CLINICAL VIGNETTE

Capnocytophaga Bacteremia and Sepsis in an Immunocompetent Adult

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Case

A 70-year-old male with chronic obstructive pulmonary disease (COPD), and allergic bronchopulmonary aspergillosis presented to the emergency department (ED) with fever, chills, dyspnea, and cough. He was tachypneic and tachycardic. Vital signs included temperature of 38.4 ℃, heart rate of 125 bpm, blood pressure of 154/94 mmHg, and respiratory rate of 30 and room air oxygen saturation was 96%. Physical examination was notable for tachycardia and end-expiratory wheezing but was otherwise benign. Laboratories were significant for a white blood cell count of 13,000/uL (normal 4,000 – 10,000/uL) and a procalcitonin of 0.3 ug/L (normal <0.1 ug/L). Chest x-ray demonstrated bilateral opacities. The patient was admitted and treated for sepsis, COPD exacerbation and presumed pneumonia. He was started on intravenous vancomycin, piperacillin-tazobactam, and fluids, steroids, and nebulizers. He rapidly improved and was discharged the following day with instructions to complete a five-day course of amoxicillin-clavulanate and prednisone.

The day following discharge, the patient's initial blood cultures resulted positive for gram negative rods, and he was instructed to return to the ED. In the ED, the patient was asymptomatic with normal vitals and physical examination. Laboratories were notable for a white blood cell count of 23,000/uL. Repeat blood cultures were drawn, prednisone was discontinued, and the patient was restarted on intravenous piperacillin-tazobactam. The initial blood cultures grew Capnocytophaga canimorsus. Upon obtaining further history, the patient disclosed that four days prior to his initial presentation, he had been bitten on his finger by a dog. He developed a bloody fungal lesion that lasted three days but resolved prior to his initial ED presentation. The blood cultures from his second presentation remained negative, so the Capnocytophaga causing his infection was assumed to be beta-lactam sensitive. He was discharged with instructions to complete a fourteen-day course of amoxicillin-clavulanate. Susceptibility testing confirmed beta-lactamase sensitivity. Following completion of antibiotic therapy, he continued to be asymptomatic, and was doing well at one year follow-up.

Discussion

Capnocytophaga species are slow-growing, facultative, anaerobic, gram-negative bacilli.[1] Certain Capnocytophaga species are part of the normal human microbiota whereas others, such as Capnocytophaga canimorsus, are commensal to the oral cavity of dogs and cats.[2] Human infection with Capnocytophaga species is rare, with 0.5-0.7 cases per million per year.[3] Previously case reports of infection by this species include bacteremia, meningitis, endocarditis, soft-tissue infection, ocular infection, mycotic aneurysm, and septic arthritis.[4-7]

As with our case, Capnocytophaga canimorsus infection is typically associated with dog bites. In one study, 68% of patients with Capnocytophaga canimorsus bacteremia had a preceding animal bite or scratch.[8] Infections with other Capnocytophaga species that normally live in the human oral cavity have also been documented in immunocompromised patients. Immunocompromised patients, including those with neutropenia, asplenia, alcohol use disorder, and cirrhosis, are at increased risk of Capnocytophaga infection.[6,8] Sepsis and septic shock due to Capnocytophaga is more likely to be seen in immunocompromised patients, but can rarely occur in immunocompetent patients, as demonstrated in our patient.

A previous case series reported 30% of Capnocytophaga species had beta-lactamase activity. These were associated with infection with species normally found in the human oral cavity.[8] Zoonotic infection with Capnocytophaga canimorsus tends to be beta-lactam sensitive. Therefore, Capnocytophaga canimorsus can typically be treated with penicillin. If there is concern for beta-lactamase production, empiric treatment with a combination beta-lactam/beta-lactamase-inhibitor or carbapenem should be utilized. Duration of treatment should be determined by the primary infection. Our patient's bacteremia was presumed to have been due to an initial soft tissue infection following the dog bite. Therefore, a fourteen-day course of therapy was sufficient.

Given the slow growth of Capnocytophaga on culture, diagnosis may be delayed. In the case presented, respiratory signs and symptoms and otherwise negative infectious workup led to initial treatment for pneumonia and COPD exacerbation. His respiratory findings were likely chronic, and the true diagnosis was not established until his blood cultures resulted and further history was obtained. This demonstrates the importance of following blood cultures for growth of fastidious organisms. This case also highlights the importance of thorough history-

taking and maintaining a broad differential to establish an accurate diagnosis.

Cognitive biases likely contributed to the initial diagnostic error. For example, anchoring bias, which is the tendency to focus on certain features of a patient's initial presentation and failure to adjust this impression as more data is obtained,[9] played a role. Given the patient's significant history of pulmonary disease and respiratory symptoms upon his presentation, he was favored to have a pneumonia and COPD exacerbation. This remained the leading diagnosis despite evidence that the patient did not have an acutely worsened cough or sputum production. We can combat anchoring bias by maintaining a broad differential and re-examining the patient's history and objective findings throughout the disease process.

In summary, Capnocytophaga rarely causes human infection, but should be considered in patients presenting with infectious symptoms following dog bites. Empiric therapy with combination beta-lactam/beta-lactamase inhibitors, such as amoxicillin-clavulanate, should be initiated. Though more common in immunocompromised patients, severe Capnocytophaga infection causing sepsis and even septic shock can occur in immunocompetent patients.

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