Case Report

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Internal Medicine Section

Rhodococcus Hoagii Masquerading as Tuberculosis: A Case Report

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ABSTRACT

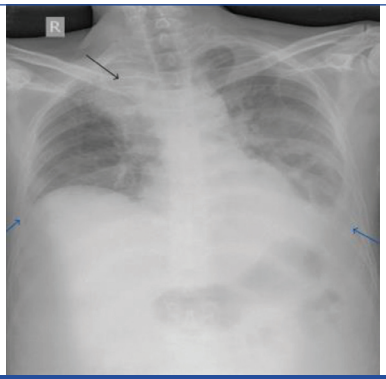
A young 35-year-old male presented with a cough, chest pain, breathlessness and bilateral lower limb swelling. He had been an alcoholic for over 21 years and had a history of Antitubercular Therapy (ATT) for six months due to tuberculous pleural effusion, which occurred a year ago. Bronchoscopy and Endobronchial Ultrasound-Transbronchial Needle Aspiration (EBUS-TBNA) revealed granulomatous lesions and acid-fast bacilli, although they were not morphologically similar to mycobacteria or Nocardia. GeneXpert-Mycobacterium Tuberculosis (MTB) and bacterial cultures were inconclusive. He was subsequently started on modified ATT. However, due to clinical worsening, a Computed Tomography (CT)-guided Fine Needle Aspiration (FNA) was performed, which revealed pus; cultures detected *Rhodococcus hoagii*. The patient was then commenced on rifampicin and ciprofloxacin. Unfortunately, his compliance with the treatment was poor and he ultimately succumbed to the infection. The present case demonstrates that *Rhodococcus hoagii* infection, a rare cause of fatal pneumonia, can mislead physicians into misdiagnosing tuberculosis.

Keywords: Acid fast bacilli, Consolidation, Endobronchial ultrasound, Fine needle aspiration, Granulomatous lesion, Tree-in bud nodules

CASE REPORT

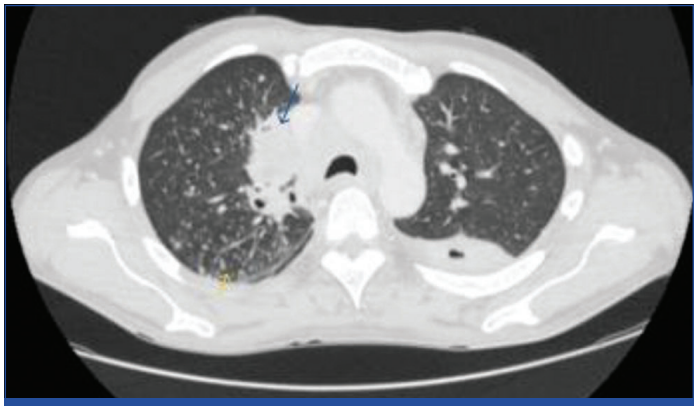
A 35-year-old male presented to the Pulmonary Medicine Outpatient Department with a 20-day history of cough, brownish sputum, right-sided chest pain and bilateral lower limb swelling. A review of symptoms did not reveal haemoptysis, fever, or weight loss. He had been consuming alcohol for over 21 years and had a left-sided pleural effusion eight months prior, which was treated with tube thoracostomy, fibrinolysis and six months of ATT. On physical examination, he had a pulse rate of 94 bpm, a blood pressure of 122/84 mmHg, oxygen saturation of 98% on room air, a respiratory rate of 18 cycles/min and bilateral pitting pedal oedema. Chest examination revealed dullness to percussion and reduced breath sounds in the right infraclavicular and bilateral infrascapular areas, with bronchial breath sounds and crepitations heard in the right infraclavicular lung fields.

A chest X-ray showed a circumscribed homogeneous opacity in the right upper zone without mediastinal shift and obliterated bilateral costophrenic angles [Table/Fig-1].

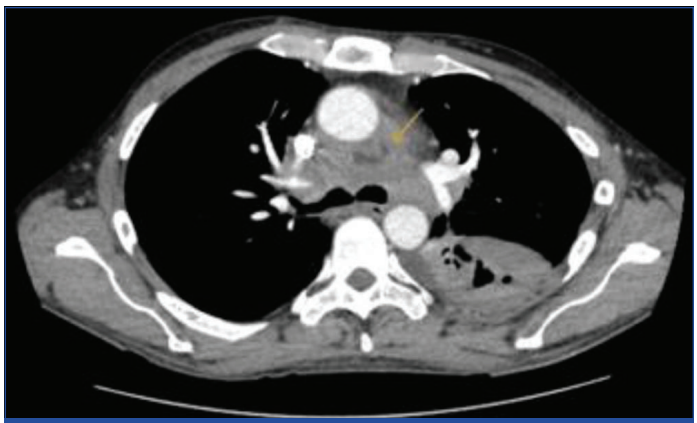


[Table/Fig-1]: Chest X-ray showing right upper zone homogenous opacity (black arrow) and bilateral obliteration of costophrenic angle (blue arrows).

