

## Fulminant *Vibrio Vulnificus* Infection as the Cause of Acute overchronic Liver Failure Case Report.

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### Abstract

*Vibrio vulnificus* (VV) is a halophilic, gram negative bacterium. It is the most deadly foodborne pathogen, accounting for almost 95% of all shellfish-related deaths. It is found naturally in estuarine waters, its main host is raw or undercooked oysters, clams and shellfish.

It mainly affects men, conditions such as diabetes mellitus, liver cirrhosis, hemochromatosis, chronic kidney disease and immunosuppression are risk factors for septicemia and soft tissue infection.

A high index of suspicion is required, since an adequate therapeutic approach can reduce mortality in sepsis, the quality can be 50% in the first 24 to 48 hours.

We present the case of a 68-year-old man with a history of liver cirrhosis and seafood consumption that developed sepsis and multiple organ dysfunction of rapid evolution, with fatal outcome despite medical management.

**Palabras Clave:** Sepsis, *Vibrio vulnificus*, Bacteremia, Falla Hepática

*Vibrio vulnificus* (VV) is a halophilic, gram negative bacterium that can cause high mortality, reaching up to 50% of cases. Its main clinical presentations are limited to wound infections, septicemia and gastroenteritis. Among the most important vibrios that cause infection in humans are *Vibrio cholerae*, *V. parahaemolyticus*, *V. vulnificus*. Other species such as *V. fluvialis* and *V. mimicus* have also been reported. VV is the deadliest foodborne pathogen in the United States (US), and possibly the world. It accounts for 95% of all shellfish-related deaths in the US. It is found naturally in estuarine waters around the world, whose main hosts are raw oysters, clams, shellfish (raw or undercooked); the former represent 93% of the cases [1,2].

VV infections occur more in men than in women (86% vs 14%), apparently due to risk factors such as cirrhosis of the liver and the practice of water sports. Until now, conditions such as diabetes mellitus, liver cirrhosis, hemochromatosis, chronic kidney disease, and immunosuppression have been associated as risk factors for sepsis and soft tissue infection [3,4]. A high index of suspicion and a complete medical history (including diet and hobbies of the patients) is required to face patients with sepsis, since the therapeutic approach with the addition of doxycycline, cephalosporins or quinolones can reduce mortality in septicemia due to VV, which can be 50% in the first 24 to 48 hours.

### Presentation of the Case:

A 68-year-old male patient with a history of controlled arterial hypertension, obesity and liver cirrhosis of alcoholic etiology in CHILD PUG class B, with recent hospitalization for upper digestive bleeding of variceal origin, who was admitted to the

## Introduction

**Keywords:** Sepsis; *Vibrio vulnificus*; Bacteremia; Hepatic Insufficiency Introduction

emergency department with a clinical picture of 12 hours of evolution consisting of multiple emetic episodes of food content, associated with intermittent unquantified febrile peaks, asthenia, adynamia and abundant liquid diarrhea without mucus or blood after the ingestion of shellfish (shrimp cocktail) and alcohol, accompanied in the last 2 hours by crydiaphoresis and dyspnea, which motivates to consult. He was admitted with active systemic inflammatory response variables (Temperature: 39.5 ° C, heart rate: 90 beats per minute, respiratory rate: 20 breaths per minute), however, hemodynamically stable (Blood pressure: 130/80 mmHg), aware, alert and oriented in the three mental spheres. As positive findings on physical examination, she presented decreased vesicular murmur in both lung bases with fine crackling rales and palpable hepatomegaly up to 3 cm below the right costal margin, without pain, without signs of peritoneal irritation, or signs of neurological targeting. After this, he presented hypotension refractory to initial management with fluid therapy, before which vasopressor support with norepinephrine was started to maintain mean arterial pressure above 65mmHg and preserve tissue perfusion. The paraclinical tests and arterial gases taken from the patient upon admission, 12 and 24 hours after hospitalization, are shown in Table 1. Given the above scenario, it was considered mixed shock: hypovolemic plus septic of gastrointestinal origin with multi-organ dysfunction syndrome (SOFA Score 7), an empirical antimicrobial therapeutic approach was started with Piperacillin-Tazobactam after taking cultures; After the water expansion, gasometric improvement was evidenced, achieving a decrease in lactate levels greater than 10% in the first 6 hours. Report of total abdominal ultrasound: liver slightly enlarged, Outlined irregular, with a diffuse micronodular appearance pattern compatible with liver cirrhosis and free fluid in a cavity of approximately 450cc, without showing other ultrasound alterations.

