The mechanisms by which hypoxaemia is produced in severe community acquired pneumonia include anatomical shunting and ventilation/perfusion mismatch.³ There is also a reduction in thoracic compliance and subsequent increase in respiratory work leading to fatigue.⁴ Continuous positive airway pressure improves oxygenation by increasing pulmonary pressure throughout the respiratory cycle, thereby increasing lung volume.⁵ This has the effect of decreasing the anatomical shunt and improving ventilation/perfusion matching; as compliance is raised the work of breathing is reduced. The use of CPAP delivered by face mask, if successful, avoids endotracheal intubation and IPPV with its associated risks including cardiovascular depression, barotrauma, and increased susceptibility to nosocomial infection.⁶⁻⁸ If, however, CPAP is not effective we emphasise that IPPV should be instituted without delay, and would recommend that CPAP only be administered under close medical supervision where this facility is immediately available.

The greatest potential complication of CPAP is the aspiration of gastric contents, so patients in whom its use is being considered must be orientated, cooperative, and not suffering from nausea or vomiting.⁹

Although CPAP is well recognised and used as an adjunct to treatment in *Pneumocystis carinii* pneumonia,¹⁰ this does not appear to be the case for community acquired pneumonia where the potential for rapid resolution and recovery are greater. We

suggest that greater consideration is given to this form of ventilatory support in patients with deteriorating pulmonary function meeting the above criteria.

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Endobronchial nocardial infection

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Abstract

The rare presentation of nocardial infection as an endobronchial "tumour" is reported. Haematogenous dissemination occurred after fiberoptic bronchoscopy and biopsy, a phenomenon not previously described in nocardial infections. This case highlights the difficulties in diagnosing pulmonary nocardial infection and the potential for invasive procedures to disseminate the disease.

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Pulmonary nocardiosis is uncommon, but this infection is becoming increasingly important with the widespread use of immunosuppressive treatment in transplant recipients and patients with malignancy.¹⁻³ The disease is caused by several species of the genus *Nocardia* which most commonly behave as opportunistic pathogens in the compromised

host.¹ Pulmonary infection is the commonest form of the disease with dissemination from the lungs to other sites occurring in up to 50% of cases.^{2,4} Pulmonary nocardiosis presenting as an endobronchial tumour is, however, uncommon with only two previously reported cases.^{5,6}

Case report

A previously healthy 70 year old male smoker presented with a three month history of non-productive cough, dyspnoea, anorexia, and weight loss. On admission he was afebrile. Chest auscultation revealed diminished breath sounds throughout the right lung associated with a monophonic wheeze. The remainder of his physical examination was normal. The neutrophil count was elevated at $24 \times 10^9/\text{litre}$. The erythrocyte sedimentation rate was 90 mm in the first hour. A chest radiograph showed a right hilar mass (fig 1) and a computed tomographic scan showed this mass to be encroaching on the lumen of the right upper lobe bronchus (fig 2). At fiberoptic bronchoscopy there was obstructing "tumour" in the right main bronchus. Endobronchial biopsy specimens showed acute and chronic inflammatory changes with no evidence of granuloma formation. There was no evidence of malignancy.

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Figure 1 Chest radiograph showing right hilar mass.

Four days after bronchoscopy he developed hypotension, a temperature of 39°C, a generalised pustular rash, and digital lesions suggestive of septic emboli.

There was no clinical evidence of cardiac, meningeal or central nervous system involvement. *Nocardia asteroides* was cultured from the bronchial washings and biopsies after four days, and subsequently from his sputum and cutaneous pustules. Intravenous co-trimoxazole (trimethoprim 480 mg and sulphamethoxazole 2400 mg twice daily) was commenced, with defervescence within 48 hours. Oral co-trimoxazole was commenced after 10 days of intravenous treatment but subsequently ceased after 20 days following the development of erythema multiforme. Minocycline, 100 mg twice daily, was substituted on day 30 and continued for a total of 10 months. After 14 months he remained well with radiographic resolution of the right hilar mass. Investigations revealed no underlying defect of humoral or cell mediated immunity.

Discussion

Nocardial infection is uncommon, with an incidence in Queensland of 2.1 new cases per year per 100 000 head of population.⁷ Fever and cough are the commonest presenting symptoms. Abscesses, necrotising pneumonias, cavitating lesions, nodules, infiltrates, and miliary patterns all occur.² The clinical and radiographic picture is variable, reflecting the host immune status and the ability of the organism to cause both suppurative and granulomatous disease.^{1,8}

The diagnosis is often delayed as *Nocardia* may take up to four weeks to grow.² Bacterial cultures of sputum and bronchoscopy specimens are often routinely discarded after 48–72 hours, so the diagnosis can be missed.⁶

This case shows the potential for bronchoscopy to disseminate nocardial infection. This phenomenon is well recognised with

Figure 2 Computed tomographic scan showing mass encroaching on lumen of right upper lobe bronchus (arrowed).

other organisms such as *Mycobacterium tuberculosis*,⁹ but has not been reported in nocardial infections. However, because sputum cultures are negative in up to two thirds of patients with pulmonary nocardiosis, invasive procedures are usually required for diagnosis.¹

Nocardial infections are usually treated with sulpha-based regimens.⁴ Sulpha drugs used alone in doses of 6–12 g daily are effective.^{2,4} More recently co-trimoxazole (trimethoprim 80 mg and sulphamethoxazole 400 mg) has been used, but treatment failures have been noted.¹⁰ Minocycline has been used successfully⁴; again, however, treatment failures have been reported. There are no comparative data available to determine duration of treatment. In pulmonary and systemic nocardiosis, without central nervous system involvement, experience suggests that treatment should be continued for 6–12 months. Infection of the central nervous system should be treated for a minimum of 12 months.^{2,4}

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