Pseudomonas Pneumonia in Smokers

Sir—Hatchette et al. [1] described a previously healthy cigarette smoker with Pseudomonas aeruginosa community-acquired pneumonia who died 36 h after admission to the hospital. Their search of the medical literature published since 1960 revealed 10 other cases of Pseudomonas pneumonia in otherwise healthy subjects, 5 of whom were smokers. The authors concluded that Pseudomonas species should be considered an etiologic agent in anyone with a history of smoking who has rapidly progressive pneumonia.

Clinical investigators should be wary of making such recommendations, and editors should be wary of printing them. First, leaving aside the platitude that physicians should always consider every diagnosis, the recommendation is not supported by the data. The number of smokers and nonsmokers with P. aeruginosa community-acquired pneumonia who were reported in the literature was the same, and mention of 6 cigarette smokers with rapidly progressive pneumonia in 40 years of medical literature (0.15 patients per year) might as well lead physicians not to consider the diagnosis. Second, the recommendation encourages abuse of antibiotics (e.g., inclusion of an antibiotic to “cover” Pseudomonas species in cases of community-acquired pneumonia). Third, such recommendations have a way of serving as grist for the mill of malpractice attorneys when rare cases do occur subsequently.

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Reference

Bacteremia and Meningitis Caused by a Macrolide-Sensitive Strain of Streptococcus pneumoniae during Treatment with Azithromycin

Sir—Kelley et al. [1] describe 4 patients being treated with azithromycin or clarithromycin who presented with breakthrough bacteremia caused by pneumococci exhibiting low-level resistance to macrolide antibiotics. We have recently treated a patient who presented with breakthrough bacteremia and meningitis caused by a macrolide-sensitive strain of Streptococcus pneumoniae while she was receiving treatment with azithromycin.

A previously healthy woman aged 65 years consulted her general physician after 2 days of cough, fever, and malaise. During the previous year, she had experienced tenderness of the left ear region in association with episodes of the common cold. Her condition was diagnosed as tracheobronchitis, and treatment was initiated with azithromycin at a dosage of 500 mg once daily. During the following 36 h, the patient experienced a headache of increasing intensity and nausea, and she was admitted to the hospital with a diagnosis of meningitis.

At the time of admission, the patient was awake, febrile, and had nuchal rigidity, but there were no clinical signs of septicemia. The leucocyte level was 20.1 × 10^9 cells/L (neutrophil count, 17.2 × 10^9 cells/L), and the C-reactive protein level was 364 mg/L (reference level, <10 mg/L). Analysis of CSF revealed a leukocyte count of 8.8 × 10^3 cells/L (97% neutrophils), a glucose concentration of 2.6 mM (serum glucose concentration, 6.7 mM), and an albumin concentration of 1.20 g/L. Microscopic analysis of a CSF sample revealed neutrophil pleocytosis and gram-positive diplococci. Findings of lung and heart stethoscopy were normal, and a thoracic radiograph also appeared normal; clinical examination by a specialist in otology the next day revealed no signs of mastoiditis or otitis media.

Immediately after the lumbar puncture was performed, treatment was initiated with iv penicillin G at a dosage of 3 × 10^6 IU 6 times per day combined with iv ceftriaxone at a dosage of 6 g once daily. Following the acute microscopic examination of a CSF sample, the treatment was changed to iv penicillin G at a dosage of 3 × 10^6 IU 6 times per day. Eighteen hours later, S. pneumoniae had grown on all culture media. Corresponding blood specimens that were obtained after the lumbar puncture but before initiation of iv therapy—and during treatment with azithromycin—yielded growth of S. pneumoniae in 2 of 2 culture flasks. The 2 isolates recovered from CSF fluid and blood had identical resistance patterns; both were fully susceptible to penicillin G (MIC, 0.016 mg/L), ceftriaxone (MIC, 0.012 mg/L), and erythromycin (MIC, 0.19 mg/L). Therefore, the pneumococci were able to grow both in the patient and in the laboratory despite being fully susceptible to azithromycin, the agent that the patient was receiving at the time when specimens were obtained for culture.

Aside from an asymmetric left-side hearing loss, the patient experienced complete clinical recovery and was discharged after completing 10 days of treatment with iv penicillin G. Azithromycin treatment of this patient failed to eliminate a macrolide-sensitive strain of S. pneumoniae, failed to prevent it from causing bacte-
remia, and failed to prevent it from infecting the meninges. This could be due to the low serum concentrations achieved during treatment with azithromycin. Due to the risk of treatment failure and further complications, azithromycin should not be chosen for empiric treatment of severe infections in which \( S. \) pneumoniae might be the etiologic agent.

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