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FROM HEPATITIS TO HYPOXIA- An atypical case of Hepatitis A presenting with Methemoglobinemia.

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Abstract
Background: Acute viral hepatitis A is typically a self-limiting illness with rare extrahepatic complications. We report a 31-year-old male who presented with fever, weakness, and dark urine, and was diagnosed with acute hepatitis A based on positive IgM serology and marked transaminitis. During hospitalization, he developed acute onset breathlessness with central cyanosis and low oxygen saturation (SpO 88%) despite normal arterial oxygen tension (PaO 99 mmHg), indicating a saturation gap. Imaging ruled out pulmonary pathology. Laboratory evaluation revealed progressive anaemia and elevated lactate dehydrogenase, suggestive of haemolysis. Methemoglobinemia was suspected and confirmed. Further workup demonstrated underlying glucose-6-phosphate dehydrogenase (G6PD) deficiency. The patient was managed with blood transfusions, intravenous vitamin C, and supportive care, with avoidance of methylene blue. This case highlights the importance of recognizing. Methemoglobinemia in patients with unexplained hypoxia and haemolysis, particularly in the setting of G6PD deficiency.

Keywords: Hepatitis A; Methemoglobinemia; G6PD deficiency; Haemolysis; Saturation gap; Cyanosis.

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INTRODUCTION
Acute viral hepatitis A, caused by the hepatitis A virus (HAV), remains a significant cause of acute hepatitis in developing countries. The disease is typically self-limiting and rarely progresses to fulminant hepatic failure, which occurs in less than 1% of cases. Most patients recover completely with supportive care.

Although hepatic involvement predominates, extrahepatic manifestations have been described, including renal dysfunction, dermatological manifestations, and haematological abnormalities. Among these, haemolysis is an uncommon complication and is usually mild and transient. However, in the presence of underlying red blood cell enzymatic defects such as glucose-6-phosphate dehydrogenase (G6PD) deficiency, haemolysis can be severe and clinically significant.

Methemoglobinemia is a rare but important condition characterized by oxidation of haemoglobin iron from the ferrous (Fe^{2}) to ferric (Fe^{3}). state, rendering it incapable of oxygen transport. This results in functional anaemia and tissue hypoxia. Clinically, it presents with cyanosis and hypoxia that is disproportionate to arterial oxygen levels. We present a rare case of acute hepatitis A complicated by severe haemolysis and methemoglobinemia in a patient with previously undiagnosed G6PD deficiency, presenting as unexplained hypoxia with a saturation gap.

CASE REPORT
A 31-year-old male with no known comorbidities presented with complaints of high-grade fever, generalized weakness, anorexia, and dark-coloured urine for 3–4 days. There was no history of drug intake, toxin exposure, or prior similar episodes. On examination, the patient was febrile, with evident icterus and tender hepatomegaly. There were no signs of hepatic encephalopathy or bleeding manifestations. Respiratory system examination was unremarkable, with normal vesicular breath sounds bilaterally and no added sounds. Initial laboratory investigations revealed normal haemoglobin and a markedly deranged liver function profile, with transaminases elevated (SGOT 6789 IU/L, SGPT 5890 IU/L) and elevated total bilirubin. Coagulation parameters (PT/INR) were within normal limits. Serological testing confirmed acute hepatitis

A (IgM anti-HAV positive). The patient was managed conservatively with hydration, nutritional support, and serial monitoring of liver function tests, complete blood count, and coagulation profile.

During the course of hospitalization, despite stable hepatic parameters and absence of coagulopathy, the patient developed sudden onset breathlessness on day 4. His respiratory rate increased to 33 breaths per minute, and oxygen saturation dropped to 88% on room air. He was started on supplemental oxygen and transferred to the intensive care unit. Clinical examination at this stage revealed central cyanosis.

Arterial blood gas analysis demonstrated metabolic acidosis; however, PaO was normal at 99 mmHg, indicating a discrepancy between measured oxygen saturation and arterial oxygen tension (saturation gap). Chest radiograph and high-resolution computed tomography (HRCT) of the chest were performed to rule out pulmonary causes such as pneumonia or acute respiratory distress syndrome and were found to be normal.

