Hospital Topics

Bronchopulmonary infection due to *Branhamella catarrhalis*

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Abstract

Over six months *Branhamella catarrhalis* was isolated in pure culture from the sputum of 81 patients with symptoms of acute respiratory tract infection. Of 38 patients who were infected in the community, over half required admission to hospital. The remaining 43 patients acquired the infection in hospital. Forty one of the 81 isolates produced β -lactamase, 24 of these being hospital acquired infections. As a result 40% of patients who were treated with ampicillin did not respond. Most patients had chronic lung diseases or lung cancer or were taking corticosteroids. Three patients died and one required assisted ventilation; strains producing β -lactamase were isolated in each case. Acute bronchitis developed in one previously healthy young non-smoker.

It is concluded that *B catarrhalis* is an important pathogen of the lower respiratory tract which should be reported, and strains producing β -lactamase should be identified. Otherwise, treatment with inappropriate antibiotics may result in increased morbidity or mortality.

Introduction

Branhamella (previously *Neisseria*) *catarrhalis* is an aerobic Gram negative diplococcus. It has generally been regarded as an oropharyngeal commensal, though it has been implicated as a pathogen in sinusitis, laryngitis, otitis media, meningitis, and endocarditis. In the past decade it has been shown to cause pneumonia in immunologically compromised patients¹ and infective exacerbations in patients with chronic lung disease.² Recently, an increasing number of strains producing β -lactamase have been described.³ In spite of this, the number of reports of respiratory disease due to *B catarrhalis* has been relatively small.⁴ We have always reported the presence of pure growths of *B catarrhalis* in sputum samples, and we noted a striking

increase in the number of such isolates during the past winter months, many being the only organism found in the sputum of seriously ill patients. This, together with a notable increase in the proportion of strains producing β -lactamase, caused us to review the antibiotic policy of our acute respiratory wards. In the belief that others will soon see this changing pattern of pathogens we report the clinical features of *B catarrhalis* infection.

Methods

Our bacteriology laboratory receives sputum samples from hospital wards, outpatient departments, and local general practices. A purulent part of each sputum specimen received was examined with a Gram film and inoculated on blood agar and heated blood plates (containing bacitracin 10 units/ml). The plates were incubated in 8% carbon dioxide for 18 hours. Only moderate or profuse growth in the primary plate was reported. *B catarrhalis* was presumptively identified by the presence of extraleucocyte and intraleucocyte Gram negative diplococci in the Gram film, by their colonial and morphological appearance, and by positive catalase and oxidase reactions. The identification was confirmed with rapid carbohydrate utilisation tests.⁵ All strains of *B catarrhalis* were tested for deoxyribonuclease activity.⁶ Production of β -lactamase was detected with chromogenic cephalosporin.⁷ This bacteriological screening of sputum has not altered for some years. Tests of sensitivity to penicillin, ampicillin, chloramphenicol, tetracycline, erythromycin, and co-trimoxazole were performed using the disc diffusion method.

A retrospective study of the case notes and radiographs of hospital patients together with information acquired from general practitioners was made for all patients who yielded a pure culture of *B catarrhalis* in sputum from November 1982 to April 1983. Mixed infections were excluded as it would have been difficult to attribute symptoms to *B catarrhalis* in the presence of known pulmonary pathogens such as *Haemophilus influenzae* or *Streptococcus pneumoniae*.

Results

The total numbers of routine sputum samples examined during the six month periods November to April in 1980-81, 1981-82, and 1982-83 were similar at 2922, 2969, and 3071 respectively. In November to April 1982-83 there were 81 pure isolates of *B catarrhalis* in sputum from 49 men and 32 women (mean age 67 years, range 22-88) (figure). Chronic pulmonary diseases in these patients included chronic bronchitis and emphysema (42), asthma (13), carcinoma bronchus (12), pulmonary fibrosis or bronchiectasis (nine), and pneumonia (nine). One patient, a healthy, non-smoking 22 year old nurse, developed acute bronchitis due to a strain of *B catarrhalis* producing β -lactamase while working on the wards. The patients included 43 smokers, 15 non-smokers, and 14 ex-smokers; smoking habits were not known in nine.

