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## Case Report

# Rare association of concurrent melioidosis and mucormycosis in a patient with diabetes mellitus: A case report of successful management.

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## Introduction

Melioidosis is a potentially life-threatening infection [1]. It is an emerging infectious disease in Sri Lanka caused by the environmental Gram-negative bacterium *Burkholderia pseudomallei*. Diabetes mellitus represents the principal recognized risk factor and is reported in approximately half of affected patients, predisposing to severe and disseminated infection [2]. The disease exhibits a broad clinical spectrum, ranging from localized infection to fulminant septicaemia and disseminated disease. Pulmonary involvement is the most frequent presentation, while hepatic and splenic abscesses are characteristic manifestations of disseminated infection [3]. Its diverse clinical and radiological manifestations allow melioidosis to mimic several important conditions, including tuberculosis, malignancy, vasculitis, and invasive fungal infections, frequently contributing to delays in diagnosis [4]. Disease incidence may increase following periods of heavy rainfall and monsoonal weather, particularly among individuals with underlying risk factors. Early recognition remains challenging because of its protean presentation, the potential for rapid progression, and the substantial mortality associated with severe disease, particularly in settings where definitive microbiological diagnosis may be limited [5].

In Sri Lanka, melioidosis is likely underdiagnosed because of limited clinical awareness and restricted access to laboratory facilities capable of identifying *B. pseudomallei* [2]. Reported Sri Lankan cases have predominantly involved patients from rural communities, with diabetes being the most frequent comorbidity [5]. The broad clinical spectrum and overlap with other diseases further emphasize the importance of considering melioidosis in patients presenting with compatible clinical features, particularly in regions where tuberculosis and other infections are prevalent [3].

Mucormycosis is a rapidly progressive invasive fungal infection that can cause rapidly angioinvasive infections that lead to tissue infarction, necrosis, and thrombosis and requires early diagnosis and treatment. Infection primarily affects immunocompromised patients, including those with hematologic malignant neoplasms, solid organ or stem cell transplants, and poorly controlled diabetes mellitus [6]. Cutaneous disease may present with erythematous or violaceous lesions that progress to necrosis, eschar, ulcers, or purpuric lesions and may result from direct inoculation or, less commonly, dissemination from another site [7]. The lungs, paranasal sinuses, orbits, and brain are among the commonly involved sites [6]. Mucormycosis may progress rapidly and requires early recognition and treatment. Diagnosis is primarily established by tissue examination, with histopathology or fungal culture being important for confirmation; radiological findings such as pulmonary consolidation, nodules, or cavitary lesions may provide additional diagnostic clues [8]. Management requires correction of the underlying predisposing factor, early antifungal therapy, and surgical debridement when feasible [6].

We report a diagnostically challenging case of concurrent melioidosis and mucormycosis in a woman with poorly controlled type 2 diabetes mellitus who presented with pulmonary mass-like lesions, hepatosplenic abscesses, mediastinal lymphadenopathy, and a necrotizing sino-nasal lesion suggestive of invasive fungal infection and was successfully managed in a resource-limited setting.

## Case Presentation

A 49-year-old woman with a 20-year history of type 2 diabetes mellitus, treated with oral hypoglycemic agents with poor medication adherence and suboptimal glycaemic control, presented with a three-month history of intermittent low-grade fever, anorexia, generalized malaise, easy fatigability, and an unintentional weight loss of approximately 8 kg over two months. Four weeks before admission, she developed a progressively worsening dry cough and exertional dyspnoea (mMRC grade III). She denied haemoptysis, pleuritic chest pain, wheezing, orthopnoea, paroxysmal nocturnal dyspnoea, or lower-limb swelling. During the same period, she developed mild intermittent dull aching pain in the right hypochondrium, without associated gastrointestinal or hepatobiliary symptoms. She also noticed a painful ulcer over the left nasal vestibule that progressively increased in size and was associated with local nasal pain.

