Pasteurella multocida peritonitis after cat scratch in a patient with cirrhotic ascites

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Abstract

Pasteurella multocida, a zoonotic agent transmitted by canines and felines, has been very rarely reported to cause bacterial peritonitis in humans. Pasteurella multocida peritonitis is associated with high mortality even with appropriate treatment, therefore its early recognition is essential. We report a case of Pasteurella multocida peritonitis following cat scratch in a patient with Child Pugh Class C alcoholic cirrhosis, culminating in multiple organ failure and death.

Case Report

A 49-year-old woman was admitted to hospital with generalized abdominal pain, fever, and drowsiness for one week. She had been diagnosed to have decompensated alcoholic cirrhosis (Child Pugh Score 12, Class C) 3 months before. In the past, she had been drinking a bottle of whisky every day (~25 standard units of alcohol) for at least 2 years, but had abstained from alcohol since the diagnosis of cirrhosis. Her left hand had been scratched by her 4 month-old pet kitten 3 weeks prior to the admission, but the abrasions had healed spontaneously within a couple of days. On physical examination, she was drowsy, febrile (38°C), and hemodynamically stable, with jaundice, spider naevi, generalized abdominal tenderness, splenomegaly (7 cm below the left costal margin), tense ascites and bilateral pitting pedal edema. She had asterisks consistent with hepatic encephalopathy, but no focal neurological signs or meningism. Healed abrasions on the distal phalanges of index and middle fingers of the left hand were noted. Cardiovascular examination did not reveal signs of infective endocarditis. Respiratory examination was unremarkable.

Laboratory tests showed: C-reactive protein 133 mg/L, neutrophil leukocytosis (hemoglobin 126 g/L, white blood cells 23.1×10⁹/L, platelets 190,000/mm³), deranged liver function tests (bilirubin 186 μmol/L, aspartate transaminase 58 U/L, alanine transaminase 45 U/L, alkaline phosphatase 165 U/L, gammaglutamyl transferase 120 U/L), poor hepatic synthetic function (albumin 19 g/L, international normalized ratio 2.2), baseline renal impairment (serum creatinine 125 μmol/L, eGFR 40 mL/min) and hyponatraemia (Na+ 120 mmol/L). Blood cultures were negative. Abdominal paracentesis yielded turbid ascitic fluid, albumin 8 g/L (serum ascitic albumin gradient 11 g/L), and white cells 13,800/mm³ (polymorphs 82%, mononuclear cells 18%). Ascitic fluid culture was positive for Pasteurella multocida. After overnight incubation the microbiology laboratory noted the growth of bacterial colonies on both the horse blood and chocolate agars, but no growth on MacConkey agar. These colonies were smooth and gray and approximately 1 mm in diameter. A positive indole and oxidase reaction combined with a gram stain revealing gram-negative cocccobacilli supported the provisional identification of Pasteurella species. Biochemical testing of the isolate on the Vitek 2™ (bioMérieux Clinical Diagnostics, Marcy l’Etoile, France) resulted in a genus level biochemical identification of Pasteurella species. An API20E™ (bioMérieux Clinical Diagnostics) identification was subsequently performed resulting in a final identification of P. multocida. Susceptibility testing was performed on the Vitek 2™ (bioMérieux Clinical Diagnostics). The ampicillin MIC was ≤2 μg/mL and the ceftriaxone MIC was ≤1 μg/mL. Based on Clinical and Laboratory Standards Institute guidelines both these antibiotics were reported as susceptible.

Specific treatment was started with intravenous ceftriaxone along with liver failure regimen, albumin infusion and supportive measures. Antimicrobial therapy was changed to intravenous ampicillin 48 hours later. Despite aggressive treatment, the patient’s condition progressively deteriorated with worsening encephalopathy, acute renal failure and gastrointestinal bleeding. She died on day 6 of hospital admission. No autopsy was performed.

Discussion

Pasteurella multocida is a zoonotic gram-negative facultative anaerobic cocccobacilli that is a member of the oropharyngeal flora of 55% dogs, and 90% of cats.1 Human infection occurs following animal bites, scratches, and even after licking. Although human infection following casual exposure has been previously reported,2 transmission via scratches from contaminated cat claws is unusual. Given the presence of encephalopathy in our patient, her history may not have been reliable, and indeed, a previous cat bite could not be ruled out. Pasteurella multocida usually causes skin, soft tissue, respiratory tract, and bone and joint infections in humans. Less commonly, life-threatening invasive infections such as intra-abdominal infections, infective endocarditis, meningitis and septicaemia have been reported in immunocompromized patients.3,4 Risk factors for invasive infection include cirrhosis, alcoholism, malignancy, diabetes mellitus, HIV infection, chronic pulmonary disease, and chronic kidney disease.5,3 Spontaneous bacterial peritonitis in patients with cirrhotic ascites is usually caused by transmigration of gram-negative enteric bacteria, and P. multocida has only rarely been reported to cause bacterial peritonitis in cirrhosis patients.5,7 Pasteurella multocida is sensitive to a broad range of antibiotics. Penicillins are the treatment of choice.5 Although susceptibility to beta-lactams is almost universal, beta-lactamase production by some isolates has been reported and thus susceptibility testing should always be performed.6 Quinolone, extended-spectrum cephalosporins, tetracyclines, and co-trimoxazole are options for patients with penicillin sensitivity.6 Even with appropriate and timely treatment overall mortality is more than 20-30%,6,5 yet improved outcomes have been reported recently as a result of better supportive care.2,4

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Conclusions

Invasive *Pasteurella multocida* infection should be suspected in patients with decompensated chronic liver disease in the context of exposure such as that of the patient described. In view of the associated high mortality, high index of clinical suspicion is required. Treatment with a beta-lactam antibiotic should be initiated immediately after collecting specimens to make a microbiological diagnosis. Since hepatic cirrhosis is an immunocompromized state, health care providers must caution cirrhotic patients regarding exposure to dogs or cats.

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