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Case Report

Bacteraemic pneumonia caused by *Neisseria lactamica* with reduced susceptibility to penicillin and ciprofloxacin in an adult with liver cirrhosis

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This report presents a case of bacteraemic pneumonia caused by *Neisseria lactamica* in an adult patient with liver cirrhosis who was successfully treated with ceftriaxone. The isolate was confirmed as *N. lactamica* by analysis of a partial sequence of the 16S rRNA gene; it had reduced susceptibilities to penicillin (MIC 0·75 μg ml^{-1}) and ciprofloxacin (MIC ⩾0·5 mg l^{-1}).

Introduction

Neisseria lactamica is a commensal species colonizing the human upper respiratory tract, and it shares this ecological niche with *Neisseria meningitidis* (Kremastinou *et al.*, 2003; Saez-Nieto *et al.*, 1985). It frequently colonizes the nasopharynx of young children (Kremastinou *et al.*, 2003; Saez-Nieto *et al.*, 1985). *N. lactamica* exhibits a higher possibility of colonization in young children than does *N. meningitidis*, passing more easily from child to child, with a shorter duration of carriage (Kremastinou *et al.*, 2003; Saez-Nieto *et al.*, 1985; Alber *et al.*, 2001). Previous reports of infections (meningitis, bacteraemia and otitis media) caused by *N. lactamica* have almost exclusively been in young children. The only previous report involving an adult was in a patient who developed meningitis following a skull trauma (Denning & Gill, 1991). None of these infections was caused by isolates with reduced susceptibility to penicillin. To our knowledge, this is the first report of an adult patient with community-acquired pneumonia and bacteraemia due to a penicillin-resistant *N. lactamica* strain.

Case report

A 42-year-old man had a four-year history of hepatitis B virus-associated Child C liver cirrhosis. He had been observed to have ascites 2 years previously, and had suffered three episodes of oesophageal bleeding, in December 2000, March 2003 and February 2004. At that time, his condition was stable, without any infection or recurrence. His previous chest radiograph was clear, although he was a smoker, smoking one pack per day for 20 years. However, he had developed a productive cough for 1 week and fever (up to 38·4 °C) 2 days prior to admission in December 2004. His white blood cell (WBC) count on admission was $10{\cdot}62 \times 10^9$ l^{-1} (neutrophils 88·2 %, lymphocytes 7·0 %) and C-reactive protein was 2·58 mg dl^{-1}. Chest radiograph showed infiltration and pleural effusion over the right lower lung. Two sets of blood cultures, including two aerobic and two anaerobic bottles (BACTEC 9240, Becton Dickinson) collected on the day of admission, all grew Gram-negative diplococci. Due to suspicion of meningococcal septicaemia and pneumonia, the patient was placed in an isolation room and ceftriaxone (1 g every 12 h) was administered intravenously. All individuals in close contact with the patient, including his wife and daughter, physicians and nurses, received rifampicin prophylaxis (600 mg every 12 h for 2 days). Throat-swab cultures and pleural-effusion specimens collected 2 days after starting ceftriaxone were both negative. A Gram-stained smear of the pleural-effusion specimen revealed a few intracellular Gram-negative diplococci. His fever subsided 3 days after starting ceftriaxone, and the treatment was continued for a 14-day course with an uneventful recovery. A chest radiograph showed improvement, except for a persistent moderate right-pleural effusion, which was suspected to be an ascites-related pleural effusion. The serial follow-up pleural effusions were all transudate. Cultures of throat-swab specimens from the patient's wife and daughter were negative for *N. lactamica*.

The organism isolated from blood culture at admission was oxidase, catalase and ONPG positive, and utilized glucose, maltose and lactose. This organism was initially misclassified as *N. meningitidis*, but was later identified as *N. lactamica* by the VITEK NHI Identification card (bioMerieux)

(identification profile 10520, 99 % identity). Partial sequencing (426 bp) analysis of the 16S rRNA gene of the isolate using the primer pair 8FPL (5′-AGAGTTTGATCCTGGCTCAG-3′) and 1492 (5′-GGTTACCTTGTTACGACTT-3′) was performed (Turenne *et al.*, 2001). The sequences were compared with published sequences in the GenBank database using the BLASTN algorithm. The closest matches were obtained with *N. lactamica* (GenBank accession no. AJ247242.2).

