

FATAL MYCOBACTERIUM ABSCESSUS PNEUMONIA IN A PATIENT WITH BRONCHIECTASIS: A CASE REPORT

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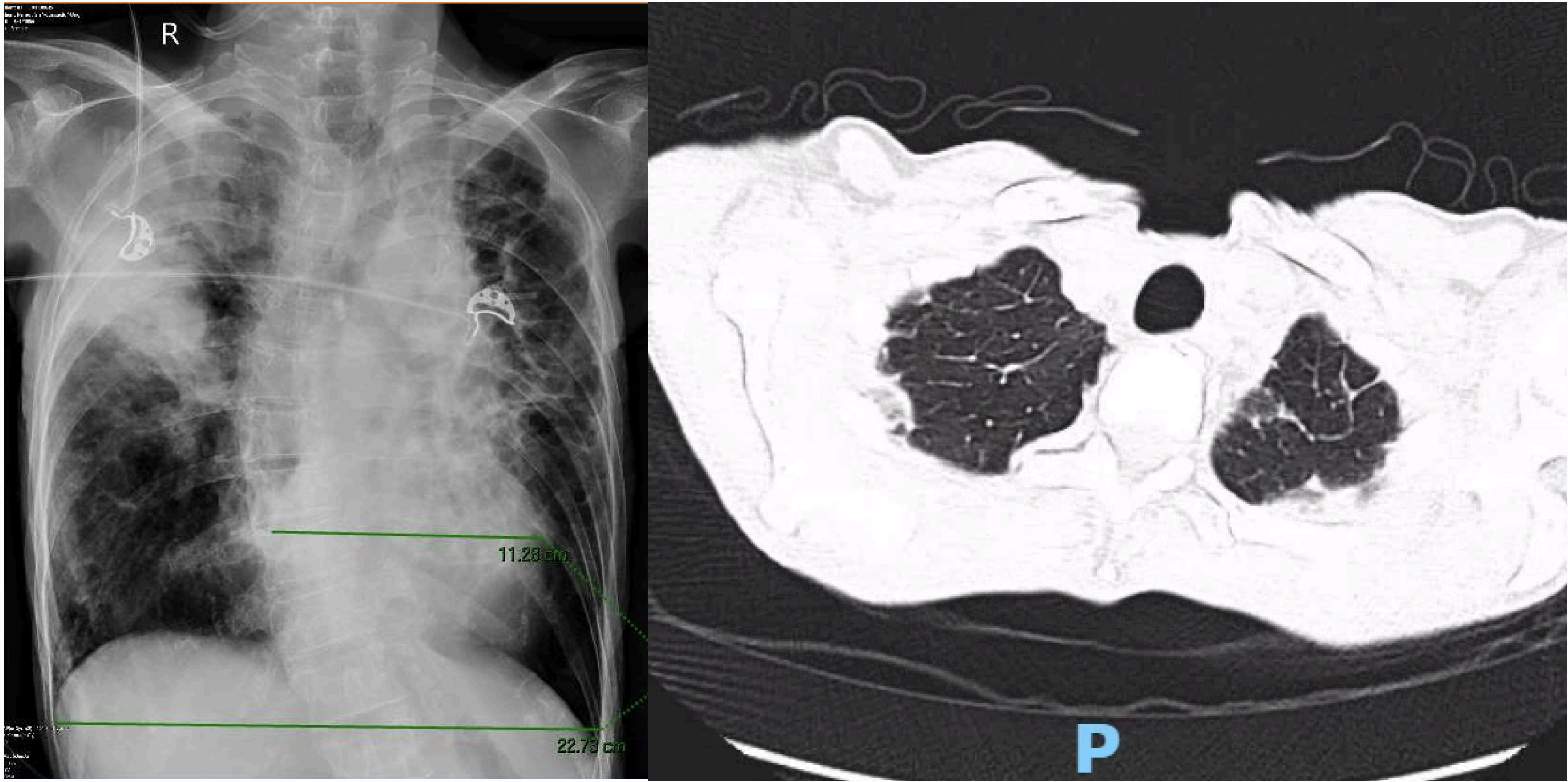
BACKGROUND

Mycobacterium abscessus is an emerging non-tuberculous mycobacterium (NTM) causing pulmonary infections, particularly in patients with underlying structural lung disease and immunosuppression. Management is difficult due to its intrinsic resistance and the toxicity of prolonged multidrug regimens.

