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Being Licked by a Dog Can Be Fatal: *Capnocytophaga canimorsus* Sepsis with Purpura Fulminans in an Immunocompetent Man

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ABSTRACT

Bite infections caused by *Capnocytophaga canimorsus* are rare. Severe and fatal infections are more frequently reported in patients with immunodeficiency, splenectomy or alcohol abuse. We describe the case of a 63-year-old man who developed flu-like symptoms and presented after some delay with severe sepsis and purpura fulminans. He was found to be infected with *C. canimorsus* without a bite injury and did not demonstrate immunodeficiency or any other typical predisposition. Despite extensive intensive care, his conditions deteriorated and he died from multiorgan failure.

LEARNING POINTS

- Pet owners with banal, for instance flu-like, symptoms should urgently seek medical advice when symptoms are unusual.
- *Capnocytophaga canimorsus* infection should be considered and empirical antibiotic therapy immediately started or adjusted in the presence of purpura fulminans in the absence of animal bites or immunodeficiency.

KEYWORDS

Capnocytophaga canimorsus, immunocompetent, purpura fulminans, sepsis

CASE DESCRIPTION

A 63-year-old previously healthy man presented with a 3-day history of fever and progressive dyspnoea. The day before he had developed facial petechiae, dysaesthesia of his right lower limb and myalgia of the lower extremities. He had been touched and licked, but not bitten or injured, by his dog, his only pet, in previous weeks. There was no hospitalisation or recent travel abroad. On examination, in addition to petechiae there were ecchymoses over the lower limbs but no open wounds. The patient was conscious and febrile (39°C), with dyspnoea at rest and a respiratory rate of 36/min, hypoxic (percutaneous oxygen saturation level 70%) and anuric but haemodynamically stable. He did not have a headache, neck stiffness or other symptoms of meningitis. Initial laboratory results showed thrombocytopenia (20×10^3/µl), lymphocytopenia (0.2×10^3/µl), elevated procalcitonin (>100 ng/ml) and CRP (205 mg/l). The partial thromboplastin time was >180 s, and the International Normalized Ratio was 3.51. The patient had acute kidney injury (creatinine 3.4 mg/dl, BUN 116 mg/dl) and signs of liver dysfunction (AST 438 U/l, ALT 142 U/l, GGT 991 U/l, bilirubin 3.96 mg/dl) as well as rhabdomyolysis (creatine kinase 1556 U/l). Arterial blood gases revealed lactic acidosis (pH 7.217, lactate 12.8 mmol/l). After the patient was referred to the ICU, an initial diagnosis of severe sepsis with purpura fulminans was made and he was immediately treated with clarithromycin and piperacillin/tazobactam to cover *Streptococci*, *Neisseria meningitidis*, *Haemophilus influenza* and *Staphylococcus aureus*, and with ceftriaxone due to the differential diagnosis of leptospirosis.

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A CT scan of the abdomen and thoracic cavity showed no sign of infection or pathological conditions.
The patient's condition deteriorated over the next 30 hours. He developed encephalopathy and paralytic ileus. Purpura and renal and liver failure progressed (*Figs. 1 and 2*). He experienced a cardiac arrest, but was successfully resuscitated, and then intubated and mechanically ventilated. Severe hypotension persisted, so we proceeded with norepinephrine (0.03 µg/kg/h), hydrocortisone (200 mg/24 h), dobutamine (0.125 µg/kg/h) and bicarbonate. The patient received erythrocyte (8 units) and platelet (2 units) concentrates, and 8 units of fresh frozen plasma. We initiated haemodialysis.

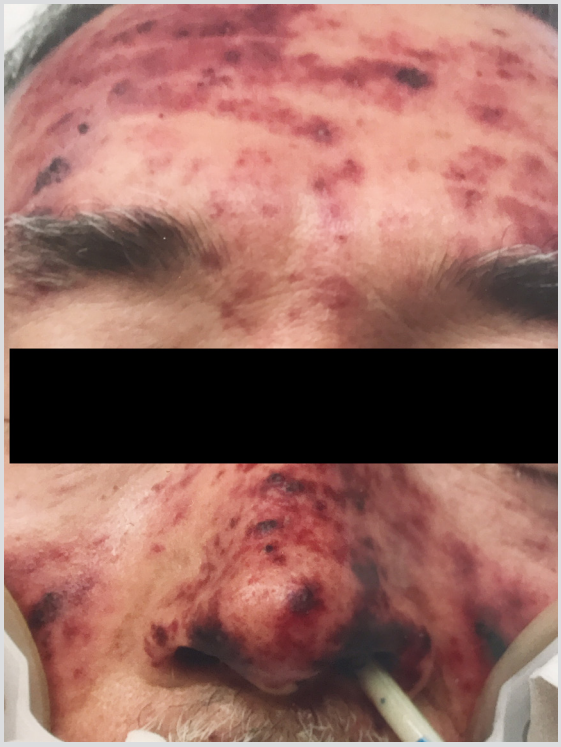

Figure 1. Patient's face with purpura fulminans unchanged a few days after admission