The isolate was negative for β-lactamase production by the cefinase disk test (Becton Dickinson). MICs of the isolate as determined by the Etest (AB Biodisk) on Mueller–Hinton agar supplemented with 5 % sheep blood were 0·75 mg l^{-1} for penicillin, 0·023 mg l^{-1} for cefotaxime, and 0·25 mg l^{-1} for ciprofloxacin. MICs of the isolate determined using the agar-dilution method were 1 mg l^{-1} for penicillin, 0·03 mg l^{-1} for ceftriaxone, and 0·5 mg l^{-1} for ciprofloxacin (National Committee for Clinical Laboratory Standards, 2005).

Our isolate had reduced susceptibility to penicillin ($\geqslant$0·5 mg l^{-1}) and ciprofloxacin ($\geqslant$0·12 mg l^{-1}) if the MIC interpretive criteria provided by the National Committee for Clinical Laboratory Standards (2005) for *N. meningitidis* were applied. However, the use of MIC criteria specific to *N. meningitidis* to interpret results for *N. lactamica* may be inappropriate. Arreaza *et al.* (2002) tested 286 isolates of *N. lactamica* obtained from two meningococcal carrier surveys and found that all isolates had penicillin MICs of 0·12–1 mg l^{-1}, and six isolates (2·1 %) had ciprofloxacin MICs of 0·12–0·5 mg l^{-1}. Mutations in the *penA* gene, or in the *gyrA* or *parC* genes, have been shown to confer resistance to penicillin or ciprofloxacin in *N. lactamica* (Arreaza *et al.*, 2002; Ito *et al.*, 2005). We did perform such sequencing on the isolate from our patient, but it was difficult to interpret the sequence data due to the lack of control strains of penicillin- and ciprofloxacin-susceptible *N. lactamica* and the fact that there are no published sequences of the *penA*, *gyrA* and *parC* genes of penicillin- and ciprofloxacin-susceptible *N. lactamica* in GenBank.

Discussion

The *N. lactamica* isolate from our patient was initially identified as *N. meningitidis*, in a similar manner to other isolates reported previously, and the patient was empirically treated with ceftriaxone. In Taiwan, colonization or infection due to *N. lactamica* has not been previously reported. In a study of *N. meningitidis* isolates causing invasive disease in Taiwan, 7 % were not susceptible to penicillin (MIC$\geqslant$0·12 mg l^{-1}), and all were susceptible to ceftriaxone (Hsueh *et al.*, 2004). Accordingly, empirical therapy for suspected invasive meningococcal disease in Taiwan should be a third-generation cephalosporin. Misidentification of the organism as *N. meningitidis* would therefore appear to have little consequence for the patient infected with *N. lactamica*. Unnecessary antibiotic prophylaxis for persons in contact with the patient and concerns about the spread of the organism in the hospital can be avoided by prompt and accurate identification of *N. lactamica*.

Previous reports of invasive *N. lactamica* infection have suggested that the upper respiratory tract is the initial portal of entry (Kremastinou *et al.*, 2003; Saez-Nieto *et al.*, 1985; Alber *et al.*, 2001; Denning & Gill, 1991). *N. lactamica* is generally considered non-pathogenic, due to the lack of 'parasite-directed endocytosis' in the non-ciliated cells of the upper respiratory tract and the absence of IgA1 protease (Denning & Gill, 1991). It is likely that the inflamed respiratory mucosa in this cirrhotic patient provided an avenue for this organism to invade lung parenchyma and enter the bloodstream.

In conclusion, we report a case of bacteraemic pneumonia caused by *N. lactamica* with reduced susceptibility to penicillin in an adult patient with liver cirrhosis. Although rare, *N. lactamica* should be considered as a potential bacterial aetiology associated with community-acquired pneumonia and bacteraemia.

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