Injury Extra (2005) **36**, 546–549

www.elsevier.com/locate/inext

CASE REPORT

Necrotising fasciitis and primary sepsis caused by *Vibrio fluvialis*—A case report

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Accepted 12 May 2005

Introduction

We report a rare case of a 47-year-old fisherman with necrotising fasciitis and progressive sepsis of the right leg caused by *Vibrio fluvialis*. Fasciotomy and debridement were performed, followed by an above-knee amputation due to progressive gangrene and sepsis.

Case report

A 47-year-old male with a history of alcoholism and hepatitis B presented to the emergency room with patchy erythematous and bullous skin lesions of the right leg, accompanied by cyanosis and tenderness of the right foot. He had sustained a contusion of the right lower leg about 4 days previously in a motor-vehicle accident. The following day, he had gone to a seashore fishpond to collect fish despite the abrasion. That night, he developed multiple bullous skin lesions on the right lower leg with swelling and tenderness. He had been seen in a local hospital where he had been given antibiotic therapy for cellulitis. However, the erythematous skin lesions extended to the whole leg with progressive cyanotic change of the forefoot. Subsequently, he was referred to our hospital due to uncontrolled sepsis.

He had patchy erythema and bullous skin lesions located all the way up to the right thigh (Fig. 1). Blood pressure was 96/62 mmHg and body temperature was 36.7 °C. Tenderness and paraesthesia were noted in the right foot. Laboratory values included haemoglobin 7.9 g/dl, platelet count 11,000/μl, leukocyte count was 21,900 cells/mm^3 with 4.5% band neutrophils. Aspartate aminoiotransferase level was 128 U/l, albumin level 2.1 g/dl, and serum creatine level 1.7 mg/dl. The erythrocyte sedimentation rate was 84 mm/h, and the C-reactive protein was 249 mg/l. Necrotising fasciitis of right leg with sepsis was diagnosed. Three hours after admission, he was taken to the operating theatre for decompression. An extensive fasciotomy and debridement with removal of necrotic skin were performed. Moderate muscle necrosis and purulent discharge were also noted in the calf muscles. Broad-spectrum antibiotic therapy with vancomycin was initiated. On the third hospital day, the blood pressure and vital signs became more stable. Subsequently, wound culture confirmed *V. fluvialis* infection by Api20E test (BioMerieux, Marcy l'Etoile, France) and positive arginine dihydrolase activity. The antibiotics were changed to ceftazidime and oxytetracycline. A repeat leukocyte count was 13,100 cells/

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1572-3461
doi:10.1016/j.injury.2005.05.016

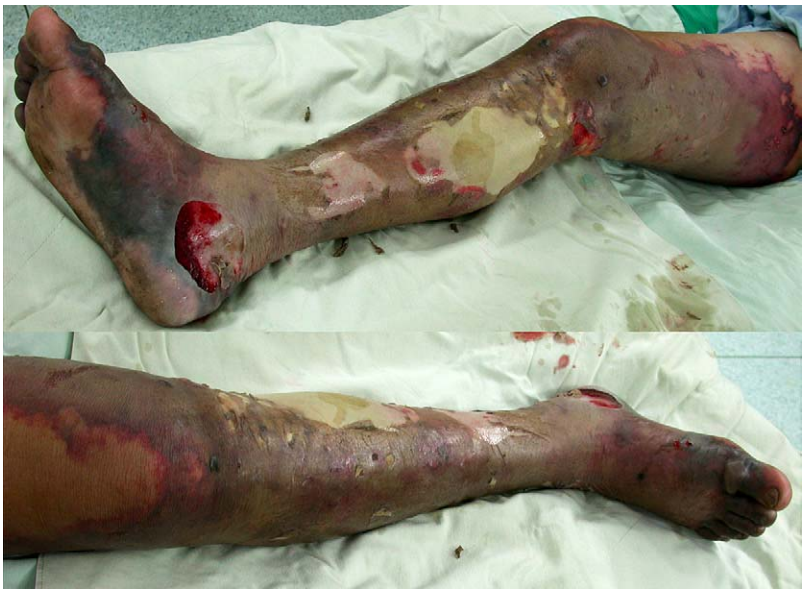

Figure 1 The right leg revealed progressive cyanotic, patchy erythematous and bullous skin lesions in the ER.

mm^3 with a differential count of 92% polymorphonuclear leukocytes, 1% lymphocytes, and 1% band forms. A second debridement was performed on the fourth day, and the culture of wound specimens of healthy-appearing tissue isolated *Pseudomonas aeruginosa*, *Acnetobacter baumannii*, and *Staphylococcus aureus*. The antibiotics were changed to imipenem and oxacillin. The wound became more painful and developed a foul odour on the sixth day, the right lower leg showed progressive digital gangrene with muscle necrosis, and the leukocyte count was 12,200 cells/mm^3 with a differential count of 82% polymorphonuclear leukocytes and 9% lymphocytes (Fig. 2). An above-knee amputation was suggested but his family refused.

