug	-	N	N	+FE	Mucomycosis

CAD	13	Black Fungal D	-	Black myco plug	-	N	N	+FE	C.Albicans
-	14	Purplish black D	-	Black WMM	-	N	N	+FE	Asp.fumigatus
Diabetes	15	Purplish black D	-	Black WMM	-	N	N	+FE	Asp.flavus
-	16	Brownish Black D	-	Black WMM	CSOM	N	N	+FE	C.Albicans
-	17	Yellowish white D	-	myco plug	-	N	N	+FE	Candida non albicans
Systemic hypertension	18	Purple black	-	Dry MM	-	N	N	+FE	Asp.fumigatus
-	19	Black D	-	Black myco plug	CSOM	N	N	+FE	C.Albicans
Diabetes	20	Black debris	-	black myco debris	-	N	N	+FE	Asp.niger
Systemic hypertension	21	Black D	-	black myco plug	-	N	N	+FE	Asp.fumigatus
-	22	Black D	-	black wet MM	-	N	N	+FE	C.Albicans
-	23	Black D	-	Black myco plug	CSOM	N	N	+FE	C.Albicans
Diabetes	24	Black D	-	Black myco plug	-	N	N	+FE	Candida non albicans
-	25	Black D	-	Black myco plug	-	N	N	+FE	Asp.flavus
Systemic hypertension	26	Brownish Black D	-	Black myco plug	-	N	N	+FE	C.Albicans
-	27	Yellow white D	-	dry mycelial mat	-	N	N	+FE	Asp.fumigatus
-	28	Brownish Black D	-	wet mycelial mat	-	N	N	-	No growth
Systemic hypertension	29	Yellowis brown D	-	dry mycelial mat	-	N	N	+FE	C.Albicans

-	30	Black Fungal D	-	black mycologic plug	-	N	N	+FE	Asp.fumigatus
-	31	Black Fungal D	-	black mycologic plug	-	N	N	+FE	C.Albicans
Systemic hypertension	32	Black Fungal D	-	black wet MM	-	N	N	+FE	Candida non albicans
CAD	33	Yellowish black	-	Dry MM	--	N	N	+FE	C.Albicans
-	34	Yellowish brown	-	dRy MM	-	N	N	+FE	Asp.niger
-	35	Purple black D	-	Black myc plug	-	N	N	+FE	Penicillium
Systemic hypertension	36	Purple black D	-	Black myc plug	-	N	N	+FE	Asp.flavus
-	37	Black debris	-	Wet MM	-	N	N	+FE	Candida non albicans
-	38	Green discharge	-	Soft debris	-	N	N	+FE	Asp.fumigatus
Diabetes	39	Yellow brown D	-	Dry MM	-	N	N	-	No growth
-	40	yellowish brown	-	Wet MM	-	N	N	+FE	C.Albicans
-	41	yellow black	-	Wet MM	-	N	N	-	No growth
-	42	Black debris	-	Wet MM	-	N	N	+FE	Candida non albicans
Diabetes	43	Black debris	-	Black myco plug	-	N	N	+FE	Mucomycosis
Systemic hypertension	44	Black F.debris	-	Wet MM	CSOM	N	N	+FE	C.Albicans
-	45	Black F.debris	-	Black myoc plug	-	N	N	+FE	Asp.fumigatus
-	46	Black F debris	-	Wet MM	-	N	N	-	No growth
-	47	White cotton	-	Soft debris	-	N	N	+FE	Candida non albicans
CAD	48	Yellowish black	-	Wet MM	-	N	N	+FE	Asp.niger
-	49	Whitish cotton	-	Cotton wooly	-	N	N	+FE	C.Albicans
-	50	Whitish cotton	-	Dry MM	-	N	N	+FE	Asp.fumigatus