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CASE REPORT

Veillonella as a cause of chronic anaerobic pneumonitis

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Summary Anaerobes are not well recognized as a cause of chronic respiratory infections. A 44-year-old man was referred for evaluation of a progressive pulmonary disease of 7-month duration characterized by hemoptysis and fever. For these complaints, based on the radiological picture, he had already received antituberculous therapy without any relief. He was also subjected to bronchial artery embolization prior to referral. Evaluation of the patient led to a diagnosis of chronic anaerobic pneumonitis. Anaerobic culture of the computed tomography-guided trans-thoracic aspirate grew *Fusobacterium* and *Veillonella* species. Within 2 weeks of therapy with oral clindamycin, there was a dramatic relief in hemoptysis. This was accompanied by remarkable radiological clearance. This report underscores the importance of *Veillonella* species as a potential respiratory pathogen. A high index of suspicion is required to diagnose chronic anaerobic pneumonitis, which can mimic pulmonary tuberculosis, especially in tuberculosis endemic regions.

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Introduction

The anaerobes are the most overlooked bacterial pathogens of the lower respiratory tract.¹ The spectrum of anaerobic pulmonary infections ranges from an acute clinical presentation resembling pneumococcal pneumonia to a chronic indolent disease, which resembles pulmonary tuberculosis.² The four well-recognized clinical categories

are pneumonitis, lung abscess, necrotizing pneumonia, and empyema.¹ Although pneumonitis due to anaerobic bacteria is generally characterized by an acute course, a chronic presentation mimicking that of pulmonary tuberculosis has been reported by us in adults³ as well as in the pediatric age group.⁴ The occurrence of this chronic form of anaerobic pneumonitis is yet to receive the recognition that it deserves.

We report herein a patient with chronic anaerobic pneumonitis due to *Fusobacterium* and *Veillonella* species, who presented with multiple episodes of hemoptysis. The rarity of such a chronic clinical presentation prompted this report.

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Case report

A 44-year-old, non-diabetic, HIV-negative male was referred to our institute for evaluation of a progressive pulmonary disease of 7-month duration. The patient complained of increasing severity of hemoptysis associated with intermittent low-grade fever. He coughed up 40–80 ml blood every 10–15 days. Hemoptysis was preceded by minimal cough, which was not associated with expectoration. He admitted to nasal symptoms in the form of intermittent sneezing and episodic rhinorrhea. There was no obvious weight loss. He worked as a taxi driver and had smoked 5 bidis per day over the past two decades. He also had a significant history of alcohol intake.

One month after the onset of symptoms and 6 months prior to referral, based on his clinical and radiological profile, he was initiated on antituberculous therapy in the form of oral rifampin 600 mg, isoniazid 300 mg, ethambutol 800 mg, and pyrazinamide 1500 mg once daily, without relief. This was in spite of several direct sputum smears and culture negative results for *Mycobacterium tuberculosis*. He was also subjected to bronchial artery embolization for his continued hemoptysis but with no relief.

General physical examination revealed a middle-aged man in no acute distress. Diaphragmatic excursions were within normal limits. Percussion note was impaired in the right infraclavicular area, and on auscultation bronchial breathing with crepitations were audible. Oro-dental hygiene was poor with multiple dental caries, a missing right upper second molar, and erosion of the enamel of the left upper premolars. However, there was no history of dental surgery, epileptic seizures, or loss of consciousness, which would have been suggestive of aspiration.

A review of three serial chest radiograms done over a period of 7 months prior to the referral revealed a non-homogeneous lesion in the right upper and mid zones with multiple areas of breakdown. A gradual increase in the size of the lesion was observed. A contrast-enhanced computed tomography (CT) of the thorax done on presentation confirmed the presence of consolidation in the right upper lobe with a central area of necrosis (Figure 1A).

