Capnocytophaga Canimorsus Septicemia Caused by a Dog Bite in an Asplenic Patient

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Introduction

Capnocytophaga canimorsus is a gram-negative bacillus, non-spore-forming, facultative aerobe that causes a zoonotic disease, most commonly in asplenic patients.[1] This organism has a long, fusiform appearance on gram stain. It is a member of the normal gingival flora of dogs and cats and can cause fulminant sepsis with disseminated intravascular coagulation (DIC). About 1.6 to 16% of bite wounds inflicted by dogs become infected.[2] Pasteurella multocida frequently is isolated from such wounds.[3,4] Occasionally, *Capnocytophaga canimorsus* also is isolated.[3] Splenectomy, alcoholism, and chronic lung disorder are significant risk factors.

Case Report

A 45 year-old Caucasian man presented to an outside facility with two days of abdominal pain, nausea, vomiting, shortness of air, and fever. He was anxious and in moderate distress. He required intubation secondary to tachypnea and hypoxia, then was transferred to our hospital.

The patient had been in Montana with his girlfriend for the prior three weeks. He had two abscessed teeth extracted while in Montana. He also had a tick bite and a dog bite on the right arm while in Montana (the dog was current on his immunizations).

The patient had a history of hepatitis C with a splenectomy four years prior secondary to idiopathic thrombocytopenic purpura (ITP). He was a heavy smoker and drinker with history of drug use, but no IV drugs. He enjoyed hunting and fishing, and had been fishing frequently.

On admission to our hospital, the patient was intubated. His temperature was 99.4^{o} F, pulse 130 bpm, respiratory rate 15 per minute, and blood pressure 65/40 mmHg. His oxygen saturation was 95% on the ventilator with assisted-controlled mode and the fraction of inspired oxygen was 100%.

The patient was confused and mildly agitated. He had a flushed face, poor oral hygiene, black eschar around his lips, and some icteric sclera. His heart sounds were regular and lungs were coarse to auscultation bilaterally. His abdomen was nontender. There were multiple tattoos on the extremities, trace edema, purplish discoloration above the umbilicus, arms, legs, feet, palms, and soles, with superimposed maculopapular rash. There was also an eschar on the right arm with mild ecchymosis located at the site of the previous dog bite.

His laboratory studies showed a white blood cell (WBC) count of 14900 with 26% bands, 63% segmented, 11% lymphocytes, hemoglobin 16.3mg/dl, and platelets 21000. His sodium level was 135 mEq/L, potassium 3.4 mEq, bicarbonate 21.1 mmol/L, glucose 110 mg/dl, blood urea nitrogen 21 mg/dL,

and creatinine 1.8 mg/dl. His transaminases were normal.

The patient was admitted to the intensive care unit, maintained on the ventilator, and started on piperacillin/tazobactam and pressors including norepinephrine and dopamine. On hospital day 2, he remained intubated, but was more alert and awake. He was febrile with a temperature of 101° F and had left-sided weakness. His WBC increased to 41000 with 53% bands. His total bilirubin was 2.0 mg/dL, AST 2515 U/L, ALT 946 U/L, alkaline phosphatase 75 U/L, and INR 1.7. A CT scan of the head showed no acute abnormalities. Antibiotics were changed to ampicillin/sulbactam, ceftriaxone, and doxycycline to cover skin infection and dog bite.

On hospital day 3, the pressors were titrated off. The patient had hemorrhagic and purpuric blisters on the hands and feet. A peripheral blood smear showed fusiform extracellular rods. Blood cultures were still negative.

Blisters and bruising worsened on hospital day 5 and extended to the arms and legs. Tick-borne serology was negative. The patient remained intubated on hospital day 6. His hands and feet became necrotic and required amputation. He underwent a bilateral below knee amputation. No overall change in patient's outlook occurred and the family agreed to comfort care. The patient died few days later. A polymerase chain reaction test was back weeks later and was positive for *Capnocytophaga canimorsus*.

Discussion

Capnocytophaga canimorsus, formerly called dysgenic fermenter 2 (DF-2), was first described in 1976.[5,6] The current name was given in 1989 and is based on the carbon dioxide requirement (capnocyto-phaga means "eater of carbon dioxide") and usual vector of transmission (canimorsus means "dog bite").

