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Fungal Microabscesses Mimicking Visceral Infarction

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Abstract

A 61-year-old man with angioimmunoblastic T-cell lymphoma receiving fourth-line therapy was admitted with febrile neutropenia. Blood cultures grew carbapenemase-producing *Klebsiella pneumoniae*, and targeted antibacterial treatment was initiated. Despite treatment, he rapidly developed septic shock and multiorgan dysfunction. Serial computed tomography (CT) imaging revealed progressive visceral hypoattenuating lesions involving the kidneys, spleen, and subsequently the liver. Initially interpreted as possible areas of hypoperfusion or infarction, their rapid progression over 48 hours raised concern for a disseminated infectious process, including fungal infection. Empirical anidulafungin was initiated for suspected invasive candidiasis, but the patient died 12 hours after intensive care unit admission. Postmortem blood cultures identified *Magnusiomyces capitatus* (formerly *Blastoschizomyces capitatus*), an organism characterised by intrinsic echinocandin resistance. Antifungal susceptibility testing revealed high echinocandin minimum inhibitory concentrations (MICs) and lower MICs for voriconazole and amphotericin B. Although the absence of histopathological confirmation prevents definitive attribution of the visceral lesions to fungal infection, this case highlights the diagnostic challenge posed by rapidly progressive visceral abnormalities in severely immunocompromised patients and the importance of early species-level identification and appropriate antifungal assessment.

Categories: Hematology, Internal Medicine, Infectious Disease
Keywords: antifungal resistance, blastoschizomyces capitatus, computed tomography, febrile neutropenia, haematological malignancy, invasive fungal infection, magnusiomyces capitatus, septic shock, visceral microabscesses

Introduction

Magnusiomyces capitatus (formerly *Blastoschizomyces capitatus*) is an emerging ascomycetous yeast responsible for life-threatening disseminated infections, particularly in patients with haematological malignancies and prolonged neutropenia [1-3]. Although rare, its incidence has increased over the past two decades, with most cases reported from Europe in patients with acute leukaemia undergoing intensive chemotherapy [2,4]. In a multicentre registry study of 90 cases, 74% of affected patients had acute leukaemia, and the overall 30-day mortality rate was 43%, rising to over 60% in the presence of septic shock [4]. Its intrinsic resistance to echinocandins poses a clinical challenge; therefore, empirical echinocandin therapy may be ineffective [1,2]. The decision to use antifungal prophylaxis in patients with haematological malignancies should be individualised according to the underlying disease, treatment regimen, duration and severity of neutropenia, and overall risk of invasive fungal infection [4,5]. Diagnosis of *M. capitatus* infection is further complicated by non-specific clinical and radiological findings. On CT imaging, visceral fungal microabscesses may manifest as multiple small hypoattenuating lesions in the liver, spleen, and kidneys, a pattern that can overlap with thromboembolic or septic infarcts, particularly in early stages [6,7].

This case describes a fatal episode of *M. capitatus* fungaemia in a patient with haematological malignancy, in whom rapidly progressive visceral lesions initially raised suspicion of a vascular aetiology. It highlights the importance of considering disseminated fungal infection in the differential diagnosis of progressive visceral lesions in severely immunocompromised patients.

Case Presentation

A 61-year-old man with angioimmunoblastic T-cell lymphoma was admitted with febrile neutropenia. He was receiving fourth-line therapy with nivolumab and azacitidine as a bridge to allogeneic stem cell transplantation, following failure of three prior lines due to hematologic and infectious complications. His medical history was also notable for dyslipidaemia and osteoarticular disease.

In the months preceding admission, he had required two hospitalisations: the first and more prolonged (65 days) because of a perineal cellulitis that complicated with persistent bacterial contamination of gluteal and perineal wound, requiring a protective colostomy, during which lymphoma treatment was interrupted; the second due to *Escherichia coli* urosepsis treated with ertapenem. His outpatient course had been further complicated by recurrent febrile episodes unresponsive to successive empirical antibacterial regimens, alongside filgrastim-refractory severe leukopenia and persistent pancytopenia requiring frequent

transfusion support. The documented total white blood cell count remained below 500 cells/µL until the day of death. The exact duration of severe leukopenia and the absolute neutrophil count were not available in the medical records.

