Short reports

Outbreak of *Moraxella catarrhalis* in a respiratory unit

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Abstract

The clinical and epidemiological features of an outbreak of nosocomial *Moraxella catarrhalis* infection in a respiratory unit are described. Six isolates from five patients were shown to be indistinguishable by immunoblotting and restriction endonuclease analysis and different from 11 other, unrelated clinical strains.

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Moraxella (Branhamella) catarrhalis is a Gram negative, oxidase positive coccus, which has been increasingly recognised as a respiratory tract pathogen during the 1980s, especially in patients with underlying lung disease.[1] It most commonly causes exacerbations of chronic bronchitis but may also cause otitis media in children, conjunctivitis, or pneumonia.

This report describes the use of a combination of restriction endonuclease analysis (REA), immunoblotting, and SDS PAGE of whole cell proteins to examine six isolates of *M catarrhalis* from a cluster of infections among patients in a respiratory unit at the Northern General Hospital, Edinburgh.

Cases

The first case to be identified in this cluster of infections was in a 52 year old man with longstanding asthma. He was admitted to the respiratory unit with right sided chest pain and increasing dyspnoea. On admission his sputum was mucoid and culture yielded commensal flora only. He was treated for an exacerbation of his asthma and investigated for a possible pulmonary embolus. Ten days later he developed a cough producing purulent sputum, from which a heavy growth of β lactamase producing *M catarrhalis* (isolate 1) was cultured. The organism was identified by hydrolysis of tributyrin. The isolate was resistant to ampicillin, but sensitive to coamoxiclav, erythromycin, and chloramphenicol. Before the bacteriological results were available he was treated empirically with oral amoxycillin 250 mg four times daily, but he failed to respond clinically. A second sputum sample, collected while he was having amoxycillin, also yielded β lactamase producing *M catarrhalis*, with the same antibiotic sensitivity pattern (isolate 2). At this point his antibiotic treatment was changed to oral ciprofloxacin, 750 mg twice daily for seven days, followed by co-trimoxazole 960 mg twice daily for a further week. With this treatment his symptoms gradually improved and he was discharged.

Four days before his discharge a heavy growth of β lactamase positive *M catarrhalis* was isolated as the sole pathogen from sputum specimens collected from four other patients on the ward (isolates 3–6). These isolates all had the same antibiogram as the isolates from the index case. All four patients had unexpectedly developed acute respiratory tract infections during the course of a single weekend, with symptoms of cough productive of large amounts of purulent sputum, but no evidence of pneumonia. The timing of these events in relation to the index case and the underlying diagnoses of the patients are shown in figure 1. The duration of their stay in hospital before the onset of these symptoms ranged from six to 25 days.

All the patients had started treatment with amoxycillin when they developed symptoms, in accordance with the unit's policy on antibiotics. Once the bacteriological results were available their treatment was changed to oral co-amoxiclav and thereafter their symptoms resolved rapidly. The discharge of one patient was delayed for four days as a consequence of this infection.

Because of the striking clustering of these cases we decided to look for more widespread carriage of *M catarrhalis*, but nose and throat swabs collected from symptomless patients were all negative. In view of the rapid response of the symptomatic patients to coamoxiclav, and the failure to find *M catarrhalis* during screening, no specific measures of infection control were instituted, and no further cases were identified.

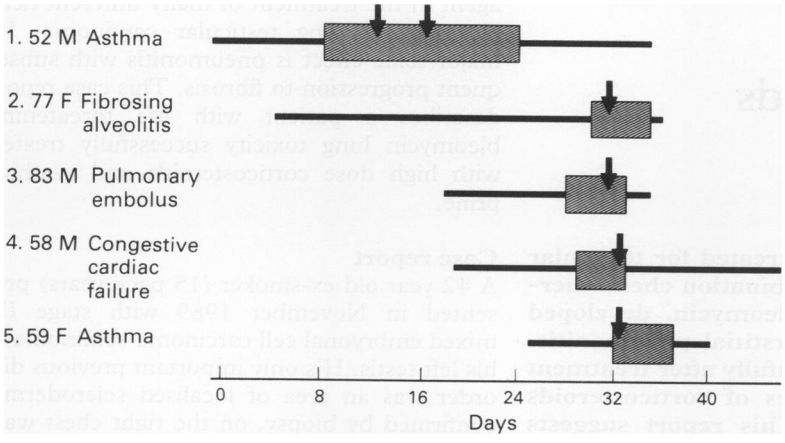

Figure 1 *Diagrammatic representation of the outbreak, with time scale in days.*

Typing of isolates

The six isolates of *M catarrhalis*, together with 11 unrelated clinical strains, were examined at Aberdeen University by immunoblotting

