

Case Report

An aquaculture business woman suffering from necrotizing fasciitis caused by *Vibrio parahaemolyticus*, case report and literature review

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Abstract: *Vibrio parahaemolyticus*, which is halophilic gram-negative bacilli and widely distributed in the aquatic environment, including river, seawater and marine sediments, is always associated with a variety of seafood. Some strains can cause human disease, primarily gastroenteritis and diarrhea. In addition, *Vibrio parahaemolyticus* can also cause wound infection or rare fatal sepsis in patients underlying basal diseases or impaired immune function. Here, we report a case of a female patient, who suffered from necrotizing fasciitis on her lower limbs caused by *Vibrio parahaemolyticus*.

Keywords: Necrotizing fasciitis, *Vibrio parahaemolyticus*, marine organisms, treatment

Case report

A 53-year-old female had to be hospitalized because of her fever and severe pain in left leg over 31 hours. She was comatose when admitted. Admission check showed her Glasgow Coma Scale (GCS) was 5, heart rate 58 beats per minute, blood pressure 118/42 mmHg, breath 45 times per minute and temperature 37.9°C. She had no jugular vein dilation and her trachea was in the middle. The lung respiratory sound was clear, without any dry or moist rale. Her pulse was regular and heart not enlarged. There was no pathologic murmur in all auscultatory valve areas. Abdominal distention and negative shifting dullness can be checked. It's obvious that she suffered from severe edema on her lower limbs, where blisters could also be found. Results of laboratory tests showed levels of leucocyte $7.8 \times 10^9/L$, neutrophil granulocyte 90.4%, hemoglobin 143 g/L, platelet $111 \times 10^9/L$, high sensitive C-reactive protein 96 mg/L, creatinine 139 $\mu\text{mol/L}$, potassium 2.5 mmol/L, fibrinogen 1.2 g/L. And coagulation function tests showed prothrombin time

17.6 s (ratio of international standard 1.50) and partial thromboplastin time 46.9 s. Results of urine routine test showed levels of occult blood (dry chemical) +++, protein (dry chemical) \pm , alanine amino transferase 39 U/L, aspartate amino transferase 45 U/L, total protein 46.9 g/L, albumin 25.7 g/L, total bilirubin 44.9 $\mu\text{mol/L}$, urea 14.09 mmol/L, creatinine 115 $\mu\text{mol/L}$, β -hydroxybutyric acid 414 $\mu\text{mol/L}$, calcium 1.73 mmol/L, creatine kinase 782 U/L, creatine kinase MB 30 U/L, lactate dehydrogenase 262 U/L, potassium 3.22 mmol/L, troponin 10.03 $\mu\text{g/L}$. The female has suffered from varicose veins for more than 10 years but receives no treatment. She has no hypertension, heart disease or diabetes mellitus.

Tracheal intubation and ventilator-assisted respiration were performed after the female was admitted to our hospital. Meanwhile, she also received therapy including intravenous drip of piperacillin/sulbactam 5 g (Q8H) for fighting infection, norepinephrine 0.12 $\mu\text{g/kg/min}$ and dopamine 20 $\mu\text{g/kg/min}$ for maintaining blood pressure, and so on. She suffered from severe

edema on her lower limbs, where tension blisters could also be found. The patient was considered of getting necrotizing fasciitis due to symptoms on her shin of left leg including ecchymosis, nigrescence, ulceration, erythema and annular extensive blisters from the middle to the bottom. Three days after she was admitted, she got fasciotomy under general anesthesia and necrotic tissue on her left leg was cleaned up. And three days after the surgery, blister fluid was authenticated as *Vibrio parahaemolyticus* via cultivation of pathogenic microbiology. Results of drug sensitivity included tigecycline S (MIC \leq 0.5), ceftriaxone S (MIC \leq 1), inipenem-cilastatin sodium S (MIC \leq 1), levofloxacin S (MIC \leq 0.25), piperacillin/tazobactam S (MIC \leq 4). In order to fight infection, injection of levofloxacin 500.0 mg (QD) was given besides intravenous drip of piperacillin/sulbactam 5 g (Q8H), which were replaced by cefoperazone/sulbactam and levofloxacin later. Debridement and negative pressure drainage were performed on the 7th, 14th, 22nd and 25th day after the surgery. And the tissue and pus gotten from the patient were detected *Vibrio parahaemolyticus* more than once. Her edema on the lower limbs generally disappeared by therapy of anti-infection and surgery. And she was discharged from hospital after recovery, which was on the 45th day after the surgery.

