



Vibrio vulnificus Septicaemia with Cutaneous Vasculitis in a Patient with Autoimmune Hepatitis: A Rare Association

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Abstract

Background: -

Vibrio vulnificus septicaemia is a rapidly progressive and potentially fatal infection, particularly in immunocompromised individuals. Autoimmune hepatitis (AIH) patients receiving azathioprine are at heightened risk of severe infections due to immunosuppressive state. *Vibrio vulnificus* infections predominantly related to marine contact or in association with seafood ingestion. Their dermal manifestation has reported to be rare.

Case summary: -

A 72-year-old female with autoimmune hepatitis on azathioprine therapy, along with various comorbidities presented with high-grade fever, altered sensorium, and hypoxia, septic shock. Initial evaluation revealed pancytopenia, acute kidney injury, elevated inflammatory markers, and gram-negative bacteraemia. Azathioprine was withheld. She was admitted to ICU - isolation, required inotropes, initiated on broad-spectrum antibiotics and received blood and blood product transfusions. Blood cultures grew *Vibrio vulnificus* (*Took longer for species identification*). antibiotics were escalated to meropenem-based therapy followed by culture-directed treatment, resulting in haematological and biochemical recovery. During hospitalization, she developed tender erythematous indurated plaques over the extremities; Her skin biopsy was suggestive of small vessel leukocytoclastic vasculitis, which responded to corticosteroid therapy with resolution of skin manifestation.

Keywords: *Vibrio vulnificus*; Septicaemia; Leukocytoclastic vasculitis; Seafood-associated infection; autoimmune hepatitis; neutropenia

Introduction

Vibrio vulnificus is recognised for causing gastroenteritis, septicaemia. [1,2] Traumatic infections can cause deep muscle infection, and cellulitis or even necrotising fasciitis.

[1] It is often found worldwide in coastal or estuarine habitats with water temperatures ranging from 9 to 31.8 degrees Celsius.

Human infections caused by *V. vulnificus* originate either through the intake of seafood or through direct penetration via open wound exposure to seawater. *V.*

vulnificus infections have been documented all throughout the world. However, less than 10 cases of *Vibrio vulnificus* infection have been reported from India. [3,5]

Case presentation: -

A 72-year-old female patient with a co-morbidity of type 2 diabetes mellitus, hypertension, hypothyroidism, medically managed ischemic heart disease, and autoimmune hepatitis on azathioprine presented to the hospital with a two-day history of low-grade fever and chills, followed by new-onset drowsiness and confusion since a few hours preceding admission.

At the time of presentation, the temperature was 102°F and pulse rate 112/min, respiratory rate 20/min and blood pressure 90/80 mmHg, oxygen saturation was 90% on room air. On examination, she was drowsy, confused but arousable and had diffused bilateral coarse crepitations.

She was admitted in the ICU, given supplemental oxygen, she required ionotropic support to maintain hemodynamics. Suspecting an infectious aetiology, empirical broad-spectrum antibiotics - intravenous piperacillin-tazobactam and doxycycline were started.

Initial laboratory investigations showed marked pancytopenia (CBC – 6 / 550 / 0.55), mild impairment of renal function (creat -2.25). Inflammatory markers were profoundly elevated (CRP 431 mg/L, PCT 14 ng/mL). Serological testing for tropical infections and thyroid dysfunction was negative. Preliminary blood cultures revealed the growth of gram-negative organism.

In due course, severe neutropenia ensued associated with sepsis. As per neutropenia protocol, she was kept in isolation and barrier nursing was followed, and azathioprine was discontinued. Antibiotics were escalated intravenous meropenem, multiple blood and blood product transfusion were given. Filgrastim and leucovorin were also given.

The CT scan of the chest, abdomen, and pelvis suggested pulmonary oedema, Changes of chronic liver parenchymal disease, and bilateral acute pyelonephritis. [Figure.1].

Further Evaluation revealed normal C3 but low C4. Autoimmune workup, including ANA profile was negative, although the IgG level was very high

(1989.36). Vasculitis markers (P-ANCA and C-ANCA) were also negative. Her urine culture showed no evidence of infection. the final blood culture showed the growth of *Vibrio vulnificus* [Figure.2], Retrospective interrogation revealed that the patient handled and consumed seafood two days prior to the onset of symptoms, suggesting the likely source of infection. Inj.meropenem was changed to inj.ceftazidime, inj.ciprofloxacin was added and regime was continued. [Figure.2].

Gradually, her various laboratory parameters improved, including complete blood count, renal function (creatinine 1.07 mg/dL), liver enzymes (SGOT 26.1 U/L, and SGPT 36.5 U/L) and inflammatory markers (CRP 106 mg/L and PCT 0.31 ng/mL), Repeat blood culture showed clearing of the organism. During hospital stay, painful erythematous maculopapular appeared over her limbs [Figure.3]. Skin lesions increased in number; Skin biopsy showed small-vessel leukocytoclastic vasculitis. [Figure.4] She responded very well to corticosteroid therapy.

3] Discussion: -

Vibrio vulnificus infection, the progression is rapid and accounts for the second-highest number of *Vibrio*-related deaths worldwide. [7] Healthy persons often experience mild disease, while those with underlying disorders are more likely to develop severe disease.

Vibrio vulnificus possesses numerous virulence factors [3],

1. **lysine decarboxylase and manganese superoxide dismutase** – acid neutralization and reduction of oxidative stress
2. A **surface lipoprotein, a flagellum and pilli** – mediate the systemic inflammatory response syndrome.
3. **Capsular polysaccharide (CPS)** – resistance to opsonization by complement and subsequent phagocytosis by macrophages
4. **hemolysin (VvhA), VvpE, an extracellular metalloprotease, RtxA1** – haemolytic activity, tissue necrosis, production of reactive oxygen species (ROS).

