**Pasteurella multocida** pneumonia complicated by *Staphylococcus aureus*  

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**Summary**

A 71-year-old woman presented with acute non-cardiogenic pulmonary oedema. She proved to have a *Pasteurella multocida* pneumonia, with blood stream invasion by the organism, and required positive pressure ventilation for 53 days.

Penicillin G., the drug of choice for this infection, failed to reverse the steady decline in her arterial oxygen tension, and it was only after treatment with chloramphenicol and prednisolone that she began to improve.

Serological tests strongly indicated the presence of a *Staphylococcus aureus* infection and the delay in giving antibiotics appropriate to this second pathogen may have been the reason for the patient's initial downhill course.

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**Key Words:** adult respiratory distress syndrome.

**Introduction**

*Pasteurella multocida* pneumonia is uncommon. We report a case notable for rapid onset of respiratory insufficiency, requirement for long-term ventilation, lack of response to penicillin G., development of *Staphylococcus aureus* secondary infection, and possible development of adult respiratory distress syndrome.

**Case report**

A 71-year-old widow lived alone with her cat. She smoked 5–10 cigarettes a day and had a history of bronchitis and hypertension.

After feeling generally unwell for 2 days, she awoke with shortness of breath and was found at home in severe respiratory distress, producing frothy pink sputum.

Her electrocardiogram showed ischaemic changes and acute pulmonary oedema was diagnosed. Two intravenous doses of frusemide 40 mg gave no improvement and she suffered an asystolic cardiac arrest. After resuscitation she was transferred, still hypotensive, to the Intensive Care Unit, where a flow-directed thermodilution-type pulmonary artery catheter was inserted.

Investigations—chest X-ray: left upper lobe consolidation; haemoglobin: 17·1 g/dl; white cell count 16·2 x 10⁶ per litre with neutrophilia; pulmonary wedge pressure 1·3 kPa (10 mmHg); central venous pressure 0·5 kPa (5 cmH₂O); blood glucose 25·3 mmol/litre; arterial Po₂ 10·9 kPa (82 mmHg) on 100% oxygen by endotracheal tube.

A provisional diagnosis was made of non-cardiogenic pulmonary oedema, possibly secondary to the left upper lobe infection. Management comprised positive pressure ventilation, fluid loading to a wedge pressure of 1·8 kPa (14 mmHg), antibiotics (cephamandol with tobramycin), and low dose insulin infusion to control the hyperglycaemia. Cardiac output after fluid loading was 6·8 litre/min. Both sputum and blood cultures showed profuse growth of a particularly mucoid, encapsulated *Pasteurella multocida* organism, and penicillin-G 2 M units 4-hourly with probenecid 500 mg t.d.s. was commenced. The adequacy and appropriateness of this therapy was confirmed when the patient's serum was shown inhibitory to the *Pasteurella* isolate at 1:512, and bactericidal at 1:32. The organism did not appear on sputum culture after day 5.

On day 32, *Pasteurella multocida* (O) serum agglutinin level was 40 units, and (H) serum agglutinin level was less than 20 units.

*Pseudomonas aeruginosa* was grown from the sputum on day 5 and intravenous tobramycin was therefore recommenced.

In the face of apparently correct treatment, her condition showed a slow but relentless deterioration. For 4½ weeks, the white cell count remained elevated associated with low grade fever. Peripheral oedema developed in association with hypo-albuminaemia (21 g/litre) in spite of high protein enteral feeding.

The most disturbing feature however, was her hypoxaemia. By day 30, her arterial Po₂ had fallen to 6·45 kPa (48·4 mmHg), despite continued intermit-
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P. multocida infections commonly require oxygen and 1.5 kPa (15 cmH₂O) positive end expiratory pressure (PEEP).

This deterioration mirrored the steady extension of consolidation through the left lung and into the right lower zone, though there was no radiological evidence of lung abscess, pleural effusion or empyema.

In the search for an underlying abnormality of immune function or a more exotic infection, several other areas of investigation were explored. Tests of immune functions showed no significant abnormality. Bronchoscopy on days 5 and 27 showed tenacious sputum, formerly purulent and growing Pasteurella multocida, latterly mucoid and clear of organisms.

Serological tests gave a strong suggestion of staphylococcal infection, although no staphylococcus was actually grown from the sputum until day 47. Antistaphylolysin titres peaked at 16 iu/litre on day 32 (normal range 0–2), then fell to 8 iu/litre on day 50 and to 2 iu/litre on day 78. Antistaphylolysin 0 titre was 200 iu (normal range less than 160) and Streptonase B, 680 units (normal range less than 85), both on day 32. The peaks of these titres corresponded within 2 days, to the patient’s lowest arterial Po₂. While waiting for the results of these tests however, we considered that a change of treatment was needed to prevent the continuing deterioration. Since chloramphenicol had previously been demonstrated to be bactericidal in vitro against the Pasteurella multocida isolate, chloramphenicol 1-2 g q.i.d. (10 days) was commenced with prednisolone 15 mg q.i.d. Within 24 hr, arterial Po₂ rose from 6-45 kPa to 11-33 kPa (48-4–85 mmHg) and the improvement was maintained, enabling stepwise reduction of inspired oxygen and PEEP.

Staphylococcus aureus grew profusely from the sputum on day 47 and this was treated with cloxacillin 1 g q.i.d. i.v. to which it had been shown to be sensitive. Ventilation was continued until day 53 (tracheostomy had been carried out on day 16). She was discharged home on day 89 still experiencing moderate exertional dyspnoea.

Discussion

Pasteurella multocida, a Gram negative coccobacillus, is part of the normal oro-pharyngeal flora in many animals, including cats, dogs and fowls.

Animal bites may cause soft tissue pasteurella infections, while respiratory infection has been associated with non-traumatic exposure to the organism. It may colonize the respiratory tract in normal humans, but under conditions of impaired pulmonary defences, it assumes an enhanced pathogenicity and may cause severe pneumonia with lung abscess and empyema. Older patients with chronic lung disease, particularly chronic bronchitis and bronchiectasis are most at risk, and animal exposure is common in such cases. Premonitory symptoms, such as dyspnoea, pleurisy or haemoptysis, may last days to weeks (Nelson and Hammer, 1981) and presentation in acute pulmonary oedema has been reported (Calverley et al., 1981). Severe cases have required up to 6 days mechanical ventilation (Calverley et al., 1981). Mortality may be 50% where septicemia is associated with the respiratory infection. Penicillin G. is reputedly the drug of choice for Pasteurella multocida infections—high dosage is necessary because of the organism’s tendency to cause tissue necrosis and abscess formation (Rose and Mathai, 1977).

Penicillin G. did not resolve our patient’s illness. Penicillin resistant strains of P. multocida have been described and lack of clinical efficacy of penicillin in the face of apparent in vitro organism sensitivity has been shown in both human (Henderson, 1963) and veterinary practice (Stevenson and Hughes, 1980).

The microbiological tests were highly suggestive that the Pasteurella multocida had given way to a Staphylococcus aureus as the main pathogen in this patient’s lungs, though interestingly, S. aureus itself was not grown from the sputum until day 47. In some way the staphylococcal infection seemed to have been masked by the Pasteurella multocida, though the anti-staphylolysin titres gave the clue as to what was really going on.

An alternative explanation may be that the initial infection precipitated adult respiratory distress syndrome. In either case, the improvement after chloramphenicol and prednisolone was extraordinarily rapid and may even have been co-incidental. The patient’s cat may have been the source of her Pasteurella multocida infection, but unfortunately the animal was destroyed before it could be tested.

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