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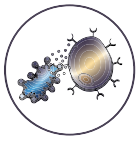

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Review Article

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Fungal Opportunist Infection: Common and Emerging Fungi in Immunocompromised Patients

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Abstract

The frequency of systemic fungal infection has significantly elevated in patients with immunosuppressive state in latest years worldwide. *Candida spp.* and *Cryptococcus spp.* are the most commonly yeasts isolated in clinical practice. The most prevalent filamentous fungi (moulds) identified are *Aspergillus spp.*, but *Fusarium spp.*, *Scedosporium spp.*, *Penicillium spp.*, and Zygomycetes are progressively more seen. Other no frequent yeasts are emerging as important opportunistic human pathogens like *non-albicans Candida* species, *Trichosporon* species, *Rhodotorula* species and *Non-neoformanscryptococcus* species. The increase in the number of cases of invasive fungal infections is a consequence of the emerging practices including: enlarging use of aggressive and exhaustive cancer chemotherapeutic regimens, immunosuppressive therapy for autoimmune illness and transplantation, and recent antimicrobial prophylactic practices.

Keywords

Opportunist infection; Common fungi; Emerging fungi

Introduction

The frequency of systemic fungal infection has considerably elevated in patients with an immunosuppressive state in current years. As diagnostic methods have been advanced and the number of immunocompromised patients (AIDS, hematopoietic stem cell transplantation HSCT, chemotherapy for cancer patients, autoimmune disorder) has increased spectacularly, clinicians and scientists have started to research more seriously into the mechanisms of disease and their correlation with vulnerable populations [1].

Until a few years ago, *Candida spp.* was retained the cause of the main part of fungal infections, but over the years a change in the common pathogens has been noticed as *Cryptococcus spp.* [2], *Aspergillus spp.*, Zygomycetes, *Fusarium spp.*, *Scedosporium spp.* [3] and *Penicillium spp.* [2]. Other infrequent yeasts are emerging as important opportunistic human pathogens such as *Non-albicans Candida* species, *Trichosporon* species, *Rhodotorula* species, *Non-neoformanscryptococcus* species [4]. The Table 1 is comparing the results of five studies regarding the distribution of different fungi in immunocompromised patients. It demonstrate that *Candida spp.* is the most frequent in Parmar et al., Jahromi et al., and Kashyap et al. studies, however *Cryptococcus spp.* and *Aspergillus spp.* are more prevalent in Chen et al. and Pagano et al. study successively. Other genuses are detected with low rate such as *Trichosporon, Fusarium spp.* and *Geotricum.*

Invasive Fungal Infections (IFIs) were initially described in 2002, with modifications occurring in 2008, including a change in nomenclature to "Invasive Fungal Disease" (IFD) by the European Organization for Research and Treatment of Cancer/Invasive Fungal Infections Cooperative Group and the National Institute of Allergy and Infectious Diseases Mycoses Study Group (EORTC/MSG) Consensus Group [5]. Confirmed IFD are separated into two categories: IFD caused by yeasts (*Candida* species and *Cryptococcus*) and IFD by molds (*Aspergillus*) and rely on blood cultures, culture or microscopic analysis from sterile anatomic sites, or researching cryptococcal antigen in cerebrospinal fluid. There are different criteria for determining proven endemic mycoses (*Coccidioides* species, *Histoplasma*, and *Blastomyces*). De Pauw et al. [6] have explained in detail the definitions of probable and possible infections which are very complex. Of note, *Pneumocystis jirovecii* infections are excluded from these definitions.

The change of IFI epidemiology feature can be related to several changes occurring in the immunocompromised host such as: the genetic variation throughout key innate or adaptive immune response genes which could affect susceptibility to, along with the outcome of, IFD. In addition to the environment witch influence the epidemiology of mold infections, in particular Invasive Aspergillosis (IA). This one can be modified by meteorological conditions, which change in regions of endemicity. As well as the defects in the immune system such as neutropenia and impaired cell-mediated immunity that predisposes individuals to mold infections [3]. So that enlarging use of aggressive and intensive cancer chemotherapeutic regimens, immunosuppressive therapy for autoimmune disorder and transplantation has generated an increase in the number of vulnerable hosts in recent years. However, we accept that the most significant variables representing just the emerging trends, specifically the increase in the frequency of invasive mycelial infections, are the modifications in transplantation practices and recent antimicrobial prophylactic protocols, which include not just antifungal but also antibacterial and antiviral agents [7].

