

# Staphylococcal pneumonia, pneumatoceles, and the toxic shock syndrome

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## Abstract

**A case of community acquired staphylococcal pneumonia is reported with the unusual complication, in an adult, of multiple pneumatoceles. Recognition of this prevented inappropriate management. The patient also developed the toxic shock syndrome. In contrast to infants with pneumatoceles, recovery of lung function has been poor.**

Community acquired staphylococcal pneumonia is uncommon. It usually follows bacteraemia from a minor skin infection and results in a range of presentations from localised pneumonia to bilateral cavitary disease and empyema.<sup>1</sup> Infection may also spread directly from the nasopharynx after influenza to produce a severe generalised necrotising pneumonia. Infants are more susceptible to staphylococcal pneumonia, 70% of cases occurring in the first year of life.<sup>2</sup> This may be due to the acquisition of particularly high nasal carriage rates at this age.<sup>3</sup> Pneumatoceles, which in the past occurred in

up to 60% of cases,<sup>2</sup> are highly suggestive of staphylococcal infection but are not generally recognised as occurring in adult life. We report a case of an adult presenting with multiple pulmonary pneumatoceles from an illness additionally complicated by the toxic shock syndrome.

## Case report

A 26 year old, previously healthy heterosexual man became unwell with fever and rigors. The following day he developed lower right sided pleuritic chest pain and the beginning of what became a generalised, fine, superficial, and non-itchy erythematous rash. He was hypotensive (blood pressure 90/50 mm Hg) with a regular tachycardia (128 beats/min) and was prescribed amoxicillin. On day four a radiograph showed peripheral upper lobe consolidation with a pleural reaction. Further investigation showed hepatorenal impairment (serum creatinine 180 (normal range 53-106)  $\mu\text{mol/l}$ ; albumin 32 (41-52) g/l; alkaline phosphatase 242 (30-118) U/l; alanine transaminase 125 (<45) U/l). Skin desquamation developed, initially on the hands and feet but subsequently becoming widespread. He remained ill and erythromycin was substituted for amoxicillin. His condition then improved and a repeat chest radiograph showed some radiographic resolution (fig 1). Fourteen days after the onset of the illness, however, he became severely breathless over a few hours and was admitted to hospital.

On admission he was unwell, had an oral temperature of 39°C, and was in respiratory distress. Surgical emphysema was evident in the neck and the clinical signs suggested a large right sided pneumothorax. A small area of traumatised and possibly infected skin was noted on the shin. The radiograph was interpreted as showing either a multiloculated pyopneumothorax or multiple intrapulmonary pneumatoceles. The presence of air bronchograms (fig 2) with peripheral lung parenchyma favoured a diagnosis of pneumatoceles and computed tomography confirmed this. A section of the scan (fig 3) shows peripheral lung parenchyma with compressed lung between two large air-fluid collections. Further scans showed multiple abscesses. An enterotoxin B producing *Staphylococcus aureus* (phage type 94/96), resistant only to penicillin, was isolated from a lung aspirate but throat, nasal, skin, and blood cultures were sterile. Viral serology excluded recent influenza.

Surgical intervention was not considered to be indicated and he was treated with intravenous flucloxacillin and oral fusidic acid for three weeks. His recovery was slow, fever persisting for six weeks, but he returned to his former employment as an electrician four months later. He remained breathless on moderate exertion. The development of considerable pleural thickening on resolution of the pneumatoceles suggested little functional recovery of the right lung and this was confirmed by isotope lung scanning (perfusion of right lung only 30% that of the left). At nine