There was no history of recurrent epistaxis, purulent or blood-stained nasal discharge, recurrent sinusitis, facial swelling or numbness, dental pain, or visual symptoms, including diplopia, proptosis, ophthalmoplegia, or visual loss. There was no history of

chronic respiratory disease, tuberculosis exposure, significant smoking, or occupational exposure to inhaled toxins. She had no history of prolonged corticosteroid therapy, chemotherapy, malignancy, HIV infection, chronic kidney disease, or other known immunocompromising conditions. Her medications included metformin, gliclazide, amlodipine, atorvastatin, and losartan. She was a lifelong non-smoker and did not consume alcohol. There was no history suggestive of connective tissue disease or other systemic autoimmune disorder. Her past surgical and family histories were unremarkable.

On admission, she appeared ill, febrile, and pale, with a BMI of 19.2 kg/m$^2$. She was tachycardic at 100 beats/min, with a blood pressure of 110/80 mmHg. A necrotic ulcerative lesion involving the left nasal ala with surrounding inflammation was noted (Figure 1 - A), accompanied by ipsilateral cervical lymphadenopathy. There was no icterus, cyanosis, clubbing, peripheral oedema, skin rash, oral ulceration, or synovitis. Respiratory examination revealed mild dyspnoea with a few crackles over the left lung fields. Heart sounds were normal, with no murmurs or pericardial rub. Abdominal examination was unremarkable. Neurological examination showed a Glasgow coma scale score of 15/15, with no focal neurological deficits or meningeal signs. Fundoscopy revealed non-proliferative diabetic retinopathy.



Figure 1A: Necrotic ulcerative lesion of the left nasal ala, suggestive of mucormycosis

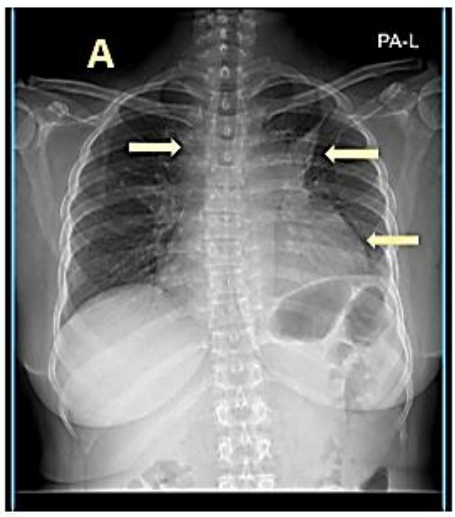


Figure 1B: Marked clinical improvement of the nasal lesion after 6 weeks of treatment.

Laboratory investigations demonstrated marked systemic inflammation, with procalcitonin of 8.81 ng/mL, CRP peaking at 199 mg/L before declining to 14 mg/L, and ESR of 110 mm/h. The initial white cell count was 14.5 × 10$^9$/L with neutrophilia, haemoglobin was 7.4 g/dL with microcytosis (MCV 74.9 fL), and platelet count was 140 × 10$^9$/L. The white cell counts subsequently increased to 18.71 × 10$^9$/L before improving to 9.36 × 10$^9$/L. Renal function remained relatively preserved with mild acute kidney injury, with serum creatinine ranging from 49–120 µmol/L. Mild electrolyte abnormalities included sodium 130–140 mmol/L and potassium 3.3–4.9 mmol/L. Liver enzymes were

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largely preserved, although ALP was elevated (157–249 U/L), with marked hypoalbuminemia ranging from 15–29 g/L. Abdominal ultrasonography demonstrated multiple focal lesions in the liver and spleen, suspicious for abscesses. Chest radiography (Figure 2) showed mild cardiomegaly, elevation of the left hemidiaphragm, right paratracheal opacity, mild left basal atelectatic change, and left upper-lobe consolidation.