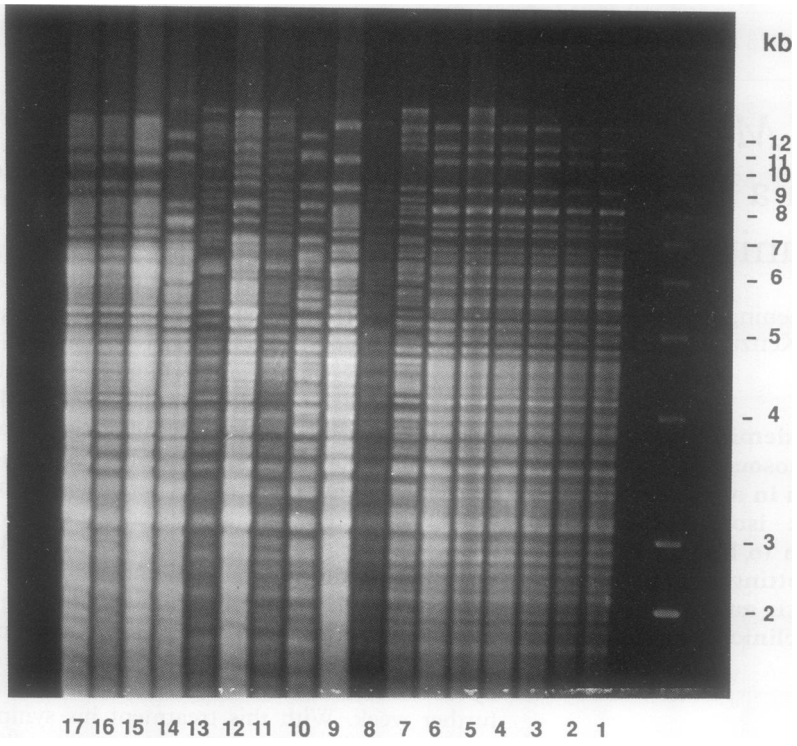

Figure 2 Restriction endonuclease analysis of bacterial DNA from 17 isolates of Morexella catarrhalis. Isolates 1–6: strains from suspected outbreak at the Northern General Hospital, Edinburgh. Isolates 7–17: unrelated clinical isolates (isolates 15–17 were from an unrelated, smaller cluster of infection in a geriatric unit).

with normal human serum and confirmed by restriction endonuclease analysis of bacterial DNA.[2] The banding patterns of the six isolates from the respiratory unit at the Northern General Hospital were indistinguishable by both methods, whereas the unrelated strains all had different profiles (fig 2). These results provide clear evidence that this cluster of cases was due to cross infection with a single strain of *M catarrhalis*.

Discussion

Nosocomial infection with *M catarrhalis* has been previously reported (McLeod *et al*[3]), but until recently there have been no reliable typing methods for investigating the epidemiology of this organism. It has not therefore been known whether nosocomial infection is caused by patients' endogenous flora or by cross infection. The first documented outbreak of cross infection with *M catarrhalis* was described in 1988, at a Veterans' Administration hospital in the United States.[4] The isolates from this outbreak were successfully typed by restriction endonuclease analysis of bacterial DNA.

The finding that cross infection with *M catarrhalis* may cause hospital acquired infections has important implications for respiratory units, where large numbers of susceptible patients may be at risk. The reservoir of infection and mode of transmission of *M catarrhalis* in this setting is still unknown but there is evidence that the organism may survive in the environment for up to 27 days in dried secretions.[5] Further work is needed to identify appropriate control measures to prevent nosocomial infection with *M catarrhalis*. Although these infections are not severe, their costs in terms of additional courses of antibiotics and prolongation of hospital stay may be substantial.

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3 McLeod DT, Ahmad F, Capewell S, Croughan MJ, Calder MA, Seaton A. Increase in bronchopulmonary infection due to *Branhamella catarrhalis*. *BMJ* 1986; 292:1103–5.
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Severe bleomycin lung toxicity: reversal with high dose corticosteroids

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Abstract

A 42 year old man, treated for testicular carcinoma with combination chemotherapy that included bleomycin, developed life threatening interstitial pneumonitis. He recovered successfully after treatment with very high doses of corticosteroids and azathioprine. This report suggests that bleomycin lung toxicity may be reversible if treated aggressively.

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Bleomycin is an effective chemotherapeutic agent in the treatment of many different neoplasms, including testicular carcinoma. Its major toxic effect is pneumonitis with subsequent progression to fibrosis. This case report describes a patient with life threatening bleomycin lung toxicity successfully treated with high dose corticosteroids and azathioprine.

Case report

A 42 year old ex-smoker (15 pack years) presented in November 1989 with stage IIb mixed embryonal cell carcinoma-seminoma of his left testis. His only important previous disorder was an area of localised scleroderma, confirmed by biopsy, on the right chest wall. There was no clinical or serological evidence of systemic sclerosis. After orchidectomy he