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CASE REPORT

NECROTIZING SOFT TISSUE INFECTION CAUSED BY AEROMONAS CAVIAE: A CASE REPORT

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ABSTRACT: Aeromonas caviae is commonly recognized as a low-virulence gram-negative bacillus. Human infections due to Aeromonas caviae may present as a variety of diseases, such as wound infection, acute gastroenteritis, septicemia, pneumonia, septic arthritis or endocarditis. It has rarely been reported as a causative organism of septicemia. We present a successfully treated case of post-traumatic necrotizing soft tissue infection with sepsis of nosocomial origin due to multidrug resistant (MDR) Aeromonas caviae in a 51-year-old immunocompetent individual. Phenotypic confirmation was done by VITEK 2 COMPACT Advance Expert System.

KEYWORDS: Aeromonas caviae, necrotizing fasciitis, MDR.

INTRODUCTION: Aeromonas caviae is an oxidase-positive, facultative anaerobic, Gram-negative bacillus of the Aeromonadaceae family. Aeromonas species, is widespread in nature and is usually found in fresh water, brackish water, moist oil, and non-fecal organic material. Aeromonas caviae (10%) is the third most common human pathogenic species of the Aeromonas genus, preceded by Aeromonas hydrophila (68%) and Aeromonas sobria (17%) according to a previous epidemiological reports.[1] Aeromonas species are most commonly known to cause diarrhea. They also cause necrotizing fasciitis and sepsis in patients with hepatic diseases, diabetes mellitus, and immunocompromised status. Most reported severe soft tissue infections have been caused by Aeromonas hydrophila.

We report a case of necrotizing fasciitis and septicemia, which progressed to multiple organ failure caused by Aeromonas caviae in an apparently immunocompetent elderly male patient. Fasciotomy was performed in the patient, with aggressive parenteral antibiotic therapy, inotropics, anticoagulants and diligent intensive care, leading to recovery and wound healing.

CASE REPORT: A 51 year old male patient presented with lacerated injury to both lower limbs extending to both the ankles and feet following a road traffic accident. He was initially admitted to a local rural hospital, received basic life support and was referred to our institution after two days of the incident. Physical examination was performed at the emergency department which revealed that he was afebrile, conscious, cooperative with a heart rate of 96/min, blood pressure 100/60 mm Hg. Extensive laceration and tissue damage were noted in both the lower limbs with locally raised temperature, redness, pain, tenderness and tissue damage. All lower limb peripheral pulses were found to be palpable. No other significant injuries were noted at the time of admission. He was admitted to the orthopedic department. He revealed no history of diabetes mellitus, chronic liver disease or prolonged chemotherapy.

On admission, his fasting and 2 hours post-prandial venous plasma glucose were respectively 94 and 123mg/dl; blood urea Nitrogen 35 mg/dl, serum creatinine 1.2 mg/dl, serum total protein

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3.6mg/dl and otherwise unremarkable liver function test. His hemoglobin was 8.6gm/dl, platelet count 1.6X10^5/µl, total leukocyte count 5800/µl with 78% neutrophils. He was seronegative for HIV, HbsAg and anti HCV in serological tests. Conservative management with regular dressing was topical chlorhexidine, cetrimide, Mupirocin ointment and intravenous Ceftriaxone and Metronidazole was initiated empirically.

For the initial 10 days, he responded well, and was afebrile, with normal vitals. Oral Coamoxiclav and Metronidazole was initiated after first seven days of parenteral therapy.

After 12 days of hospital stay, he started to develop a fever of 103^{0}F with tachycardia and hypotension. The wound started developing new purulent secretion, blackish discolouration and signs of fresh inflammation, including pain and rise in temperature. On the 14th day of admission, the total leukocyte count escalated to 20,600/ µl with 88% of Neutrophils and 19% band cells. His haemoglobin and total erythrocyte counts reduced to 7.7mg/dl and 2.85X10^6/µl respectively. A clinical diagnosis of necrotizing fasciitis following degloving lacerated injury was made and extensive fasciotomy and debridement along with removal of necrotic tissue were performed. The drained thick and blood tinged pus sample along with peripheral venous Blood were sent for culture and sensitivity.

Direct Gram's staining of the pus revealed plenty of pus cells and gram negative bacilli. Aerobic culture on sheep blood agar showed β-hemolytic non-swarming, grey, moist, oxidase positive colonies. On MacConkey's agar lactose fermenting, flat, spreading colonies with irregular margin with a "central dot" were seen, which were catalse positive. On wet mount the bacilli were actively motile. Gram stain from the colonies showed Gram negative short slender, non-sporing bacilli. The organism exhibited suicidal phenomenon in glucose fermentation broth, was anaerogenic, and positive for Indole production, aesculin hydrolysis, Arginine hydrolase. Lysine and Ornithine decarboxylase reactions were negative. The colonies were also put up for identification in the VITEK2 COMPACT Advance Expert System™ (Biomerieux Co., France) using a GNID™ card.

The isolate was identified to be Aeromonas caviae. The antimicrobial susceptibility was studied with Mueller–Hinton agar using the standard disc diffusion method according to Clinical and Laboratory Standards Institute recommendations.[2] The isolate was sensitive to amikacin, gentamicin, aztreonam, cefepime, piperacillin-tazobactam, imipenem and meropenem and resistant to ceftazidime, ceftriaxone, cotrimoxazole, levofloxacin and tobramycin. Blood culture was put on BacT/ALERT 3D™ (Biomerieiux, France) showed positive result after 13.4 hrs. From the positive blood culture bottle it was plated on sheep blood agar and MacConkey's agar, growth of similar colonies noted, further put for antimicrobial susceptibility and phenotypic identification tests which yielded similar results.