On admission, blood cultures were drawn and empirical meropenem initiated. Blood cultures were positive for *Klebsiella pneumoniae* producing Klebsiella pneumoniae carbapenemase (KPC), and therapy was sequentially optimised to ceftazidime-avibactam and subsequently meropenem-vaborbactam, given persistent fever and elevated inflammatory markers.

An initial contrast-enhanced abdominal CT performed on day 3 demonstrated bilateral renal cortical hypoenhancing lesions, predominantly involving the upper poles, initially interpreted as possible areas of hypoperfusion or parenchymal infarction. Additional hypoattenuating splenic lesions were identified, the largest measuring 19 mm (Figure *1A*, *1B*). No focal hepatic lesions were described on this initial examination.

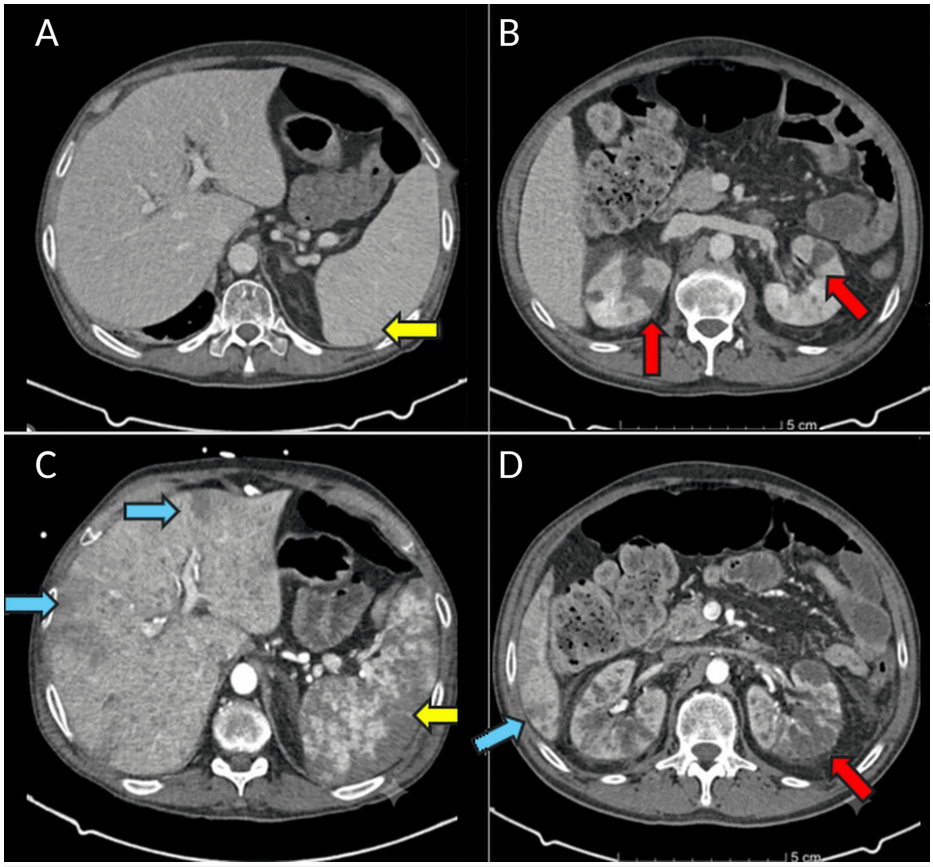


FIGURE 1: Disseminated Magnusiomyces capitatus Infection

Panel A: Initial abdominal CT scan showing a hypoattenuating splenic lesion in the inferior pole (yellow arrow).

Panel B: Initial abdominal CT scan showing bilateral renal cortical hypoenhancing lesions, predominantly involving the upper poles (red arrows).

