

Necrotising pneumonia and empyema caused by *Clostridium bifermentans*

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The *Clostridia* are unusual causes of primary pleuropulmonary infection in the absence of penetrating chest injury or surgery.¹ We describe here the first reported case of primary pleuropulmonary infection caused by *Clostridium bifermentans*, in this case associated with pulmonary thromboembolism.

Case report

A 41-year-old woman was admitted to the hospital with a one-week history of increasing fatigue, weakness, and breathlessness. She had a persistent cough, haemoptysis, and a sharp pleuritic chest pain on the right the evening before admission.

Her temperature was 36.3°C (oral), the respiratory rate was 30/min, the pulse was 130/min with atrial fibrillation, and the blood pressure was 110/66 mmHg. Examination did not reveal conjunctival petechiae, mucous membrane lesions, ulcerations, splinter haemorrhages, or Janeway lesions. Murmurs consistent with mitral stenosis, mitral regurgitation, aortic stenosis, and aortic regurgitation were present. There was no calf tenderness, Homans' sign was absent, and neurological examination was within normal limits.

The total leucocyte count was 15 800/mm³ (neutrophils 70% and the SGOT and LDH were both raised. Chest radiograph demonstrated generalised cardiomegaly, prominent vasculature to the upper

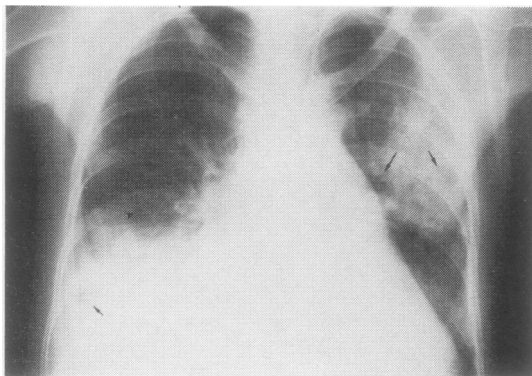


Fig 1 Chest radiograph on the ninth hospital day showing discrete lucent areas within the right lower lobe and left upper lobe infiltrates.

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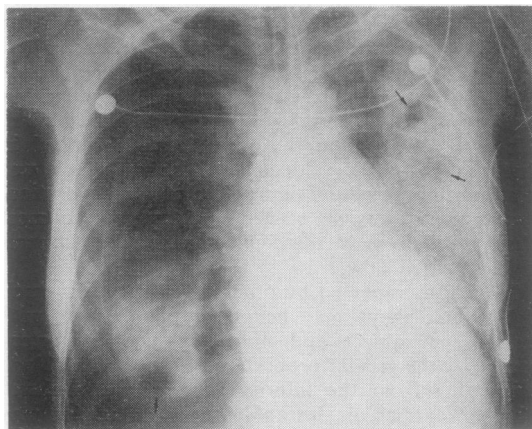


Fig 2 Chest radiograph on the tenth day demonstrating the cavitory lesions in the right lower and left upper lobes. The cavity in the right lower lobe appears contiguous with the pleura and a large pneumothorax is present on the right side.

lobes, a right lower lobe infiltrate, and an elevated right hemidiaphragm. Lung scan was compatible with emboli to the left upper and lower lobes. The patient was started on treatment with oxygen, diuretics, digitalis, and heparin.

The third day the patient continued to be dyspnoeic and the chest radiograph demonstrated a new infiltrate in the upper left lung. The patient remained in congestive heart failure and on the sixth day had an increase in temperature to 38°C orally. There was no change in sputum colour or quantity. All investigations to prove a coexisting infection were negative. By the ninth hospital day the patient was noted to be confused and mildly disoriented and the chest film showed a decrease in the right lower lobe basilar infiltrate but a persistent right pleural effusion. In addition, an area of lucency in the left lung infiltrate as well as in the right lower lung zone was noted (fig 1). The total leucocyte count was 52 000/mm³ (neutrophils 87%).

The next day the patient suffered a cardiopulmonary arrest, and was placed on a mechanical ventilator. Chest radiography demonstrated two radiolucent areas in each of the lung fields within both left upper and right lower lung infiltrates. One of the radiolucent cavities on the right appeared to com-

municate with a large pneumothorax (fig 2). She became febrile, 38.2°C, hypotensive, and succumbed to multiple arrhythmias. Two separate specimens of blood as well as the pleural fluid grew *Cl bifermentans*.

Postmortem examination showed several segmental pulmonary arteries occluded by recent thromboemboli and both lungs showed multiple areas of infarction. The left pleural cavity contained 300 ml of bloody fluid. The heart contained mural thrombi in both atria but there were no vegetations on the cardiac valves. An area of acute necrotising pneumonia was observed in the left lower and left upper lobes. Gram stain and histological examination of these areas revealed gram positive bacilli while post-mortem culture from this region and from heart blood grew *Cl bifermentans*.

Discussion

The *Clostridia* are spore-forming anaerobes normally found in the gastrointestinal tract, the female genital tract and, rarely, on the surface of the skin and in the mouth. *Cl bifermentans* is a rare pathogenic strain and has been isolated from open wounds with Clostridial myonecrosis, and a single outbreak of food poisoning caused by the organism has been reported.² Bartlett and Finegold,³ in their review of 143 cases of anaerobic pleuropulmonary infections, cited *Clostridia* as the organism bacteriologically recovered in two out of 45 cases of lung abscess, in seven out of 45 cases of pneumonitis, in two out of 47 cases of empyema, and in one of the eight cases of necrotising pneumonitis.

In the current case the initial presentation was consistent with pulmonary thromboembolism. The gradual increase of leucocyte count from 15 800/mm³ to 52 000/mm³ with significant numbers of immature forms and the radiological picture of left upper lobe

and lower lobe infiltrate with an bronchogram suggested a coexisting bacterial pulmonary infection. The patient did not have fever during the hospital course except on days six and ten. This lack of physical signs is quite consistent with the "silent" state of clostridial bacteraemia previously described by Gorbach and Thadepalli.⁴ Two blood cultures and one pleural field culture subsequently grew *Cl bifermentans* in pure culture. In retrospect, the radiolucent areas were indicative of underlying necrotising pneumonitis and probably led to one of the preterminal events, the pneumothorax.

The genesis of the superimposed clostridial infection in this case was unclear. It is difficult to assume that the patient had haematogenous spread of the organism as the infection was confined to the lungs. No primary site of septic thromboemboli was located at necropsy. There was no history of aspiration to account for the infection, although one could postulate a subclinical aspiration episode. This case thus represents the first reported occurrence of pleuropulmonary infection from this unusual but potentially pathogenic organism.

References

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