

Reaching a Diagnosis of Pulmonary Melioidosis in a Resource Limited Setting

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Abstract

We report chronic pulmonary melioidosis in a 69-year-old diabetic farmer in Suriname, initially misidentified as *Burkholderia cepacia* complex infection by MALDI-TOF mass spectrometry. Standard commercial databases lack *Burkholderia pseudomallei* spectra due to biosafety restrictions and limited endemicity in database development regions. The patient presented with hemoptysis and a cavitating upper-lobe consolidation mimicking tuberculosis. Clinical suspicion based on occupation, radiological findings, and known presence of *Burkholderia thailandensis* (a less virulent species within the *B. pseudomallei* complex) in Surinamese soil led to empiric melioidosis treatment, with dramatic response. This case highlights critical gaps in diagnostic capabilities for melioidosis in emerging endemic regions and underscores the need for increased clinical awareness as climate change potentially expands the geographic distribution of *B. pseudomallei*.

Keywords

Burkholderia cepacia, *Burkholderia pseudomallei*, *Burkholderia thailandensis*, Tuberculosis

1. Introduction

With a population of less than 1 million (<https://population.un.org/wpp/>), Suriname reported a tuberculosis incidence of 29 cases per 100.000 population in 2023 (<https://data.who.int/countries/740>). We describe a case that initially presented as straightforward pulmonary tuberculosis (PTB) but ultimately yielded a microbiological diagnosis of *Burkholderia cepacia* complex (Bcc) infection, a condition not previously reported in Suriname. Bcc is typically encountered in patients with cystic fibrosis (CF) [1] [2], but our patient has neither CF nor does he have any

bronchiectasis [3] [4] on CT imaging. The clinical and radiological features, however, were highly suggestive of chronic pulmonary melioidosis caused by *Burkholderia pseudomallei*, which is notoriously misidentified as Bcc by standard microbiological systems. These findings prompted the preparation of this case report after written consent was obtained from the patient.

2. Case Presentation

A 69-year-old male watermelon farmer presented to the emergency department of our hospital in June 2025 with his first episode of hemoptysis, following three weeks of malaise, anorexia, and weight loss. He denied fever, shortness of breath, chest pain or risk factors for pulmonary embolism. His medical history included hypertension, type 2 diabetes mellitus, cardiac stents, and gout. He is being treated with metformin, glibenclamide, enalapril, amlodipine, aspirin and allopurinol. Physical examination was unremarkable and the body temperature was 36.3 degrees Celsius. Laboratory tests showed normal blood clotting, normal serum values of urea, creatinine, sodium, potassium, ASAT and ALAT. Hemoglobin was 7.3 mmol/L; white blood cell count $12 \times 10^9/L$; platelet count $213 \times 10^9/L$; CRP 0.6 mg/dL. Non-fasting blood glucose was elevated at 16.3 mmol/L and a HIV test was negative. A chest X-ray showed a consolidation in the left upper lobe (**Figure 1**, Panel A).



Figure 1. Panel A: consolidation in left upper lobe before treatment with amoxicillin/clavulanic acid. Panel B: involution of the pulmonary consolidation after 5 weeks trimethoprim/sulfamethoxazole.

Suspicion of a Community Acquired Pneumonia led to prescription of amoxicillin/clavulanic acid and the patient was next day referred to the pulmonologist for further evaluation. Supplementary history revealed no known exposure to tuberculosis and minimal lifetime smoking (1 cigarette monthly for 30 years). Sputum collected at the emergency department showed no acid fast bacilli (AFB) on auramine stain, the GeneXpert ultra test was negative for *Mycobacterium tuberculosis* (MTB) and culture showed growth of mixed flora. A follow up chest x ray after completion of the weeklong amoxicillin/clavulanic acid treatment showed no improvement [5]. A subsequent CT scan of the chest revealed a consolidation of $4.7 \times 4.2 \times 3.4$ cm with central hypodensity representing an abscess, posteriorly in the left upper lobe (**Figure 2**). The other parts of the lungs were totally normal

and no upper abdominal abnormalities were detected on the CT scan (**Figure 3**).