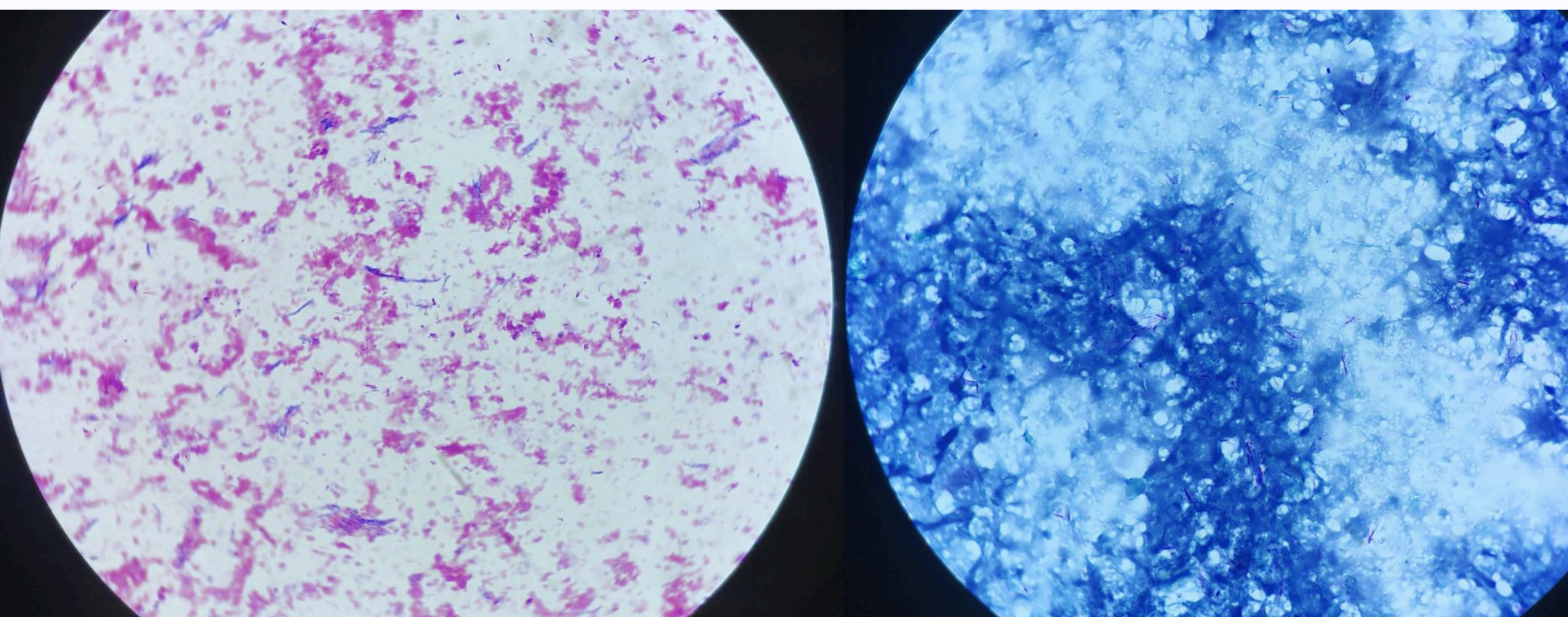
CLINICAL PRESENTATION

We present the case of a 71-year-old Filipino male with a history of chronic bronchiectasis and autoimmune necrotizing myopathy who was admitted for worsening cough, dyspnea, and intermittent low-grade fever.

He was initially treated empirically for nosocomial pneumonia with multiple antimicrobial regimens, but his condition failed to improve. Subsequent chest imaging revealed multifocal infiltrates with cavitary changes, consistent with non-resolving pneumonia.



Figures 1 and 2. Chest x-ray and CT scan findings of the patient demonstrating multifocal infiltrates and bronchiectatic changes.



Figures 3 and 4. Chest x-ray and CT scan findings of the patient demonstrating multifocal infiltrates and bronchiectatic changes.

Despite broad-spectrum antimicrobial therapy, the patient remained ventilator-dependent. Bronchoalveolar lavage cultures later yielded *Mycobacterium abscessus subsp. abscessus*. Targeted therapy was initiated with intravenous amikacin, oral clarithromycin, and linezolid based on in vitro susceptibility testing.

While his condition initially stabilized, he subsequently developed progressive metabolic acidosis with rising lactate by the fourth week of treatment. He ultimately succumbed to refractory lactic acidosis and respiratory failure.

DISCUSSION AND CONCLUSION

This case illustrates the diagnostic and therapeutic challenges of NTM infections in chronic lung disease, where symptoms often mimic unresolving pneumonia. *M. abscessus* is especially difficult to treat due to multidrug resistance and the need for prolonged, toxic regimens. Linezolid can cause hematologic and metabolic complications, including mitochondrial toxicity, while amikacin adds nephrotoxic risk. In this patient, death was likely due to linezolid-induced mitochondrial toxicity with refractory lactic acidosis.

These challenges underscore the importance of early recognition of drug toxicity and judicious therapy, particularly in the Philippines, where chronic lung disease is common and safer alternatives are limited.