Case Report

A case of pharyngitis caused by *Streptococcus pneumoniae*

Vincenzo Savini,¹ Marco Favaro,² Carla Fontana,² Nicola Pietro Consilvio,³ Assunta Manna,¹ Marzia Talia,¹ Chiara Catavittello,¹ Andrea Balbinot,¹ Fabio Febbo,¹ Giovanni Di Bonaventura,⁴ Nicola Di Giuseppe⁵ and Domenico D'Antonio¹

Correspondence
Vincenzo Savini
vincsavi@yahoo.it

¹Unità Operativa Complessa di Microbiologia e Virologia Clinica, Dipartimento di Medicina Trasfusionale, Ospedale Civile Spirito Santo, Pescara, Italy

²Dipartimento di Microbiologia Clinica, Policlinico Torvergata, Roma, Italy

³Clinica Pediatrica, Ospedale Clinicizzato SS Annunziata, Chieti, Italy

⁴Laboratorio di Microbiologia Clinica, Centro Studi Invecchiamento (Ce.S.I.), Università Degli Studi Gabriele D'Annunzio, Chieti, Italy

⁵Dipartimento di Otorinolaringoiatria e Chirurgia Cervico-Facciale, Ospedale Civile Spirito Santo, Pescara, Italy

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.

Received 19 September 2007
Accepted 15 January 2008

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% CO₂. 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 (MLS_B 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 & Isturitz, 2002; Johnston *et al.*, 1998; Reinert *et al.*, 2003).

Acknowledgements

The authors are grateful to colleagues at the Clinical Microbiology Laboratories, Policlinic of Tor Vergata, Rome, Italy, and to Mrs Annarita Perfetti (Clinical Microbiology and Virology Complex Operating Unit, Department of Transfusion Medicine, Spirito Santo Hospital, Pescara, Italy) for their precious aid.

References

- Balsalobre, L., Hernandez-Madrid, A., Llull, D., Martin-Galiano, A. J., Garcia, E., Fenoll, A. & de la Campa, A. G. (2006). Molecular characterization of disease-associated streptococci of the mitis group that are optochin susceptible. *J Clin Microbiol* **44**, 4163–4171.
- Carbon, C. & Isturitz, R. (2002). Narrow versus broad spectrum antibacterials: factors in the selection of pneumococcal resistance to beta-lactams. *Drugs* **62**, 1289–1294.
- Esposito, S., Blasi, F., Bosis, S., Droghetti, R., Faelli, N., Lastrico, A. & Principi, N. (2004). Aetiology of acute pharyngitis: the role of atypical bacteria. *J Med Microbiol* **53**, 645–651.
- Gonzales, R., Bartlett, J. & Besser, R. (2001). Principles of appropriate use for treatment of acute respiratory tract infections in adults: background, specific aims, and methods. *Ann Intern Med* **134**, 479–486.
- Ip, M., Chau, S. S., Chi, F., Qi, A. & Lai, R. W. (2006). Rapid screening of fluoroquinolone resistance determinants in *Streptococcus pneumoniae* by PCR-restriction fragment length polymorphism and single-strand conformational polymorphism. *J Clin Microbiol* **44**, 970–975.
- Johnston, N. J., De Azavedo, J. C., Kellner, J. D. & Low, D. E. (1998). Prevalence and characterization of the mechanisms of macrolide, lincosamide, and streptogramin resistance in isolates of *Streptococcus pneumoniae*. *Antimicrob Agents Chemother* **42**, 2425–2426.
- Reinert, R. R., Luetticken, R., Brysker, A. & Al-Lahham, A. (2003). Macrolide-resistant *Streptococcus pneumoniae* and *Streptococcus pyogenes* in the pediatric population in Germany during 2000–2001. *Antimicrob Agents Chemother* **47**, 489–493.
- Sakata, H. (2005). Study of *Streptococcus pneumoniae* bacteremia in children. *Kansenshogaku Zasshi* **79**, 1–6.
- Tan, T. Q. (2002). Update on pneumococcal infections of the respiratory tract. *Semin Respir Infect* **17**, 3–9.
- Watt, J. P., O'Brien, K. L., Katz, S., Brondson, M. A., Elliott, J., Dallas, J., Perilla, M. J., Reid, R., Murrow, L. & other authors (2004). Nasopharyngeal versus oropharyngeal sampling for detection of pneumococcal carriage in adults. *J Clin Microbiol* **42**, 4974–4976.