

Case Report

***Vibrio Cholerae* Necrotizing Fasciitis**Ibrahim Lasheen¹, Ahmed A Al-Aqeel¹, Sami Asfar^{2,3}¹Departments of Internal Medicine and ²Surgery, Mubarak Al-Kabeer Hospital, ³Department of Surgery, Faculty of Medicine, Kuwait University, Kuwait

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ABSTRACT

We report a rare cause of necrotizing fasciitis in our geographical area. The patient was already compromised with chronic liver disease and diabetes mellitus.

KEYWORDS: liver cirrhosis, necrotizing fasciitis, surgical debridement, *Vibrio cholerae*

INTRODUCTION

Necrotizing fasciitis (NF) is a severe life-threatening infection. It is caused by group A Beta hemolytic streptococci (*Streptococcus pyogenes*) and most commonly is polymicrobial as a synergistic infection of aerobic and anerobic organisms. It usually presents as a community-acquired infection in immunologically compromised patients (diabetes, chronic liver disease, malignancy etc).

Vibrio species have recently been recognized to cause NF in warm coastal countries in the Far East (Japan, Hong Kong, Thailand, Taiwan), Gulf of Mexico, South America, Australia and New Zealand^[1]. A literature search revealed only one series of *Vibrio* species causing NF from countries on the Mediterranean Sea in 1996^[2]. Arabian Gulf countries share the coasts of the gulf in this warm geographical area. As far as we are aware, no case of NF due to *Vibrio* species has been reported before from this area. We present the first report of such a case in a diabetic patient who also had chronic liver disease.

CASE REPORT

The patient was a 55-year old male who was known to have type 2 diabetes mellitus and chronic liver disease. He presented during August 2001 (summer time) with a two days history of fever, dyspnea and severe pain in the left leg. He stated that for the last three weeks he had developed progressive abdominal distension and swelling in both lower limbs. On clinical examination, he looked ill with acidotic type of breathing, a BP of 110/60 mmHg, a pulse rate of 110/min and a temperature 38° C. There was moderate amount of ascites and bilateral lower

limb edema. The left leg was warm and swollen with an area of redness of about 5 x 5 cm in the skin over his calf.

A complete blood picture showed: Hemoglobin 10.2 gm/dl, platelets 44 x 10⁹ /L and white blood cells 13.4 x 10⁹ /L. Arterial blood gases showed metabolic acidosis: pH 7.22 (range 7.34-7.43). Biochemistry was as follows: HCO₃ 12 mmol/L (range 22-30), blood urea 7.0 mmol/L (range 2.5 - 6.6), serum creatinine 177 µmol/L (range 60-120), serum ammonia 111 µmol/L (range 11-55). His INR was 2, total serum bilirubin 76 mmol/L (range 2-20), AST 139 iu/L (range 10-40), serum albumin 16 gm/L (range 35-47) and blood glucose 18 mmol/L (range 3.9-6.1). Ultrasound of the abdomen showed a small sized cirrhotic liver with an irregular outline and dilated portal veins with splenomegaly and moderate ascites. Samples of blood and ascitic fluid were sent to the clinical microbiology laboratory for culture.

Over the next day, a bluish discoloration developed around the erythematous area of the skin (Fig. 1a) and his leg pain became more intense. Necrotizing fasciitis was suspected. An urgent ultrasound of his leg showed patent deep veins but necrotic tissue in the deep fascia, in keeping with the diagnosis of NF. The patient at this stage had become more septic and was shifted to the ICU where he was intubated because of respiratory distress. Extensive urgent surgical debridement of the leg was performed (Fig. 1b), excising all necrotic skin and subcutaneous tissue down to the healthy deep fascia.

