CASE REPORT

Vibrio vulnificus and V. parahaemolyticus necrotising fasciitis in fishermen visiting an estuarine tropical northern Australian location

Anna Ralph a,*, Bart J. Currie b,1

a General Medicine and Infectious Diseases, Northern Territory Department of Health and Community Services, Alice Springs Hospital, PO Box 2234, Alice Springs, NT 0871, Australia
b Menzies School of Health Research, Charles Darwin University and Northern Territory Clinical School, Flinders University, Royal Darwin Hospital, PO Box 41096, Casuarina, NT 0811, Australia

Accepted 27 June 2006

Summary Three cases of severe necrotising fasciitis due to Vibrio vulnificus (two cases) and Vibrio parahaemolyticus (one case, fatal), have occurred in Caucasian tourists while fishing at a remote tropical northern Australian estuarine area. Infections were acquired over a 4-year period during the tourist fishing season (April to July 2000–2003), when water temperatures range from 23 to 30 °C. They are notable for their geographical clustering in the remote western aspect of the Gulf of Carpentaria, an area characterised by sedimentary stratiform zinc-lead-silver deposits and a major mining operation. Patients presented with classical bul- lous cellulitis with necrotising fasciitis, accompanied by severe sepsis. Underlying risk factors were identified in each patient; in one instance, previously unrecognised haemochromatosis was diagnosed. Likely reasons for Vibrio occurrence in this particular ecological niche are discussed.

Introduction Infections caused by marine Vibrio species arise due to a combination of specific favourable environmental and host factors. In Australia, sporadic and infrequent cases of infection due to V. vulnificus, V. parahaemolyticus, V. damsela, V. alginolyticus, and non-01 V. cholerae have been reported in the literature.1–8 These include only two recognised cases of serious infection from the north of Australia, comprising fatal non-01 V. cholerae sepsis, in Aboriginal patients from the Kimberley region of North Western Australia.6 Mandatory laboratory reporting of Vibrio food poisoning in the Northern Territory has detected four cases over the last 7 years. The three cases reported

0163-4453/530 © 2006 The British Infection Society. Published by Elsevier Ltd. All rights reserved. doi:10.1016/j.jinf.2006.06.015
here, and an additional fourth recently described case from the same locality, demonstrate an apparent emerging geographical focus (Fig. 1) for acquisition of *Vibrio* infections in the Northern Territory.

**Case 1**

A 55 year old man with alcoholic cardiomyopathy, chronic liver disease and chronic airways disease, presented in July 2000 with septic shock and severe blistering leg cellulitis. The left leg was dusky and dark red below the knee, with fluid-filled blisters and areas of necrotic skin. The toe had first become swollen 3 days prior, after he had been catching fish and prawns in a tidal river (Site 1, Fig. 1).

On arrival he was shocked and had acute renal failure with creatinine of 259 μmol/L (normal range 60–120 μmol/L). Initial treatment included ticarcillin plus clavulanate, clindamycin, inotropes, non-invasive ventilation, and continuous veno-venous haemofiltration. Within 24 h of hospitalisation, guillotine amputation of the leg was required as the limb was no longer viable. Gram stain of intraoperative tissue demonstrated Gram negative bacilli, which were identified as *V. vulnificus*. Antimicrobial therapy was changed to intravenous doxycycline and ceftazidime (Fig. 2). He was managed in the intensive care unit for 20 days, and required extensive rehabilitation thereafter. He died of his underlying co-morbidities 2 years later.

**Case 2**

In May 2001, a 63 year old man with type 2 diabetes, congestive cardiac failure, atrial fibrillation (for which he was warfarinised), chronic airways disease, and a lower limb ulcer (present for approximately 1 week), went fishing in seawater with his legs fully immersed (Site 3, Fig. 1). Approximately 12 h later, he required emergency transfer to hospital with delirium, septic shock, transient myocardial ischaemia, acute renal failure, pancytopenia, hypoglycaemia, coffee-grounds vomiting and an exquisitely tender, swollen, red left lower leg.

He was treated initially with cephazolin, gentamicin, metronidazole, dextrose, noradrenaline and morphine, and was transferred to the intensive care unit for ongoing inotropic support and continuous veno-venous filtration. Gram stain of fluid aspirated from the left foot revealed Gram negative rods. Antibiotics were changed to meropenem, gentamicin and doxycycline 200 mg intravenously twice daily.

The limb was urgently debrided, with removal of necrotic fascia from the left shin and dorsum of the right foot. *V. parahaemolyticus* was isolated from the initial aspirates, and from intraoperative tissue specimens. Blood cultures were sterile. The post-operative course was complicated by progressive renal failure, liver dysfunction and gastrointestinal bleeding. He died 18 days later due to multi-organ failure.

**Case 3**

A 38 year old man with no past medical history presented in April 2003 with an exquisitely painful, swollen lower limb, fever and hypotension. The foot had become painful and swollen on the second day of a fishing holiday. He had visited two locations (Site 2, Fig. 1) by boat, and his legs had frequent contact with water. Symptoms progressed rapidly, such that on arrival in hospital 24 hours later, multiple large bullae were seen extending from the foot to the upper medial thigh, with mottling and swelling of

![Figure 1](https://example.com/figure1.png)  
*Figure 1*  
surrounding tissue. He was mildly pancytopaenic and coagulopathic, with acute renal failure and mildly abnormal liver function.

