Pseudomonas pneumonia in status asthmaticus

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Summary

A case of *Pseudomonas pneumonia* developing in a patient on treatment for status asthmaticus is described. The use of broad spectrum antibiotics, high dose steroids and humidifiers or nebulizers may be of aetiological importance. *Pseudomonas pneumonia* is increasing in prevalence although it has not previously been described in association with status asthmaticus. It carries a high mortality and its prevention is, therefore, clearly important.

Case report

A 36-year-old housewife was admitted to hospital in status asthmaticus. She had had asthma since the age of 3 but had been well controlled on a salbutamol inhaler only. She was treated on admission with steroids (hydrocortisone and subsequently prednisolone 50 mg daily), aminophylline, salbutamol, ampicillin and oxygen through an East-Radcliffe humidifier. She responded well to these measures and 12 days later was considered ready for discharge home. However, that night she felt unwell and developed pleuritic pain over the right side of the chest. There were no signs on examination apart from diminished air entry at the right base anteriorly. Six hours later the pain became severe and she had a small haemoptysis. On examination she was pale, sweaty and cyanosed with a thready pulse of 108/min and systolic blood pressure of 10.64 kPa. The diminished air entry at the right base was still apparent. There was no pyrexia, ECG was normal, WBC $3.9 \times 10^9/l$, Pao_2 12.1 kPa, $Paco_2$ 5.0 kPa and pH 7.4. Chest X-ray showed patchy shadowing over the right mid zone. In view of the severe shock it seemed that pulmonary embolism was the most likely diagnosis, the pleurisy and haemoptysis which preceded the shock being accounted for by an earlier, smaller embolus which had led to the infarct. A pulmonary angiogram was therefore performed, but this merely

showed changes consistent with obstructive airways disease and no evidence of an embolus. A repeat chest X-ray 6 hr after the initial X-ray showed further extension of the right mid-zone shadowing and a diagnosis of fulminating pneumonia was made. Empirical treatment with ampicillin, flucloxacillin and gentamicin was started and, in view of further deterioration in her condition, general resuscitative measures, including intravenous fluids, isoprenaline, digoxin, sodium bicarbonate, further steroids and intermittent positive pressure ventilation, were instituted. However, there was little response and she died from a cardiac arrest approximately 28 hr after the onset of the initial symptoms. At post-mortem both lungs were found to be congested, haemorrhagic and oedematous with no evidence of pulmonary embolism. In the central portion of the right lower lobe there was a greyish firmer area stretching across the lung. Histology showed the alveoli to be stuffed with Gram-negative bacteria but there was no surrounding vital reaction. Swabs from the lung grew profuse *Pseudomonas aeruginosa*; no viruses were isolated.

Discussion

P. aeruginosa is becoming increasingly recognized as a cause of pneumonia (Pierce *et al.*, 1966; Editorial, 1971; Rose, Heckman and Unger, 1973; Pennington, Reynolds and Carbone, 1973; Crane and Lerner, 1975). In 1973 Pennington *et al.* described thirty-six patients with *Pseudomonas pneumonia* of whom twenty-nine (81%) died. This compares with mortalities of 100% and 72% reported by Rose *et al.* (1973) and Crane and Lerner (1975) respectively. In the series of Rose *et al.*, the lower lobe was involved in seventeen of the nineteen cases. Flick and Cluff (1976) in a review of 108 cases of *Pseudomonas* bacteraemia found the respiratory tract to be the major portal of entry. They noted a proportional increase in middle-aged patients compared with an earlier review. Of their cases 49%

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died from the *Pseudomonas* infection and 61% of these deaths were on the same day as the positive blood culture. Shock was common and found to be an 'ominous sign'. Pierce *et al.* (1966) in their series reported shock before death in twenty-four out of forty-one patients confirmed at post-mortem to have *Pseudomonas* pneumonia.

A number of factors seem to be of aetiological importance: (1) previous broad spectrum antibiotics, (2) immunosuppressive agents (including prednisolone) and (3) the use of ventilators, reservoir nebulizers or humidifiers. All these factors may have been important in the present case although it seems unlikely that her short period on the ventilator was significant. The absence of pyrexia or leucocytosis is characteristic of *Pseudomonas* infection (especially in the patient on steroids) and may lead to diagnostic difficulties. Whilst *Pseudomonas* pneumonia is well described in chronic obstructive airways disease (Editorial, 1971; Rose *et al.*, 1973; Crane and Lerner, 1975), it has not previously been reported as a complication of the routine management of status asthmaticus. A number of points should be considered. *Pseudomonas* pneumonia is a rapid, fulminant process with little response to antibiotics and a very high mortality (Pennington *et al.*, 1973). Prevention is clearly better than cure. Superinfection with *Pseudomonas* is a risk with both humidifiers and nebulizers though it is more common with the latter as the particles produced are smaller and may therefore reach the peripheral airways and alveoli. If

humidifiers or nebulizers must be used then it is essential that meticulous decontamination of the equipment is undertaken every 24 hr, as recommended by Crane and Lerner (1975). The routine use of broad spectrum antibiotics in status asthmaticus is questionable and it may be better to reserve these for specific, proved infections. High dose steroids are undoubtedly life-saving but the dosage should be reduced as soon as possible. Nursing in an isolation room may also be a sensible precaution. Finally, the patient on treatment for status asthmaticus would seem to be at special risk of developing *Pseudomonas* pneumonia and this diagnosis should be considered in any such patient whose condition suddenly deteriorates.

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