A Fatal Case of *Vibrio vulnificus* Cellulitis with Septicaemia

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**CASE REPORT**

A 53-year-old Malay man presented with a one-day history of severe epigastric abdominal pain with diarrhoea and fever. No history of contact or consumption of seafood or recent activity in the sea was elicited. He had progressive bilateral pedal oedema four months prior to admission. Thorough investigations had ruled out cardiovascular dysfunction or an infective cause.

On admission, although conscious, alert and afebrile, his heart rate was 116 beats per minute with a blood pressure of 70/45 mmHg and respiratory rate of 30 per minute. Examination of the cardiovascular and respiratory systems found no other abnormality. He was dehydrated and had a decreased urine output. No organomegaly was detected but a localised tenderness at the epigastric and right hypochondriac regions together with decreased bowel sounds were noted. Per rectal examination showed yellowish stools. The patient had multiple small blisters up to the knees and crepitus at both lower limbs. An abdominal ultrasound followed by CT thorax and abdomen to rule out dissecting aneurysm only revealed dilated bowels. In the CT scan room, the patient's Glasgow Coma Scale deteriorated to 7/15 and he became haemodynamically unstable. He was resuscitated and transferred to the intensive care unit (ICU) and treated as severe septic shock, requiring inotropic support and mechanical ventilation. Full septic workout was done and he was started on broad-spectrum antibiotics (meropenem and clindamycin). The blisters on the lower limbs progressed into extensive necrotic bullae overnight. A diagnosis of necrotizing fasciitis was made, wound debridement and fasciotomy of the lower limbs were performed.

His total white cell count was within normal limits, but the neutrophil level was elevated with normal lymphocyte level, suggestive of bacterial infection. The haemoglobin was 12.9 g/dL and the platelet count was 52 x 10⁹/L. Coagulation profile was deranged with elevated liver enzymes. His serum creatinine was raised and blood urea was at the high normal range. His random blood sugar was within normal range. Radiological examination revealed bilateral haziness of the lungs. ECG showed sinus tachycardia with no other changes.

The blood specimen grew gram-negative rods after 5 hours of incubation. The antibiotics were continued but his condition deteriorated into multiorgan failure and he died 36 hours after admission despite aggressive support in ICU. *Vibrio vulnificus* was later confirmed by biochemical tests and API® 20NE (bioMérieux) identification kit. The organism was found to be sensitive to meropenem but resistant to clindamycin. Blister fluid aspirated for culture and sensitivity became infected.

**DISCUSSION**

A study on potentially pathogenic Vibrio species in seafood marketed in Malaysia between 1998 and 1999 found an overall incidence of 6.0% for *Vibrio vulnificus* in samples that included shrimps, squids, crabs, cockles and mussels. As the most virulent of the noncholera vibrios, *Vibrio vulnificus* has various virulence factors that facilitate development of clinical disease. These include capsular materials and extracellular enzymes such as metalloproteinase, lecithinase, lipase, caseinolytic protease, deoxyribonuclease, mucinase and elastase. Transmission of the organism is via ingestion of contaminated raw seafood or water and open wound exposure to seawater. No evidence of person-to-person transmission has been reported. In this previously healthy man with undetermined exposure to the organism, an accurate detailed history could not be elicited since he died before the culture results confirmed the organism. However, his symptoms of having abdominal pain and diarrhoeal illness could be incipient.

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According to the 422 infections reported to the Centers for Disease Control and Prevention (CDC) between 1988 and 1996, incidence of wound infections (45%) and septicaemia (43%) were far more common than diarrhoeal illness (5%). Patients may present with fever that rapidly progresses to sepsis which is often followed by skin or soft tissue infection that progresses from cellulitis to haemorrhagic bullae and necrotizing fasciitis. Primary septicaemia due to this organism can be rapidly fatal especially in the immunocompromised host.

Laboratory results on admission may indicate severe bacterial infection. Blood culture is required in the presence of sepsis, while additional specimens for cultures may include stools and tissues. This organism grows on MacConkey agar and thiosulphate-citrate-bile-salts agar (TCBS) and is identified by standard biochemical tests.

A combination of an antibiotic from the tetracycline group such as doxycycline with a third generation cephalosporin such as ceftazidime or cefotaxime is the first choice of treatment. However, monotherapy with ciprofloxacin is an alternative. Surgical debridement and drainage of abscesses are essential and patients with sepsis will require more intensive therapy. Despite the advances in management, the mortality rate for Vibrio vulnificus septicaemia is between 60-75%. At present, thorough cooking of seafood or avoiding consumption of raw and undercooked seafood are the most effective means of prevention.

CONCLUSION
Vibrio vulnificus should be suspected in the presence of sepsis, progressive skin and soft-tissue infections with history of raw seafood consumption or contact with seawater. However, when exposure history could not be found, the clinical syndrome appears to be distinct and should prompt this diagnosis, particularly with the initial finding of gram-negative rather than gram-positive organisms. Communication between the clinician and microbiologist is essential to ensure prompt diagnosis. Early surgical intervention and culture-directed antimicrobial therapy should be aggressively pursued in these patients, with supportive therapy in the intensive care setting.

REFERENCES