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Histopathological diagnosis of invasive pulmonary candidiasis mimicking bronchogenic carcinoma in an immunocompetent adult: a case report

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Case Report

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Abstract

Background.

Invasive pulmonary candidiasis is an uncommon and frequently disputed diagnosis because Candida species commonly colonize the respiratory tract without causing tissue invasion. Definitive diagnosis, therefore, relies on histopathological demonstration of fungal invasion with concurrent inflammation. We report a case of invasive pulmonary candidiasis presenting as an endobronchial mass mimicking bronchogenic carcinoma in an adult male without known immunosuppression.

Case presentation.

A 49-year-old Ugandan male presented with a two-month history of dry cough, chest pain, progressive worsening dyspnea, intermittent fever, and non-bloody diarrhea. The fever was moderate grade and intermittent without rigors or night sweats. He developed septic shock and hypoxemic respiratory failure requiring intensive care unit admission. Radiological evaluation demonstrated left lower lobe consolidation with minimal parapneumonic effusion. Bronchoscopy revealed a narrowed bronchial lumen due to an endobronchial mass, raising suspicion for bronchogenic carcinoma. Histopathological examination of bronchoscopic biopsy specimens demonstrated non-branching pseudohyphae consistent with Candida species, confirming invasive pulmonary candidiasis. Blood, sputum, urine, and pleural fluid cultures were negative. The patient improved clinically following antifungal therapy and was discharged home on oral antifungal therapy.

Conclusion.

This case highlights the diagnostic challenge of invasive pulmonary candidiasis and underscores the indispensable role of histopathology in distinguishing true tissue invasion from colonization. In patients with persistent pneumonia and endobronchial lesions, invasive fungal infection should be considered even in the absence of classical immunosuppression.

Background

Pulmonary involvement by Candida species remains a controversial entity because Candida commonly colonizes the upper and lower respiratory tract, particularly in hospitalized patients(1, 2). Consequently, respiratory cultures alone are insufficient for diagnosis(3). True invasive pulmonary candidiasis is rare and requires histopathological evidence of fungal invasion into lung tissue for confirmation(3). Failure to recognize this entity may lead to delayed or inappropriate management, especially when radiologic or bronchoscopic findings mimic malignancy or bacterial pneumonia(4).

Case presentation

A 49-year-old male farmer from Kikuube district, western Uganda, who had only for four days been a casual labourer working with the China Petroleum Pipeline project in Hoima District, western Uganda,

presented on 22 January 2026 with dry cough, chest pain of two months duration, progressive dyspnea for four days, fever, and diarrhea. The fever was moderate grade and intermittent without chills, rigors, or night sweats. He had been unwell for approximately two months before presentation and was referred from a peripheral health facility.

He had no known history of diabetes mellitus, hypertension, chronic lung disease, malignancy, or immunosuppressive conditions. There was no history of active smoking. There was no significant weight loss. He had recently engaged in manual labour involving digging and filling soil bags during pipeline construction.

Clinical examination.

On admission, he was anxious and acutely ill-appearing. Vital signs revealed hypotension (blood pressure 97/56 mmHg), tachypnea (respiratory rate 29 breaths/min), and severe hypoxemia with oxygen saturation of 70% on room air, improving to 90% on 10 L via non-rebreather mask. Pulse rate was 91 beats/min, temperature 36.1°C, and Glasgow Coma Scale score was 15/15. Use of accessory muscles of respiration was noted. Cardiovascular and abdominal examinations were unremarkable.

A preliminary diagnosis of sepsis with severe pneumonia, acute respiratory distress, metabolic acidosis, and possible pulmonary embolism was made.

Investigations.

Baseline laboratory investigations included complete blood count, liver and renal function tests, venous blood gas analysis, blood glucose, procalcitonin, blood cultures, urine culture, and HIV testing. Blood, urine, and sputum cultures showed no growth. HIV testing was negative.

Electrocardiography and echocardiography were normal. CTPA demonstrated left lower lobe consolidation consistent with pneumonia with minimal parapneumonic effusion, and no evidence of pulmonary embolism.

Due to persistent symptoms and radiologic findings, bronchoscopy was performed. This revealed narrowing of the bronchial lumen by an endobronchial mass, raising suspicion for bronchogenic carcinoma. Bronchoscopic biopsy specimens were obtained for histological analysis.

Histopathology diagnosis.

Microscopic examination demonstrated combined non-branching pseudohyphae and budding yeast infiltrating tissue with marked tissue necrosis and inflammation, consistent with invasive Candida species. These findings established a diagnosis of invasive pulmonary candidiasis, rather than mere colonization.