An elevating incidence of invasive fungal infections might simply reflect greater laboratory expertise in the diagnosis and identification of fungi especially molds.

Organisms

Candida spp.

Candida spp. is currently one of the five principal causes of blood infections in hospitals, and the rise in candidemia cases has been reported essentially among patients taking antibiotics, under immunosuppressive therapy or parenteral nutrition and patients exposed to multiple invasive procedures. Various studies evaluate mortality attributed to *Candida spp.* to be 38%, although it can fluctuate from 50 to 60% [8]. In the United States *Candida* species are classified as the fourth most prevalent cause of hospital-acquired

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Table 1: Distribution of fungi detected in immunocompromised patients in different studies.

	Candida spp	*Aspergillos spp*	*Cryptococcus spp*	*Fusarium spp*	*Trichosporon*	*Geotricum*	Other
Parmar et al. 2012	55%	3%	4%	-	-	-	-
Chen et al. 2012	16.7%	16.7%	50.0%	-	-	-	
Pagano et al. 2006	32.5%	57.6%	1.4%	2.8%	1.3%	-	4.3%
Jahromi et al. 2005	69.4%	13.9%	4.2%	2.8%	1.4%	-	8.4%
Kashyap et al. 2012	18.3%	6.9%	0.6%	0.3%	0.3%	0.6%	-

BSI (bloodstream infection). The ARTEMIS Global Antifungal Surveillance Program found that *C. albicans* was the most prevalent (63-70%) candidal cause of invasive fungal infections, succeeded by *C. glabrata* (44%), *C. tropicalis* (6%), and *C. parapsilosis* (5%) [9]. The causes of this high rate of infection are unclear, a wide variety of factors might be implicated, such as limited resources offered for medical care and training programs, obstacles in developing countries to implement infection control in hospitals, reduced numbers of health-care personnel to assist patients in critical care units, and not using so much antifungal protocols for treatment of patients with high risk [10].

Candida can cause a diverse disease. Diagnosing clinically invasive Candida infection is difficult: as the majority of symptoms are non-specific, and generally are those of sepsis, and positive cultures can only be delayed in the course of the infection. Treating with delay or no treating are independent predictors of mortality in invasive candidiasis [2]. In healthy hosts, Candida species generally constitute commensal flora of the gastrointestinal tract and genital tract, but they are also opportunistic pathogens that have the potential to cause numerous superficial and systemic infections. They transform into pathogens if the host's resistance to infection is reduced locally or systemically. In particular, neutropenia, neutrophil dysfunction, and disruption of mucosal barriers are the principal risk factors for disseminated infections. In patient with immunosuppressive state, two major portals of entry for disseminated *Candida* infection are reported which are the translocation from the gastrointestinal tract and intravascular catheters. Without doubt one of the important reasons for candida's virulence is its flexibility in adaptation to several different habitats, and the formation of biofilms that increases its capacity to adhere to surfaces and leads to infection [4].

Cryptococcus spp.

Cryptococcosis is due to a complex group of basidiomycetes, generally yeasts of 2-10 mm in diameter. This complex is named *Cryptococcus neoformans* and is classified in two varieties: *C. neoformansvarneoformans* (serotypes A and D, genotypes VNI–IV) and *C. neoformansvargattii* (serotypes B and C, genotypes VGI–IV) [11].

Cryptococcus neoformans is the most frequent cause of cryptococcosis, and is usually obtained by inhalation. Pulmonary cryptococcosis could be asymptomatic or can be revealed by non-specific symptoms like cough, fever or pleural symptoms. Meningitis is a common feature of infection, particularly in HIV-seropositive patients and solid-organ transplant receiver. All cerebrospinal fluid (CSF) specimens from HIV-seropositive patients, transplant receivers and patients with sarcoidosis that show anomalous biochemical parameters or increased leukocytes without satisfactory explanation need to be tested for cryptococcal antigen and cultures incubated for an extended period to facilitate isolation of the organism [2].

