

High Altitude Liver Failure: An Infrequent Trigger

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Rapid ascent to high altitude imposes a state of relative hypoxia, commonly affecting the brain and lungs, which is referred to as high altitude cerebral edema and pulmonary edema, respectively. However, this is not limited to these organs and could involve others as well.¹ We encountered one such case wherein liver was predominantly involved causing acute liver failure.

A 54-year-old gentleman, a known case of chronic liver disease of autoimmune etiology for 1 year had a visit to Amarnath Yatra by walking. On day 1 of the pilgrimage trip, he had complaints of worsening dyspnea and vague abdominal pain for a day. He denies any history of trauma, insect bite, or contact with contaminated water. His personal history was unremarkable. One month prior to current admission, his conjugated bilirubin was 2.6 mg/dL, SGOT was 116 IU/L, SGPT was 56 IU/L, and his kidney function was within range (creatinine 0.84 mg/dL). He was immediately taken to a nearby hospital for emergency care and was airlifted to Hyderabad and reached our center almost 36 hours after the onset of symptoms. On arrival to our emergency services, he was drowsy and arousable. His blood pressure was 100/60 mm Hg on noradrenaline at 0.05 µg/kg/min, heart rate was 80 beats/min, temperature was 36.8 °C, respiratory rate was 17 bpm, and saturation on room air was 98%. In addition, he had muscle pain and soreness, diminished skin turgor, dry oral mucosa, and high colored urine. Fluid resuscitation was done, and he was started on empirical antibiotics after sending cultures, which later reported as sterile. Urine analysis showed occult blood with proteinuria. Blood analysis revealed high anion gap metabolic acidosis with a normal anion gap metabolic acidosis with delta ratio of 0.66. Cardiac evaluation revealed moderate left ventricular dysfunction. Tropical workup panel (dengue, malaria, leptospirosis, scrub typhus) was negative. His liver parameters were suggestive of hepatocellular pattern with conjugated hyperbilirubinemia (8.2 mg/dL) with SGOT at 511 IU/L, SGPT at 153 IU/L, and CPK at 9500 IU/L. His renal parameters suggested acute kidney injury (KDIGO-3) and hyperkalemia (5.6 mg/dL), with serum calcium 8 mg/dL and phosphorus at 7.6 mg/dL. Invasive lines were secured and his shock was worsening and managed appropriately with vasopressor support. A CT abdomen report indicated chronic liver disease with mild ascites, edematous cecum and ascending colon. He was planned for an emergent liver transplant, but liver function was downtrending due to acute liver failure by encephalopathy,

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coagulopathy, and multi-organ dysfunction, and he succumbed on day 8 of ICU stay.

Introspecting the etiology of his acute decompensation, we could explain it as high altitude (>17,000 ft) induced hypobaric hypoxia with physical exertion as the inciting event leading to rhabdomyolysis.^{2,3} This is in turn secondary to release of ALT from the skeletal muscles, in addition to the effects of hypoxia on the liver, precipitating acute liver failure and dyspnea, from which the patient was symptomatically better upon descending.² Understanding that these subset of patients are prone to develop sepsis and septic shock, we attribute the rhabdomyolysis to have triggered acute decompensation further possibly complicated by sepsis.⁴

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