The hemoglobin level was 10.8 g/dl with a total leukocyte count of $10.96 \times 10^9/l$ and a differential count of polymorphs 77%, lymphocytes 11%, and eosinophils 5%. A tuberculin test with 5 TU was negative. Sputum samples obtained on multiple occasions were negative for acid-fast bacilli (AFB) by direct smear microscopic examination. Sputum cultures did not show the presence of any aerobic pathogen or *M. tuberculosis*. Spirometry was within normal limits. All three segmental openings of the right upper lobe as visualized on fiberoptic bronchoscopy, contained mucopurulent secretions along with boggy edematous mucosa that bled on touching. Bronchial aspirate was negative for AFB and the culture did not yield any aerobic pathogen or pathogenic fungus. A CT-guided transthoracic fine needle aspiration was done. Microscopic examination of the Gram-stained smear revealed the presence of Gram-negative bacilli, morphologically resembling *Fusobacterium*. The CT-guided transthoracic aspirate was subjected to aerobic and anaerobic cultures. Aerobic pathogens were not isolated. Anaerobic culture grew *Fusobacterium* and *Veillonella* species, which were sensitive to chloramphenicol, clindamycin, coamoxylav, imipenem, and metronidazole.

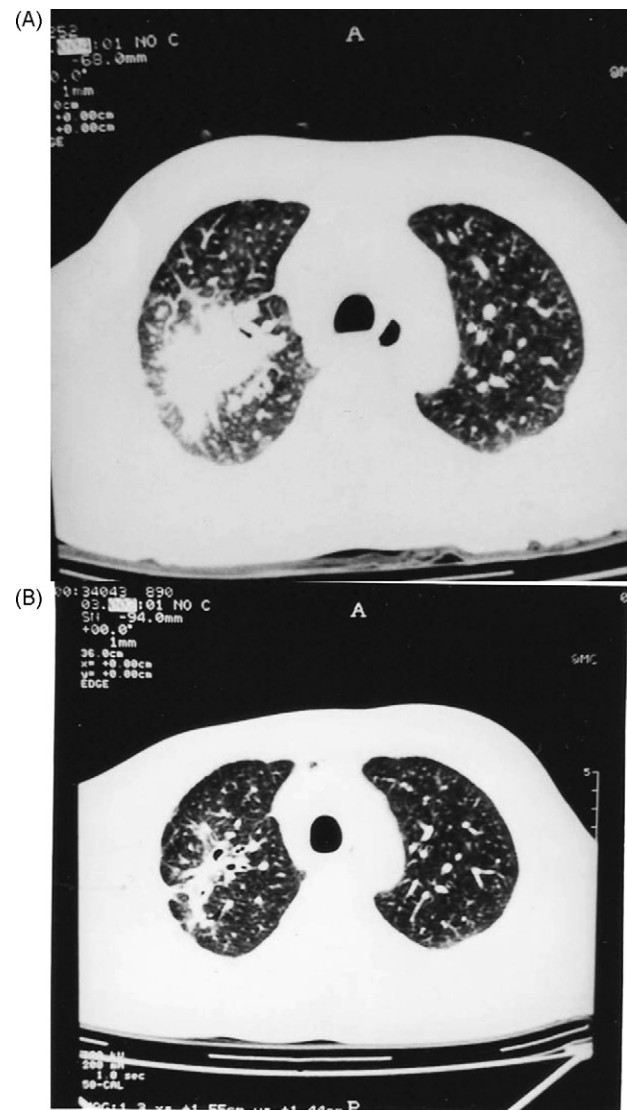


Figure 1 (A) Computed tomogram of the thorax showing an irregular area of consolidation in the right upper lobe with central area of necrosis. (B) Computed tomogram of the thorax, taken after 8 weeks of therapy, showing resolution of the consolidation to a large extent, with residual lesions.

On the basis of a positive anaerobic culture of the transthoracic aspirate, a diagnosis of chronic anaerobic pneumonitis was made. The patient was initiated on oral clindamycin 300 mg 6-hourly. Within two weeks, symptomatic improvement was dramatic, with cessation of hemoptysis. The patient became afebrile. Therapy was continued for 8 weeks. Although there was a remarkable radiological clearance, residual lesions remained. This was also confirmed on CT (Figure 1B).