Panel C: Follow-up CT scan performed 48 hours after the initial examination, demonstrating progression of the hepatic and splenic lesions, including multiple predominantly subcentimetric nodular lesions throughout the hepatic parenchyma (blue arrows) and splenic involvement (yellow arrow).

Panel D: Follow-up CT scan performed 48 hours after the initial examination, demonstrating progression of bilateral renal parenchymal hypoenhancing lesions (red arrows), with additional hepatic involvement (blue arrows).

Despite targeted antibacterial therapy, the patient developed progressive multiorgan dysfunction, including haemodynamic instability requiring vasopressor support, hypoxaemic respiratory failure requiring high-flow nasal oxygen and non-invasive ventilation (FiO_2 >50%, PaO_2/FiO_2 ratio 165), acute kidney injury, hepatic dysfunction with rising direct hyperbilirubinaemia, and encephalopathy.

Given this clinical evolution, persistent fever, severe immunosuppression, and progressive multiorgan

involvement, CT was repeated 48 hours later. The follow-up scan demonstrated marked progression of the renal and splenic lesions, together with new multiple predominantly subcentimetric nodular hypoattenuating lesions involving the hepatic parenchyma (Figure *1C*, *1D*). The radiological interpretation favoured an infectious process and specifically raised the possibility of fungal infection.

The follow-up CT documented patency of the major intra-abdominal arterial and venous structures, including the renal arteries and veins, coeliac trunk, mesenteric vessels, portal vein, and splenic vein.

Additional blood cultures were collected, and empirical intravenous anidulafungin was initiated because of persistent fever and clinical suspicion of invasive candidiasis in the setting of severe immunosuppression and prolonged leukopenia (200 mg loading dose, followed by 100 mg daily).

The patient was transferred to the intensive care unit but died 12 hours after admission. Postmortem blood cultures were positive for *M. capitatus* (formerly *Blastoschizomyces capitatus*). Antifungal susceptibility testing revealed high minimum inhibitory concentrations (MICs) for echinocandins, with values of 4 µg/mL for anidulafungin and >4 µg/mL for both micafungin and caspofungin. The MICs for voriconazole and amphotericin B were 0.125 µg/mL and 1 µg/mL, respectively. These results should be interpreted cautiously, as established species-specific clinical breakpoints are not available for *M. capitatus*.

No histopathological examination or tissue culture of the visceral lesions was performed. Furthermore, serum and bronchoalveolar lavage galactomannan testing were not performed during the hospital stay.

Discussion

This case illustrates two interrelated challenges in invasive fungal infections among patients with haematological malignancy. First, early visceral fungal lesions may be radiologically indistinguishable from thromboembolic or septic infarcts in their early stages. In this patient, multiple hypoattenuating lesions in the liver, spleen, and kidneys evolved over 48 hours into a diffuse micronodular pattern. The speed and distribution of progression, despite targeted antibacterial therapy, were important clues to an additional disseminated infection. Multiphasic contrast-enhanced CT has been shown to improve sensitivity for detecting hepatic fungal microabscesses, with lesions best visualised on the arterial phase; their typical appearance - multiple sub-centimetre hypoattenuating foci without rim enhancement - differs from pyogenic abscesses but may overlap with embolic infarcts in the early phase [6,7]. In the present case, the acquisition phase was not available in the original radiological reports, and therefore no specific phase-related interpretation can be made. Furthermore, the rapid progression observed on serial imaging should be considered a potential diagnostic clue rather than an established or specific radiological pattern of *M. capitatus* infection, particularly given the limited published imaging evidence for this organism.