On day 10, an above-knee amputation was performed owing to severe digital gangrene and continued muscle necrosis. The patient improved following repeated debridement and had delayed closure with skin grafts to cover the stump wound. He was discharged 3 weeks after the amputation.

At 1-year follow-up, he had received rehabilitative training with an orthosis and there had been no recurrent infection in this stump.

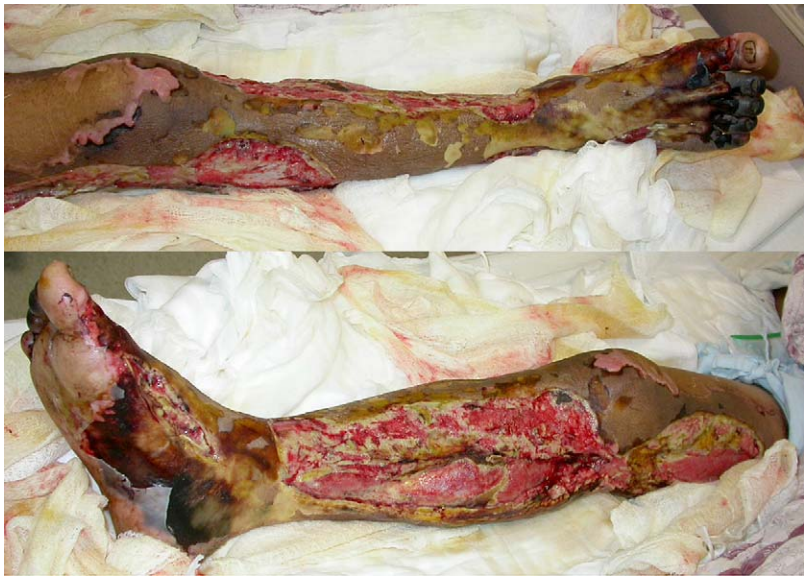

Figure 2 Three days later after fasciotomy, severe digital gangrene and continued muscle necrosis were noted.

Discussion

Several *Vibrio* species, including *Vibrio cholerae*, *V. cholerae* non-O1, *Vibrio parahaemolyticus*, *V. fluvialis*, *Vibrio furnisii*, *Vibrio hollisae*, *Vibrio mimicus*, and *Vibrio metschnikovii*, are pathogenic and capable of causing diarrhoea in human.[7,8,11] *V. fluvialis* is a halophilic bacterial pathogen and a facultative anaerobe that has been isolated from humans with diarrhoea and from the environment, such as sewage, river water, estuarine water, shellfish, crustaceans, and fish.[5–7] It has been shown to produce an enterotoxin and clinical symptoms of gastroenteritis similar to those of *Vibrio cholerae* O1 and non-O1 strains.[6,14]

Necrotising fasciitis is a surgical emergency. Early recognition and prompt aggressive debridement of all necrotic tissue is critical for survival.[12] Group A beta-haemolytic streptococci (*Streptococcus pyogens*) is the organism that usually causes fulminating necrotising fasciitis. It affects people with diabetes, minor or major trauma, or some degree of immunocompromise.[12] Recently, *Vibrio* species have been reported as causing necrotising fasciitis and septicaemia in the Gulf of Mexico and Southeast Asia, associated with minor trauma and exposure to fish, raw oyster, shellfish, crabs or seawater, especially in the summer months.[2,8,11]

The mortality of *Vibrio* necrotising fasciitis was reported up to 50% in patients who present with hypotension on admission, especially *Vibrio vulnificus* infection, and most of them died within 48 h of admission with a fulminating course.[2,11] Chuang stated that underlying chronic illness such as cirrhosis, alcoholic liver disease, gouty arthritis, chronic renal failure, diabetes mellitus, or the use of chronic steroids was present in 55% of patients with primary wound infection and 94.7% of patients with primary bacteraemia.[4]