A Contrast-Enhanced Computed Tomography (CECT) scan of the thorax revealed multiple areas of consolidation with tree-in-bud nodules in the right upper [Table/Fig-2] and left lower lobes, bilateral pleural effusion, homogeneous enhancement of the mediastinal lesion [Table/Fig-3] and liver nodularity with ascites, suggesting parenchymal liver disease.



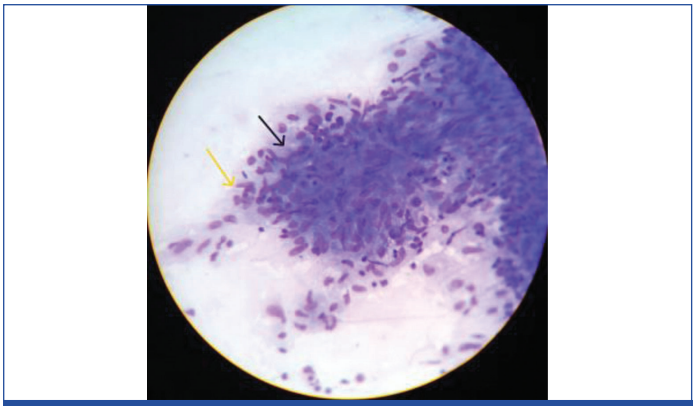
[Table/Fig-2]: CECT Thorax lung window showing right upper lobe homogeneous opacity (blue arrow) and tree-in bud nodules (yellow arrow).



[Table/Fig-3]: CECT Thorax mediastinal window showing homogeneously enhancing soft-tissue density in the visceral compartment of mediastinum (yellow arrow).

On day 2, flexible bronchoscopy with Bronchoalveolar Lavage (BAL) was performed, followed by endobronchial ultrasound with Transbronchial Needle Aspiration (TBNA) from the mediastinal lesions.

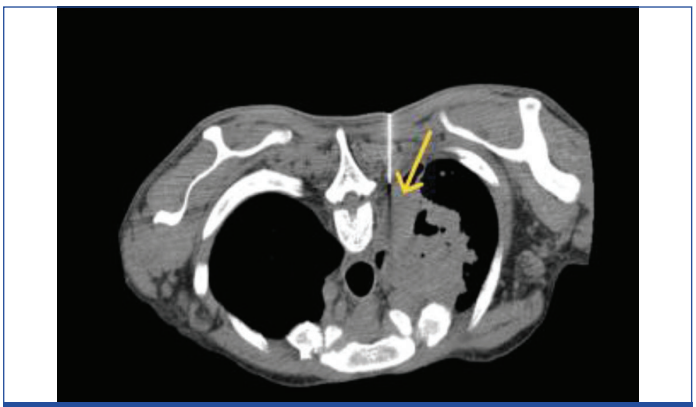
The TBNA cytology revealed a granulomatous lesion [Table/Fig-4]. Ziehl-Neelsen staining with 20% sulphuric acid did not show acid-fast bacilli; however, acid-fast bacilli resembling coccobacilli were observed using Kinyoun's modification of acid-fast staining with 1%



[Table/Fig-4]: TBNA cytology stained with Methylene blue (40x) showing granulomatous inflammation yellow arrow pointing to epithelioid cells against a necrotic background (black arrow).

sulphuric acid [1]. These acid-fast bacilli did not morphologically resemble *Nocardia spp*. The Cartridge-Based Nucleic Acid Amplification Test (CBNAAT) and bacterial culture on the BAL sample did not detect *Mycobacterium tuberculosis* or yield any respiratory pathogen.

On day 4, based on clinical suspicion and histopathological evidence, he was started on modified ATT (Streptomycin 15 mg/kg, Levofloxacin 15 mg/kg and Ethambutol 15 mg/kg) once daily due to his deranged liver function, with a plan to initiate ATT Fixed-dose Combination (FDC) as per the National Tuberculosis Elimination Programme (NTEP) once liver functions improved [2]. On day 10, the patient's condition further deteriorated, requiring oxygen supplementation. Pulmonary embolism was suspected and a CT Pulmonary Angiogram (CTPA) was performed, which showed no evidence of embolism but revealed further radiological worsening of the lung lesions. A CT-guided transthoracic FNA was then performed [Table/Fig-5].



[Table/Fig-5]: CT-guided FNA from right upper lobe lesion (yellow arrow indicating the needle track).

The FNA revealed pus, which was sent for bacterial culture and sensitivity testing. The culture showed large, irregular, mucoid, non haemolytic colonies on blood agar [Table/Fig-6], identified as *Rhodococcus hoagii* by Matrix-Assisted Laser Desorption/Ionisation Time-of-Flight Mass Spectrometry (MALDI-TOF MS).