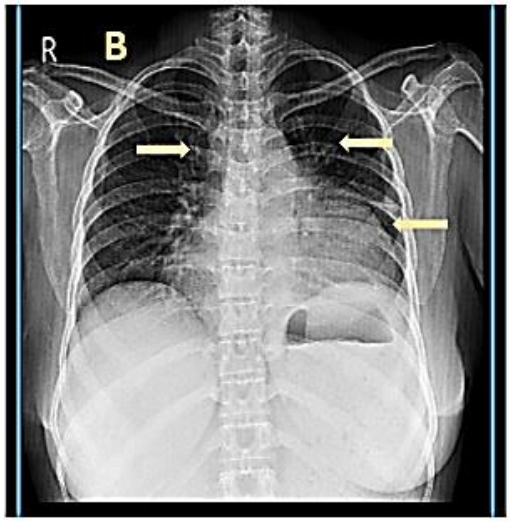




Figure 2A: Initial chest radiograph showing consolidation involving the left upper and lower lung zones, with right paratracheal opacity.
Figure 2B: Follow-up chest radiograph demonstrating radiological improvement following 4 weeks of treatment.

Contrast-enhanced CT of the chest, abdomen, and pelvis (Figure 3) demonstrated multiple focal hepatic and splenic lesions, left lower-lobe consolidation, pleural effusion, intrapulmonary nodules, necrotic paratracheal lymphadenopathy, and pericardial effusion. The radiological differential diagnoses included melioidosis, disseminated mucormycosis, and septic emboli with multifocal abscess formation.

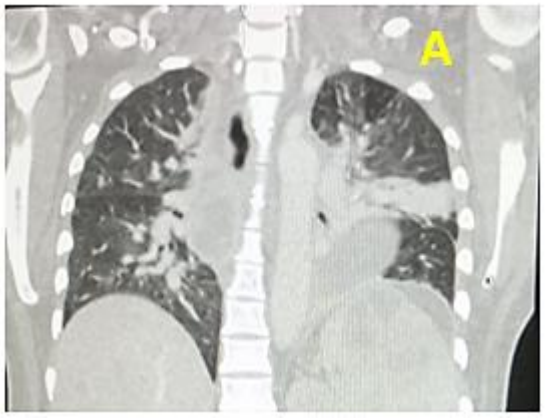






Figure 3. Contrast-enhanced CT of the chest, abdomen, and pelvis.

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(A) Coronal lung-window image showing multifocal pulmonary consolidation, including patchy and lobar areas of consolidation, with multiple small pulmonary nodules and cavitary lesions. (B) Axial abdominal image demonstrating multiple hypodense lesions involving the liver and spleen, consistent

Initial microbiological investigations were negative, However, the melioidosis antibody titre was markedly elevated at >10,240. A subsequent blood culture isolated *Burkholderia pseudomallei* (Figure 4).



Figure 4: Blood culture isolate of *Burkholderia pseudomallei* demonstrating characteristic growth consistent with melioidosis

The painful nasal ulcer raised suspicion of an invasive fungal infection. NCCT of the brain & sinus demonstrated bilateral maxillary, ethmoidal, and sphenoidal sinus disease with hyperdense material within the involved sinuses (Figure 5).

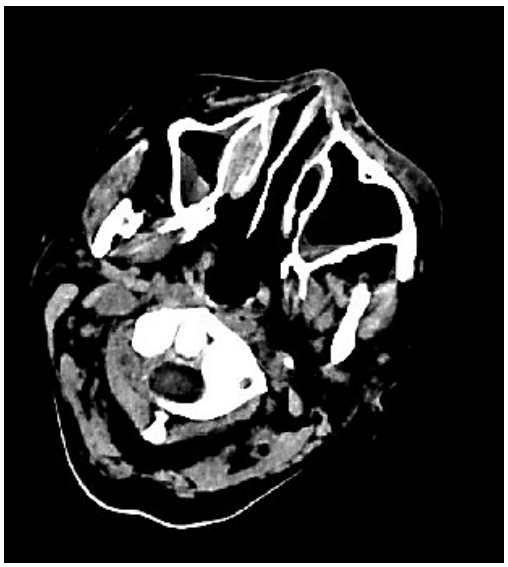


Figure 5. NCCT of the paranasal sinuses showing sinusitis with hyperdense material within the involved sinuses, raising suspicion of fungal sinusitis.

