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Unusual presentation of more common disease/injury

Cirrhosis, cellulitis and cats: a 'purrfect' combination for life-threatening spontaneous bacterial peritonitis from *Pasteurella multocida*

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Summary

Pasteurella multocida is a Gram-negative coccobacillus that colonises the upper airways of many animals, in particular, dogs and cats. It acts as an opportunistic infection in humans following an animal bite or scratch and is associated with soft tissue infections, septicaemia and pneumonia, particularly in patients with a compromised immune response, such as patients with liver failure. Spontaneous bacterial peritonitis (SBP) is a serious complication of cirrhosis with a death rate of 10–15%. We report a case of a 47-year-old man with cirrhosis who presented with life-threatening *P multocida* SBP and bacteraemia secondary to a lick from a cat to a cellulitic leg wound. This case highlights the potential severity of an infection from domestic animals and an otherwise innocuous organism in an immunocompromised host.

BACKGROUND

Pasteurella multocida is a facultative Gram-negative organism that colonises the upper respiratory tract of up to 90% of cats and 70% of dogs. Superficial soft tissue infections and abscesses are most commonly reported in healthy individuals; however, in immunocompromised hosts, *P multocida* can manifest as severe systemic infection. Cases of this organism causing sepsis, osteomyelitis, lower respiratory tract infections, meningitis and endocarditis have all been reported in the literature.[1] Patients with end-stage liver disease seem to be more vulnerable to infections by *P multocida*, as evidenced by a small number of previous case reports.

CASE PRESENTATION

A 47-year-old man, with a background of Child Pugh C cirrhosis secondary to hepatitis C virus and heavy alcohol intake, presented to the emergency department with grade 3 encephalopathy, fever (38.1°C), and hypotension (85/38 mm Hg) on a background of chronic left leg cellulitis and lymphoedema. The cellulitis had led to several previous admissions, and a range of organisms had been isolated including methicillin-sensitive *Staphylococcus aureus*, methicillin-resistant *S aureus* and *Pseudomonas aeruginosa*. In the months leading up to his presentation, he had been on rotating antibiotics, consisting of clindamycin, ciprofloxacin and cephalexin, in an effort to control his chronic cellulitis. He had no history of spontaneous bacterial peritonitis (SBP).

On presentation, the patient was hypotensive, febrile and drowsy. Both legs were swollen with evidence of left leg cellulitis extending from the calf to the groin. He had prominent ascites but no abdominal pain. He became unstable over the next 2 h with progressive septic shock and acute-on-chronic liver failure manifest as acute renal dysfunction, hypoglycaemia, deteriorating conscious state requiring intubation and circulatory collapse requiring inotropic support with norepinephrine (peak infusion rate 12 mcg/min).

INVESTIGATIONS

Blood results were consistent with multiorgan dysfunction with a C reactive protein of 137.2 mg/l (normal <5 mg/l), creatinine 180 μmol/l (baseline 57 μmol/l, normal range 30–110 μmol/l), serum lactate 13.6 mmol/l (normal 0.5–1.6 mmol/l), blood glucose 2.4 mmol/l (normal 3.9–5.8 mmol/l) and international normalised ratio 2.7 (baseline 1.7). He underwent a diagnostic paracentesis, which drained turbid yellow fluid. Unfortunately, the sample had clotted and, therefore, a cell count could not be performed.

Gram-negative bacilli were seen on a Gram stain of both blood cultures and ascitic fluid. On day 3, culture results from both blood and ascitic fluid had isolated *P multocida* sensitive to penicillin, cephalosporins, gentamicin and ciprofloxacin.

TREATMENT

The patient required admission to the intensive care unit for ventilation and circulatory support. On the basis of the initial Gram stain, he was empirically started on intravenous piperacillin/tazobactam. With antibiotic therapy and administration of albumin, the patient's encephalopathy and fevers improved, his creatinine normalised and he was extubated after 3 days of ventilatory support. Following the results of the blood/ascitic fluid culture and sensitivities, antibiotic therapy was changed to intravenous benzylpenicillin and then to oral amoxicillin/clavulanic acid,

upon discharge for a further 2 weeks, following which he was started on long-term norfloxacin (400 mg daily) for SBP prophylaxis.

OUTCOME AND FOLLOW-UP

On further questioning, the source of *P multocida* was believed to be the patient's pet cat, who reportedly had a disturbing habit of regularly licking the serous exudate from the patient's chronic left leg cellulitis. Following discharge, he was referred for work up for liver transplantation and given the appropriate advice with regard to the safe cohabitation with domestic animals.

DISCUSSION

SBP is a common complication of cirrhosis with a reported incidence of 7–30% per year in patients with ascites.[2] The most common causative organisms include *Escherichia coli*, *Klebsiella pneumonia* and *Enterococci*.[3 4] A review of the literature revealed 12 reported cases in English of *P multocida* causing SBP. Alcoholic cirrhosis was the cause of liver disease in nine of the reviewed cases. Other causes for cirrhosis included hepatitis C virus and cryptogenic cirrhosis.[5–15]

The key step in the pathogenesis of SBP is thought to be bacterial translocation. This involves passage of bacteria through the gut lumen to the mesenteric lymph nodes and ultimately to the ascitic fluid or blood.[16] Evidently, haematogenous spread of bacteria to ascitic fluid also has a role as seen in this case study where bacteraemia from suspected inoculation of a cellulitic wound lead to SBP. Of the other reported cases of *P multocida* SBP, three recounted a recent bite or scratch from a domestic animal.[5 9 11] Seven patients had frequent exposure to domestic animals without known trauma, and one case reported no known animal exposure precipitating the illness.[13]

While there is no gold standard for diagnosis of SBP, a polymorphonuclear leucocyte count of ≥250/mm^3 is highly indicative.[4] Culture bottles of ascitic fluid are also important and have been shown to isolate the causative organism in 93% of cases.[17]

If SBP is suspected, third generation cephalosporins are regarded as the treatment of choice.[3 4] These have a broad antimicrobial spectrum and should cover up to 98% of common causative organisms.[18]

In regards to the treatment of *P multocida*, when isolated in human infections, it is usually susceptible to a broad range of antimicrobials including penicillins. A recent study examined the antimicrobial sensitivities of 192 strains of *Pasteurella spp*. All strains were sensitive to cefotaxime, minocycline and fluoroquinolones such as ciprofloxacin and levofloxacin. They were also sensitive to penicillins with the exception of one strain of *P multocida*, which conferred β-lactamase activity. The organism's sensitivity to penicillin was restored with the addition of clavulanic acid.[19] Therefore, even if *P multocida* is suspected as a cause of SBP, third generation cephalosporins are still the most effective antibiotics to use until culture results are known. Evidently, once results of cultures are known, antibiotics should be tailored and penicillins are very effective in treating most *Pasteurella spp*.

Learning points

- Patients with end-stage liver disease have significantly impaired immune responses, leaving them vulnerable to overwhelming sepsis from otherwise innocuous organisms.
- Domestic animals can, under the appropriate clinical circumstances, pose a risk to an immunocompromised host, particularly if adequate hygienic practices are not adhered to.
- Questioning regarding exposure to domestic animals should form part of the clinical history when assessing immunocompromised hosts, allowing early institution of empiric antimicrobial therapy in the appropriate clinical scenario.
- While translocation of enteric gut organisms to regional lymphatics is the pivotal step in spontaneous bacterial peritonitis, primary bacteraemia, with secondary infection of ascitic fluid can also occur, as seen in this case.

Competing interests None.

Patient consent Obtained.

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