

Fulminating chromobacterial septicaemia presenting as respiratory distress syndrome

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Respiratory distress syndrome occurs in a variety of clinical situations of which sepsis is one important precipitating factor. We have observed and successfully treated with high PEEP (positive end-expiratory pressure) a rare case of chromobacterial septicaemia complicated by an acute respiratory distress syndrome. We describe here the first reported case of a patient in this country surviving this illness with pulmonary involvement.

Case report

A 3-year-old white male had been healthy until six days before admission when he developed lethargy, high fever, and bug-bite-like nodular skin lesions on his left hip. This occurred while he and his family were on holiday in Florida. He was admitted to a local hospital and found to have leucocytosis ($24.8 \times 10^9/l$) with a left shift. The discharge from the skin eruptions was cultured and revealed gram-negative rods which were identified as chromobacteria. He was given gentamicin (7 mg/kg/day) and ampicillin (100 mg/kg/day), which was soon replaced by carbenicillin (600 mg/kg/day) when the chromobacteria proved to be ampicillin-resistant. The patient remained febrile, pustules and ulcerative lesions appeared on his extremities, his right index finger became swollen and gangrenous, and he developed grey, watery diarrhoea.

The patient was then transferred to the University of Kentucky Medical Center at his parent's request. On arrival, his body temperature was $41^\circ C$, pulse 140/minute, respiration 25/minute, and blood pressure 120/70 mm Hg. His chest was clear and chest radiographs were normal. A grade II/VI systolic ejection murmur was audible at the apex and left sternal border. White blood count was $22.1 \times 10^9/l$ and haematocrit 40%. Prothrombin time and partial thromboplastin time were within normal limits. Liver function tests showed an increased serum glutamic oxaloacetic transaminase. Tenderness and pain in the knee joints was noted.

On the second day after transfer, gentamicin was increased to 13 mg/kg/day and Bactrim (cotrimoxazole) 100 mg/kg/day was added. When the patient developed rapidly-worsening tachypnoea, chest radiographs showed the sudden appearance of a bilateral diffuse nodular-patchy infiltration that suggested a possible septic embolisation to the lungs (fig 1). While breathing room air, the patient had an arterial oxygen tension of 22 mm

Hg (2.93 kPa), an arterial carbon dioxide tension of 42 mmHg (5.60 kPa), and an arterial pH of 7.3. Chest radiographs taken shortly thereafter had changed to near total bilateral homogeneous infiltration (fig 2). The patient quickly deteriorated and suffered cardiopulmonary arrest. He was resuscitated three times and placed on mechanical ventilation.

At this time, with F_{iO_2} at 1.0, tidal volume at 250 ml, PEEP as high as 15 cm H_2O , and respiratory rate of 26/minute, arterial blood gases remained constant at pH 7.2-7.3, P_{CO_2} 54-60 mmHg (7.20-8.0 kPa), and P_{O_2} 25-35 mmHg (3.33-4.67 kPa). Calculated static lung compliance was below 10 ml/cm H_2O . A Swan-Ganz catheter was inserted. The pulmonary artery pressure was 40/25 mmHg with a mean of 30 mmHg, and the pulmonary capillary wedge pressure was 13 mmHg. PEEP was then increased to 20 cm H_2O ; arterial oxygenation improved to 76 mmHg (10.1 kPa), and static compliance increased to 21 ml/cm H_2O . The patient's pupils began to react. Despite barotraumatic pneumomediastinum, pulmonary interstitial emphysema, and subcutaneous emphysema, the patient's respiratory function was satisfactory with an arterial oxygen saturation of 87-98%, a P_{aCO_2} of 38-46 mmHg (5.07-6.13 kPa), and a stable pH. F_{iO_2} and PEEP were gradually decreased, and the patient was weaned with intermittent mandatory ventilation (IMV) on the eleventh day of artificial ventilation (fig 3). During this period, he had three positive blood cultures for chromobacterium violaceum

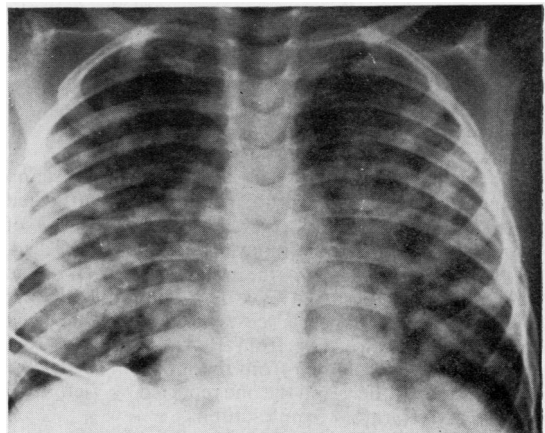


Fig 1 Bilateral diffuse nodular-patchy infiltration suggesting a possible septic embolisation to the lungs.