A functional endoscopic sinus surgery (FESS) tissue biopsy from the nasal mucosa was obtained. Direct microscopy demonstrated fungal filaments suggestive of mucormycosis (Figure 6).



Figure 6: Direct KOH microscopy of the nasal lesion showing broad, aseptate fungal hyphae with right-angle branching, consistent with mucormycosis..

However, histopathological examination showed ulcerated nasal mucosa with nonspecific changes and only possible evidence of fungal infection. Bronchoscopy demonstrated narrowing of the left upper-lobe bronchus without a definitive endobronchial mass. Echocardiography showed preserved left ventricular systolic function with an ejection fraction of 60% with no features of infective endocarditis. Tumour markers were within normal limits, including CEA 0.92 ng/mL, CA 19-9 <1.4 U/mL, CA 125 20 U/mL and AFP 0.945 ng/mL. The combination of *B. pseudomallei* bacteraemia, multiple visceral abscesses and fungal filaments on direct microscopy of the nasal lesion confirmed concurrent melioidosis and mucormycosis. The diagnosis required integration of clinical, radiological, microbiological, and pathological findings, while excluding tuberculosis, malignancy, and ANCA-associated vasculitis.

The patient was treated with intravenous meropenem as intensive-phase therapy for melioidosis, followed by prolonged oral cotrimoxazole eradication therapy. Liposomal amphotericin B was initiated for mucormycosis, with close monitoring of renal function and electrolytes. The nasal lesion was assessed by the otorhinolaryngology team, with appropriate local management. Glycaemic control was optimized, and anaemia and electrolyte abnormalities were corrected. Follow-up imaging demonstrated improvement in the disseminated lesions with appropriate management. Abdominal ultrasonography

showed only a tiny residual hepatic abscess, with no splenic lesions visualized. Repeat CT showed reduction in the hepatic abscesses, while the previously identified right lung nodule was no longer seen. Follow up blood culture was negative. HIV and VDRL screening were negative.

She demonstrated progressive clinical and biochemical improvement following 8 weeks of antibiotic therapy, with microbiological clearance and resolution of the hepatic and splenic abscesses and improvement of the nasal lesion (Figure 1 - B). She was discharged on continued eradication therapy with close follow-up. This case highlights that concurrent melioidosis and mucormycosis can be successfully managed in a resource-limited setting through early recognition, prompt initiation of targeted antimicrobial and antifungal therapy, optimization of glycaemic control, and multidisciplinary care. Despite two invasive infections, evaluation for other underlying immunocompromising conditions was unrevealing, with poorly controlled diabetes mellitus identified as the only predisposing factor.

## Discussion

This case highlights the important association between diabetes mellitus and susceptibility to severe opportunistic infections. Diabetes is the strongest risk factor for melioidosis and is associated with impaired neutrophil function, reduced phagocytosis, and altered cellular immunity [1]. The presence of multiple hepatic and splenic abscesses provided an important diagnostic clue in our patient. Similar findings were described in a patient with disseminated *B.* bacteraemia and multiple splenic abscesses, in whom delayed diagnosis contributed to disease progression [9].