As saprophytes, *Non-neoformanscryptococci* are occasionally reported as human pathogens. However, infections caused by *non-neoformanscryptococcal* have been noticed as sporadic cases in patients with immunosuppressive state, specifically those with advanced HIV infection and patients with cancer who are being candidate to transplant surgery [12]. *Cryptococcus laurentii* and *Cryptococcus albidus* produce 80% of cases. However, *Cryptococcus curvatus*, *Cryptococcus humicolus*, and *Cryptococcus uniguttulatus* may cause opportunistic infections in human host's [11].

Aspergillus spp.

Aspergillus is a very old genus of fungi, with a wide variety of species, most of which are of medical importance. It is a ubiquitous and thermophilic filamentous fungus. Human host's contamination occurs mainly by inhalation. The subsequent clinical condition depends on the balance between the infectious charge (i.e. the number of conidia inhaled) against the immune status of the host. Acute invasive aspergillosis (IA) is a damaging opportunistic infection. In spite of advances in therapy, IA is correlated to significant morbidity and mortality, varying from 30% to 70% in transplant receivers [13].

Patients exposed to IA are those with prolonged neutropenia, advanced AIDS, hematopoietic stem cell transplant (HSCT) receivers, and solid organ transplant recipients (particularly lung transplant recipients) [14]. The commonest causal agent of IA is the *Aspergillus fumigatus*, with airborne conidia resulting in almost wide-ranging and constant exposure in almost all individuals [15]. *Aspergillosis* can affect practically any organ in the patient with immunosuppressive state, but sinopulmonary disease is the most frequent.

Angioinvasion of hyphae causing vascular thrombosis and tissue infarction and coagulative necrosis is feature of invasive aspergillosis in neutropenia, but is also detected in non-neutropenic allogeneic HSCT receivers with GVHD (Graft Versus Host Disease). Early diagnosis of aspergillosis in seriously immunocompromised patients is still difficult. Bronchoalveolar (BAL) cultures have an estimated sensitivity of 50% in focal pulmonary lesions and certain diagnosis often needs an invasive procedure (e.g. thoracoscopic biopsy) [16].

Computed tomography (CT) of the chest has been recommended for the early diagnosis of IPA, as this method enables better screening of the pattern and distribution of known abnormalities than radiography, in addition to the revelation of infiltrates not apparent on chest radiography. The more characteristic CT finding in circumstances of IPA is a "halo sign" which is ground-glass

attenuation around focal nodules, corresponding pathologically to hemorrhage around a focus of pulmonary infarction [17].

Other diagnostic methods that have been applied include culture, histopathology, and serologic techniques; galactomannan in serum by immunoenzymatic assays has proven to be efficient when determined in multiple serial samples in numerous circumstances, such as early diagnosis in probable invasive infection (with concomitant improvement in outcome), and therapeutic follow-up to monitor treatment efficacy [18]. The measurement of plasma (1-3)-β-D glucan concentration is an additional important screening method revealing deep mycoses such as IPA. As these blood tests identify circulating fungal components, their specificities regarding fungal infections are about 90% [17].

Penicillium species

Among more than 200 species of *Penicillium, Penicillium marneffei* is the only dimorphic pathogenic organism [19]. *Penicillium marneffei* (PM) was initially identified in 1973, leading to human infection in a patient with Hodgkin Lymphoma. In the HIV/AIDS epidemic, it has been reported as one of the major opportunistic infection after tuberculosis and cryptococcosis in endemic region of Southeast Asia and Southern China. The most probable mode of PM transmission is inhalation of airborne conidia [20]. Penicilliose appears late during the course of HIV infection, usually at CD4 lymphocyte counts <100 cells/mL [21], reaching the blood stream, skin, liver, spleen, lymph nodes, bone marrow, lung and gastrointestinal tract [22]. In HIV infected patients, the skin lesions as umbilicated papules that appear like those of *molluscum contagiosum*are frequent, while in individuals not infected with HIV, abscesses are the more prevalent manifestation of cutaneous involvement [23]. Laboratory identification is done by microscopy as well as culture of skin lesions, blood, lymph node, and other body fluids [24].