Discussion

Pneumonitis due to anaerobic bacteria is not commonly suspected, and the chronic form is considered even less.^{3,4} Anaerobic infections are difficult to diagnose due to the invasive procedures required. This is further compounded by the tedious nature of isolation techniques. More often

than not, treatment is commenced without a prior confirmed laboratory diagnosis. This could be due to the ubiquitous nature of anaerobes, raising doubts about their pathogenicity.⁵

Anaerobic pneumonitis is usually characterized by an acute course. The median duration of symptoms prior to presentation in two of the three large series of patients with anaerobic pneumonitis was 3 days,^{1,2} while in the third it was 4.5 ± 0.7 days.⁵ In contrast, our patient presented with a prolonged and persistent illness that was strikingly similar to that of chronic pulmonary tuberculosis. The inordinate delay in the diagnosis was attributed to this remarkable clinico-radiological similarity for which he received antituberculous therapy. Our earlier patients with chronic anaerobic pneumonitis were also prescribed antituberculous therapy prior to referral.^{4,6}

An anaerobic culture of the transthoracic aspirate in our patient yielded *Fusobacterium* and *Veillonella* species. In an earlier series of 44 patients¹ with anaerobic pneumonitis, *Peptostreptococcus* was isolated in 22 patients, *Bacteroides melaninogenicus* (now known as *Prevotella melaninogenicus*) was isolated in 19 patients, and *Fusobacterium nucleatum* was isolated in 17 patients. These three organisms are the most common organisms isolated and are known as the 'big three'.²

In humans, *Veillonella* species are a part of the normal anaerobic flora of the oral cavity, upper respiratory tract, small intestines, and vagina, and are usually considered to be of low virulence.⁷ Bartlett and Finegold¹ also isolated *Veillonella* species in six patients with anaerobic pneumonitis; three isolates were grown in pure culture. They also cultured this organism in four patients each with lung abscess and empyema. Brook⁷ reviewed the 83 isolates of *Veillonella* species that were recovered in 83 children over a period of 20 years. Of the 16 pulmonary isolates, 12 were from aspiration pneumonia, two from children with cystic fibrosis, and one each from empyema and ventilator-associated pneumonia. *Veillonella parvula* was also cultured after percutaneous aspiration from a lung abscess in a 7-year-old girl.⁸

Ever since the classical study of Altemeier⁹ in 1938, putrid odor of sputum is considered to be the hallmark of an anaerobic infection. However, in anaerobic pneumonitis, this may not be a consistent feature as it has only been reported in 9%,¹ 18%,⁵ and 5%² of patients in the three earlier series. None of our reported patients with chronic anaerobic pneumonitis had foul smelling sputum.^{3,4,6}

Chronic anaerobic pneumonitis is an indolent disease with a protracted course. In our earlier patients who also had

chronic anaerobic pneumonitis, we were unable to culture any aerobic organism. In all these patients, there was a small number of anaerobic pathogens in the culture focus. It is possible that the chronicity is due to the selective but paucibacillary presence of anaerobes, in contrast to other forms of anaerobic pleuropulmonary infections where there is associated polymicrobial aerobic growth. Although our patient responded favorably to anti-anaerobic therapy, we have previously reported isolates from a patient with anaerobic lung abscess that were resistant to penicillin and metronidazole.¹⁰

In high tuberculous prevalent areas, the chronicity of the presentation without fetid sputum may result in diagnostic confusion with pulmonary tuberculosis, as occurred in our patient. In addition, our case also highlights the fact that *Veillonella* species are potential respiratory pathogens in humans. A high index of suspicion would not only help the physician to make an early diagnosis but also help to initiate appropriate therapy, thereby reducing morbidity.

Conflict of interest: No conflict of interest to declare.

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