Of the 38 patients who acquired the infection in the community, 24 had *B catarrhalis* in their sputum on admission to hospital; 12 were attending hospital outpatient departments, and two were

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attending general practices. Among these 38 patients 17 had strains that produced β -lactamase; 12 of these 17 had been treated unsuccessfully with ampicillin. Forty three patients became infected in hospital, and in 24 of these the sputum contained strains producing β -lactamase. In January 64% of all the strains produced β -lactamase. All the isolates were sensitive to chloramphenicol, erythromycin, and co-trimoxazole. A retrospective study of 50 strains of *B catarrhalis* (stored at -70°C) showed that all were sensitive to clavulanic acid and amoxycillin (Augmentin) and to cefuroxime but resistant to trimethoprim.

Number of patients per month with *B catarrhalis* bronchopulmonary infection, January 1981 to April 1983.

Discussion

Most strains of *B catarrhalis* are commensals, but well documented reports confirm that some are potential pathogens, and latterly attention has been drawn to their capacity for producing β -lactamase.³ This β -lactamase has minimal cephalosporinase activity and is inactivated by clavulanic acid.⁸ Its production is due to a chromosomal determinant, is not plasmid associated, and is therefore not transferable.³ Our bacteriology laboratory has always reported *B catarrhalis* in sputum as a potential pathogen when there are numerous pus cells with extraleucocyte and intraleucocyte Gram negative diplococci, a moderate or profuse growth of *B catarrhalis*, and no other known respiratory pathogens. The results from culture together with the Gram film assist greatly in the clinical assessment of whether antibiotic treatment should be started. Transtracheal aspiration of sputum to prevent oropharyngeal contamination is uncomfortable and, in our experience, unnecessary.⁹

Initially, we ignored reports of *B catarrhalis* until it became apparent that patients' symptoms did not respond to ampicillin and that no other pathogens had been isolated. Moreover, patients admitted with *B catarrhalis* infection had usually not responded to a course of ampicillin but were soon better with erythromycin or co-trimoxazole. More disturbing was the development of purulent sputum in inpatients already taking ampicillin, from whom strains of *B catarrhalis* producing β -lactamase were almost inevitably isolated. This striking increase in the incidence of isolation of *B catarrhalis* is partly explained by the high proportion of patients who became infected on the wards, a feature of *B catarrhalis* infection not previously documented. There was also an unexplained increase in the community, which perhaps may be due to local changes in antibiotic prescribing habits in general practice and hospital practice leading to the growth of resistant, more virulent

strains. The true incidence of *B catarrhalis* is unknown but is probably underreported. In our experience the concurrence of other pathogenic bacteria such as *H influenzae* or *S pneumoniae* with *B catarrhalis* is uncommon. As *B catarrhalis* is a potential pulmonary pathogen capable of producing β -lactamase it should be reported both in pure culture and when mixed with other pathogens, as not only may an inappropriate antibiotic be prescribed but the β -lactamase may protect the other pathogenic bacteria from the action of ampicillin.

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A 53 year old woman has a 10 year history of the sicca syndrome—dry mouth and minimal dryness of the eyes—but no other part of the body is affected. In the past year she has had three attacks of parotitis affecting both parotid glands and associated with a high fever and exacerbation of the dryness of the mouth. This has gradually responded to either penicillin or a tetracycline. Can a further attack be prevented? Is there any treatment to encourage the flow of saliva to relieve dryness of the mouth?

Sicca syndrome is a widespread clinical problem, most frequently recognised as dryness or irritation in the eyes but also affecting other mucus producing surfaces in the body, including the mouth (leading to dental caries and difficulty in swallowing), trachea and larynx, oesophagus, vagina, and bronchial tree. Although the dry eyes are usually helped by methyl-cellulose eye drops ("artificial tears"), treatment of the dry mouth is at best unsatisfactory. Of all the sialogogues advocated, none is wholly satisfactory. Pure lemon juice is worth trying, if only because of convenience. In severe cases parotid dryness leads to recurrent infection and sialectasis. Little can be done to prevent attacks other than to maintain adequate hydration and oral hygiene—especially when surgery is carried out. Parotid calculi may require surgery. Sicca syndrome is also a feature of about 30% of patients with various connective tissue diseases such as rheumatoid arthritis. Furthermore, it may be the only clinical feature of these diseases, manifest, for example, in relatives of patients with connective tissue diseases or subclinically as hyperglobinaemia, raised erythrocyte sedimentation rate, positive rheumatoid factor, anaemia, or leucopenia. The risks of such individuals developing more serious disease are not clearly known, and it is doubtful whether treatment at this stage would be beneficial—GRAHAM R V HUGHES, consultant physician and reader in rheumatology, London.