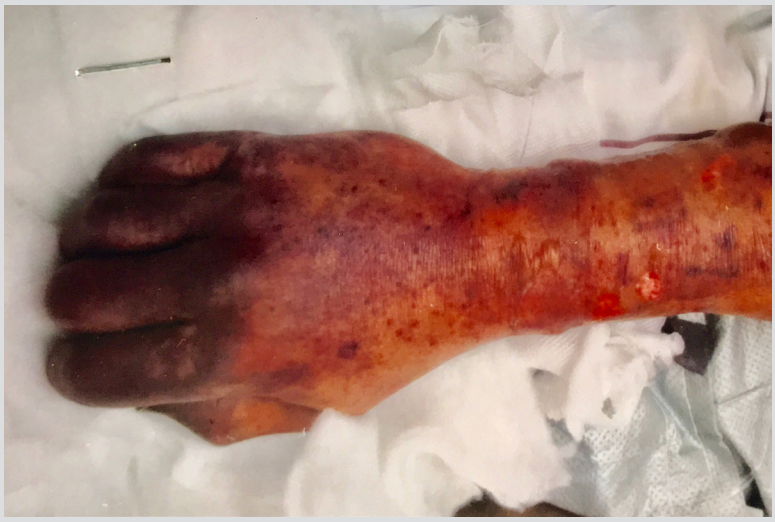

Figure 2. Patient's right forearm and hand after 1 week with unchanged purpura fulminans and emerging gangrene of the finger

On the 4th day of hospitalization, blood cultures yielded the Gram-negative bacillus *Capnocytophaga canimorsus*. Therefore, we added ciprofloxacin to the antibiotic regimen. The patient's history and clinical and laboratory examinations indicated no immunodeficiency, asplenia or alcohol abuse. While fever resolved and procalcitonin levels fell on the 8th day, CRP was rising and *Candida albicans* was isolated in repeated blood cultures. We began empirical treatment with fluconazole.
The patient developed progressive epidermolysis of the entire body. Signs of systemic infection persisted after 10 days of antibiotic treatment. In contrast, liver function, coagulation parameters and creatine kinase levels had normalized. CRP and procalcitonin levels rose again rapidly, and on day 11 the patient developed a temperature of 41°C. In order to treat a possible hospital-acquired infection with methicillin-resistant *Staphylococcus aureus*, we added vancomycin to the antibiotic regime.
A CT scan of the chest showed atypical centrilobular and peribronchovascular consolidation, probably due to pneumonia. This was possibly associated with pulmonary aspergillosis, as *Aspergillus fumigatus* was detected in the patient's tracheal secretion, and we switched antimycotic treatment to voriconazole. Antibiotic treatment was changed to meropenem and penicillin G. Additionally, a CT scan of the abdomen showed complete splenic infarction, and all extremities turned gangrenous. As a cranial CT scan at this point showed signs of severe hypoxic cerebral oedema, a joint decision to de-escalate therapy was made together with relatives. The patient died after 16 days of treatment.

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DISCUSSION

Our report of a patient with fatal septic shock due to *C. canimorsus* describes several noteworthy features which may be important to clinical practice. *C. canimorsus* is a Gram-negative rod and facultative anaerobic bacterium that physiologically inhabits the oral cavity, especially of dogs and cats[1, 2]. *C. canimorsus* infection is most frequently transmitted by dog bites[3]. Infections by *C. canimorsus* are generally rare, ranging from self-limiting, local skin infections to septic shock. Severe and fatal infections have been reported in patients with immunodeficiency, splenectomy or alcohol abuse[3, 4]. Transmission by bite and frequent reports of immunodeficiency may indicate that a higher bacterial concentration and underlying preconditions are usually required to cause especially severe *C. canimorsus* infection. The occurrence of purpura fulminans is an early ominous sign of a progressively severe course[5].

Our patient did not show any immunodeficiency, splenectomy or alcohol abuse. In addition, he only touched and was licked by his dog in the weeks prior to infection. Therefore, we assume there was a low bacterial concentration during transmission and no specific susceptibility to severe *C. canimorsus* infection. Despite this, the patient developed septic shock with fatal multiorgan failure. Additionally, he already had purpura fulminans on admission.

Very rarely, severe *C. canimorsus* infections without biting or scratching have been reported [6,7]. Only one patient had no immunodeficiency. *C. canimorsus* infection is fatal in approximately 25% of patients[3]. However, this high mortality rate is based on collections of case reports and case series which are limited by selection and publication bias and lack of differentiation between immunocompetent and immunodeficient patients. Purpura fulminans is also very rare and most commonly associated with severe infections[8]. Sepsis due to *Neisseria meningitidis* or *Streptococcus spp*., as well as *Haemophilus influenza*, *Staphylococcus aureus* and, very rarely, *C. canimorsus* is frequently complicated by purpura fulminans[8].

What are the clinical implications of this case report? Pet owners with flu-like symptoms should urgently seek medical advice when their symptoms exceed those of a simple viral infection, which in this case were severe dyspnoea and petechiae. Physicians confronted with such patients should ask about contact with dogs and cats. They should consider *C. canimorsus* infections also in the presence of purpura fulminans and the absence of animal bites or scratches, and any immunodeficiency. In such cases, the clinician should immediately start empiric treatment with a penicillin in combination with a beta-lactam inhibitor until a definite diagnosis is established.

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