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Aeromonas hydrophila: myofascial necrosis and sepsis

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Abstract. An alcoholic patient with ascites was admitted to the intensive care unit for gastrointestinal bleeding. He subsequently developed spontaneous myonecrosis of the extremities culminating in sepsis syndrome. This was a unique, non-traumatic presentation of *Aeromonas hydrophila* soft tissue injury.

Key words: *Aeromonas hydrophila* – Soft tissue infection – Gangrene

Although infrequently encountered in the ICU setting, primary soft tissue infection is associated with significant morbidity and mortality. Previously, Wright had described gas gangrene, and sepsis attributed to the "Bacillus of Welch" (Clostridium) in 1917, while Meleney identified necrotizing fascitis in 1924 [1, 2]. Soft tissue infection may involve superficial, localized processes such as impetigo, erysipelas, folliculitis, and cellulitis due to *S. pyogenes* or *S. aureus* [3]. Systemic conditions may manifest as invasive disease such as ecthyma gangrenosum due to *P. aeruginosa*; necrotizing fascitis – Meleney's or Fournier's gangrene postoperatively; and myonecrosis most often caused by *Clostridium septicum* or *perfringens* [3, 4]. Non-clostridial gangrene occurs secondary to *E. coli*, *Proteus*, *Pseudomonas*, *Klebsiella*, and *Enterococcus*; and is also associated with high mortality [5].

Aeromonas hydrophila has been implicated as an unusual causes of myonecrosis usually occurring after traumatic injuries [6, 7]. This case involves an alcoholic patient admitted to the ICU for gastrointestinal bleeding, who developed spontaneous myonecrosis of his lower extremities, culminating in a sepsis syndrome.

Case report

A 41-year-old, white male presented to the ICU after a one-week hospitalization for hematemesis and melena. Past medical history included chronic alcohol abuse with two prior episodes of gastrointestinal bleeding. Prior to transfer, he had been managed conservatively but had a 9-unit transfusion requirement. Endoscopy revealed Barrett's epithelium in the distal esophagus and no varices were noted. Initial assessment revealed an alert patient with temperature of 37 °C, blood pressure of 122/70 mmHg, heart rate of 88/min, and respirations of 20/mm. The stigmata of alcoholic lever disease were apparent including scleral icterus and spider angiomas. Abdominal examination found a distended, non-tender abdomen consistent with ascites and a rectal examination positive for blood.

Laboratory evaluation found evidence of gastrointestinal bleeding and a coagulopathy presumed secondary to hepatic dysfunction. Hematologic indices were a hemoglobin of 8.7 g/dl, hematocrit of 25.3% without leukocytosis. There were 51,000/mm^3 platelets, a PT of 16.2, and PTT of 32.1 s. Electrolytes, BUN, and creatinine were within normal limits. Liver function tests were significant for a total bilirubin of 12.8 and direct fraction of 7.5 mg/dl with a normal alkaline phosphatase, parenchymal enzymes, ammonia, and hepatitis screen. Arterial blood gas, chest and abdominal radiographs, and electrocardiogram were unremarkable.

The patient was admitted to the ICU for hemodynamic monitoring. Endoscopy found esophageal varices which were sclerosed, and the patient was started on Pitressin (0.5 μ/min). Paracentesis was performed and was significant for 6000/mm^3 leukocytes with 86% neutrophils, and the patient was started on ampicillin and gentamicin for presumed spontaneous bacterial peritonitis. The patient's course was uneventful over 48 h, except for blood product replacement. However, routine physical exam found two, small hemorrhagic bullae located on the posterior calves associated with crepitance, along with left upper extremity involvement. Hemodynamic monitoring revealed an increased cardiac index of 9.7 l/m^2, a decreased systemic vascular resistance index of 427 dynessec/cm^{-5}/m^2 and a C(a-v)O_2 of 1.6 vol.%. The patient was noted to have had adequate tetanus immunization. Clindamycin was added to the antibiotic regimen. Dopamine was started, and an urgent surgical consultation was obtained. Radiographic evaluation found rapidly progressive (1 h) evidence of soft tissue gas formation (Figs. 1 and 2).

The patient underwent rapid clinical deterioration consistent with sepsis over the next hour, and operative intervention was undertaken. Intraoperatively, a rapidly progressive myonecrosis involving bilateral lower and unilateral upper extremities was noted. After much deliberation with the patient and family, a triple amputation was performed.

Bacteriologic assessment found a Gram-negative motile gas-producing organism, *Aeromonas hydrophila*, in multiple blood specimens, while intravascular catheter cultures were sterile. Although, a prior specimen from the outlying hospital was positive for *Serratia marcescens* or *Citrobacter freundii* organisms similar to *Aeromonas* and are often misidentified. Paracentesis fluid was sterile, and surgical pathology found *A. hydrophila* and extensive leukocyte infiltrates, vascular thrombosis, and myofascial coagulative necrosis. Antibiotic sensi-