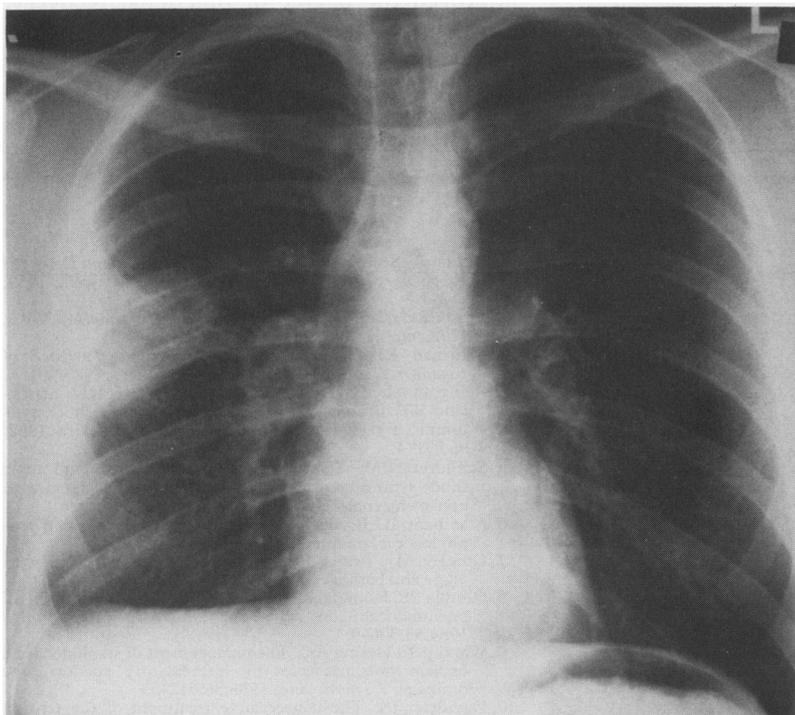
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**Figure 1** Posteroanterior radiograph 12 days after the onset of illness showing peripheral mid zone consolidation with a pleural reaction. This represents an improvement on the film taken on day 4.

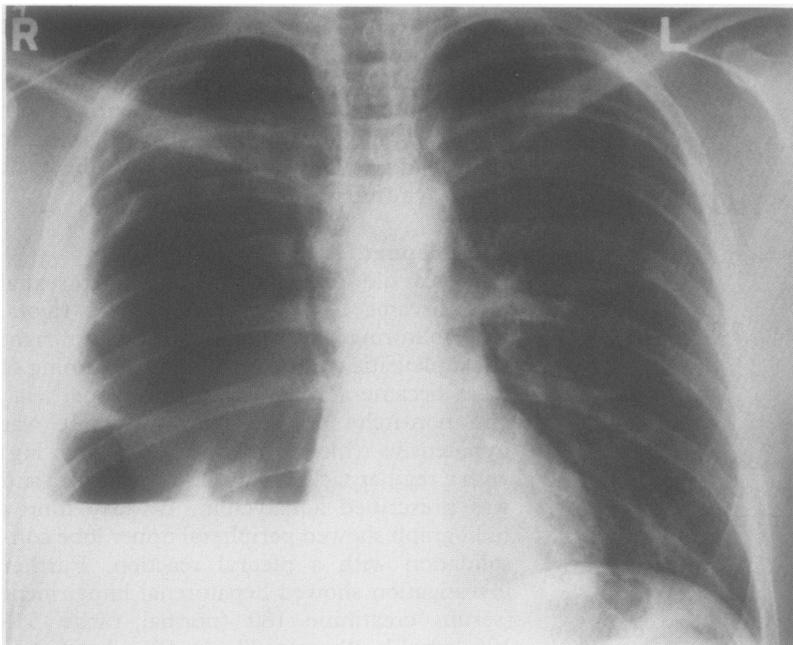


Figure 2 Posteroanterior radiograph showing multiple air-fluid collections within the right hemithorax. Air bronchograms of the upper and lower lobe segmental bronchi are visible and lung parenchyma can be seen peripherally at the right apex.

months lung function testing showed a restrictive ventilatory impairment (FEV<sub>1</sub> 2.8 l/s, FVC 3.1 l) with a reduction in carbon monoxide transfer (65% predicted).

