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Otomycosis: Main challenging microbial agent causing otitis externa in northern Iran

Behrad Roohi[1], Shadman Nemati[2], Abbas Alipour[3], Leila Faeli[1], Sabah Mayahi[4], Iman Haghani[1], Tahereh Shokohi[1,4]
[1]Invasive Fungi Research Center, Communicable Diseases Institute, Mazandaran University of Medical Sciences, Sari, Iran
[2]Department of Otolaryngology and Head and Neck Surgery, Otorhinolaryngology Research Center, School of Medicine, Guilan University of Medical Sciences, Rasht, Iran
[3]Department of Community Medicine, Faculty of Medicine, Mazandaran University of Medical Sciences, Sari, Iran
[4]Department of Medical Mycology, School of Medicine, Mazandaran University of Medical Sciences, Sari, Iran

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Objectives: Otitis externa is one of the most common ear diseases, caused by bacterial and fungal agents. Accumulation of epithelial cells, cellular detritus, waxy substances, and microorganisms in the ear canal provide a proper environment for fungal growth, leading to annoying congestion. In this study, we aimed to identify the fungal causative agents and determine their susceptibility to the antifungal drugs were examined.

Methods: All patients with suspected otitis externa, referred to Amir-al Momenin Referral Centre from October 2020 to November 2021 entered to study. A total of 200 patients' ear discharge and debris collected samples were examined by direct examination and cultured on SDA supplemented with chloramphenicol. Definitive identification of grown fungi was made by PCR-RFLP and sequencing of ribosomal DNA. *In vitro* susceptibility testing of the fungal isolates against eleven different antifungal agents was analyzed using the CLSI broth microdilution methods.

Results: A total of 101 (50.5%) cases were confirmed with otomycosis. Most patients were in their fifth decade of life ($n = 35$, 34.6%), female ($n = 57$, 56.4%), and had unilateral ear involvement ($n = 78$, 77.2%). The most common symptoms were earache (56.4%) and itching (51.4%). Most underlying predisposing factors are overaggressive using cotton swabs (65.3%) and history of topical antibiotics (48.5%). Among the 117 fungal isolates, *Aspergillus* section *Nigri* was the most common agent 58 (49.57%), followed by *Aspergillus* section *Flavi* 19 (16.23%), *C. parapsilosis* 14(11.96%), *Aspergillus* section *Fumigati* 12 (10.25%), *C. orthopsilosis* 6 (5.12%), *C. albicans* complex 5 (4.27%), *Mucor* spp. 2 (1.71%), and *Syncephalastrum* spp. 1 (0.85%). Mainly, all tested antifungals were active against the most isolates of *Aspergillus*, aside from tioconazole, nystatin, and terbinafine, which showed low *in vitro* effects. Also, nystatin and itraconazole showed higher GM MICs in against all *Candida* species isolates. Conversely, amphotericin B (GM = 0.07129) in *Aspergillus* and voriconazole (GM = 0.03686) in *Candida* showed the highest antifungal activity. Regarding ECV values represented by ECOFFs, one of each *A. niger* (MIC 8 μg/mL), *A. flavus* (MIC 2 μg/ml), and A. *fumigatus* (MIC 2 μg/ml) isolates were resistant to itraconazole.

Conclusion: In this study, inconsistent with previous ones, fungal agents have overcome bacterial ones as the etiology of otitis externa. This result may relate to misuse or improper use of topical steroids, antibiotics, and inappropriate control of infection. In addition, our sample collection site was an ENT referral center and many patients have been admitted to this center due to treatment failure and suffered from recurrence. *Aspergillus* section *Nigri* isolation domination was seen, in agreement with other results. The MICs distribution of *Aspergillus* species isolates against triazole antifungals are close to ECVs defined by the CLSI and likely outrun it over time. We recommend that physicians request drug susceptibility testing before antibiotic therapy, to prevent the development of resistance.

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A masquerading case of *Cryptococcus neoformans*

Divyashree S[1], Tushar Sontakke[1], Satish Kulkarni[1], Ashok Pandey[1], Archana Thakur[1], Panchami Debbarman[6], Shalini Yadav[1]
[1]MGM New Bombay Hospital, Vashi, Navi Mumbai, India

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Introduction: Cryptococcal infections are commonly seen in immunocompromised hosts, especially HIV-infected patients and patients on immunosuppressive therapy. *Cryptococcus* shows a strong tropism for the central nervous system however cutaneous tropism is not uncommon. Here we describe an HIV-negative immunocompromised patient who developed disseminated cryptococcosis with the predominant presentation being painful thigh lesions.

Case Details: A 56-year-old lady presented with skin lesions and burning pain over both thighs for 1 month and a high-grade fever for 15 days. Her thigh lesions were initially treated elsewhere as tinea corporis. Her previous medical history was significant for poorly controlled diabetes, and chronic liver disease (secondary to autoimmune hepatitis or AIH). Her ongoing medications included prednisolone, anti-diabetics, and tenofovir. At presentation, the only significant physical findings were large irregular areas of central hypo-pigmentation with peripheral hyper-pigmentation and superficial small blisters over both thighs. These areas showed signs of inflammation. The empiric antimicrobial therapy included piperacillin-tazobactam and fluconazole (in view of prior urine culture growing *Candida* species). Over the next 48 h, fever continued, thigh pain worsened and the lesions on thighs blistered with violaceous discoloration. Her blood cultures sent at admission grew *Candida kruzei,* resistant to fluconazole, sensitive to voriconazole, amphotericin and echinocandins. Beta-D Glucan was also elevated. Fluconazole was discontinued, and anidulafungin was initiated. A repeat culture (prior to the start of anidulafungin) was still positive for C. *krusei*, but a subsequent blood culture (after the start of anidulafungin) was negative. Echocardiography did not show features of endocarditis. After a transient improvement, she worsened again with breakthrough fever, hypotension, and worsening of thigh lesions with eschar formation. Wound debridement was done, and antibiotics were escalated to carbapenem and polymyxins. Tissue cultures (sent during wound debridement) grew carbapenem-resistant klebsiella pneumonia. With these, the fever and her wounds were better; but the fever still persisted and she described a persisting severe burning pain over both the thigh wounds which did not respond to several analgesics.

A few days later, the fever worsened, and she had hypotension and disorientation. Blood and urine cultures were repeated. Repeat Serum beta D glucan levels were elevated. CSF examination was not possible in view of severe coagulopathy. The blood and wound cultures both grew *C. neoformans* (confirmed on MaldiTOFF). Antibiotics and anidulafungin were discontinued; liposomal amphotericin and flucytosine were started. Patient had significant improvement in sensorium, fever, and thigh pains within the next 72 h. Later, liposomal amphotericin was switched to conventional amphotericin because of financial constraints. This was followed by acute kidney injury and a flare of AIH. Amphotericin and flucytosine were withheld for a few days; a pulse of methylprednisolone was needed for AIH. Following resolution of acute kidney injury and AIH flare, conventional amphotericin B and flucytosine were restarted (and given for cumulative 3 weeks). This was followed by fluconazole consolidation. At hospital discharge, although there were raw areas over the thighs, these were healthy and she was otherwise well. A total of 3 months later, skin grafting was successfully performed.

Conclusions: *Cryptococcus* can be a great masquerader, and can mimic a variety of conditions. A high index of suspicion is required to clinch the diagnosis.