On day 14, ATT was stopped and the patient was started on rifampicin 600 mg and ciprofloxacin 500 mg once daily for two months. His condition clinically improved after seven days of treatment. On day 21, oxygen supplementation was tapered off and he was discharged on room air. The patient was on regular follow-up; however, after 15 days of discharge, he became non compliant with the treatment and discontinued his medications. Subsequently, his condition worsened and he succumbed to the infection.

DISCUSSION

The genus *Rhodococcus* belongs to the family *Nocardiaceae* and the order *Actinomycetales*, which also includes the genera

[Table/Fig-6]: Large, irregular, mucoid, non haemolytic colonies of *R.hoagii* on blood agar (yellow arrow).

Mycobacterium and *Nocardia*. *Rhodococcus hoagii* was formerly known as *Rhodococcus equi*, *Corynebacterium equii* and *Prescottella equii*. It is a Gram positive, non motile, non spore forming, encapsulated coccobacillus [3]. It can exhibit a weak acid-fast reaction, which may lead to a misdiagnosis as *Mycobacterium*.

R. hoagii is a zoonotic pathogen primarily known to cause pneumonia in foals and it rarely infects humans. Infection can be acquired through inhalation, ingestion, or inoculation into a wound [4]. The isolation of *R. equi* increases on dry, warm and windy days, suggesting that aerosolised soil particles contaminated with *R. equi* may be a mode of transmission [1]. The first recorded human case of *R. hoagii* (previously *R. equi*) occurred in 1967 in a 29-year-old male with autoimmune hepatitis who presented with fever and cavitary pneumonia [5].

The pathogenesis of *R. hoagii* relies on its ability to survive within and destroy macrophages. The bacterium interferes with phagosome-lysosome fusion, allowing it to evade destruction by macrophages. It also induces non specific lysosomal degranulation, leading to the destruction of both macrophages and surrounding tissues [6]. The virulence of *R. hoagii* is attributed to a surface antigen, VapA, associated with an 85-90 kbp plasmid [7]. The specific histological appearance of this infection is a necrotising granulomatous reaction dominated by macrophages with granular cytoplasm, containing numerous coccobacillary forms [8].

R. hoagii can present with both pulmonary and extrapulmonary manifestations. The clinical presentation usually develops insidiously over days to weeks. Common pulmonary symptoms include a non productive cough, dyspnoea and pleuritic chest pain. Radiographically, consolidation with cavitation is the most common feature, with cavitation occurring in nearly 75% of cases and a predilection for the upper lobes in 55% [9]. It can also present as a lung abscess, empyema, pneumothorax, or mediastinitis [10,11].

Matrix-Assisted Laser Desorption/Ionisation-Time of Flight Mass Spectrometry (MALDI-TOF MS) is a valuable diagnostic tool in clinical laboratories, offering rapid and accurate identification of bacterial, mycobacterial and fungal pathogens. Currently, there are no established Clinical Laboratory Standards Institute (CLSI) guidelines for antibiotic testing and treatment of *R. hoagii*. Managing *R. hoagii* infections is often challenging due to frequent relapses and treatment failures. It is recommended to initiate antimicrobial therapy with a combination of atleast two agents to reduce the risk of developing resistance. Preferred agents include macrolides (azithromycin, clarithromycin, or erythromycin), fluoroquinolones (levofloxacin, moxifloxacin, or ciprofloxacin) and rifampicin. Vancomycin, carbapenems, linezolid, or aminoglycosides are also options. Some studies have shown that imipenem, vancomycin and rifampicin are the most effective [12,13]. Treatment duration should be extended (atleast 2 months) due to the high incidence of relapses and mortality associated with shorter regimens [14]. Surgical resection of the infected tissue may be considered as an

adjunctive treatment in cases where an isolated lung abscess is present and medical therapy alone is unlikely to be effective [15].

R. hoagii represents a rare but significant human infection, primarily affecting immunocompromised individuals. Its clinical presentation can closely mimic that of *Mycobacterium tuberculosis*, emphasising the need for a high index of suspicion in its diagnosis. Prompt identification, aided by advanced techniques like MALDI-TOF MS, is crucial for distinguishing this pathogen from other infectious agents. Effective management entails an extended course of treatment with a combination of atleast two antibiotics. Recognising the clinical and diagnostic challenges posed by this organism is essential for improving outcomes and preventing potentially fatal complications.

CONCLUSION(S)

In tuberculosis-endemic countries, when a previously treated TB patient presents with chest symptoms and necrotising granulomatous lesions, *Rhodococcus hoagii* should be considered a potential cause if Ziehl-Neelsen staining and GeneXpert results are inconclusive. Kinyoun's modified acid-fast staining and MALDI-TOF MS should be employed to achieve an accurate diagnosis.

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