Case Report

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Fatal Necrotizing Soft Tissue Infection by *Aeromonas hydrophila*

Microbiology Section

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ABSTRACT

Aeromonas infections in healthy individuals are self limiting, but those in patients with immuno-compromised conditions are frequently associated with significant morbidity and mortality. The current case report describes a fatal case of necrotizing soft tissue infection by *Aeromonas hydrophila* in an immuno-competent patient.

Keywords: *Aeromonas hydrophila*, Soft-tissue infection

CASE REPORT

A 28-year-old female, residing in the rural Alwar district of Rajasthan, India presented to the Emergency Department of a tertiary care hospital in Jaipur, India with complaints of breathlessness and swelling in right arm with pain. On admission, the patient was found to have a body temperature of 101ºF, accompanied by hypotension (71/45 mmHg), tachycardia (160/min), tachypnea (44/min) and oxygen saturation of 64%. Examination of the right upper limb revealed a tender edematous swelling on the entire arm extending up to the digits, with multiple bleb formations and ecchymosis over the skin. A provisional diagnosis of septic shock with cellulitis of the right arm was made.

The patient provided a recent history of acute gastroenteritis, for which she had received an I.V. infusion at a rural health care centre. The I.V. line was established on the right arm. The patient subsequently developed swelling and pain in the same arm. Due to progressive clinical deterioration in her condition over the next five days, the patient was admitted and treated conservatively for septic shock in a local hospital in Alwar, before being referred to our hospital. The treatment details of the same were not available with the patient. There was no history of any comorbidity such as hypertension, tuberculosis, diabetes mellitus or asthma.

Following admission, the patient was immediately intubated and put on mechanical ventilation. Antibiotics (Piperacillin/Tazobactam and Amikacin) and ionotropes were started as initial treatment for septic shock. Her laboratory investigations revealed marginally increased renal and hepatic function tests:(Serum BUN- 49mg/dl, creatinine -2.8mg/dl, SGOT-115 u/l, Alkaline phosphatase-171u/l, total bili-rubin-6.15 mg/dl, prothrombin time-28.8seconds), accompanied by thrombocytopenia (platelet count-40 thousand/microlitre) and leucocytosis (TLC-22.4 thousand/microlitre). The blood and urine cultures were sterile.

A surgical consultation, taken for the right arm, suggested gangrene on the right upper limb with compartment syndrome. An axial fasciotomy with releasing incision was performed under sedation in the Intensive Care Unit (ICU). During the procedure, a deep wound sample was collected from the necrotic tissue and sent for culture and sensitivity to the microbiology lab. After the procedure, Levofloxacin, Teicoplanin and Clindamycin were added to the existing antibiotic regimen. Over the next few hours, the patient developed conjunctival haemorrhage in the left eye and ecchymotic patches over the left ankle and left wrist. Repeat laboratory investigations revealed a further deranged coagulation profile, liver function tests and renal function tests. The haemoglobin had dropped to 6.5 g/dl and the platelet count to 15 thousand/microlitre. Fibrinogen degradation products of >160 < 320 microgram/ml (reference range < 5 mcg/ml) were noted. The patient then developed ventricular tachycardia with hypotension. In spite of all resuscitative measures, the patient succumbed to the infection within two days of hospitalisation, severe sepsis with multi-organ failure being the immediate cause of death.

In our laboratory, oxidase positive non-lactose fermenting colonies on Mac-conkey agar and β hemolytic colonies on sheep blood agar were obtained from the culture of the necrotic tissue. The isolate was identified as *Aeromonas hydrophila group* using the Microscan® Walkaway SI automated identification and susceptibility system (Seimens Healthcare Ltd.) It was sensitive to Amikacin, Cefepime, Ciprofloxacin, Gatifloxacin, Gentamicin, Levofloxacin, Moxifloxacin and Tetracycline and demonstrated resistance to Ampicillin, Carbapenems and all second and third generation Cephalosporins. The identity was reconfirmed on the mini API system (Biomerieux) using the API GN ID® strips.

DISCUSSION

Aeromonads are gram negative bacilli rods in the family *Aeromonadaceae.* These organisms are oxidase positive, motile by means of a polar flagellum and are facultative anaerobes [1]. *Aeromonas species* are a known cause of a wide variety of human illnesses, including intestinal and extra-intestinal diseases and syndromes, ranging from systemic and local infections in both immunocompetent and immunocompromised hosts [2].

