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Case report/Cas clinique

## *Saprochaete capitata* (*Geotrichum capitatum*), an emerging fungal infection in kidney transplant recipients

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### ABSTRACT

We are reporting the case of an 82-year-old Yemeni patient, renal transplant recipient who was admitted to our institution and who subsequently developed disseminated infection with *Saprochaete capitata*. This pathogenic fungus is rarely reported in patients with solid organ transplants. *Saprochaete capitata* is an emerging fungal pathogen, ubiquitously spread in the environment. This is the second case to our knowledge of infection with *Saprochaete capitata* in a renal transplant patient. Our patient was treated for multiple nosocomial infections with prolonged antibiotic courses. He succumbed to the infection with *Saprochaete capitata* after several weeks spent in the intensive care unit.

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### 1. Introduction

The field of solid organ transplant showed significant advances since the first kidney transplant between living identical twins in 1954 [1]. The progress is related mainly to the better use of immunosuppressive agents, prophylactic antimicrobials, and the expansion of preemptive and empirical antimicrobial therapy [2]. Despite these advances, infections remain one of the most challenging obstacles that face kidney transplant recipients with infections related to immunosuppressive therapy occurring later in the post-transplant period [3].

Fungal infections are a major complication post-solid organ transplantation. *Candida* species and *Aspergillus* species remain the most common cause of invasive fungal infections in this group of patients [4]. Rubin et al. categorized invasive fungal infections (IFI) following renal transplant into two groups: classic opportunistic infections (*Aspergillus*, *Candida*, and *Cryptococcus* infections and zygomycoses) and disseminated primary or reactivation infection of “geographically restricted” mycoses, associated with specific geographic and environmental exposures, those include histoplasmosis, coccidioidomycosis, blastomycosis, and paracoccidioidomycosis [5].

#### 1.1. Case presentation

This is an 82-year-old man from the Republic of Yemen with end-stage renal disease (ESRD) attributed to hypertensive nephro-

sclerosis who received a living related kidney transplant in 2003. He lived in Yemen and visited Lebanon periodically for regular clinic follow-ups with his transplant nephrologist at the American University of Beirut Medical Center (AUBMC). He suffered multiple comorbidities, including diabetes, dyslipidemia and benign prostate hypertrophy. His maintenance immunosuppression regimen consisted of tacrolimus, mycophenolate mofetil and prednisone as well as prophylaxis with trimethoprim, sulfamethoxazole for *Pneumocystis jirovecii* pneumonia.

In September 2016, he presented to the intensive care unit (ICU) with lower gastrointestinal bleed. He had had multiple hospitalizations since June 2016 for pneumonia, urinary tract infection for which he received several courses of antibiotics. He also developed high-grade CMV viremia (46500 DNA copies/ml of blood) and was treated with valganciclovir. On the day of his admission, he underwent a subtotal colectomy with an ileosigmoid anastomosis. His post-operative course was complicated by Methicillin Resistant *Staphylococcus aureus* (MRSA) and *Pseudomonas aeruginosa* pneumonia that were treated with linezolid and ceftazidime for 14 days. On the sixteenth day of his hospitalization, he developed *Candida glabrata* fungemia treated with caspofungin. Ten days later the patient developed septic shock secondary to cholecystitis with acute respiratory failure necessitating mechanical ventilation. He was hemodynamically unstable and received vasopressor support and meropenem. In view of the general status, it was opted to treat him conservatively. He remained in a critical condition despite negative blood cultures. A bile culture sent on the 39th day of hospitalization revealed *Saprochaete capitata* (*S. capitata*). Therefore, liposomal amphotericin B was started. Unfortunately, despite initiating antifungal therapy he passed away from refractory septic

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shock and multiorgan failure. A review of all the cultures revealed *S. capitata* growing from different sites including the deep tracheal aspirate, the laparotomy wound, bile and urine.