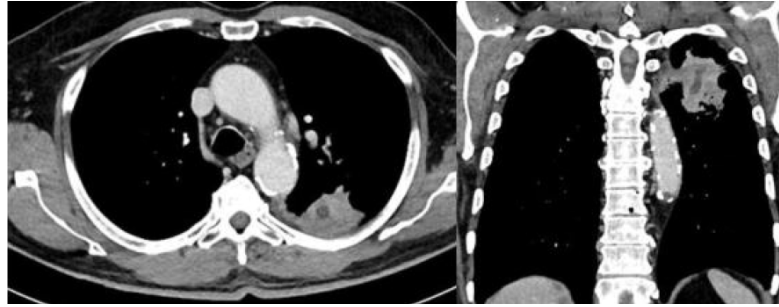


Figure 2. Pulmonary consolidation in the left upper lobe with abscess formation.

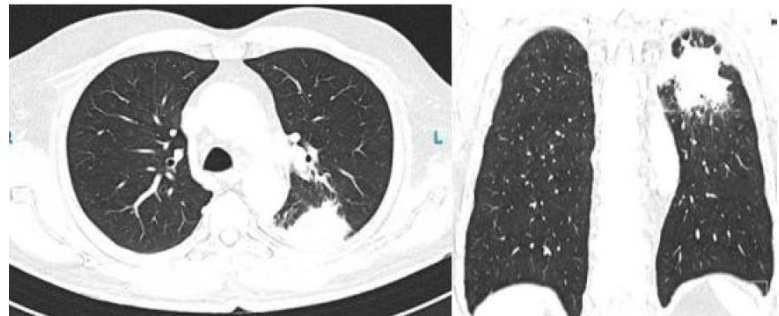


Figure 3. Consolidation in the left upper lobe while other parts of the lungs are totally normal.

Malignancy was considered unlikely as no enlarged lymph nodes in the thoracic cavity were seen, no signs of distant metastases were detected and the other parts of the lungs were normal. Given the absence of major symptoms, a working diagnosis of slow-resolving pneumonia was made, and a wait-and-see approach was adopted. One month later at follow up visit the patient had experienced another episode of hemoptysis, and a repeat chest X-ray showed an increase in the size of the pulmonary consolidation. Subsequently fiberoptic bronchoscopy was performed, which revealed no blood or lesions in the tracheobronchial tree. Lack of X-ray fluorescence and EBUS equipment made endobronchial biopsy of the consolidation impossible. The bronchial aspirate obtained during bronchoscopy showed no AFB, but bacterial culture grew colonies identified as *Burkholderia cepacia* and *Achromobacter xylosoxidans* (using a *Bruker MALDI Biotyper), both susceptible to trimethoprim/ sulfamethoxazole and meropenem. Upon reviewing the culture results, the pulmonologist and the Medical Microbiology Department concluded that the patient's occupation, clinical presentation, imaging findings, and the isolation of *B. cepacia* complex were highly suggestive of melioidosis [6]-[9], a conclusion drawn 2 months after first presentation with hemoptysis. As such the patient was presumed to have chronic pulmonary melioidosis and because he was in a very good clinical condition, not warranting hospitalization, the initial intravenous intensive phase of treatment was omitted. Outpatient treatment for chronic pulmonary melioidosis was initiated with trimethoprim/sulfamethoxa-

zole, starting in August 2025 for a planned 20-week course [7], with monthly radiologic and laboratory monitoring. By September 2025, chest radiography showed clear reduction of the pulmonary consolidation (Figure 1, Panel B). Currently the patient is asymptomatic and gaining weight.

Commercial systems like the Bruker MALDI Biotyper and the bioMérieux VITEK MS historically lack *Burkholderia pseudomallei* spectra in their standard databases, primarily because melioidosis is uncommon in the regions (North America and Europe) where these systems are developed. Furthermore, *B. pseudomallei* is classified as a Risk Group 3 (RG3) or Security Sensitive Biological Agent (SSBA). Handling this organism in a standard clinical laboratory requires enhanced biosafety measures (Biosafety Level 3, or BSL-3), which is a significant barrier to including it in routine clinical workflows and standard IVD databases. This absence often results in its misidentification (frequently as *Burkholderia thailandensis* or *Burkholderia cepacia* complex species) or no identification at all when using the standard IVD libraries in routine clinical laboratories [10]-[12].

