what the patients need—train students to treat patients, not the rare diseases seen in medical centers.

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REFERENCE

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Dr Cesario Responds

TO THE EDITOR: Dr Taylor and I have no disagreement. My comment regarding clinical judgment and cost-effectiveness was made to indicate that I think they are mutually compatible. They have, in fact, similar implications merely viewed from two different perspectives—hence, opposite sides of the same coin.

Nor do I have any disagreement with the ability of family practitioners as excellent teachers and role models. I appreciate the many fine teachers who are practitioners of family medicine and am eager to include practicing physicians in our training programs because they constitute important role models for students.

I would, however, strongly disagree with the notion that internal medicine has been reinventing the wheel. We have sought to maintain standards of academic credibility and have done critical investigations when necessary to yield new knowledge on which clinical judgment or educational methods could be based.

It is not practical under the current system to suggest that many physicians can still competitively do clinical work, research, and instruction all at the same time when the competition for research dollars has become so fierce. The demands of the clinic are such now that considerable time is necessary to satisfy the expectations of practice groups and of patients. This is not to say that it is impossible for practitioners to do good research or to participate in outstanding projects. What is difficult, however, is for them to constantly maintain the funding and the support necessary to pursue the type of research that will yield essential new information to advance our understanding of the functions, the diseases, and the therapies needed to heal the human body.

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Capnocytophagal Pneumonia in a Healthy Man

TO THE EDITOR: *Capnocytophaga* species (DF-1) are facultatively anaerobic, slender gram-negative rods found as part of the oral flora. Serious capnocytophagal infections usually occur in immunocompromised patients.[1] We report a case of capnocytophagal pneumonia in an otherwise healthy young man.

Report of a Case

Malaise, sore throat, and nonproductive cough developed in a 25-year-old sailor. The patient's cough worsened, and severe paroxysms of coughing developed, followed by emesis and gasping respirations. Subsequently, he had shortness of breath at rest, fever to 38.9°C, and produced purulent sputum. On the third day of his illness, the patient was admitted to the sick bay of his ship with clinical signs of pneumonia, and empiric treatment with ceftriaxone, 1 gram every 12 hours, was initiated.

The patient was transferred to our facility four days after the onset of his illness. He appeared ill and had a temperature of 39.7°C. Bronchial breath sounds were present in the posterior basal lung fields. With the patient breathing room air, blood gas measurements showed an arterial pH of 7.48, a P_{O_2} of 50 mm of mercury, and a P_{CO_2} of 33 mm of mercury. A chest x-ray film showed diffuse interstitial and alveolar infiltrates with lower lobe predominance. Gram's stain of sputum revealed sheets of neutrophils and numerous slender, fusiform, gram-negative rods.

Therapy was initiated with the combination drug ticarcillin disodium and clavulanate potassium, 3.1 grams every four hours; tobramycin sulfate, 5 mg per kg of body weight per day; and erythromycin, 1 gram every six hours. Three sputum specimens obtained for culture during the first two days of hospital admission grew the *Capnocytophaga* species (DF-1). The *Capnocytophaga* organism was strongly β-lactamase-producing by Cefinase disc testing (BBL Cefinase, Becton Dickinson, Cockeysville, Maryland), and antibiotics were changed to the combination product ampicillin sodium and sulbactam sodium. The fever subsided over the next three days, and antibiotics were changed to an oral combination of amoxicillin and clavulanate potassium. His cough and hypoxemia resolved, and he was discharged on the fifth hospital day.

Discussion

Capnocytophaga species (DF-1) are unusual causes of infection in immunocompetent hosts. In a review of 16 cases of capnocytophagal infections in immunocompetent persons, most of the patients had serious underlying illnesses including malignancy, trauma, or surgical therapy, chronic renal insufficiency, and cholangitis.[1] Many of the patients were elderly, and multiple organisms were common. An oral source of infection was implicated in these patients.

We postulate that our patient initially had an upper respiratory tract infection, and then aspiration pneumonia developed as a complication of severe coughing spasms and posttussive emesis. Subsequently, ceftriaxone selected for the β-lactamase-producing *Capnocytophaga* species. The failure of a third-generation cephalosporin to eradicate β-lactamase-producing *Capnocytophaga ochracea* has been documented in a neutropenic patient.[2]

A recent report by Roscoe and Clarke documented the substantial frequency of β-lactamase-resistant *Capnocytophaga* species in a Canadian hospital. Of 19 isolates in their series, 6 were β-lactamase producing. Several investigators have suggested that because of the increasing frequency of β-lactamase-positive organisms, standard β-lactam agents may not be adequate therapy for oral infections in neutropenic patients.[2,3]

Our case shows that β-lactamase-producing strains can produce clinically important infection in normal hosts and should be considered in the treatment of community-acquired aspiration pneumonias. Therapy with β-lactam β-lactamase inhibitor combination agents is effective in these patients.

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3. Roscoe D, Clarke A: Resistance of *Capnocytophaga* species to β-lactam antibiotics (Letter). Clin Infect Dis 1993; 17:284-285

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