## 2. Discussion

*S. capitata* is a rare fungal pathogen, mostly reported in hematological malignancies [6]. The genus *Geotrichum* has been reported in Europe and the United States, it is a well-known pathogenic fungus in the Mediterranean area [7] that has also been reported recently from South East Asia [8]. Like the majority of invasive fungal infections, an immunocompromised state is the most recognized risk factor for infection with *Geotrichum capitatum* [9]. A few reports in the literature have connected it with others risk factors such as contaminated milk [10] or polytrauma [11]. The most common organ site of involvement is the lung as most patients present with respiratory infections. There is a major difference in the clinical spectrum of disease between *S. capitata* and invasive *Candida*: pulmonary infections are of major importance in geotrichosis unlike disseminated candidiasis where pneumonia is uncommon [12]. It is believed that *S. capitata* may colonize the lung during chemotherapy-induced neutropenia before disseminating through the blood to cause systemic infections [12] or alternatively, lead to a systemic blood infection which further disseminates to the lungs and liver [9] [13] [14]. The mortality rate attributed to *S. capitata* can be as high as 52 to 57 % and is greater than that associated with candidemia (36 %). The organism is relatively easy to grow in cultures from different body fluids. Laboratory data reveal cross-reactivity with *Aspergillus galactomannan* GM [15].

In the right clinical setting, it is appropriate to suspect invasive Geotrichosis in a patient with a positive GM test and pulmonary lesions if *S. capitata* is recovered from respiratory culture.

This case sheds the light on an emerging fungus that is notorious for causing infections in patients with hematological malignancies [16]. *S. capitata* has rarely been implicated as a pathogen in solid organ transplant recipients, as per the few case reports published recently [8,17]. To our knowledge, this is the second case report of such an infection in a solid organ transplant patient. Our patient received immunosuppressive therapy for years, albeit reduced during his ICU stay. Deepened by multiple courses of antibiotics, CMV reactivation and a prolonged ICU stay this severe immunosuppression justifies the invasive fungal infection.

We were not able to obtain GM and other fungal blood markers on our patient. This was a major limitation to our management. Had we suspected an invasive fungal infection earlier, the outcome could have been altered. Nevertheless, since the fungus grew from multiple sites this made our suspicion for an invasive fungal infection very high and antifungal therapy was initiated. Another important observation is the persistent low procalcitonin levels; this is particular to invasive fungal infections [18]. Procalcitonin is a marker of invasive bacterial infection which is used as an indicator for response to therapy in the ICU. Our patient suffered from a refractory septic shock and succumbed to an overwhelming sepsis despite the initiation of amphotericin B. There is no consensus regarding the optimal therapeutic regimen for the treatment of invasive infections caused by *S. capitata* [19]. In the absence of susceptibility breakpoints, either liposomal amphotericin B (alone or in combination with 5-fluorocytosine) or voriconazole have been suggested as the gold standard of therapy for this infection [20]. None of these susceptibility-testing methods were available to us to tailor antifungal therapy.

There are still some uncertainties and controversies in the diagnosis of *S. capitata*. In the clinical setting of immunosuppression

following transplantation, *S. capitata* may be the causative agent of IFI in transplant patients if recovered from a sterile site [6]. This can be expected in a patient receiving echinocandins, as *S. capitata* is resistant to this class of anti-fungals. With the increasing use of echinocandins in the oncology and transplant worlds, there is a breakthrough of infections with this *S. capitata*. These infections have been associated to the use of echinocandins for prophylaxis or treatment in one study [21]. The same conclusion was not confirmed by a recent review from India [8]. The role of echinocandins in favoring and promoting the emergence of this pathologic organism remains to be clarified as more studies are needed [22]. Rapid diagnosis in our case was challenging because the initial presentation mimicked any other invasive nosocomial infectious process [23] in addition to the limitations listed above: rapid diagnosis and susceptibility testing.

## Disclosure of interest

The authors declare that they have no competing interest.

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