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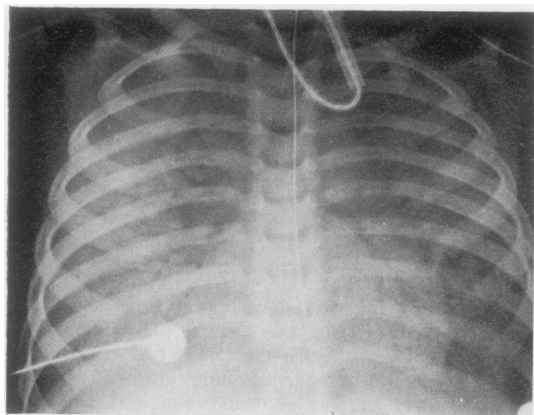


Fig 2 Near total homogeneous infiltration with air bronchogram.

and developed positive chromobacterial osteomyelitis of the left knee (fig 4), which was drained surgically. The child also suffered right ankle cellulitis and spontaneous amputation of the right index finger. The skin lesions which had begun as pustules progressed to dry gangrene.

Susceptibility tests on the chromobacterium proved it to be resistant to ampicillin, carbenicillin and cephalothin and sensitive to chloramphenicol, polymixin B, gentamicin, kanamycin, tobramycin, tetracycline, and Bactrim. The patient continued to receive gentamicin and Bactrim, but carbenicillin was discontinued when the organism was found to be resistant to it. His general condition improved remarkably, and he became afebrile after drainage of the left knee lesion. Gentamicin was stopped after 26 days of therapy because of increasing evidence of toxicity, namely hearing impairment and decreased creatinine clearance. At that time, serial cultures of blood and wounds had been negative for three weeks and one week, respectively. Bactrim remained the only antibiotic prescribed until the patient's discharge from the hospital. The chest radiograph was clear on discharge with the exception of a small cystic lesion in the left lower lobe.

Discussion

It was not until Black first reported in 1938 its infection of a human being that chromobacterium was considered a pathogenic organism.¹ At present, only 19 cases of human infection have been reported,¹⁻⁸ of which 14 died, three survived, and in two the outcome is not known. Almost all cases occurred in tropical areas. Including the present case, only six cases have been reported in the United States, five from Florida, and one from Louisiana.^{1-3 6 8} All died from the infection except the patient presented here and the one reported by Victorica,⁸ who had no respiratory manifestations.

Skin lesions, liver abscesses, pulmonary involvement, and sepsis appear to be characteristic of most patients with chromobacterial infections.¹⁻⁸ Interestingly, even

though respiratory failure has not been claimed as the primary cause of fatality, a varying degree of pulmonary involvement has been mentioned in several cases.³⁻⁷ Nunnally⁵ and Ognibene⁷ described acute respiratory crises of dyspnoea and cyanosis associated with extensive infiltrative processes. The necropsy report of another patient showed an excessively heavy lung weight suggesting the possibility of a "wet lung" syndrome.³ The exact mechanism of respiratory involvement accompanying sepsis is still uncertain. Gross disturbance of clotting systems with intravascular coagulation and direct

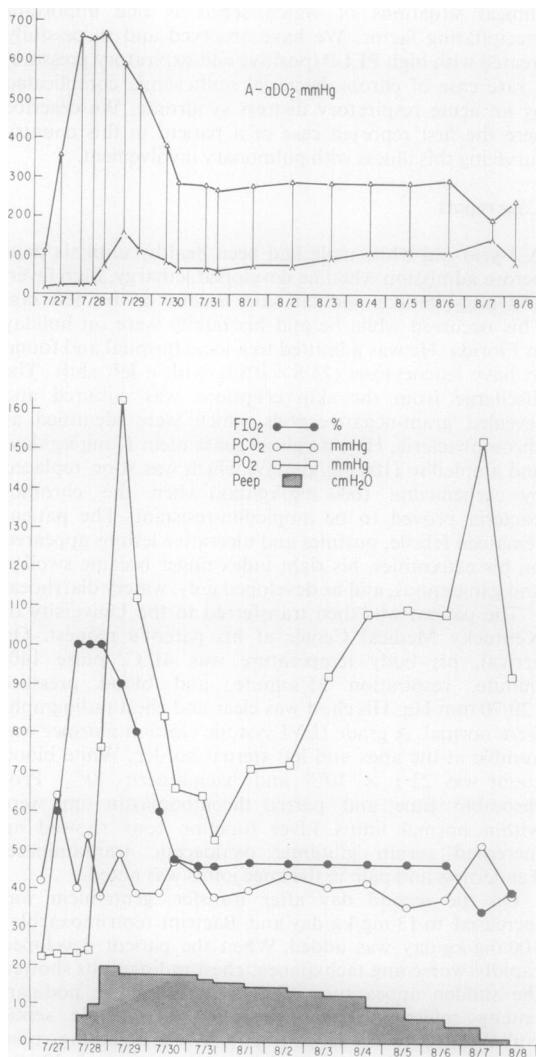


Fig 3 Change of arterial blood gases during the course of respiratory insufficiency. Note severe hypoxaemia and wide $A-aDO_2$ before institution of high PEEP. Conversion: 1 mmHg = 0.133 kPa.



Fig 4 Osteomyelitis of the left knee with extensive moth-eaten lesion in the distal femur.

endothelial damage with increased capillary permeability have been described as two important factors.⁹⁻¹¹

In the case presented here, sepsis was followed by the development of intractable hypoxaemia with a wide A-aDO₂ shunt, remarkable decrease in lung compliance, a picture of non-cardiogenic pulmonary oedema on the chest radiographs, and response to a high PEEP level, all of which clinically suggested a respiratory distress syndrome.¹²

Based on this experience, although not clearly defined, we suspect that acute respiratory distress syndrome may

have been one of the significant causes contributing to the death of the previously reported cases.

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