Signs of sepsis may be present initially or develop subsequently, Skin findings, including erythema, ecchymosis, bullae, usually appear within 24 hours following infection. skin lesions present as severe

cellulitis with fluid-filled bullae that become haemorrhagic and progress rapidly to ulceration, necrotizing fasciitis with myonecrosis.[7] A person with gastroenteritis following consumption of material infected with *V. vulnificus* will present with focal or diffuse mild-to-moderate abdominal pain, anorexia, nausea, and vomiting that spontaneously resolves. Less common manifestations of *Vibrio vulnificus* infection include keratitis, pneumonia, pyogenic spondylitis, meningoenzephalitis, bacterial peritonitis, and endophthalmitis.[7]

A culture may be negative due to technical challenges in growing *Vibrio vulnificus*.

Surgical intervention for necrotizing skin infections has great impact on morbidity and mortality.

The CDC recommends combination therapy with intravenous ceftazidime and either a quinolone or a tetracycline.[7]

One effective antibiotic regimen consists of doxycycline 100 mg orally or intravenously twice daily for 1 to 2 weeks, combined with a third-generation cephalosporin, 1 to 2 g intravenously every 8 hours. Ciprofloxacin can be added to achieve a higher antibiotic serum concentration.[7] Recommendations for children include a third-generation cephalosporin combined with doxycycline, ciprofloxacin, or trimethoprim-sulfamethoxazole with an aminoglycoside.

Conclusion:

This case illustrates -

1. Under-reporting and Lack of awareness about the risk of *V. vulnificus* infection from seafood consumption or marine water exposure.
2. The risk of severe infection in immunocompromised individuals.
3. The significance of timely recognition and management of *Vibrio vulnificus* septicemia.

This is a case of rare but serious *Vibrio vulnificus* septicemia in elderly, immunocompromised host -

and it may reflect an altered host response due to immunosuppression. early broad-spectrum and then culture based antibiotic therapy, aggressive supportive care may be associated with recovery and potentially reducing the historically high mortality. This case emphasizes that *V. vulnificus* infection should be considered even in the absence of classical symptoms, when risk factors are present and relevant history of seafood exposure is noted.

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Figure 1. CT Chest Abdomen Pelvis plain

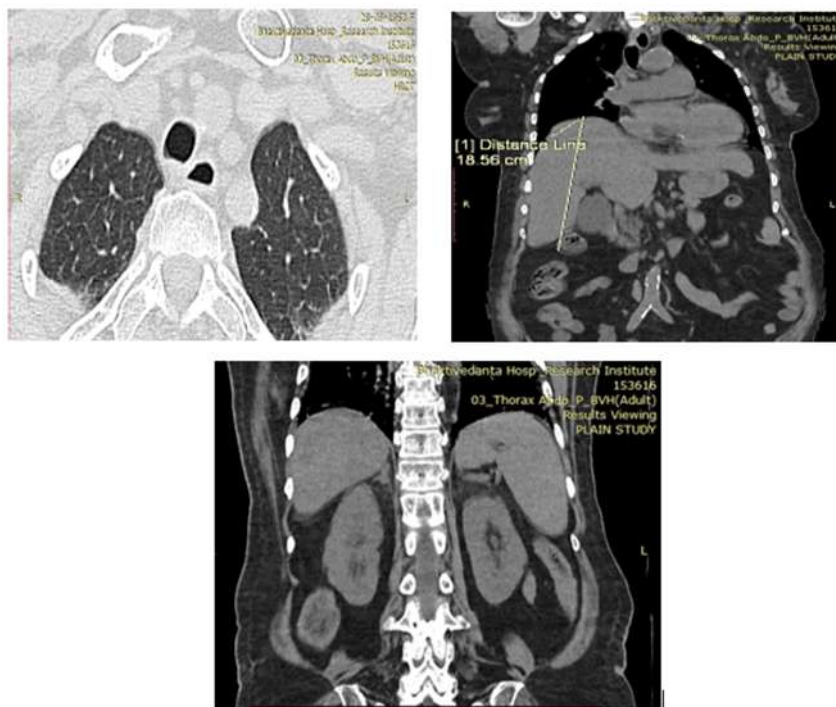


Figure 2. blood culture and sensitivity report

Identification Information		Analysis Time: 5.80 hours	Status: Final		
Selected Organism		93% Probability Vibrio vulnificus			
ID Analysis Messages		Bionumber: 5625711140720202			
Susceptibility Information		Analysis Time: 13.62 hours	Status: Final		
Antimicrobial	MIC	Interpretation	Antimicrobial	MIC	Interpretation
Amoxicillin/Clavulanic Acid	<= 2	S	Meropenem	<= 0.25	S
Piperacillin/Tazobactam	<= 4	S	Amikacin	32	I
Cefuroxime	4	S	Gentamicin	4	S
Cefuroxime Axetil			Ciprofloxacin	<= 0.06	S
Ceftriaxone			Tigecycline		
Cefoperazone/Sulbactam	<= 8	S	Fosfomycin		
Cefepime	0.25	S	Colistin	>= 16	R
Ertapenem			Trimethoprim/Sulfamethoxazole	<= 20	S
Imipenem	<= 0.5	S			

Figure 3. Erythematous and indurated plaques over the lower limb associated with tenderness



Figure 4. Histopathology of skin lesion showing leukocytoclastic vasculitis (H&E).