Differential diagnosis

The main differential diagnoses considered included:

Bronchogenic carcinoma

Bacterial pneumonia with endobronchial obstruction

Invasive pulmonary aspergillosis

Pulmonary mucormycosis

Histopathological morphology excluded filamentous fungi such as Aspergillus (septate, acute-angle branching hyphae) and mucormycosis (broad, aseptate hyphae), supporting Candida infection.

Treatment and outcome

The patient required intensive care management, including high-flow oxygen, intravenous fluids (crystalloids), vasopressor support with noradrenaline, broad-spectrum antibiotics, and electrolyte correction. Following histopathological diagnosis, antifungal therapy was initiated. He was initiated on oral itraconazole 200 mg once daily in addition to antibiotics.

He showed significant clinical improvement, was weaned off oxygen, and was no longer in respiratory distress by day 5 after initiating Itracozole, which was day 10 of admission. He was discharged to continue taking oral Itraconazole at home after 15 days in the hospital.

Discussion

Invasive pulmonary candidiasis (IPC) is rare and often over- or misdiagnosed when based solely on respiratory cultures (4). Although Candida species frequently colonize healthy people's mucosal and dermal surfaces, invasion can happen when immune or mechanical defences are compromised (5). The incidence of invasive pulmonary candidiasis is rising in both immunosuppressed and immunocompetent patients (1, 6). This case illustrates a classic diagnostic pitfall: an endobronchial lesion mimicking malignancy, forming a nodule within the bronchus (7), with negative cultures but definitive histopathological evidence of fungal invasion. IPC may induce sepsis or ultimately septic shock, both indistinguishable from bacterial infection (8). Fungal cultures may be negative even in proven invasive disease; histopathology remains the prerequisite for definitive diagnosis(3). The absence of traditional immunosuppressive risk factors in this patient further underscores the importance of tissue diagnosis (3).

At admission, the laboratory tests indicated severe systemic inflammation and sepsis. The full blood count demonstrated marked leucocytosis with neutrophilia (total white blood cell = 20.02 $\times 10^9$/L; absolute neutrophils = 13.7 $\times 10^9$/L), accompanied by absolute monocytosis and relative eosinopenia. Acute phase reactants were significantly elevated, with procalcitonin peaking at 25.54 ng/mL, and CRP

was markedly raised (> 270 mg/L), consistent with high-risk severe sepsis or septic shock as shown in some studies (9, 10).

Despite this intense inflammatory response, initial microbiological investigations were negative. HIV serology was non-reactive, malaria testing was negative, and both aerobic and anaerobic blood cultures subsequently showed no growth after five days of incubation, arguing against bacteraemia as a cause of sepsis, which is consistent with culture-negative invasive pulmonary candidiasis (2). Renal function was preserved, while electrolyte abnormalities (hypocalcaemia and hypomagnesemia) were noted, consistent with critical illness–related metabolic derangements. Cardiac biomarkers (CK-MB and troponin) were transiently elevated, but without corroborative ECG evidence of acute coronary pathology.

Microbiological evaluation of blood specimens did not yield a pathogen and this was consistent with study in Eastern Uganda that showed culture negative sepsis in up to 47% of patients(11). Respiratory specimen such as sputum microscopy and culture was negative for bacteria, fungi, and acid-fast bacilli, with Bartlett's score indicating an adequate sample but no identifiable infectious agent. Urine analysis and culture were unremarkable. These findings further deepened the diagnostic dilemma of culture-negative sepsis in a patient with progressive pulmonary disease (3, 12)

Histopathology remains the gold standard for diagnosis of IPC, particularly in resource-limited settings where advanced fungal diagnostics may be unavailable(3, 13). This case reinforces that IPC should be considered in patients with persistent pneumonia and atypical bronchoscopic findings, even when cultures are negative.

Conclusion

Histopathological demonstration of tissue-invasive Candida is essential for the diagnosis of invasive pulmonary candidiasis. This case emphasizes that invasive pulmonary candidiasis can masquerade as bronchogenic carcinoma and occur in patients without overt immunosuppression. Early biopsy and pathological evaluation are critical to ensure timely, accurate diagnosis and appropriate antifungal therapy.

Abbreviations

CK-MB
Creatinine kinase-MB
CRP
C-reactive protein
CTPA
Computed tomography pulmonary angiography
ECG
Electrocardiogram
HIV

Human immunodeficiency virus
IPC
Invasive pulmonary candidiasis

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Competing interests

The authors declare no competing interests.

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Availability of data and materials

The data generated or analyzed during this study are included in this published article. Patient identifiers were omitted.

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