3. Discussion

This case describes a 69-year-old diabetic male farmer presenting with malaise, hemoptysis, a left upper-lobe pulmonary consolidation with normal blood clotting parameters. The differential diagnosis was pulmonary infection or pulmonary malignancy. Cultures identified *B. cepacia*, a complex of gram negative bacteria with a ubiquitous soil and water distribution [1]. However, several factors argue against true Bcc infection. First, Bcc is an opportunistic pathogen predominantly affecting patients with CF or the immune compromised [1] [2]. Our patient is HIV negative and being from East Asian descent has no signs of CF [13] and has no bronchiectasis [3] [4]. Although he has type 2 diabetes mellitus which can impair the immune system [14], we have no clinical signs to consider him significantly immune compromised [15]. Second, the radiological finding of a nodular infiltrate with abscess formation in an upper lobe of a diabetic patient with hemoptysis is a classic presentation of chronic pulmonary melioidosis [6]-[9]. Third, a major limitation of our MALDI-TOF system is the inability to reliably identify *B. pseudomallei* (the causative micro-organism of melioidosis), increasing the likelihood of misidentification as Bcc or related species [10]-[12]. Another factor is the presence of *B. thailandensis* in Surinamese soil, a gram-negative bacteria which is a less virulent species within the *B. pseudomallei* complex [16]. Given the patient's occupation, environmental exposure is plausible [17]. Finally, the patient's dramatic clinical and radiological response to targeted melioidosis antibiotic therapy with trimethoprim/sulfamethoxazole strongly supports the diagnosis. Treatment will be given for at least 20 weeks [7]. *B. pseudomallei* is an intracellular gram-negative saprophyte transmitted by percutaneous route via contaminated environmental water and soil [7]. The main presentation is pulmonary melioidosis, while skin infection, genito-urinary infection, bacteremia and sepsis are less common [6]. *Achromobacter xylosoxidans* was co-isolated in the

bronchial aspirate of our patient. This is a soil- and water-associated aerobic gram negative rod, increasingly recognized as a pathogen in CF patients [18]. As our patient has no CF, we consider the presence of *Achromobacter xylosoxidans* in his bronchial aspirate a matter of contamination or an innocent bystander. While we cannot entirely rule out malignancy, we consider it unlikely because treatment with trimethoprim/sulfamethoxazole antibiotics resulted in mass involution of the pulmonary consolidation, and as such to be considered evidence against malignancy. Also weight gain and improvement of his wellbeing and minimal tabaco exposure argue against the presence of malignancy. Although we see progressive improvement in our patient, our case report has limitations. These limitations include the absence of prior imaging, the lack of a pulmonary biopsy to histologically rule out malignancy, and the inability to confirm *B. pseudomallei* with a reference laboratory as the bronchial aspirate was not stored. Still, we are convinced our patient has pulmonary melioidosis.

4. Conclusion

Cavitating pulmonary melioidosis can be misdiagnosed for pulmonary tuberculosis [19], especially in TB endemic regions. In areas where melioidosis is non endemic, the lack of adequate diagnostic tools [20] is also a factor which could delay correct diagnosis and treatment. Physicians must be aware of the limitations of standard microbiological identification systems and consider the evolving epidemiology of diseases like melioidosis, potentially influenced by factors such as climate change [21]. As Suriname is vulnerable to climate change with rising water levels, and the presence of *B. thailandensis* is confirmed, (<https://www.undp.org/suriname/projects/suriname-global-climate-change-alliance-gcca#>), we would like to advocate the inclusion of *B. pseudomallei* spectra in regional diagnostic laboratory databases and set up targeted environmental surveillance to better map endemic areas.

Credit Author Contributions

F. M.: Data Curation, Resources, Writing-reviewing. A. A.: Data Curation, Reviewing. E. IJ.: Data Curation, Resources, Writing-reviewing. I. T.: Data Curation, Resources, Writing-reviewing. F. G.: Conceptualization, Data Curation, Original Draft Preparation, Supervision, Resources.

Conflicts of Interest

We have no conflict of interest to report, nor have we received any funding for the preparation of this case report.

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