Following antimicrobial sensitivity report IV inj Amikacin was started and patient responded well. There was subsidence of fever and negative blood culture after 3 days with blood counts returning to normal levels. Daily dressing of wound was done. Subsequent wound swab culture after 7 days became negative. Patient later referred for split skin grafting on the bare area of legs.

DISCUSSION: Aeromonas are nonsporulating gram-negative rods that are ubiquitous inhabitants of fresh water sources. These hardy organisms multiply and grow under a variety of conditions and temperatures. Most species are motile and catalase and oxidase positive and reduce nitrates to nitrites.[1] At present, the family Aeromonas comprises 17 species. At least ten of these have been

implicated in human disease, but A. hydrophila, A. caviae, and A. veronii biovar sobria have been most commonly isolated from human infections. The type species is A. hydrophila.[3]

The pathogen is responsible for opportunistic infections in people with compromised immune systems, usually in association with malignancies and liver cirrhosis.[1,3,4] Diabetes mellitus, alcoholism, malnutrition, renal failure, and severe peripheral arterial occlusive disease have also been reported as predisposing factors. Nosocomial outbreaks associated with aeromonads have been reported. These organisms are important causes of skin and soft-tissue infections and aspiration pneumonia following contact with water and after floods. High incidence of antimicrobial resistance, including third-generation cephalosporins and the fluoroquinolones, is found among Aeromonas sp. isolated from clinical sources in some developing countries in Asia.[5] We suspected that contamination of wound during dressing may have been the source of infection in this case. The patient's relatives or locals carrying Aeromonas in their slippers or water contamination may be the source.

Aeromonas species can produce many virulence factors, including hemolysin, cytotoxin, aerolysin, enterotoxin, endotoxin, protease, adhesins, leukocidin, and lipases.[1,6] Aeromonas hydrophila is the most commonly reported pathogen that causes Aeromonas necrotizing fasciitis and septicemia; these conditions often occur after soft tissue trauma with exposure to contaminated water or nonfecal organic materials and produce skin lesions similar to those observed in infections caused by Vibrio species.[7,8] Aeromonas can produce myonecrosis with gas gangrene resembling that caused by Clostridia. Brenden and Huizinga reported endotoxins of Aeromonas hydrophila intramuscularly inoculated in mice caused the pathogenesis of sepsis; moreover, endoxemia appeared to damage the liver, kidneys, and pulmonary function, resulting in septic shock and multiple organ failure.[9]

This was a case of multidrug resistant Aeromonas caviae necrotizing fasciitis with fatal sepsis in patients with lacerated injury, probably of nosocomial origin. Early diagnosis, immediate fasciotomy, appropriate empiric antimicrobial therapy with aminoglycosides, and intensive care should be given to patients. When patients present with a rapid onset of necrotizing skin necrosis and progressive sepsis, an Aeromonas infection should be considered in the differential diagnosis.

REFERENCES:

1. Janda JM, Duffey PS: Mesophilic aeromonads in human disease: Current taxonomy, laboratory identification, and infectious disease spectrum. Rev Infect Dis 1988; 10: 980-97.
2. CLSI. Performance Standards for Antimicrobial Susceptibility Testing; Twenty fourth Informational Supplement. CLSI document M100-S24. Wayne, PA: Clinical and Laboratory Standards Institute.
3. 2014Winn WCjr, Allen SD, Janda WM, Koneman EW, Procop GW, Schreckenberger PC, Woods GL, editors. Koneman's color atlas and textbook of diagnostic microbiology. 6th ed. Washington: Lippincott Williams & Wilkins; 2006: 1443-75.
4. Ko WC, Chuang YC: Aeromonas bacteremia: Review of 59 episodes. Clin Infect Dis 1995; 20:1298-1304.
5. Ghenghesh KS, Ahmed SF, El-Khalek RA, Al-Gendy A, Klena J: Aeromonas in Developing Countries. J Infect Developing Countries 2008; 2(2): 81-98.

DOI: 10.14260/jemds/2015/374

6. Wu CJ, Wu JJ, Yan JJ, Lee HC, Lee NY, Chang CM, Shih HI, Wu HM, Wang LR, Ko WC. Clinical significance and distribution of putative virulence markers of 116 consecutive clinical Aeromonas isolates in southern Taiwan. J Infect Dis. 2007; 54: 151–8.
7. Simon TP, Rajakulendran S, Yeung HT. Acute hepatic failure precipitated in a patient with subclinical liver disease by vibrionic and clostridial septicemia.Pathology.1988; 20: 188–190.
8. Tsai YH, Hsu RW, Huang TJ, Hsu WH, Huang KC, Li YY, Peng KT. Necrotizing soft-tissue infections and sepsis caused by Vibrio vulnificus compared with those caused by Aeromonas species.J Bone Joint Surg Am.2007; 89: 631–6.
9. Brenden RA, Huizinga HW. Pathophysiology of experimental Aeromonas hydrophila infection in mice.J Med Microbiol.1986; 21: 311–7.

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