Necrotizing Fasciitis in the Context of Hepatorenal Syndrome: A Case Report

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ABSTRACT

Hepatorenal syndrome (HRS) is the impairment of kidney function secondary to advanced liver disease. One potential mechanism is kidney dysfunction induced by bacterial spread[2].

Here we report a 30-year-old female with advanced liver disease who developed HRS following the onset of necrotizing fasciitis.

This case demonstrates the possible induction of HRS via exogenous skin wound infection.

INTRODUCTION

- Hepatorenal syndrome (HRS) is the impairment of kidney function secondary to advanced liver disease[1].
- Kidney dysfunction results from hypoperfusion due to vasoconstriction of renal arteries[2].
- Splanchnic arterial vasodilation could be an intermediate mechanism, but exact etiology is not yet known. HRS remains a diagnosis of exclusion[3,4].
- The hematogenous spread of bacteria in the setting of concurrent liver trauma is proposed to be one of the mechanisms[2,5,6].
- Here we report a case of HRS following the onset of necrotizing fasciitis (NF) in a patient with preexisting advanced liver disease, suggesting that in the absence of liver trauma, spread from skin wounds may contribute to the onset of HRS.

HOSPITAL COURSE

A female in her early 30's with a past medical history of hypothyroidism, ascites and pancreatitis presented to the emergency department due to spontaneous bleeding from wounds on her bilateral flanks and legs. Blood loss was reported to be around 1 L.

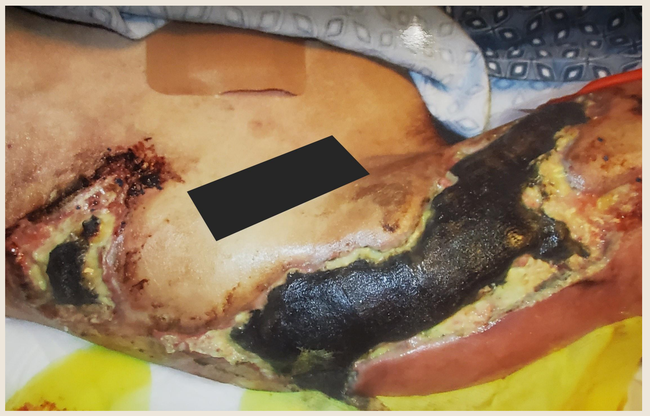
Patient has a history of alcoholism. She reported drinking 3 alcoholic beverages daily. Her home medications were morphine, fentanyl, furosemide, spironolactone, levothyroxine, and omeprazole.

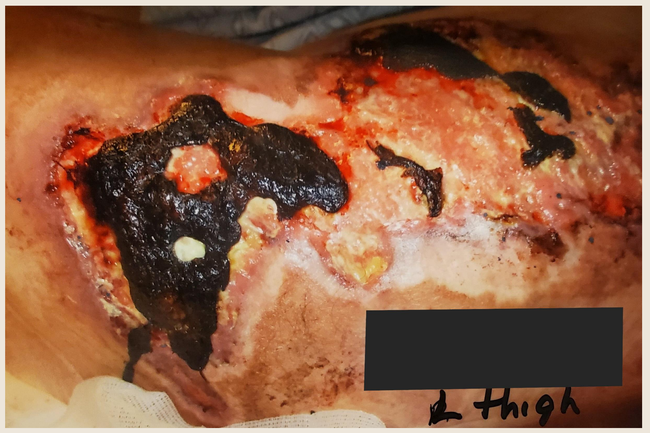
During the hospital stay, she was found to have probable alcohol cirrhosis, ascites, necrotizing fasciitis over her bilateral flanks and upper thighs (Images 1 and 2), for which she received surgical debridement and antibiotic treatment (ceftriaxone and daptomycin).

Her creatinine level rose sharply on hospital day 6 to 1.56 mg/dL from 0.72 mg/dL (Day 5). On hospital day 8, patient had a bleeding event. Her kidney function continued a downward trend despite intravenous fluid, midodrine, and cessation of diuretics. This presentation is suggestive of type 1 HRS.

DIAGNOSTICS

- CT angiogram showed skin interruptions, superficial ulcers and generalized anasarca bilaterally in the affected regions.
- Creatinine level was 0.59 mg/dL on admission, 0.72 on Day 5, 1.56 on Day 6.
- GFR was >89 since admission, and dropped sharply to 41 on Day 6, 12 on Day 18.
- Wound cultures identified Enterococcus Faecalis, Staphylococcus Aureus and Klebsiella Oxytoca.





Images 1 and 2. Skin lesions found on patient's bilateral flanks and thighs of widths between 3cm to 17cm. Wound culture positive for Enterococcus faecalis, Staphlococcus aureas, and Klebsiella oxytoca.

DISCUSSION

- The etiology of hepatorenal syndrome is not clear. Proposed mechanisms include bacteria spread from GI track.
- In this case, we provide further evidence to the proposed mechanism of bacterial induced hepatorenal failure of skin origin.
- External skin wounds may serve as a nidus for the development of HRS for patients with preexisting advanced liver disease, who are predisposed to impaired wound healing.
- Surgical debridement addresses wound healing, but does not improve kidney function.
- Acute bleeding event occurred closely to onset of rise in creatinine.
- Bacterial hematogenous spread vs. contiguous spread from adjacent sites is a subject worthy of exploration.

CONCLUSIONS

- Skin lesions and infections in patients with pre-existing advanced liver disease should be met with higher level of precaution with regards to hepatorenal syndrome.
- Early surgical debridement and barrier treatment may be desired as prophylaxis for HRS.

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