A case of pharyngitis caused by *Streptococcus pneumoniae*

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Throat cultures from an adult pharyngitis patient yielded *Streptococcus pneumoniae* as a single organism, with a very high bacterial count. The isolate was found to be macrolide and fluoroquinolone resistant, and the same strain was cultured from the patient’s denture washing solution. Ceftriaxone therapy, a gradual reduction in the bacterial count and progressive clinical improvement proceeded at the same pace, so we labelled this clinical case as a pneumococcal pharyngitis.

Introduction

*Streptococcus pneumoniae* is known to be responsible for acute otitis media, acute sinusitis, acute bacterial exacerbations of chronic obstructive pulmonary disease, community-acquired pneumonia, acute meningitis, cellulitis, arthritis and bacteraemia (Sakata, 2005; Tan, 2002).

Case report

A 79-year-old woman suffered from a sore throat and dry cough for 2 months. Only a conservative therapy (pain control) was previously administered, so she came to our department with worsening symptoms. We recorded a very red pharynx, tonsils that were slightly enlarged and an absence of tonsillar exudate. Only two slightly enlarged and painful cervical lymph nodes were observed. The patient’s temperature had never been elevated during the previous 3 months. After obtaining a throat swab, we started azithromycin (500 mg every 24 h, for 3 days). Nevertheless, she returned with persistent symptoms, in spite of complying with the antibiotic treatment regimen (as ensured by her daughter).

Cultures grew *S. pneumoniae* (around $10^7$ c.f.u. ml$^{-1}$) as a single organism, and the isolate was found to be macrolide and fluoroquinolone resistant. Interestingly, a pneumococcal strain with the same susceptibilities to antibiotics was cultured from the water solution in which the patient’s dentures were washed.

Parenteral ceftriaxone was started (1 g every 24 h). After 1 week of therapy, a great clinical improvement was recorded, and a second culture showed a remarkable reduction in the *S. pneumoniae* bacterial count (around $10^3$ c.f.u. ml$^{-1}$). The patient made a full recovery after a 2 week therapy and no relapses were recorded within 2 months. Total eradication of the organism from the throat was also achieved. In fact, nasopharyngeal and oropharyngeal cultures performed after 7, 30 and 60 days from the end of the antimicrobial treatment were found to be negative.

Methods

Nasopharyngeal and oropharyngeal swabs (Watt *et al.*, 2004) were plated onto CNA (colistin–nalidixic acid) agar (Biolife), sheep blood agar (Biolife) and Sabouraud dextrose agar (Biolife). Sabouraud plates were incubated at 36 °C in ambient air, whereas CNA and sheep blood plates were incubated at 36 °C in ambient air, anaerobically and in an atmosphere of 5% CO$_2$. All plates were examined after 24, 48 and 72 h.

After 24 h of incubation, α-haemolytic colonies belonging to the same streptococcal species had grown, under each of the three atmospheric growth conditions. The isolate was identified as *S. pneumoniae* (Vitek2; bioMérieux), and confirmation of the identification was...
provided by inhibition of the isolate by optochin. Optochin-susceptibility testing was performed in ambient air, anaerobically and in an atmosphere of 5% CO₂, to prevent misidentification of Streptococcus pseudopneumoniae as true S. pneumoniae (Balsalobre et al., 2006).

The isolate exhibited resistance to erythromycin (MIC > 8 μg ml⁻¹), clarithromycin (MIC 8 μg ml⁻¹), ciprofloxacin (MIC ≥ 4 μg ml⁻¹) and levofloxacin (MIC ≥ 4 μg ml⁻¹), but susceptibility to penicillin (MIC ≤ 0.125 μg ml⁻¹), cefotaxime (MIC 0.06 μg ml⁻¹), ceftriaxone (MIC 0.06 μg ml⁻¹), tetracycline (MIC < 1 μg ml⁻¹) and cotrimoxazole (MIC ≤ 10 μg ml⁻¹) (Vitek2; bioMérieux).

Potential pharyngeal pathogens other than S. pneumoniae were not isolated, such as β-haemolytic streptococci, γ-haemolytic streptococci, Neisseria spp., Gemella spp., Moraxella spp., Haemophilus spp., Corynebacterium spp., strictly anaerobic organisms (particularly Fusobacterium necrophorum) and yeasts (particularly Candida albicans) (Esposito et al., 2004).

Conclusions and Discussion

Physicians often dismiss pharyngitis as viral when tonsillar exudate and fever are absent, so patients do not receive antibiotics (Gonzales et al., 2001). S. pneumoniae throat infections can have a similar presentation to viral ones and they should be suspected in patients with persistent and/or worsening sore throat and/or dry cough.

In the case reported here, S. pneumoniae was cultured as a single organism from oropharyngeal swabs. Furthermore, numerous pneumococcal colonies grew. Finally, antibiotic therapy, a gradual reduction in the S. pneumoniae bacterial count and progressive clinical improvement proceeded at the same pace. Hence, we considered S. pneumoniae as responsible for the pharyngitis case we have described.

S. pneumoniae antibiotic resistance has spread all over the world, and an increase in macrolide and fluoroquinolone resistance has also been reported. Macrolide resistance in S. pneumoniae is generally caused by the genes ermB or mefA. ermB encodes a 23S methylase, which confers resistance to 14-, 15- and 16-membered-ring macrolides, lincosamides, and streptogramin B (MLS₉ phenotype). The mefA gene encodes an efflux pump that is responsible for resistance to only 14- and 15-membered-ring macrolides (Reinert et al., 2003). Fluoroquinolone resistance in S. pneumoniae is mainly due to mutations in the QRDRs (quinolone-resistance-determining regions) of the genes (particularly parC and gyrA) encoding the A and B subunits of DNA gyrase and topoisomerase IV. Mutations to the parE and gyrB genes have been described to a lesser extent (Ip et al., 2006).

No data exist so far about the establishment of pneumococcal reservoirs on dentures. We think this possibility should always be considered, and daily accurate cleaning of orthodontic appliances is suggested, to avoid the establishment of a pneumococcal carrier state due to denture colonization. Furthermore, S. pneumoniae cells surviving in toothbrushes or any orthodontic appliances probably come into contact with submaximal antibiotic concentrations and this could contribute to the selection of resistance (Carbon & Istruritz, 2002; Johnston et al., 1998; Reinert et al., 2003).

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References