Discussion

The diagnosis of necrotizing fasciitis is mainly based on six diagnostic criteria proposed by Fisher [9]: 1. extensive thanatosis of subcutaneous superficial fascia accompanying with widely stealthy tunnel, which spreads into its neighbor tissue; 2. moderate to severe systemic poisoning symptoms accompanying with obtundation; 3. without impact on muscle; 4. without clostridia in wound or blood culture; 5. without angioedema in important blood vessel; 6. extensive leukocyte infiltration founded in pathological test of debridement organization, and focal necrosis and microvascular thrombosis of fascia and neighbor tissue. The incipient symptoms of necrotizing fasciitis, which mainly includes fever, chill, asthenia and pain, are nonspecific. Patients with these symptoms are always mistakenly diagnosed as virus infection, pulled muscle or cellulitis. The results of laboratory tests for necrotizing fasciitis are also nonspecific including leukocytosis, incre-

ased blood glucose, increased creatinine, coagulation disorders and metabolic acidosis [10]. Soft tissue gas detected by plain or computed tomography occurred in 57% of patients [11]. Physical examination reveals that crepitus occurs in about 37% of patients [11].

In the view of etiology, necrotizing fasciitis can be classified into three types. The first one is caused by aerobic gram-negative bacteria, anaerobic gram-negative bacteria and gram-positive bacteria. The diabetic patients who also suffer from necrotizing fasciitis always belong to this type. The second one, which is a most common type, is mainly caused by group A streptococcus pyogenes and sometimes may be associated with secondary infection to staphylococcus. About 50% of the patients with streptococcal necrotizing fasciitis don't have marked relation to focus of infection or route of infection [12]. The third one is caused by *Vibrio* bacterial infection and it's mostly because people expose their wound to seawater contaminated by *Vibrio* bacteria. Although it's really a rare occasion, the clinical progress of this type always goes faster than the above two. Infected microorganisms release endotoxin and exotoxin which can cause damage to endotheliocyte. This damage can cause inflammation, edema, diminution of blood flow and necrosis of endotheliocyte [8]. With the development of anaerobic bacteria culture technique, anaerobic bacterium is verified as a significant pathogenic bacterium. And necrotizing fasciitis is always the result of the cooperation of aerobic bacteria and anaerobic bacteria. Bacteriological examination is significant to the diagnosis of necrotizing fasciitis. The best test sample is taken from the margin of progressive lesions and vesicular fluid and smear examination is taken. The culture of the sample are divided into aerobic and anaerobic culture.

Since mid-1990s, a new type of infection caused by *Vibrio parahaemolyticus* has been markedly increased, which may be associated with a better condition in shellfish culture for *Vibrio parahaemolyticus* [13]. Report shows that almost 3% of the patients infected by *Vibrio parahaemolyticus* died [14]. Symptoms of mild *Vibrio vulnificus* infection are swell, pain, ecchymosis and blister while symptoms of severe infection are hemorrhagic bulla, necro-

sis, gangrene and/or subcutaneous hemorrhage. Sometimes severe *Vibrio vulnificus* infection even can cause hematosepsis and eventually lead to multiple organ failure and death [15]. *Vibrio* bacteria can secrete extracellular toxin which result in the damage of soft tissue. Fasciae necrosis will reduce the blood flow of subcutaneous fascia layer, where naturally lacks blood supply. This reduction of blood flow also leads to the reduction of antibiotic concentration, thus the antibiotics can't perform antibacterial action normally. Therefore, for the treatment of necrotizing fasciitis, it's necessary to perform debridement and at the same time confirm the pathogen. Besides, patients should be treated with appropriate antibiotics during the treatment of debridement. For the patients with mild *Vibrio vulnificus* infection but without necrotizing fasciitis, tetracycline or fluoroquinolones might be sufficient for the cure of this infection and it usually takes 5 to 7 days. The patients with septicemia, potential septicemia or necrotizing fasciitis should receive combined antibiotic therapy employing third generation cephalosporin plus tetracycline or its analogues [17], which usually takes 2 weeks or more for the cure.

Our patient was engaged in marine products. She brought on an attack of necrotizing fasciitis after working 10 hours in humid condition. The samples taken from the infection site of the patient were identified as *Vibrio parahaemolyticus* for many times. We used automatic microorganism identification instrument (VITEK II) and mass spectrometer (Brukerbiotyper) to make sure that it was *Vibrio parahaemolyticus* which caused necrotizing fasciitis. Our patient was finally cured and discharged from hospital after surgery and therapy of third generation cephalosporin plus fluoroquinolone. She didn't suffer from immune function impairment of systemic or local tissue, diabetes, hypertension or nephropathy. However, she has suffered from varicose veins of lower limbs for more than 10 years. Maybe the pressure from veins forces microcirculatory system to behave abnormally and, in consequence, it leads to a series of abnormal activities including activation of vascular endothelial cell, exudation of macromolecule and erythrocyte, activation of immune system and leukopenesis. Followed by these activities is chronic inflammation, which

leads to the damage of tissue including tissue necrosis and anabrosis [18].

Necrotizing fasciitis is an internal emergency as well as surgical emergency. The diagnosis of necrotizing fasciitis is easy to be ignored because of the lack of specific clinical symptoms and laboratory indexes. The purpose of this paper is to help early diagnosis of necrotizing fasciitis, which may include confirming pathogeny through pathogenic microbiology examination early and making a therapeutic schedule appropriately.

Disclosure of conflict of interest

None.

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