

The Fermented Filament-Otomycosis

Anubha Bajaj*

Histopathologist in A.B. Diagnostics, New Delhi, India

***Corresponding Author:** Anubha Bajaj, Histopathologist in A.B. Diagnostics, New Delhi, India.

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Preface

Otomycosis is an exceptionally delineated fungal infection emerging within stratified squamous epithelium of external ear. Infective otomycosis is commonly delineated within inhabitants of tropical or subtropical region and may be challenging to discern. Fungal infection in otomycosis occurs due to diverse species of saprophytic fungi, moulds, yeasts or exceptionally, dermatophytes. Predominant fungi engendering otomycosis are comprised of *Aspergillus* or *Candida* spp.

Otomycosis emerges as an acute to chronic, secondary fungal infection arising within the auricle and external auditory canal. Extension of fungal infection can incriminate the tympanic membrane. However, fungal infection expanding into the middle ear is exceptional.

Otomycosis as a superficial mycotic infection implicating extraneous acoustic canal can be singularly diagnosed by competent clinical examination. Typically, acute or subacute infection displays superficial scaling, inflammation, pruritus or severe discomfort within the external ear.

Disease characteristics

Otomycosis is primarily engendered by *Aspergillus* or *Candida* or *Malassezia* spp or *dermatophytes*.

Commonly, otomycosis appears in individuals with impacted cerumen or subjects utilizing hearing aids. A moist environment is created which facilitates accumulation and colonization of fungi. Antibiotic therapy adopted for treating otitis externa can initiate the condition [1,2].

An estimated 2% to 7% instances with clinical signs and symptoms of otitis externa are engendered by concurrent fungal infection [1,2].

Fungal organisms can appear as authentic infective agents or fungal spp may simply colonize external ear cutis on account of compromised, localized host immunity arising secondary to bacterial infection [1,2].

Predisposing factors engendering otomycosis emerge as a humid environment, impacted cerumen, ear instrumentation, enhanced topical antibiotics or steroids, immunocompromised host, employment of hearing aids with occlusive ear moulds or individuals subjected to open cavity mastoidectomy [1,2].

Disease pathogenesis

Otomycosis appears to be induced by cerumen impaction which augments fungal growth. Also, autoinoculation of external auditory canal engenders otomycosis, especially in subjects with untreated dermatomycosis [3,4].

External auditory canal functions as a habitation and route of transmission of fungal organisms generation otomycosis. Additionally, tropical conditions, trauma, bacterial otitis, anomalies of the ear and employment of hearing aids contribute to emergence of otomycosis.

Tropical climate with exposure to dust and fungal contaminants, predominantly discerned in individuals of low socioeconomic status augments disease occurrence [3,4].

Inappropriate utilization of fluoroquinolone or neomycin- polymyxin B ear drops enhances possible occurrence of otomycosis [3,4].

Post mastoidectomy subjects or individuals with preceding surgical intervention of mastoid cavity contributes to occurrence of otomycosis. Thus, it is posited that disease eradication can be challenging with occurrence of mastoid cavity [3,4].

Besides, diabetes mellitus, steroid administration, human immune deficiency virus (HIV) infection, chemotherapy, diverse malignancies and invasive therapeutic procedures can induce otomycosis [3,4].

Previous surgical intervention within the ear predisposes to otomycosis. Repetitive drainage of ear or topical antibiotics can modify bacterial microflora within external auditory canal with consequent super infection with nosocomial fungi [3,4].

Altered anatomy of external auditory canal as engendered with canal wall down surgical procedures may alter cerumen production or proportionate ear canal humidity, factors which augment fungal propagation [3,4].

Perforation of tympanic membrane occurs due to avascular necrosis of the membrane. Also, mycotic thrombosis within adjacent blood vessels may be discerned. Membrane perforation can appear as a consequence of fungal inoculation within medial aspect of external auditory canal or as a result of direct extension of disease from adjoining cutaneous surfaces [3,4].

Clinical elucidation

Fungal infection of the ear is generally unilateral. Characteristic clinical symptoms appear as inflammatory pruritus, scaling and otalgia. Commonly, otomycosis represents with distinctive clinical symptoms such as pruritus, discharge or pain in the ear [5,6].

