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CASE REPORT

Hyperammonemic Encephalopathy in *Staphylococcus aureus* Necrotizing Fasciitis

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This study described a rare case of necrotizing fasciitis presenting as neural dysfunction. The patient's serum ammonia level was significantly elevated; however, no liver function impairment was observed. Hyperammonemia is frequently related to the liver's ammonia metabolism failure. In literature reviews, infections also induce critical complications in specific circumstances. Herein, a rare case of *Staphylococcus aureus* necrotizing fasciitis with a hyperammonemic encephalopathy presentation was offered.

Key words: *Staphylococcus aureus*, necrotizing fasciitis, hyperammonemic encephalopathy

INTRODUCTION

Necrotizing fasciitis is a rare disease that often occurs in the extremities. This soft-tissue infection has a high mortality rate when primarily involving the superficial fascia, subcutaneous fat, and deep fascia.[1] *Staphylococcus aureus* is one of the causative organisms of necrotizing fasciitis; however, infections are rare. Prolonged hyperammonemia is a fatal condition associated with brain injury, but is uncommon in necrotizing fasciitis patients. Here, a rare *S. aureus* fasciitis case was reported in a patient who initially presented with a seizure attack.

CASE REPORT

A 79-year-old hypertensive man was sent to the emergency department due to a 2-day gradually declining disturbance of consciousness with no cerebral vascular, chronic liver, or congenital metabolic disease history. Initial oxygen saturation was 98% on room air. Body temperature was 37.8°C, heart rate was 103 bpm, respiratory rate was 21 bpm, and blood pressure was 95/50 mmHg. The Glasgow Coma Scale (GCS) score was E2M3V2. Pupils were symmetrical, 2.0 mm in size with normal light reflex. No focal neurological deficits were observed upon arrival. Physical examination revealed a poorly healing wound over the right second distal phalanx and right lower limb swelling, erythema, and crepitus was found. Lower limb computed tomography (CT) showed gas accumulation in the deep fascia from the right sciatic foramen to the right foot. Laboratory testing revealed leukocytosis (18, 600 WBC/uL, neutrophil = 86.2%) and an elevated serum C-reactive protein (25.4 mg/dL). Serum aspartate transaminase, alanine transaminase, total bilirubin, direct bilirubin, and r-glutamyl transpeptidase were within normal range. Abdominal ultrasound was negative for liver cirrhosis. Tonic seizure attacks 2 h after arrival. Unenhanced cranial CT was performed but showed negative findings. Brain magnetic resonance imaging (MRI) revealed water diffusion restriction at the deep and superficial gray matter, including the bilateral cerebral cortex, cingulate gyrus, insular cortex, basal ganglia, and thalamus on diffusion-weighted imaging [Figure 1]. After the MRI findings, serum ammonia was checked and revealed a hyperammonemic state, with the 728 μmol/L level (normal range, 0–34 μmol/L). The patient was diagnosed with acute hyperammonemic encephalopathy and right lower limb necrotizing fasciitis. Broad-spectrum antibiotics were administered and emergent debridement was performed. The serum ammonia level returned to normal after 2 days and the GCS score improved to E3M5Vt. The patient was transferred to the respiratory care ward for a ventilator

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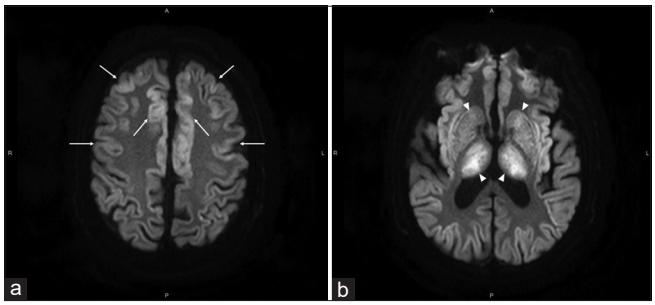

Figure 1: (a) Brain magnetic resonance imaging showing symmetrical and diffuse water diffusion restriction at the deep and superficial gray matter, including the bilateral cerebral cortex, cingulate gyrus, and insular cortex (arrow). (b) Basal ganglia and thalamus (arrowhead) on diffusion-weighted imaging

weaning program. Blood culture and the necrotic tissue were *S. aureus* positive. Recurrent pneumonia occurred and the patient succumbed 2 months later; although, the necrotizing fasciitis was controlled.

DISCUSSION

Hyperammonemia is frequently related to the liver's ammonia metabolism failure.[1,2] In nonhepatic etiology, massive muscle damage provides the nitrogen source and increases urea formation.[2] *S. aureus* is urease positive and catalyzes urea to ammonia.[2-4] Ammonia's toxic effects on the brain parenchyma may lead to progressive drowsiness, seizures, and coma, and may also progress to cerebral herniation.[5] Early hyperammonemia detection is vital in preventing permanent neurologic complications. MRI is a valuable diagnostic tool to detect hyperammonemic states.[1] Symmetric cingulate gyrus and insular cortex involvement on brain MRI is a common acute hyperammonemic encephalopathy imaging finding.[1] This is the first report of necrotizing fasciitis complicated with hyperammonemic encephalopathy. This study was presented to remind clinicians that hyperammonemic encephalopathy can be an initial presentation in severe skin and soft-tissue infections.

CONCLUSION

This case reports a specific pattern of MRI findings and alerts clinical specialists and radiologists that hyperammonemia can lead to significant brain injury and long-term sequelae. Necrotizing fasciitis will also lead to hyperammonemic encephalopathy. Early clinical feature recognition, laboratory data, and hyperammonemic encephalopathy imaging studies are important in providing prompt management and preventing fatal complications.

Declaration of patient consent

The authors certify that all appropriate patient consent forms have been obtained. The patient has provided his consent for his images and other clinical information to be reported in the journal. The patient acknowledges that his name and initials will not be published and due efforts will be made to conceal his identity; however, anonymity cannot be guaranteed.

Data availability statement

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

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Conflicts of interest

There are no conflicts of interest.

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