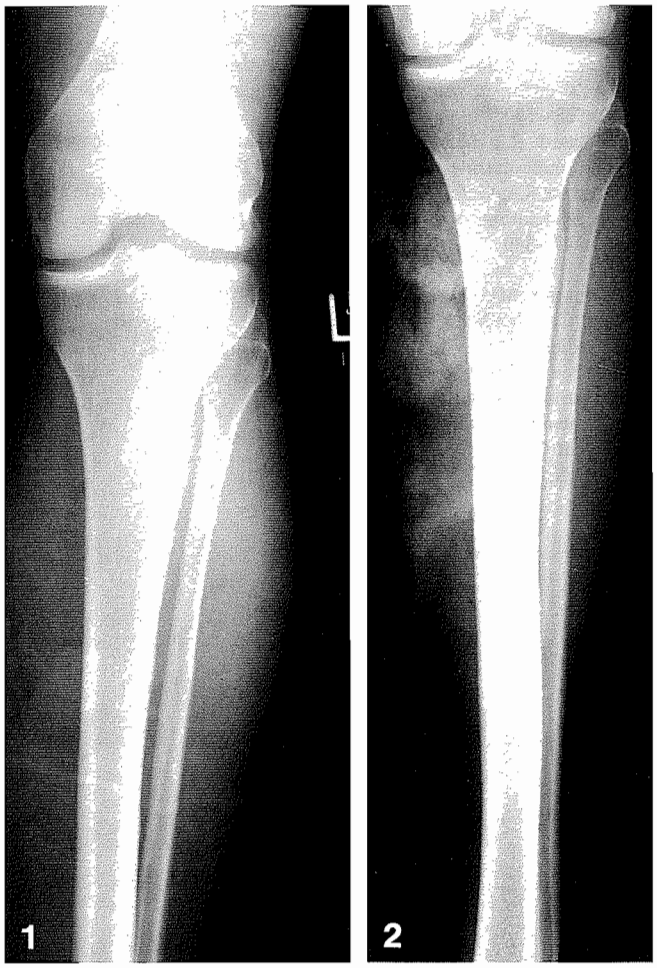

Fig. 1. Radiograph of left calf showing early soft tissue gas formation

Fig. 2. Radiograph of left calf 1 h later showing progression of gas formation

tivity patterns required the substitution or piperacillin for ampicillin, along with gentamicin and clindamycin. After a two-week hospital course, the patient succumbed to hepatorenal syndrome, sepsis, and suffered a bradycardic arrest as therapy was withdrawn.

Discussion

Aeromonas hydrophila is a nonfastidious, motile, gram negative bacillus that exhibits beta hemolysis [6, 8]. Fermentation and gas production allows differentiation from non-fermenting *Pseudomonas* species; while the positive oxidase and indole reaction allows differentiation from other Enterobacteriaceae; and decarboxylase reaction delineates the *Vibrionaceae* [6, 8, 9]. This is significant since this organism was initially misidentified. *Aeromonas* species are found in both fresh and saltwater habitats along with soil samples on the Eastern seaboard of the US [8, 10]. This low virulence opportunist can become a human pathogen based on exposure to well water or contaminated hospital delivery system [9, 10]. The most common subtype is *A. sobria* and found in 77% and *A. hydrophila* isolated in 23% of *Aeromonas* sepsis patients [11].

Patients predisposed to *Aeromonas* infection are usually immunocompromised to some extent [6]. Malignancy is the most common association, specifically hematologic in 36% and colonic in 26%, followed by diabetes mellitus (19%) and cirrhosis (17%) [4]. This association between liver dysfunction manifested as cholestasis, ascites, peritonitis, or Laennec's cirrhosis, as was found in this case has been described previously by Conn in 1964 [4]. Suggested mechanisms for sepsis include transmural intestinal bacterial translocation, failure of hepatic hemofiltration, and lymphatic stasis [12].

Soft tissue infections secondary to aquatic exposure are most often due to marine halophilic *Vibrio* species [13]. *Vibrio vulnificus* causes necrotizing cellulitis in 22% of cases, as well as gastroenteritis and sepsis [13]. *Aeromonas* soft tissue infection has been described in the form of cellulitis, fascitis, and osteomyelitis in non-immunocompromised patients, usually after traumatic injury in an aquatic environment or after freshwater ingestion [7, 8, 14]. However, Dryden suggested only 15% (2/13) of patients encountered had this history [11]. Disease syndromes include gastrointestinal infection manifested as diarrheal illness due to proctocolitis [8, 9].

Secondly, skin and soft tissue involvement is described with severe local infiltrate and necrosis secondary to hematologic dissemination after traumatic injury [8, 11]. Sepsis has been described in 40 cases to date with a mortality of 46% to 57% [11].

Diagnosis is made by evidence of leukocytosis, usually a lymphocytosis with atypical forms [9]. Serology offers a 70% sensitivity for *Aeromonas* antibody agglutination reactions [9]. Microbiologic assessment finds that the organism may be isolated from stool, bile, blood, or soft tissue sources by gram stain or culture. Vascular involvement may be demonstrated by a bleeding diathesis featuring thrombocytopenia or a disseminated intravascular coagulation profile. Therapy involves broad spectrum antibiotics including antipseudomonal coverage for the immunocompromised and antistaphylococcal coverage for the normal host along with an aminoglycoside. Clinically, *Aeromonas* was found to be sensitive to gentamycin (100%), trimethoprim-sulfamethoxazole (100%), chloramphenicol (97%); intermediate to erythromycin and resistant to first-generation cephalosporins (12%), carbenicillin (6%), and ampicillin (3%) [6, 9]. Definitive care is afforded by rapid aggressive surgical debridement of devitalized tissue. Adjunct therapy includes a possible role for antitoxin or hyperbaric oxygen therapy.

Although the association of *Aeromonas hydrophila* and liver disease has been described, the major extent of the spontaneous myofascial involvement in an untraumatized patient is unique.

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