Diarrhoea, fever, shock and bullous skin lesions after ingestion of raw oysters

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A 38-year-old man presented to the emergency department with a 2-day history of cramping abdominal pain, severe vomiting, diarrhoea, and fever with chills. He reported eating a large quantity of raw oysters at a local restaurant, one day prior to the onset of symptoms. The patient had a significant history of daily alcohol consumption (12 cans of beer and one bottle of wine) for many years. On examination, the patient appeared toxic, with a systolic blood pressure of 90 mmHg, pulse of 116 beats/min, respiration rate of 22 breaths/min and an oral temperature of 39°C. Right basal crackles were present on auscultation of the chest. Cardiovascular examination showed tachycardia with normal heart sounds. Abdominal examination revealed a tense abdomen without evidence of ascites, with diffuse tenderness and hyperactive bowel sounds. The patient's skeletal muscles were tender to palpation and movement. Joint examination revealed no evidence of synovitis. A few hours after admission, multiple 1–2 cm skin lesions were noted predominantly on the truncal area (figure). Over the next 24 hours, these evolved into haemorrhagic bullae with purpuric centres.

Laboratory findings were significant for severe leucopenia (1000 cells/mm³), hypoalbuminaemia (1.7 g/dl), abnormal liver function tests (total bilirubin 5.3 g/dl; lactate dehydrogenase 1544 IU/l; aspartate transaminase 615 IU/l; alanine transaminase 465 IU/l), elevated creatine phosphokinase 14 600 IU/l, prolonged prothrombin time 15.9 s, INR 1.6, and lactacidaemia (4.6 mEq/l). Empiric antibiotics using a third generation cephalosporin and doxycycline were started immediately after blood cultures were drawn. However, within 8 hours of admission the patient became diaphoretic, tachypnoeic, hypoxic, and his mental status deteriorated.

Figure Skin showing early target lesions and bullae over the truncal area

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Questions

- 1 What is the diagnosis and what are the predisposing factors?
- 2 What is the treatment and prognosis?

Answers

QUESTION 1

This patient had *Vibrio vulnificus* septicaemia after ingestion of contaminated raw shell-fish. Individuals with pre-existing liver disease are at 80 times greater risk for illness and at over 200 times greater risk of death from *V. vulnificus* oyster-associated infection.¹ Patients with cirrhosis of the liver, haemochromatosis and immunocompromised states are especially susceptible.

QUESTION 2

Current recommendations include intravenous administration of doxycycline (100 mg q 12 h) and ceftazidime (2.0 g q 8 h). Early and aggressive treatment is recommended, as the case fatality rate for patients with septicaemia has been shown to increase with greater delays between illness onset and initiation of antibiotic treatment.² Fatality rates exceed 50% and are greater than 90% in patients who develop shock, even with appropriate treatment.^{1,3}

Outcome

One day after admission, blood cultures grew *V. vulnificus*, although stool cultures were negative. The organism was sensitive to all antibiotics tested, including third generation cephalosporins, tetracycline and gentamicin. Despite immediate resuscitation with intravenous fluids and appropriate antibiotics, the patient rapidly developed fulminant septicaemia, with refractory hypotension requiring vasopressors. He subsequently developed adult respiratory distress syndrome, requiring mechanical ventilation. The patient died on day 16 after a complicated hospital course.

Discussion

V. vulnificus, a halophilic, lactose-fermenting, marine organism, is known to cause two distinct clinical syndromes. The first is primary bacteraemia with secondary seeding of the soft tissues. This usually occurs in patients with chronic liver disease and a history of recent ingestion of raw oysters. The disease is rapid in onset with high fever, chills and shock as well as haemorrhagic bullous skin lesions. The second syndrome is characterised by primary wound infection after exposure to sea-water.³ The organism is aptly named 'vulnificus' (Latin for 'wounding'), since it may cause extensive soft-tissue destruction.³