Besides, fungal infections manifest with characteristic features of otitis externa. Initially discerned clinical symptoms appear as deafness, pruritus, otalgia, otorrhoea and tinnitus. During the course of disease evolution pruritus, pain, erythema, swelling, ear discharge, fullness of the ear and gradual hearing loss are delineated [5,6].

Clinical manifestations as pain, loss of hearing, aural fullness appear due to accumulation of fungal debris within the external auditory canal. Pruritus is a pathognomonic symptom of otomycosis [5,6].

Upon physical examination, otomycosis demonstrates dense aggregates of fibrinous debris, watery ear discharge and miniature, well circumscribed foci of granulation tissue confined to the external acoustic canal or tympanic membrane [5,6].

Histological elucidation

Mycotic infection is associated with inflammation, superficial epithelial aggregates of debris constituted of fungal hyphae and exuding suppuration. Superimposed stratified squamous epithelium demonstrates acanthosis, parakeratosis and focal aggregates of intraepithelial neutrophils [7,8].

Fungal hyphae and yeast forms are accumulated within superficial parakeratotic plaques. Sub-epithelial aggregates of chronic inflammatory cells such as lymphocytes and macrophages are characteristic [7,8].

Uncomplicated otomycosis is usually devoid of accumulated fungal organisms within subjacent epithelium. Deep seated fungal infections are exceptional and engendered by organisms such as cryptococcus or Blastomyces [7,8].

Investigative assay

Aspergillus and *Candida* spp are the commonest fungal pathogens associated with otomycosis. Clinical discernment of *Candida* infection can be challenging as the fungi are devoid of a characteristic appearance and the condition may manifest as otorrhoea unresponsive to aural antimicrobial agents although the organisms can be suitably identified by fungal culture [7,8].

Therapeutic options

Appropriate elimination or regulation of predisposing factors and adoption of pertinent antifungal therapy are necessitated for comprehensive eradication of otomycosis. Cogent treatment strategies are debridement of localized, infected tissue, cessation of topical antibiotics and administration of local and systemic antifungal agents [9,10].

Otomycosis can be a challenging disease to treat and requires extended therapy of up to 8 weeks or beyond along with meticulous monitoring.

Suitable topical antifungal agents in combination with frequent mechanical debridement is optimal and recommended for rapid resolution of clinical symptoms which generally occurs within a period of two weeks in appropriately treated instances [9,10].

Nevertheless, reoccurring or residual disease is commonly discerned. Efficacious therapy of otomycosis can be extensive and prolonged. Around 60% subjects employ topical antibiotics such as neomycin, polymyxin-B, ciprofloxacin or hydrocortisone and oral antimicrobial agents for treating presumed otitis media prior to precise disease discernment [9,10].

Pertinent therapeutic agents employed in conjunction with meticulous mechanical debridement of fungal elements discerned within the external auditory canal are generally efficacious. Agents such as clotrimazole, miconazole, ketoconazole, itraconazole, lanconazole, voriconazole, fluconazole, bifonazole, econazole, terbinafine, nystatin or salicylic acid appear competent in treating otomycosis. Incriminating fungi can be resistant to fluconazole [9,10].

Topical clotrimazole is associated with brisk resolution of fungal infection along with decimated proportionate disease reoccurrence [9,10].

Caspofungin is a contemporary antifungal agent with significant fungicidal activity, predominantly employed for treating invasive aspergillosis and candidiasis. However, certain species of *Candida* can be resistant to caspofungin, fluconazole or itraconazole [9,10].

Terbinafine is contemplated to be a superior antifungal agent for treating otomycosis arising due to *Aspergillus* or *Candida* spp [9,10].

Oral antifungals can be adopted for treating severe disease and instances with inadequate response to topical therapy. Nevertheless, oral antifungals may be inefficacious when ingested in the absence of adequate localized treatment [10,11].

Majority of individuals ameliorate with precise, preliminary therapy. Proportionate disease reoccurrence is significant. Disease eradication can be challenging within post-mastoidectomy subjects or immunocompromised individuals [10,11].