Fusariosis species

One of the most frequent mold IFIs reported in immunocompromised patients is due to *Fusarium spp* septated fungi. Three species important clinically have been identified: *F. solani*, *F. oxysporum*, and *F. verticillioidis*. In immunocompetent patients, fusariosis is generally a superficial infection (keratitis and onychomycosis) or leading to locally invasive skin or soft-tissue infection. However, in immunocompromised patients Fusariosis causes fungemia (>60%), with or without endocarditis, skin lesions (60-80%), sinusitis, and lung disease [25]. Risk factors for grave fusariosis include prolonged neutropenia and T-cell immunodeficiency, particularly in hematopoietic stem cell transplant recipients with severe graft-versus-host disease. The prognosis is mediocre and is identified largely by level of immunosuppression and extent of infection, with basically a 100% mortality rate in persistently neutropenic patients with disseminated disease [26].

Mucormycosis species

The subphylum Mucoromycotina (formerly Zygomycetes) is opportunistic fungi producing acute rhinocerebral and pulmonary diseases with thromboses, infarction and blood vessel invasion. Mucormycoses exist throughout the world. The disease is exceptional in both sexes, but has started to become more prevalent and classified on the third place among opportunistic fungal infections after candidiasis and aspergillosis [27]. The most frequent species are *Mucor*, *Rhizopus*, *Rhizomucor*, and *Lichtheimia* (before known as *Absidia*), while *Cunninghamella Bertholletia* and *Apophysomyces elegans* also are common causes [1]. Most important mucormycosis risk factors include profound and prolonged neutropenia and chronic high-dose corticosteroid use, in addition to tissue iron excess, uncontrolled diabetes, hypertriglyceridemia, voriconazole prophylaxis (mainly in HSCT), or previous skin and soft-tissue trauma [28].

Trichosporon species

Trichosporon is one of a basidiomycetous yeast genus producing septate hyphae, arthroconidia, yeasts, and pseudohyphae. Invasive fungal infection due to Trichosporon species is the second commonest cause of yeast fungaemia in patients with malignant haematological disease (after Candida species). The principal Trichosporon species causing invasive fungal infections are *Trichosporonasahii, Trichosporonasteroides, Trichosporoncutaneum, Trichosporoninkin, Trichosporonmucoides* and *Trichosporonovoides* [29]. In addition to that *T. asteroid*s and *T. cutaneum* may cause superficial skin infections, *T. ovoides* produces white piedra of the scalp, and *T. inkin* induces white piedra of the pubic hair [30,31].

Invasive Trichosporon infection has been progressively moreobserved during 30 years ago. The majority of cases appear in patients with haematological diseases, especially those patients with acute leukaemia [32].

Rhodotorula species

One of emerging opportunistic pathogens is Rhodotorula genus, especially in immunocompromised patients. *Rhodotorula mucilaginosa* (also called as *Rhodotorula rubra*) is the most frequent cause of *Rhodotorula* species fungaemia, followed by *Rhodotorula glutinis* and *Rhodotorula minuta* [33]. In the ARTEMIS surveillance project, *Rhodotorula* species were the fourth most prevalent noncandidal yeast identified from clinical specimens (4.2% of 8821 isolates) [9]. Patients having cancer (including those undergoing bone marrow transplantations) and patients with AIDS are more susceptible to systemic *Rhodotorula* infection. Patients who have had abdominal surgery, cirrhosis, autoimmune diseases, or burns are also exposed to this infection. A majority of cases of *Rhodotorula* infection are fungaemia caused by catheters, endocarditis, and meningitis [33].

Scedosporium spp.

As emergent opportunistic pathogens, *Scedosporium spp*. are progressively noticed as origins of resistant life-threatening infections in patients with immunosuppressive state. It also leads to a wide variety of conditions, such as mycetoma, saprobic involvement and colonization of the airways, sinopulmonary infections, extrapulmonary localized infections, and disseminated infections. Invasive infections with *scedosporium* are also involving central nervous infection ensuing near-drowning accidents. The commonest sites of infection are the lungs, sinuses, bones, joints, eyes, and brain. *Scedosporium apiospermum* and *Scedosporium prolificans* are the two main medically significant species of this genus [34]. In advanced HIV infection, patients can acquire infections with *Scedosporium spp*. while neutropenia. Contrary to *cryptococcosis* and *histoplasmosis*, *scedosporiosis* usually will not appear at the beginning during HIV disease [35]. Current progressin molecular taxonomy has advanced the understanding of the genus *Scedosporium* and has revealed a wider variety of species than previously identified. Microbiological diagnosis of *Scedosporium spp*. recently relies on culture and morphological characterization. Molecular methods for clinical

microbiological identification of *Scedosporium spp*. are nowadays investigational [35].