Second, *M. capitatus* is frequently associated with haematological malignancy, profound neutropenia, fungaemia, and haematogenous dissemination [1-4]. In this patient, the underlying haematological malignancy and prolonged, severe leukopenia represented important established risk factors. In vitro studies consistently report high echinocandin MICs and comparatively lower MICs for voriconazole, although susceptibility testing remains method-dependent and species-specific breakpoints are lacking [1,2]. Contemporary guidance for rare yeast infections supports species-level identification and favours an amphotericin B formulation, with or without flucytosine, or voriconazole for invasive *Magnusiomyces* infection; treatment should be individualised according to infection site, prior prophylaxis, toxicity, and susceptibility results [1]. Consequently, MIC values should not be interpreted as categorical susceptibility results in the absence of validated species-specific clinical breakpoints.

Anidulafungin was initiated empirically in this case because of persistent fever and clinical suspicion of invasive candidiasis in a severely immunocompromised patient. This decision preceded the postmortem identification of *M. capitatus*. The subsequent microbiological findings highlighted an important therapeutic limitation, as this organism is intrinsically resistant to echinocandins. Anidulafungin would therefore not be considered appropriate targeted treatment once *M. capitatus* had been identified.

The immunological context of this case merits attention. The patient was receiving nivolumab, an immune checkpoint inhibitor (ICI), as part of his fourth-line regimen. Although ICIs have been investigated as potential adjuncts in refractory fungal infections by reversing T-cell exhaustion, their net effect on antifungal immune surveillance in the setting of severe treatment-associated leukopenia remains uncertain [8]. In this case, no causal relationship between nivolumab exposure and susceptibility to fungal infection can be established. The underlying haematological malignancy and prolonged severe leukopenia remain the most clearly documented clinical risk factors.

The concomitant KPC-producing *K. pneumoniae* bacteraemia provided a plausible explanation for the initial clinical deterioration and represented an important potential confounding factor in the interpretation of the patient's subsequent course. This bacterial infection may have contributed significantly to septic shock and multiorgan dysfunction, and the relative contribution of bacterial and fungal infections cannot be established with certainty.

Nevertheless, progression during active antibacterial therapy should prompt reassessment for coinfection, including invasive fungal disease. In this case, anidulafungin was started before species identification, but the isolate had a high MIC for anidulafungin. Microbiological confirmation became available only after death, precluding a switch to an antifungal agent with potential activity against this organism. Recent case series continue to show severe disease and high mortality in neutropenic patients, including breakthrough infections during echinocandin exposure [9,10].

The principal limitation is the absence of histopathological confirmation of the visceral lesions. The attribution of the CT findings to disseminated *M. capitatus* infection is supported by fungaemia, compatible host factors, and rapid multiorgan progression, but remains inferential. Positive postmortem blood cultures confirm fungaemia but do not independently establish the aetiology of each visceral lesion. The coexisting Gram-negative bacteraemia is an additional confounding factor.

Conclusions

Disseminated *M. capitatus* infection poses a diagnostic challenge in immunocompromised patients. Progressive visceral hypoattenuating lesions on serial CT imaging, initially interpreted as possible infarcts, may represent a diagnostic clue to an underlying disseminated infectious process, including fungal infection. However, this observation should not be regarded as an established or specific radiological pattern, particularly in the absence of histopathological confirmation.

The intrinsic resistance of *M. capitatus* to echinocandins highlights the importance of early species-level identification and antifungal susceptibility testing. When invasive *Magnusiomyces* infection is suspected, early infectious diseases and clinical mycology consultation is essential to support timely reassessment of antifungal therapy. Treatment decisions should be guided by current rare-yeast recommendations, previous antifungal exposure, organ function, and susceptibility results when available, rather than by Candida-specific assumptions. Histopathological confirmation should be pursued whenever feasible.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Maria João Vilela, Ana Costa, Carolina Nogueira

Acquisition, analysis, or interpretation of data: Maria João Vilela, Ana Costa, Carolina Nogueira

Drafting of the manuscript: Maria João Vilela, Carolina Nogueira

Critical review of the manuscript for important intellectual content: Maria João Vilela, Ana Costa, Carolina Nogueira

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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Maria João Vilela and Carolina Nogueira contributed equally to the work and should be considered co-first authors.

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