_Vibrio_ infection was suspected clinically, and he was treated with intravenous doxycycline 100 mg twice daily and meropenem after obtaining blood and blister fluid for culture. The leg was urgently debrided from the dorsum of the left foot to the groin. _V. vulnificus_ was isolated from blister fluid and intraoperative tissue specimens. The leg was debrided 24–48 hourly for the next 10 days. Left forefoot amputation was required on day 6 of admission due to peripheral gangrene. The illness was complicated by myocardial infarction and refractory hypertension. Split skin grafting of the limb was undertaken a month later, and the patient made a full recovery.

Investigation was made of underlying risk factors. There was no evidence of diabetes, but iron studies were found to be grossly abnormal (ferritin >30 000 μg/L (normal range 15–200 μg/L), iron 28 μmol/L (normal range 12–27 μmol/L), transferrin 0.99 g/L (normal range 2.0–3.6 g/L), total iron binding capacity 25 μmol/L (normal range 42–76 μmol/L), saturation ratio 1.13 (normal range 0.2–0.5)). He was subsequently found to be homozygous for the C282Y gene mutation, consistent with haemochromatosis. He had no known family history of haemochromatosis, and once sepsis had resolved, he had no evidence of impaired hepatic function or other end-organ involvement. Cardiac function also returned to normal as documented echocardiographically.

**Discussion**

These cases are typical of _V. vulnificus_ and _V. parahaemolyticus_ necrotising fasciitis clinically, epidemiologically and geographically. _Vibrio_ infection associated with high morbidity and mortality therefore needs to be considered in visitors to this area, and appropriate public health warnings provided to those at risk. Infection with _Vibrio_ sp. may be the defining event leading to the diagnosis of an underlying risk factor, as in the instance of haemochromatosis in Case 3. The history of sea water contact and the bullous appearance assist in differentiating from other causes of necrotising fasciitis, such as Group A Streptococci (GAS). GAS necrotising fasciitis occurs at elevated frequency in the Top End of the Northern Territory (incidence 3.8 per 100 000 general population; 5.8 per 100 000 in the indigenous population), in keeping with the high community prevalence of GAS infection and post infectious sequelae.9

During preparation of this manuscript, a fourth case of _Vibrio_ infection has occurred in the same locality. Markey10 reports that a 19 year old indigenous female, treated with oral steroids for autoimmune hepatitis, complicated by cirrhosis and portal hypertension, presented with overwhelming sepsis and died within 24 h of illness onset, after swimming in a tidal river in same vicinity as these cases. Non-toxigenic _V. cholerae_ was isolated from her blood cultures.

The geographical clustering of these cases of unusual infection is particularly notable given the small number of visitors annually to this remote coastal location. Although water conditions seem favourable for _Vibrio_ growth throughout much of Australia’s northern waters, it would appear that a local environmental factor must specifically favour _Vibrio_ growth in this region. Aspects of marine climate conducive for _Vibrio_ growth include appropriate temperature, salinity, pH and the presence of zooplanktons.11–14 Despite these stipulations, _Vibrio_ species are characterised by their ability to adapt to altered marine environments, such as polluted waterways.13 This ability to evolve in accordance with external pressure is likely...
related in part to the presence of a superintegron, in a process analogous to the development of antibiotic resistance by organisms under antibiotic pressure. This has been demonstrated in soil organisms found in an area contaminated by heavy-metal containing mine tailings. The orebody in this northern Australian location is said to be one of the largest known deposits of zinc and lead in the world. Concentrations of zinc and lead in the river downstream from the mine have been noted to be twice the levels recorded upstream, and as high as 100 μg/L early in the wet season (three times the 80th percentile Australian and New Zealand Environment Conservation Council water quality trigger values). Other heavy metals including copper are similarly found in high concentration downstream. These levels are likely to be diluted in the further downstream estuarine regions where the Vibrio infections occurred, although actual heavy metal concentrations in this area are unknown.

On exposure of the host to the organism, the internal host milieu is similarly important in determining the ongoing survival of Vibrio. Specifically, iron overload, as demonstrated in Case 3, and likely present also in Case 1 as a consequence of alcoholic liver disease, provides a permissive environment for V. vulnificus proliferation, since the organism is impaired in its ability to extract iron from transferrin. Improved survival of V. vulnificus therefore occurs in blood with higher ferritin concentration and transferrin iron saturation. The influence of environmental heavy metals on Vibrio growth ex vivo is less well described. It would appear that zinc at least is among its important constitutive minerals, as zinc-containing metalloproteases, contributing to pathogenic potential, are elaborated by Vibrio. A link between Vibrio infections and local environmental perturbation is speculative only, but it would appear possible that altered heavy metal water content in the area could have the potential to affect Vibrio population density and hence risk of human infection.

The epidemiology of infection with environmental organisms will continue to evolve in the current era of climate change, as both small and large scale influences alter local microbial ecology, and bring about changes in the patterns of human–pathogen interactions. Understanding microbial environmental growth requirements is therefore important not only in explaining current patterns of disease occurrence, but in anticipating future change.

Acknowledgements

We would like to thank Dr Phillip Carson, Dr Dianne Stephens, and Professor Nick Anstey for providing clinical care, Dr Gary Lum and the microbiology laboratory staff at Royal Darwin Hospital for bacterial identification, and Mr David Ralph for geological and mining advice, and for creating Fig. 1.

References