There have been several descriptions of *Aeromonas hydrophila* skin and soft tissue infections in literature including localized cellulitis, ecthyma gangrenoseum, Clostridium like gangrenous cellulitis, localised myonecrosis, acute rhabdomyolysis, and necrotising fasciitis [3,4]. The most common clinical presentation is that of cellulitis with a good prognosis if bacteremia is not produced [5]. *Aeromonas hydrophila* soft tissue infections have been found associated with fish and reptile bites. The organism is known to inhabit in mouth, fangs or venom of the snakes and is inoculated into the human tissue due to the bite. The local necrotising myotoxic, oedema inducing action of the snake venom, along with the use of a tourniquet to contain the venom favours the local multiplication of the bacteria, thereby leading to a soft tissue infection [6].

Literature review of *Aeromonas hydrophila* soft tissue infections has found that these infections occur following motor vehicle crashes, farmyard injuries, puncture injuries and mud football injuries involving wound contamination from aquatic and non-aquatic environments [7]. The infection predominantly affects lower limbs of middle aged males with previous history of injury. Other sites of infections include the scalp; hands and arms [6].

In our case, *A. hydrophila* was cultured from the necrotic tissue that was obtained during fasciotomy, incriminating this organism as the causative agent of the necrotizing soft tissue infection. The patient did not give any history of exposure to contaminated water or associated water related injury. However, the patient did suffer from acute gastroenteritis, following which an I.V. line medication was administered. It is most likely that the acute gastroenteritis was due to *A. hydrophila*. It is possible that organism might have subsequently gained entry by local inoculation followed by insertion of an intravenous line. A case of myonecrosis due to *A. hydrophila* following insertion of an intravenous canula has been reported in literature [8]. Firm association between aeromonads and the use of indwelling devices has also been demonstrated [9].

The pathogenicity of *Aeromonas* infection can be attributed to the action of several extra cellular toxins that lead to a very short incubation period and a rapid progression of infection. Certain *A. hydrophila* strains contain a gene called Aerocytotoxin Enterotoxin (ACT) that releases a toxin (aerolysin) that can cause tissue damage. This toxin is an extra-cellular soluble hydrophilic protein which has both hemolytic and cytolytic properties. Aerolysin binds to specific glyco- receptors on the surface of eukaryotic cells before inserting into the lipid bilayer and forms holes [10]. Several other virulence factors that lead to subcutaneous and skeletal muscle damage have also been identified and muscle destruction has been demonstrated in an animal model [3].

Some investigators suggested that early surgical intervention is essential to save the life of patients with *Aeromonas* septicemia with deep soft tissue infection. Certain reports also point that delays in surgical removal of necrotic tissue have resulted in high mortality [11]. In our case, the infection probably mimicked cellulitis in its earlier stages which led to conservative treatment at the local hospital, without aggressive surgical intervention. As a result, organism multiplication in the necrotic tissue and liberation of toxins progressed to toxemia and sepsis, followed by severe toxic shock. Unfortunately, this particular patient was already in severe toxic shock by the time she reported to our hospital.

Another major concern regarding *Aeromonas* infections is their potential resistance to Penicillin, Ampicillin, Carbenicillin and Cefazolin [12]. Thus, standard empirical therapies, that are effective against *streptococcal* or *staphylococcal* soft tissue infections, do not provide coverage for *Aeromonas* infection. The organism is usually found to be susceptible to broad spectrum cephalosporins aminoglycosides, carbapenems, chloramphenicol, trimethoprim – sulfamethoxazole and quinolones [10]. Our patient's isolate was resistant to carbapenems and all second and third generation cephalosporins. Certain *Aeromonas* species have been found to possess at least 3 inducible chromosomally mediated β-lactamases. Reports suggest that susceptible strains might develop resistance during clinical antibiotic therapy with extended-spectrum beta-lactam agents. Emergence of resistance in *Aeromonas* strains resulting from the selective pressure exerted by a β-lactam is a rare event. However, it is likely that the heavy local colonisation of *Aeromonas* species on ischemic and damaged tissue results in a high inoculum of bacteria immersed in a sub- inhibitory concentration of antibiotic, thus favouring the emergence of resistant mutants [13]. In our case, though the patient had not received treatment with extended-spectrum beta-lactam agents at our hospital, she did provide a history of treatment at a local hospital prior to being admitted at our centre. However, the details of the treatment provided at the other hospital were not available.

CONCLUSION

This case report proves that an *Aeromonas hydrophila* soft tissue infection may rapidly progress to fatal sepsis with multi-organ failure. Hence, its early identification with prompt surgical intervention along with appropriate antibiotic therapy may be critical for a better outcome.

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