Complications associated with otomycosis are serous otitis media, perforation of the tympanic membrane or external auditory canal osteitis. Otomycosis may be associated with tympanic membrane perforation during initial representation which may also arise during therapeutic intervention and heals with resolution of infection. Perforation of tympanic membrane and serous otitis media usually meliorate with cogent treatment. External auditory canal osteitis may appear concurrent to diabetes mellitus [10,11].



Figure 1: Otomycosis demonstrating aggregates of debris imbued with fungal hyphae admixed with inflammatory suppuration [12].

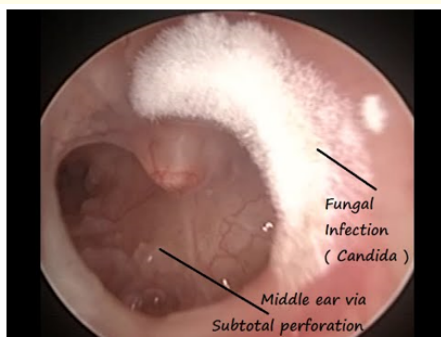


Figure 2: Otomycosis exhibiting subtotal perforation of the tympanic membrane admixed with cotton-wool like aggregates of candida [13].

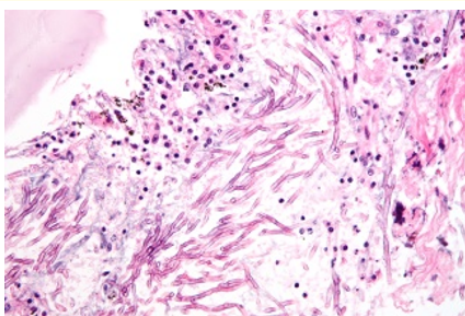


Figure 3: Otomycosis exhibiting septate hyphae of aspergillosis intermixed with an acute and chronic inflammatory cell infiltrate composed of neutrophils, lymphocytes and plasma cells [14].

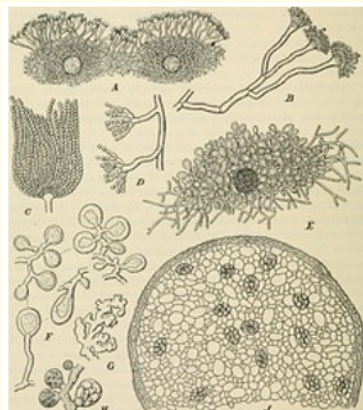


Figure 4: Otomycosis enunciating budding fungal hyphae, mycelial and sporulated forms incriminating the external acoustic canal [15].

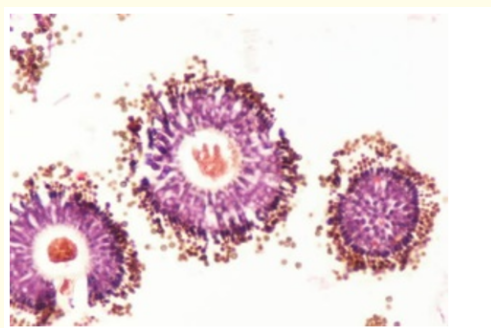


Figure 5: Otomycosis exemplifying spores and budding fungal hyphae with commingled inflammatory granulation tissue [16].

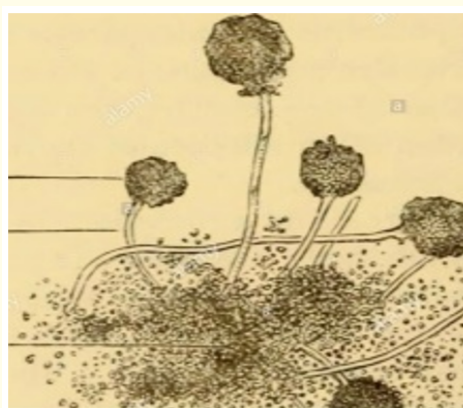


Figure 6: Otomycosis delineating fungal spores and budding hyphae commingled within inflammatory granulation tissue [17].

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12. Image 1 Courtesy: Ijorl.com.
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