V. vulnificus is known to inhabit coastal waters and estuaries throughout the world. This bacteria is found in sea-water as well as contaminated sea-food, particularly oysters, fish, shell-fish and crustaceans. Like other vibrios, *V. vulnificus* is concentrated in filter feeders, such as oysters.³ Studies have found that more than 50% of the oyster lots sampled in the US contain *V. vulnificus*. Infections are seasonal, with the peak onset of the illness from April to October in the Gulf Coast areas of the North American continent.⁴ *V. vulnificus* infec-

Learning points

- *Vibrio vulnificus* infection is usually seen in coastal areas, but can occur anywhere with ingestion of raw sea-food, particularly oysters
- it can cause serious and fatal infection in people with chronic liver disease
- fever, shock and bullous skin lesions should raise suspicion for the diagnosis
- fatality rates exceed 50% and are more than 90% in patients who develop shock
- early institution of appropriate antibiotics and surgical debridement can decrease mortality
- prevention relies upon educating patients and thorough cooking of sea-food

tions occur most commonly in persons exposed to sea-water along the Gulf of Mexico and the Southern Atlantic and Pacific coasts. Infections have also originated from other American coastal waters and from Europe, Asia, Australia and South America.⁵ A water temperature above 20°C and a saline content of 0.7–1.6‰ is required for colonisation.⁶

The organism is the most virulent of the vibrios, which may account for the high mortality in infected patients. The presence of a polysaccharide capsule may increase the organism's resistance to phagocytosis and to the bactericidal activity of human serum. *V. vulnificus* also produces a cytotoxin–haemolysin, collagenase, phospholipases and a protease that lyses elastin, thus increasing tissue penetration.³ High frequency of infection is seen in elderly men with an underlying liver disease. This is especially true of patients with cirrhosis and haemochromatosis who have an elevated serum iron concentration.⁷ Iron is essential for bacterial growth, and the ability to obtain iron from the host is essential for pathogenicity.⁸

This patient had primary bacteraemia with secondary seeding of the soft tissues as a consequence of ingesting contaminated raw oysters. The clinical course was characteristic with rapid onset of high fevers, chills and shock as well as the development of haemorrhagic bullae. He also developed myositis which is often seen with this infection. This case displayed many of the classic features seen in primary *V. vulnificus* septicaemia and had a fatal outcome.

Early treatment with antibiotics, debridement and amputation when necessary may improve survival. The duration of the antibiotic therapy depends on the clinical response of the patient. Surgical debridement and good wound care facilitate the healing of the necrotic lesions.¹

Final diagnosis

Fatal *Vibrio vulnificus* septicaemia after ingestion of raw oysters.

Keywords: *Vibrio vulnificus*; septicaemia; oysters; poisoning

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A rare but important cause of focal hepatic lesions

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A 57-year-old man presented with a 3-month history of right hypochondrial pain which was a constant dull ache often radiating to the back. There was no history of nausea, vomiting, gastro-intestinal bleeding, jaundice or weight loss. Clinical examination was unremarkable apart from hepatomegaly; the liver was felt 2 cm below the right costal margin with a vertical span of 10 cm and was smooth, firm and non-tender. There were no signs of chronic liver disease. A basic haematological and biochemical screen was normal, with the exception of an isolated rise in γ -glutamyl transpeptidase (γ -GT) at 104 IU/l (normal 10–50 IU/l). He had not taken any hepatotoxic or enzyme-inducing drugs. His admitted alcohol intake was less than 15 units per week. An abdominal ultrasound (figure 1) and computed tomography (CT) (figure 2) were done. A liver biopsy was done under ultrasound guidance (figure 3). The patient was well after 6 months and had improved with symptomatic treatment.

Figure 1 Abdominal ultrasound

Figure 2 Abdominal CT scan

Figure 3 Liver histology

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Questions

- 1 What are the findings on the abdominal ultrasound?
- 2 What are the findings on the abdominal CT scan?
- 3 What does the liver biopsy show?