However, the diagnosis in our patient was challenging because the combination of pulmonary nodules, mediastinal lymphadenopathy, pleural effusion, weight loss, and hepatosplenic lesions initially raised the possibility of metastatic malignancy [3]. Similar diagnostic difficulties have been reported in Asian cohorts, where pulmonary melioidosis may mimic bronchogenic carcinoma and tuberculosis [10]. The Sri Lankan National Melioidosis Surveillance Programme demonstrated the widespread distribution of melioidosis in Sri Lanka and emphasized the importance of maintaining clinical suspicion in patients with diabetes presenting with prolonged fever and deep-seated abscesses [6]. The present case further illustrates the diagnostic challenges associated with melioidosis, particularly when its clinical manifestations overlap with malignancy and invasive fungal infection.

Mucormycosis is an important opportunistic fungal infection, particularly among patients with haematological disorders, malignancy, diabetes mellitus, and those undergoing transplantation. The increasing incidence in these populations reported [11]. although due to high morbidity and mortality, requiring early diagnosis, prompt surgical intervention, and timely antifungal therapy. Liposomal amphotericin B is the preferred first-line treatment, with isavuconazole and posaconazole as alternatives. However, limited access to contemporary antifungal agents in resource-limited settings can complicate management and adversely affect outcomes [12].

The necrotizing sinonasal lesion added further diagnostic uncertainty. Direct microscopy demonstrated broad aseptate fungal filaments suggestive of mucormycosis. There are cases described in Sri Lankan patient with pulmonary mucormycosis in whom overlapping clinical and radiological manifestations contributed to diagnostic delay [13]. Although fungal invasion demonstrated histologically in our patient, the initial findings raised sufficient concern for mucormycosis to warrant empirical treatment with liposomal amphotericin B in accordance with international recommendations [6]. The overlap between pulmonary melioidosis and invasive fungal disease is further illustrated by the report entitled "Reversed Halo Sign: Don't Forget Melioidosis," which highlighted the potential for pulmonary melioidosis to resemble invasive fungal disease radiologically [8]. This diagnostic overlap is particularly relevant in a patient presenting with pulmonary abnormalities together with other manifestations of disseminated infection [14].

Concomitant infection with *B. pseudomallei* and mucormycosis has been reported, including a case of melioidosis with cutaneous mucormycosis following corticosteroid exposure for COVID 19 infection, although the outcome was unfavourable [8]. The concurrent occurrence of both infections in patients with diabetes is exceptionally rare [7]. and presents substantial diagnostic and therapeutic challenges. Disseminated coinfection is associated with high mortality, emphasizing the need for early recognition, accurate diagnosis, and timely, appropriate antimicrobial and antifungal treatment [15]. In lower-middle-income settings, socioeconomic and healthcare-related factors may further contribute to diagnostic and management challenges, emphasizing the need for practical diagnostic approaches and improved recognition of disseminated disease and coinfections [9].

Overall, this case highlights the diagnostic and therapeutic complexity of concurrent disseminated melioidosis produced a clinical/radiological picture that mimicked malignancy and invasive fungal disease, while a sinonasal lesion simultaneously raised genuine concern for mucormycosis in a patient with poorly controlled diabetes mellitus. The combination of microbiologically confirmed melioidosis, multiple visceral abscesses, and a necrotizing lesion containing fungal filaments, together with overlapping clinical and radiological features, necessitated repeated complementary microbiological and histopathological investigations. Despite the challenges of a resource-limited setting, early recognition, targeted antimicrobial and antifungal therapy, optimization of glycaemic control, and multidisciplinary management resulted in successful clinical and radiological improvement.

## Conclusion

Concurrent melioidosis and mucormycosis is a rare and diagnostically challenging presentation in a patient with poorly controlled diabetes mellitus. The overlapping clinical and radiological features may mimic disseminated malignancy and invasive fungal disease, potentially delaying diagnosis. In endemic settings, melioidosis should be considered in patients with diabetes who present with prolonged fever, pulmonary lesions, and hepatosplenic abscesses. Early microbiological and pathological evaluation,

prompt initiation of targeted antimicrobial and antifungal therapy, optimization of glycaemic control, and multidisciplinary management can facilitate successful outcomes even in resource-limited settings.

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