It presents torpid evolution due to altered state of consciousness secondary to mixed encephalopathy: hepatic and septic and hypoxemic respiratory failure (PaO<sub>2</sub> / fiO<sub>2</sub> index: 142), before which the airway is secured by means of orotracheal intubation. With the results of the laboratory control that are listed in table 1, it was considered: 1) acute liver failure superaggregated with chronic liver failure (ACLF score grade 3, CLIF-C ACLF of 72 points, with probability of death in the first month 95%). 2) Worsening of chronic kidney disease with dialysis urgency data: refractory hyperkalemia, metabolic encephalopathy and anuria. Renal replacement therapy was started and, in conjunction with gastroenterology, medical treatment (thiamine, vitamin K, L-ornithine + L-aspartate, rifaximin, lactulose) was optimized and an urgent liver transplant protocol was requested. In addition, antimicrobial coverage was increased with carbapenems (Meropenem), taking into account progressive clinical deterioration and preliminary report of blood cultures with isolation of gram-negative bacilli, despite the comprehensive treatment received by the patient, he had no favorable clinical evolution, dying after 48 hours of admission to hospital. Postmortem, it was possible to confirm the strain of *Vibrio vulnificus* causing the clinical picture of sepsis and multi-organ failure.

## Discussion

The virulence of VV basically depends on three factors: its polysaccharide capsule that prevents phagocytosis by the immune system, the production of pore-forming toxin RtxA1, and the body's iron concentration. The growth of the organism in human serum is directly related to the percentage of transferrin saturation, reaching its maximum peak with saturation levels greater than 70% [6].

Taking into account the severity of the infection by this germ, survival will depend on the initial clinical suspicion and the prompt initiation of targeted treatment in patients at high risk of infection [5].

Among the risk factors associated with mortality from *Vibrio vulnificus* infection, those that have been described as main are: alcoholic liver cirrhosis, chronic viral hepatitis, alcoholism, hemochromatosis, diabetes mellitus, thalassemias major, disease chronic kidney disease, the use of TNF inhibitors and blood malignancies such as lymphomas. On this occasion, the patient had alcoholic liver cirrhosis as a risk factor for severity [2,5].

Its diagnosis is confirmed by culture. *Vibrio vulnificus* will grow without major problem in conventional culture media, such as blood agar. First-line antibiotic treatment is based on the combination of Minocycline or Doxycycline plus Ceftriaxone or Ceftazidime, improving the probability of survival with the early initiation of antibiotic treatment [5,7]. In the therapeutic approach of the case in question, no addition of these antimicrobials was made, since VV infection was not suspected at the time of initiating empirical antibiotic treatment. In this lies the disclosure of this clinical case to generate an alert and think about VV in clinical cases with rapid evolution to sepsis, liver cirrhosis and multiple organ dysfunction with a history of exposure to shellfish and seawater.

What is interesting about this case is the rapid clinical evolution with systemic compromise, which led to the presentation of acute liver failure in addition to chronic liver failure (ACLF) [8]. In the medical literature it has been proposed as an alternative in the evolution of the natural history of patients with decompensated liver cirrhosis, whose presentation is a clinical syndrome characterized by sudden worsening of liver function, with a history of disease chronic liver disease, and this is accompanied by life-threatening multisystemic dysfunction [8] [9].

Other prognostic factors include: age, leukocyte count at admission, and CLIF-C ACLF organ function score. The latter, compared with traditional risk stratification models (Child Pugh, MELD), has been shown to be superior, discriminating between patients at high risk of death who have acute liver failure over-aggregated to chronic, because it includes data from extra and intrahepatic function. However, the role played by the Child Pugh and MELD scores in defining the requirement for liver transplantation should not be underestimated [10]. The CLIF-C ACLF score was 72 points with a 95% probability of death in the first month, unfortunately in our clinical case death occurred within the first 48-72 hours, a situation that is also correlated

with the high rate of mortality in a patient with septicemia due to VV, as it was in our case.

#### Additional:

Paraclinical	Admission	12 hours	24 hours	Reference value
Leukocytes:	2080		11,700	
neutrophils:	74.50%		91.6	
Hemoglobin	12.1		12.5	
VCM	107		110	
HCM	34.5		35.1	
Platelets	64800		74,500	
Glycemia	80	127	85	Mg / dl
Sodium	141		133	Meq / L
Potassium	4.3		6	Meq / L
Chlorine	106		101	Meq / L
Calcium C	10.6			Meq / L
BUN	20		48	Mg / dl
Creatinine	1.45		4.03	Mg / dl
Bilirubin T	4.42		5.28	Mg / dl
Bilirubin D	2.68		3.61	Mg / dl
Bilirubin I	1.74		1.67	Mg / dl
Albumine	2.83			
AST	47		71	
ALT	24		30	
LDH	260		279	
TP	15.7		21.2	11.5 Seconds
INR	1.36		1.82	
pH	7.44	7.37	7.22	7.35-7.45
pCo2	25	30.9	37.6	
HCO3	17	17.7	15.3	

Lactate	7.2	6.5	6.2	
pO2	90	58.9	96.8	
BE	-5	-6.3	-9.7	

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