Current Advances in Identification of Fungal Species

Identifying fungal clinical isolates correctly and timely is a significant component in the treatment of patients with invasive fungal infections; especially immunocompromised one. Recent fungal detection methods, such as biochemical analyses, germ tube examination, chlamydospore examination on rice Tween 80 agar, and evaluation of colonial morphologies on chromogenic agar, constitute conventional and efficient approaches, but these routine methods can necessitate 48 to 72 hours or longer to be able to deliver definitive identifications, resulting in considerable delays in correct patient management [36,37].

PCR constitutes one of the more recent progresses in fungal genomics lowing identification of clinical isolates. It is demonstrating to be far excellent when compared to conventional biochemical identification panels [38]. Pyrosequencing is a considerably not expensive, remarkably rapid DNA sequencing method that requires novel chemistry to sequence pre-selected regions of the genome to short fragments (>70-bp) [39,40]. The important function of pyrosequencing has been proved in large comparative studies focusing on differentiating closely-related and distinct pathogenic fungi and at diagnosing uncommon yeast species [39-41]. In Borman et al. [39] study over 450 yeast isolates were processed to conventional identification, molecular identification based on 28S rRNA gene sequencing, as well as pyrosequencing. Pyrosequencing efficiently permitted fast and unambiguous identification of >98% of isolates, which include 40 various species of pathogenic yeasts. In other study of Boyanton et al. [42], it is reported that DNA pyrosequencing was efficient in realizing the exact identification of 8 distinct, including *C albicans, C dubliniensis, C glabrata, C guilliermondii, C krusei, C lusitaniae, C parapsilosis*, and *C tropicalis*. All these studies prove the usefulness of this perfect current method.

Treatment and Prevention

To improve outcomes for patients with invasive fungal infections, complementary strategies are suggested such as the use of immunomodulators and combination therapies.

Adjunctive interferon γ could be suggested for refractory *cryptococcosis*. The function of interferon γ in invasive candidiasis and other emerging yeast infections is not well defined. Even if combination therapy is well established for treatment of cryptococcal meningitis, its rolein invasive candidiasis is unclear. Combination therapy is highly recommended for candida endocarditis and other difficult-to-treat cases. Only some data are offered for other emerging opportunistic yeast infections, while combination regimens could be useful as salvage therapy [4].

Antifungal prophylaxis is strongly suggested for high risk patients undergoing pancreas, liver and small-bowel solid organ transplantations, for chemotherapy-induced neutropenia patients, receivers of stem cell transplantation with neutropenia, and it may be recommended for adults in intensive care units who are at high risk for invasive candidiasis. Common use of anti fungal prophylaxis and therapy has possibly moved the epidemiological features of invasive fungal infections to these emerging yeast infections [4].