Capnocytophaga canimorsus, an anaerobic non-spore forming gram-negative rod, rarely but regularly, has been isolated from dog or cat bite infections (Figure 1). This organism has a long, fusiform appearance on gram stain (Figure 2). It can cause fulminant sepsis with disseminated intravascular coagulation (DIC), meningitis, endocarditis, acral gangrene, disseminated purpura, and rare ocular infections.[6,7] Persons at increased risk of developing *C. canimorsus* infections include patients who have undergone a splenectomy, are immunosuppressed, and those who abuse alcohol.[6] More than 40% of the patients have no obvious risk factors.

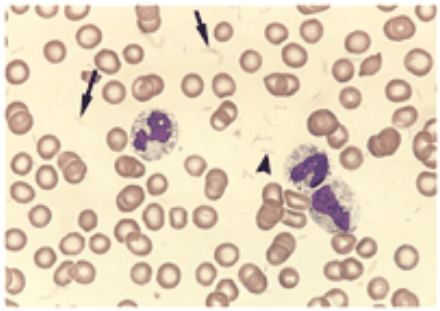

Figure 1. Peripheral blood smear showing fusiform rods 2-4 um in length (arrows) and in pairs (arrow head) mostly extracellular. (Used with permission.[8])

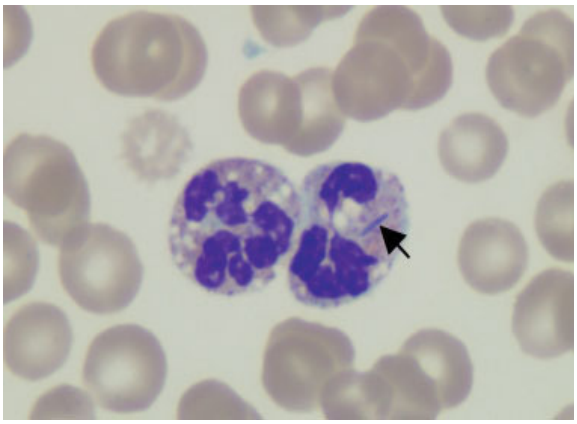

Figure 2. Wright-Giemsa stain, x100 oil immersion, showing intracellular elongated rod (arrow). (Used with permission.[9])

The clinical presentation of *C. canimorsus* infection usually involves sepsis. However, there are other clinical findings frequently present.[7,10] A maculo-papular rash is present in 13% of cases and is often purpuric (in 37%), with erythema multiforme also being described in several cases. Disseminated intravascular coagulation commonly has been associated with *C. canimorsus* septicemia (34 to 36% of cases).[7,10] Gangrenous involvement of the bite site and other areas are encountered in 15% of cases. It can involve digits, as well as entire limbs. As in our patient, gastrointestinal complaints including abdominal pain (26%), vomiting (31%), and diarrhea (26%) are commonly associated with C. canimorsus infection.[10]

Although *C. canimorsus* frequently is present in dog bite wounds, it rarely results in clinical infections.[11] This is most likely due to its slow growth, low virulence, and susceptibility to antibiotics frequently used for post-dog bite prophylaxis. Therefore, most cases of systemic infection appear in immunocompromised patients.

In one comprehensive review, 33% of systemic infections occurred in asplenic patients, 24% in alcoholics, and 5% in other immunocompromised patients.[7] Almost 41% of infections occurred in patients without any known risk factor.[7] While *C. canimorsus* has low virulence, it has a high mortality rate once systemic infection has developed. Persons with risk factors such as a history of asplenia, alcoholism, or hematologic malignancy receive antibiotic prophylaxis following animal bites.[12]

C. canimorsus infection classically is treated with penicillin G.[13] However, the increasing prevalence of beta-lactamase-producing strains of *Capnocytophaga* warrant broader first-line coverage with antibiotics such as beta-lactam/beta-lactamase inhibitors. *Capnocytophaga* is typically resistant to aminoglycosides and narrow-spectrum cephalosporins. Other active antibiotics for *C. canimorsus* include imipenem, doxycycline, rifamycin, ofloxacin, ciprofloxacin, erythromycin, and clindamycin.[13]

In conclusion, this case of fatal *C. canimorsus* emphasized that the features of a dog bite coupled with a preexisting condition of splenectomy should alert physicians to suspect this unusual organism. Furthermore, all asplenic patients after a dog bite should undergo antibiotic prophylaxis with amoxicillin/clavulanate. Since these bacteria grow slowly, laboratories also should be alerted to its potential presence, since it otherwise would be discarded as a contaminant or misidentified. Finally, physicians should inform patients with splenectomy or other immunocompromising conditions that dog ownership or bite are important risk factors for *C. canimorsus* infection.

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