### Discussion

Non-gynaecological toxic shock syndrome accounts for 12% of all cases and usually complicates staphylococcal infection after surgery.<sup>4</sup> Non-menstrual cases are more commonly associated with production of enterotoxin B than of toxic shock syndrome toxin 1, and this accounts for the syndrome of prostration, desquamative erythema, and multi-organ disease.<sup>5</sup> The toxic shock syndrome has recently been reported in *S aureus* pneumonia,<sup>6</sup> though in this case the diagnosis was presumptive. The infrequency of staphylococcal pneumonia may account for the lack of previous reports. *S aureus* is rarely found in

blood in the toxic shock syndrome, but it is likely, though unproved, that blood cultures would have been positive at the onset of illness in our patient. It is not clear why the toxic shock syndrome is so rarely associated with positive blood cultures.

Although Crofton and Douglas<sup>1</sup> recognise the development of pneumatoceles in young adults, we have found only two individual case reports,<sup>7,8</sup> and in neither case were the intrapulmonary cysts as dramatic as in our patient. Although we cannot be certain that our patient did not have a multiloculated pyopneumothorax, the history and radiological findings are much more in favour of pulmonary pneumatoceles, a condition in which chest drains would have been inappropriate. Single or multiple pneumatoceles were considered diagnostic of staphylococcal pneumonia in infancy,<sup>2</sup> though there are recent reports of this complication with other bacterial pathogens. The older reports document the development of these high pressure air spaces typically in early convalescence, with spontaneous resolution and good functional recovery.<sup>2,9,10</sup> Although cases of subsequent bronchiectasis have come to be recognised, this suggests that normally little lung is destroyed. Alternatively, the growth potential in infants may account for the good prognosis. Partial obstruction to the drainage of an area of cavitation, a flap valve mechanism leading to progressive inflation, and enlargement of the microabscess probably account for the development of pneumatoceles.<sup>7</sup> The absence of collateral ventilation, such as the pores of Kohn, might explain why pneumatoceles typically develop in infancy because interalveolar ventilation has not developed at this age.<sup>11</sup> Considerable lung destruction was apparent in the present case and surgical intervention is unlikely to have improved the degree of functional recovery. Surgery is not indicated in this condition unless the mediastinum is substantially displaced or, possibly, rupture occurs into the pleural space.<sup>9,10</sup>

We thank Dr S Ekyinn and Dr B Ayers for their helpful advice in the preparation of this report.

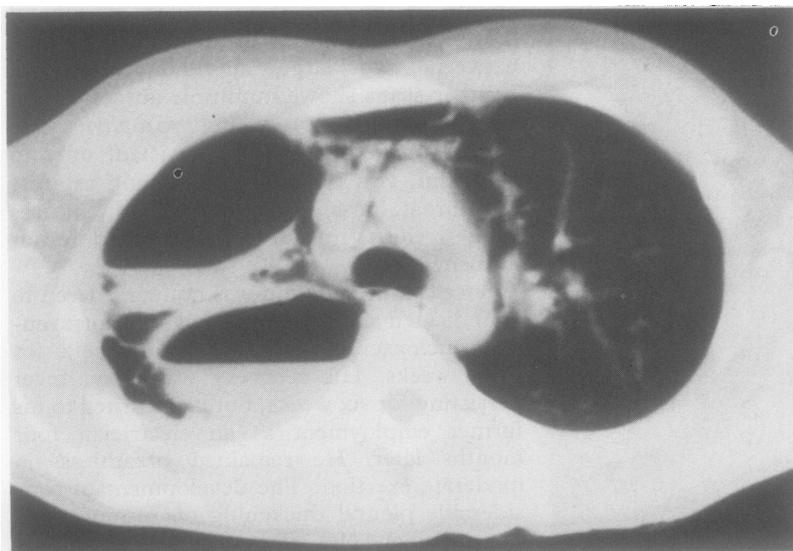


Figure 3 Axial computed tomogram at a level just proximal to the carina showing two large pneumatoceles with lung parenchyma peripheral to the more posteriorly placed air cyst.

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