References

1. Sifuentes-Osornio J, Corzo-León DE, Ponce-de-León LA (2012) Epidemiology of Invasive Fungal Infections in Latin America. Curr Fungal Infect Rep 6: 23-34.
2. Enoch DA, Ludlam HA, Brown NM (2006) Invasive fungal infections: a review of epidemiology and management options. J Med Microbiol 55: 809-818.
3. Pagano L, Akova M, Dimopoulos G, Herbrecht R, Drgona L, et al. (2011) Risk assessment and prognostic factors for mould-related diseases in immunocompromised patients. J Antimicrob Chemother 66: i5-i14.
4. Miceli MH, Díaz JA, Lee SA (2011) Emerging opportunistic yeast infections. Lancet Infect Dis 11: 142-151.
5. Barber CE, Barnabe C (2012) Another consequence of severe lupus: invasive fungal disease. J Rheumatol 39: 1772-1774.
6. De Pauw B, Walsh TJ, Donnelly JP, Stevens DA, Edwards JE, et al. (2008) Revised definitions of invasive fungal disease from the European Organization for Research and Treatment of Cancer/Invasive Fungal Infections Cooperative Group and the National Institute of Allergy and Infectious Diseases Mycoses Study Group (EORTC/MSG) Consensus Group. Clin Infect Dis 46: 1813-1821.
7. Singh N (2001) Trends in the epidemiology of opportunistic fungal infections: predisposing factors and the impact of antimicrobial use practices. Clin Infect Dis 33: 1692-1696.
8. Mondelli AL, Niéro-Melo L, Bagagli E, Camargo CH, Bruder-Nascimento A, et al. (2012) Candida spp.: manual identification (reference method) and automated identification (Vitek system platform). J Venom Anim Toxins 18: 335-339.
9. Pfaller MA, Diekema DJ, Gibbs DL, Newell VA, Meis JF, et al. (2007) Results from the ARTEMIS DISK Global Antifungal Surveillance study, 1997 to 2005: an 8.5-year analysis of susceptibilities of Candida species and other yeast species to fluconazole and voriconazole determined by CLSI standardized disk diffusion testing. J Clin Microbiol 45: 1735-1745.
10. Das R, Ranganathan R (2012) An Overview of Changing Trends in Systemic Fungal Infections. WebmedCentral Microbiology 3: WMC003386.
11. Lin X (2009) Cryptococcus neoformans: morphogenesis, infection, and evolution. Infect Genet Evol 9: 401-416.
12. Khawcharoenporn T, Apisarnthanarak A, Mundy LM (2007) Non-neoformans cryptococcal infections: a systematic review. Infection 35: 51-58.
13. Pfeiffer CD, Fine JP, Safdar N (2006) Diagnosis of invasive aspergillosis using a galactomannan assay: a meta-analysis. Clin Infect Dis 42: 1417-1427.
14. Fouad L, Fathi MS, ElMaraghy AA (2013) The Clinical relevance of Aspergillus isolation from respiratory tract samples & detection of Aspergillus galactomannan antigen in serum of patients with acute exacerbation of COPD. Nat Sci 11: 40-43.
15. Barton RC (2013) Laboratory Diagnosis of Invasive Aspergillosis: From Diagnosis to Prediction of Outcome. Scientifica 2013: 1-29.
16. Segal BH, Walsh TJ (2006) Current approaches to diagnosis and treatment of invasive aspergillosis. Am J Respir Crit Care Med 173: 707-717.
17. Kami M, Tanaka Y, Kanda Y, Ogawa S, Masumoto T, et al. (2000) Computed tomographic scan of the chest, latex agglutination test and plasma (1AE3)-beta-D-glucan assay in early diagnosis of invasive pulmonary aspergillosis: a prospective study of 215 patients. Haematologica 85: 745-752.
18. Cruz Ch R, Piontelli L E (2011) [Invasive fungal disease in patients from five hospitals in the Valparaíso region, Chile: 2004 to 2009]. Rev Chilena Infectol 28: 123-129.
19. Chariyalertsak S, Sirisanthana T, Supparatpinyo K, Praparattanapan J, Nelson KE (1997) Case-control study of risk factors for Penicillium marneffei infection in human immunodeficiency virus-infected patients in northern Thailand. Clin Infect Dis 24: 1080-1086.
20. Chitasombat M, Supparatpinyo K (2013) *Penicillium marneffei* Infection in Immunocompromised Host. Curr Fungal Infect Rep 7: 44-50.
21. Carey J, Hofflich H, Amre R, Protic J, Perlman DC (2005) Penicillium marneffei infection in an immunocompromised traveler: a case report and literature review. J Travel Med 12: 291-294.

22. Le T, Wolbers M, Chi NH, Quang VM, Chinh NT, et al. (2011) Epidemiology, seasonality, and predictors of outcome of AIDS-associated Penicillium marneffei infection in Ho Chi Minh City, Viet Nam. Clin Infect Dis 52: 945-952.

23. Wortman PD (1996) Infection with Penicillium marneffei. Int J Dermatol 35: 393-399.

24. Vanittanakom N, Cooper CR Jr, Fisher MC, Sirisanthana T (2006) Penicillium marneffei infection and recent advances in the epidemiology and molecular biology aspects. Clin Microbiol Rev 19: 95-110.