Necrotic tissue was also sent to the clinical microbiology laboratory. Intravenous fluids (80 ml dextrose in saline per hour), fresh frozen plasma (6

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Fig. 1a: Area of diffuse erythema with bluish discoloration spots at the center of the lesion



Fig. 1b: Extensive debridement deep to the healthy tissue

units) and platelet concentrates (6 units) were given in addition to broad-spectrum antibiotics (ceftazidime and imipenem). Following surgery, the results of ascitic fluid aspirate, blood culture and the excised tissue became available and all showed *Vibrio cholerae* non O1. This was first identified biochemically by API and VITEK identification system.

Serological confirmation showed that the stain did not agglutinate anti-serum O1 or anti-serum O139. Unfortunately, in spite of all intensive measures and inotropic support, the patient continued to deteriorate, developed multi-organ failure and died on the sixth day of admission.

DISCUSSION

Necrotizing fasciitis (NF) is a rare, rapidly progressive, and potentially fatal infection of the superficial fascia and subcutaneous tissue^[3], with sparing of skin and muscles. Giuliani *et al*^[4] divided bacteriologic culture results in necrotizing fasciitis into two distinct groups: Type I is polymicrobial and includes non-group A streptococci plus anaerobes and/or facultative anaerobes. In type II, the pathogen is group A Beta hemolytic *Streptococcus* alone or in combination with a *Staphylococcus*.

A distinct type of necrotizing fasciitis is caused by marine vibrios, particularly *Vibrio vulnificus*, *Vibrio parahaemolyticus*, and *Vibrio alginolyticus*^[3]. The usual portal of entry is a puncture wound caused by a fish or a cut wound exposed to sea water in tropical areas^[3]. *Vibrio* infections occur in hot summer months, in contrast to *Streptococcus pyogenes* infections, which tend to be contracted in wintertime^[5].

Patients are often over the age of 50 and compromised with other conditions, particularly chronic liver disease or diabetes^[6]. The clinical features of NF caused by *Vibrio* species are different

from that caused by the classical pathogens^[7]. The predominant skin lesion in *Vibrio vulnificus* presents as edema and subcutaneous bleeding with ecchymosed and purpuric skin rather than superficial necrosis as seen in NF caused by the classical pathogens^[5]. Systemically, there is fever, rigors and in severe cases, hypotension. Sepsis with *Vibrio vulnificus* is a more prominent feature and this leads to rapid multi-organ failure within 24 hours^[6]. Unlike other types, the mortality is almost 90 – 100% in NF caused by *V. Vulnificus*^[5].

Our case is the first report of *Vibrio* NF from the Arabian Gulf region. He conforms to previous reports as he presented in summertime, he was immunocompromised and he deteriorated very rapidly, but we failed to establish a history of skin puncture or ingestion of raw marine products (*e.g.*, oyster).

In both, *Vibrio*-NF and non-*Vibrio* NF the diagnosis is usually clinical when the presenting symptoms are out of proportion to the local signs. Radiological studies, including plain radiographs, CT scan, ultrasound and MRI are helpful in confirming the diagnosis^[8-10].

The goal of empiric antibiotic therapy is to assure broad coverage of aerobic Gram positive and Gram negative organisms and anaerobes. This includes the combination of a β -lactam, an aminoglycoside and anaerobic cover with either clindamycin or metronidazole^[4]. Antimicrobial therapy should then be tailored to the culture and sensitivity results. Tetracycline and ciprofloxacin are the drugs of choice for *Vibrio cholerae*. Early aggressive surgical debridement and fasciotomy have been associated with improved survival^[5]. Hyperbaric oxygen (HBO) should be considered as an adjunct treatment in patients with necrotizing fasciitis. Recent studies suggest that continuous hemofiltration and adsorption of bacterial toxins may ameliorate the clinical course of the patients

with sepsis^[11]. The early application of these measures for patients with *Vibrio*-NF may lead to a better outcome^[5].

CONCLUSION

Vibrio cholerae can be the cause of necrotizing fasciitis in countries in the Arabian Gulf region. This is especially so in patients suffering from chronic liver disease. Perhaps such patients must be advised to seek medical care as soon as they have any skin lesion associated with pain. Moreover, primary care physicians should have a high index of suspicion, if faced with such immunocompromised patients.

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