

Enterobacter agglomerans: a new cause of primary pneumonia

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Enterobacter agglomerans (formerly known as *Erwinia herbicola lathyri*) is a Gram-negative bacillus belonging to the tribe Klebsiellae of the family Enterobacteriaceae. Members of the genus *Enterobacter* are distinguished from those of *Klebsiella* by their motility and inability to split urea. The organism is a plant pathogen¹ and was considered unimportant clinically until the mid-1960s, when it was identified in hospital-acquired infections in debilitated patients (especially those receiving broad-spectrum antibiotics) as causing postoperative wound infections and urinary tract infections after instrumentation.² Attention was focused on the organism in the early 1970s in the United States, when it was implicated in an epidemic of septicaemia caused by contamination of bottle caps of intravenous fluid containers.³ Infections acquired outside hospital have usually been associated with contaminated agricultural wounds.⁴ The organism is generally regarded as opportunistic, of low virulence, and with little intrinsic invasiveness. We describe a patient with a fulminating primary pneumonia acquired outside hospital that was caused by *Enterobacter agglomerans*.

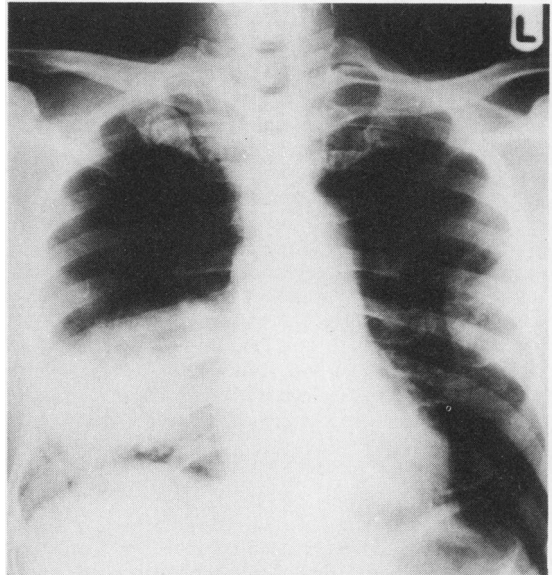
Case report

The patient was a 58-year-old man who smoked 20 cigarettes a day and had a 10-year history of chronic bronchitis (daily cough and sputum production with winter exacerbations). His symptoms, however, were mild in that he had required only one hospital admission with an acute exacerbation of chronic bronchitis 10 years previously and had worked full-time as a carpenter, with full exercise tolerance and no dyspnoea. His alcohol intake was heavy and he had had diabetes for 10 years but this was well controlled by diet alone. He presented to hospital with a three-day history of cough, haemoptysis, right pleuritic chest pain, and dyspnoea. Members of his family confirmed that there was no history of drinking bouts, unconsciousness or contact with ill people.

On examination he was very ill, dyspnoeic, and hypotensive, but was afebrile. He was jaundiced with slight hepatomegaly, but there were no physical signs of chronic liver disease. Venous pressure was not raised and there was no oedema. Heart sounds were normal. He had the physical signs of consolidation in the right lung and a chest radiograph confirmed consolidation in the right middle and lower lobes (fig).

Arterial blood gas sampling showed him to be hypoxic with a good ventilatory drive (P_{O_2} 6.5 kPa (49 mm Hg), P_{CO_2} 2.53 kPa (19 mm Hg), pH 7.43, total bicarbonate 12 mmol(mEq)/l, and base excess -8 mmol(mEq)/l); so he was treated with 40%

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Chest radiograph showing consolidation in the right middle and lower lobes.

oxygen by face mask. The right atrial pressure was 9 cm saline. A diagnosis of septicaemia due to lobar pneumonia was made and he was given ampicillin and gentamicin intravenously.

Shortly after admission the patient had a respiratory arrest. He was intubated immediately and ventilated mechanically but he remained hypoxaemic despite high inhaled oxygen concentrations, and hypotension persisted despite maintenance of an adequate venous pressure with colloid and administration of inotropic drugs. Ten hours after admission he developed cardiac asystole and died.

Investigations On admission the haemoglobin concentration was 16.1 g/dl, the WBC was $1.4 \times 10^9/l$ (60% lymphocytes, 13% monocytes, 27% neutrophils) and the platelet count $35\,000 \times 10^9/l$. The prothrombin time was 25 seconds (control 12 s) and the kaolin partial thromboplastin time was 116 s (control 47 s). The blood urea concentration was 12.7 mmol/l (76.5 mg/100 ml); the electrolytes were normal; and the blood glucose concentration was 6.6 mmol/l (119 mg/100 ml).

Bacteriology Three consecutive blood cultures, a sputum specimen, and a postmortem lung specimen yielded pure cultures of a motile Gram-negative bacillus, which gave the following reactions in the API 20E series of tests: ONPG +;

arginine dehydrolase -; lysine decarboxylase -; ornithine decarboxylase -; citrate +; H₂S -; urea -; tryptophane desaminase -; indole -; VP +; gelatine -; glucose +; mannitol +; inositol -; sorbitol +; rhamnose +; sucrose -; melibiose +; amygdaline +; arabinose +; oxidase -. These reactions identified the organism as *Enterobacter agglomerans*. The organism was sensitive to gentamicin, trimethoprim, colistin, and cephaloridine and resistant to ampicillin, ticarcillin, sulphafurazole, and tetracycline.

Necropsy There was bilateral fibrinous pleurisy and the trachea and main bronchi contained brown, purulent material. The right middle and lower lobes were extensively consolidated, there were areas of haemorrhage, and the texture of the lung substance resembled necrotic liver. There were early similar changes in the left lower lobe. Histological examination showed an acute necrotising bronchopneumonia with haemorrhagic areas and many Gram-negative organisms were visible. The pericardium showed mild inflammatory changes and the pericardial sac contained a little cloudy fluid, but the cardiovascular system was otherwise normal. The liver (weight 2030 g) had a normal external appearance but histological examination showed an excess of fibrous tissue in the portal tracts and the hepatocytes showed moderate large-vacuole fat with no Mallory's hyaline. There was some acute centrilobular necrosis and cholestasis and the overall appearances were thought to be consistent with septicaemia. The bone marrow appeared normal on histological examination, and the naked-eye and microscopic appearances of the other organs were also normal.

Discussion

Enterobacter agglomerans has only rarely been implicated with confidence as a human pathogen; when found in sputum it is usually in combination with known pathogens and is presumed to be a commensal or a contaminant.^{4,5} There is no doubt of the pathogenicity of the organism in this case as it was isolated in pure growth from blood, sputum, and postmortem lung specimens. *Enterobacter agglomerans* has been implicated in indolent lung⁶ and brain⁷ abscesses but there are apparently no reports of a primary pneumonia caused by this organism. The

incidence of bacteraemia due to *Enterobacter agglomerans* is low, a total of 38 cases having been reported to the Communicable Disease Surveillance Centre in the five years 1975-9 (unpublished report 30 May 1980). This represented 7% of all bacteraemias due to members of the genus *Enterobacter* reported in this period.

We can only speculate on the factors that predisposed the patient to this illness as we were unable in the time available to investigate his immune function. His normal bone marrow indicates that the neutropenia and thrombocytopenia were due to increased peripheral consumption secondary to septicaemia. The history of chronic bronchitis, alcoholism, and diabetes may be relevant, as may variations in the virulence of the organism.

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