25. Nunes Mdo C, Barbosa FB, Gomes GH, Bráulio R, Nicoliello MF, et al. (2011) Fatal right-sided endocarditis caused by Fusarium in an immunocompromised patient: a case report. Mycoses 54: 460-462.

26. Nucci M, Anaissie E (2007) Fusarium infections in immunocompromised patients. Clin Microbiol Rev 20: 695-704.

27. Perusquía-Ortiz AM, Vázquez-González D, Bonifaz A (2012) Opportunistic filamentous mycoses: aspergillosis, mucormycosis, phaeohyphomycosis and hyalohyphomycosis. J Dtsch Dermatol Ges 10: 611-621.

28. Sun HY, Singh N (2011) Mucormycosis: its contemporary face and management strategies. Lancet Infect Dis 11: 301-311.

29. Fleming RV, Walsh TJ, Anaissie EJ (2002) Emerging and less common fungal pathogens. Infect Dis Clin North Am 16: 915-933.

30. Walsh TJ, Groll A, Hiemenz J, Fleming R, Roilides E, et al. (2004) Infections due to emerging and uncommon medically important fungal pathogens. Clin Microbiol Infect 10: 48-66.

31. Marty FM, Barouch DH, Coakley EP, Baden LR (2003) Disseminated trichosporonosis caused by Trichosporon loubieri. J Clin Microbiol 41: 5317-5320.

32. Girmenia C, Pagano L, Martino B, D'Antonio D, Fanci R, et al. (2005) Invasive infections caused by Trichosporon species and Geotrichum capitatum in patients with hematological malignancies: a retrospective multicenter study from Italy and review of the literature. J Clin Microbiol 43: 1818-1828.

33. Tuon FF, Costa SF (2008) Rhodotorula infection. A systematic review of 128 cases from literature. Rev Iberoam Micol 25: 135-140.

34. Cortez KJ, Roilides E, Quiroz-Telles F, Meletiadis J, Antachopoulos C, et al. (2008) Infections caused by Scedosporium spp. Clin Microbiol Rev 21: 157-197.

35. Rollot F, Blanche P, Richaud-Thiriez B, Le Pimpec-Barthes F, Riquet M, et al. (2000) Pneumonia due to Scedosporium apiospermum in a patient with HIV infection. Scand J Infect Dis 32: 439.

36. Cetinkaya Z, AltindiÅŸ M, Aktepe OC, KarabiÃ§ak N (2003) [Comparison of different methods for the identification of Candida species isolated from clinical specimens]. Mikrobiyol Bul 37: 269-276.

37. Hospenthal DR, Beckius ML, Floyd KL, Horvath LL, Murray CK (2006) Presumptive identification of Candida species other than *C. albicans, C. krusei,* and *C. tropicalis* with the chromogenic medium CHROMagar Candida. Ann Clin Microbiol Antimicrob 5: 1.

38. Abu-Elteen KH, Hamad MA (2012) Changing Epidemiology of Classical and Emerging Human Fungal Infections: A Review. Jordan Journal of Biological Sciences 5: 215-230.

39. Borman AM, Linton CJ, Oliver D, Palmer MD, Szekely A, et al. (2010) Rapid molecular identification of pathogenic yeasts by pyrosequencing analysis of 35 nucleotides of internal transcribed spacer 2. J Clin Microbiol 48: 3648-3653.

40. Montero CI, Shea YR, Jones PA, Harrington SM, Tooke NE, et al. (2008) Evaluation of Pyrosequencing technology for the identification of clinically relevant non-dematiaceous yeasts and related species. Eur J Clin Microbiol Infect Dis 27: 821-830.

41. Ghannoum MA, Jurevic RJ, Mukherjee PK, Cui F, Sikaroodi M, et al. (2010) Characterization of the oral fungal microbiome (mycobiome) in healthy individuals. PLoS Pathog 6: e1000713.

42. Boyanton BL Jr, Luna RA, Fasciano LR, Menne KG, Versalovic J (2008) DNA pyrosequencing-based identification of pathogenic Candida species by using the internal transcribed spacer 2 region. Arch Pathol Lab Med 132: 667-674.

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