**BACKGROUND**

Patients with liver disease are susceptible to infection by vibrio vulnificus with exposure to raw or uncooked seafood or preexisting wounds to seawater. Vibrio is a gram-negative bacterium that can cause serious wound infections, septicemia, and gastroenteritis. Primary septicemia with secondary skin and soft tissue infection (SSTI) is usually in people with underlying risk factors such as liver cirrhosis, especially alcohol (EtOH)-induced or hepatitis B and C induced, diabetes mellitus, and chronic renal failure, all of which are immunocompromised states. The case illustrates that with risk factors, a high suspicion for liver disease is present or developed with a history of vibrio infection.

**CASE**

A 65-year-old male with a previous history of right knee osteoarthritis, EtOH abuse, and tobacco abuse presents as a transfer from an outside hospital with right lower extremity pain, swelling, blistering, and ecchymosis. The patient stated that he was hit in the right shin a week prior with a frisbee while at the beach. **Figure 1.** The patient was in septic shock due to vibrio bacteremia and the injury that developed into vibrio necrotizing SSTI of the right lower extremities. He required seven debridements and washouts, with the final washout requiring the insertion of a wound vacuum. He was also started on ceftriaxone and doxycycline for the skin infection and bacteremia, which was recommended by infectious disease. He was also incidentally found to have COVID-19, only vaccinated with one dose of the COVID-19 vaccine; however, he did not require treatment. In addition, he was found to have elevated liver function enzymes and thrombocytopenia on admission. Underlying liver disease was suspected considering the patient's long-term history of EtOH use. He completed a full course of antibiotics and was eventually discharged with a pain regimen, home health assistance, and surgery follow-up. However, he returned to the ED from the surgery clinic with concern for worsening abdominal distension for three weeks since discharge from previous admission. An ultrasound was done a few days prior to outpatient, suggesting cirrhosis with ascites and mild splenomegaly with possible portal hypertension. A CT abdomen and pelvis showed paraoesophageal varices and portal hypertension. He was admitted to the hospitalist service for a diagnostic and therapeutic paracentesis, started IV furosemide twice daily and spironolactone, started on vitamins for EtOH abuse, iron supplements, and GI was consulted for acute decompensated liver cirrhosis with ascites. During the paracentesis, 2225 ml was removed and found to be hepatitis B and C negative, and he was continued on furosemide and spironolactone at discharge with a GI follow-up. He was instructed to stop EtOH use to be eligible for a liver transplant.

**DISCUSSION**

According to the literature, a bacterial infection is one of the known precipitants to induce acute decompensated cirrhosis and EtOH-related liver injury. Patients with cirrhosis often have dysregulated immune functions, decreased complement activity, and aberrant phagocytic and chemotactic mechanisms that make them more susceptible to vibrio vulnificus infection. US CDC data further supports that chronic liver disease increases the risk of developing an infection by a factor of 80 and increases death by a factor of 20 compared to those patients without a history of chronic liver disease. Therefore, the immune response induced by vibrio likely incited the liver disease that was present all along.

**REFERENCES**


**ACKNOWLEDGEMENTS**

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