Infections due to *V. fluvialis* most commonly present as gastroenteritis and diarrhoea.[5–7] *V. fluvialis* produces various extracellular toxic factors, such as lipase, protease, and haemolysin.[6,9,13] Baffone reported that *V. fluvialis* has weak adhesiveness and no bacterial cytotoxicity, but Wong found it had strong haemolytic and proteolytic activity.[1,13] Two cases of fatal infection due to *V. fluvialis* have been reported.[5,10] However, *V. fluvialis* rarely causes wound infection with primary septicaemia.[8,10]

Of the human *Vibrio* pathogens, *V. vulnificus* is now recognised as causing the most rapidly fatal infection. It can cause severe wound infection and sepsis in immunocompromised patients who have underlying hepatic disease or diabetes. *V. vulnificus* has extracellular toxic factors, such as haemolysin and protease.[2] *V. vulnificus* haemolysin can disrupt various eukaryotic erythrocytes, as well as mast cells. The *V. vulnificus* protease also enhances vascular permeability by activating the Hageman factor—plasma kallikrein—kinin cascade and/or exocytotic histamine release from mast cells, forming a haemorrhagic lesion that finally provokes severe dermonecrotis. Thus, the protease is the most probable candidate for oedema formation and bacterial invasion. Further, by cooperating with haemolysin, *V. vulnificus* protease is thought to play a role in supplying iron, an essential element for bacterial growth.[2]

In our patient, the route of infection was soft tissue seeding through the pre-existing abrasions during immersion in a seashore fishpond. His history of alcoholism and hepatitis B produced a relative immunocompromised state, which may have allowed the rapid spread of *Vibrio* infection and septic shock, followed by gangrene. However, the clinical course of *V. fluvialis* necrotising fasciitis was less fulminant than that of *V. vulnificus* infection in our hospital.[11]

Early detection and treatment of necrotising fasciitis are vital. This case suggests that the diagnosis of *Vibrio* necrotising fasciitis should be considered when a patient has a history of contact with seawater or seafood.

References

1. Baffone W, Citterio B, Vittoria E, et al. Determination of several potential virulence factors in *Vibrio* spp. isolated from sea water. Food Microbiol 2001;18:479—88.
2. Chakraborty S, Nair GB, Shinoda S. Pathogenic Vibrios in the nature aquatic environment. Rev Environ Health 1997;12:63—80.
4. Chuang YC, Yuan CY, Liu CY, et al. *Vibrio vulnificus* infection in Taiwan: report of 28 cases and review of clinical manifestations and treatment. Clin Infect Dis 1992;15:271—6.
5. Klontz KC, Cover DE, Hyman FN, et al. Fatal gastroenteritis due to *Vibrio fluvialis* and nonfatal bacteremia due to *Vibrio mimicus*: unusual *Vibrio* infections in two patients. Clin Infect Dis 1994;19:541—2.
6. Kothary MH, Lowman H, Mccardell BA, et al. Purification and characterization of enterotocigenic El Tor-like hemolysin produced by *Vibrio fluvialis*. Infect Immun 2003; 71:3213—20.
7. Lesmana M, Subeki DS, Tjaniadi P, et al. Spectrum of *Vibrio* species associated with acute diarrhea in North Jakarta, Indonesia. Diagn Microbiol Infect Dis 2002;43:91—7.
8. Morris Jr JG. Cholera and other types of vibriosis: a story of human pandemics and oysters of half shell. Clin Infect Dis 2003;37:272—80.
9. Rahim Z, Aziz KMS. Factors affecting production of haemolysin by strains of *Vibrio fluvialis*. J Diarrhoeal Dis Res 1996;14:113—6.
10. Tacket CO, Hickman F, Pierce GV, et al. Diarrhea associated with *Vibrio fluvialis* in the United States. J Clin Microbiol 1982;16:991—2.

11. Tsai YH, Hsu RWW, Huang KC, et al. Systemic Vibrio infection presenting as necrotising fasciitis and sepsis — a series of thirteen cases. J Bone Joint Surg 2004;86A:2497—502.
12. Wong CH, Chang HC, Pasupathy S, et al. Necrotising fasciitis: clinical presentation, microbiology, and determinants of mortality. J Bone Joint Surg 2003;85A:1454—60.
13. Wong HC, Ting SH, Shieh WR. Incidence of toxigenic Vibrios in foods available in Taiwan. J Appl Bacteriol 1992;73:197—202.
14. Yoshii Y, Nishino H, Satake K, et al. Isolation of *Vibrio fluvialis*, an unusual pathogen in acute suppurative cholangitis. Am J Gastroenterol 1987;82:903—5.