Further laboratory evaluation revealed a progressive decline in haemoglobin from 12 g/dL to 8.2 g/dL, along with leukocytosis (total leukocyte count 16,000/mm³) and elevated lactate dehydrogenase levels, suggestive of ongoing haemolysis. Peripheral smear findings were consistent with haemolytic anaemia. Workup for other infectious causes, including leptospirosis, scrub typhus, dengue, and other viral infections, was negative.

In view of persistent hypoxia, central cyanosis, and PaO–SpO discordance in the absence of pulmonary pathology, methemoglobinemia was suspected and subsequently confirmed on co-oximetry. Further evaluation revealed severe G6PD deficiency, explaining the susceptibility to oxidative stress–induced haemolysis and methemoglobin formation.

The patient was managed with supportive care, including packed red blood cell transfusions and intravenous vitamin C as an antioxidant therapy. Methylene blue was avoided due to the risk of worsening haemolysis in G6PD deficiency. The patient showed gradual improvement, with resolution of hypoxia, stabilization of haemoglobin levels, and recovery of liver function and was shifted to ward and discharged.

DISCUSSION

Acute hepatitis A is predominantly a benign illness; however, rare complications can pose significant diagnostic and therapeutic challenges. Haemolysis associated with acute viral hepatitis has been reported but is typically mild and self-limiting. Severe haemolysis, as seen in this case, is usually associated with underlying red blood cell disorders such as G6PD deficiency. G6PD deficiency is one of the most common enzymatic disorders of red blood cells and predisposes patients to oxidative haemolysis under stress conditions such as infections. Viral infections, including hepatitis A, can act as triggers for oxidative stress, leading to haemolysis.

Methemoglobinemia results from oxidation of haemoglobin, impairing its oxygen-carrying capacity. Normally, enzymatic pathways maintain methemoglobin levels below 1%. In conditions of increased oxidative stress or enzymatic deficiency, these pathways are overwhelmed, leading to accumulation of methemoglobin.

A key diagnostic feature of methemoglobinemia is the presence of a saturation gap, where pulse oximetry shows low oxygen saturation despite normal PaO on arterial blood gas analysis. This discrepancy should prompt consideration of dyshemoglobinemias, including methemoglobinemia and carboxyhemoglobinemia.

In the present case, the absence of pulmonary pathology, presence of haemolysis, and persistent cyanosis led to suspicion of methemoglobinemia, which was confirmed. The coexistence of G6PD deficiency further complicated management, as methylene blue—the standard treatment—is contraindicated in such patients due to the risk of exacerbating haemolysis.

Management in such cases relies on supportive therapy, including oxygen supplementation, blood transfusions, and antioxidant therapy such as vitamin C. In severe or refractory cases, exchange transfusion or dialysis may be required. This case underscores the importance of maintaining a high index of suspicion for methemoglobinemia in patients with unexplained hypoxia, particularly when accompanied by haemolysis and a saturation gap.

CONCLUSION

This case highlights a rare but important complication of acute hepatitis A in the form of methemoglobinemia associated with G6PD deficiency. The presence of unexplained cyanosis with normal arterial oxygen levels should alert clinicians to the possibility of dyshemoglobinemias. Early recognition is essential to avoid inappropriate management and to initiate appropriate supportive therapy. Screening for underlying enzymatic defects should be considered in cases of severe haemolysis in viral infections.

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3. Infectious Diseases. 2020 Sep;26(9). UpToDate [Internet]. www.uptodate.com. Available from:https://www.uptodate.com/contents/methemoglobinemia Acute viral hepatitis A is a common cause of acute hepatitis in developing countries and is usually self-limiting. Fulminant hepatic failure occurs in less than 1% of cases.
4. Extrahepatic manifestations are uncommon and include haematological, renal, and neurological complications.
5. Haemolysis is a recognized but rare complication of acute viral hepatitis and is typically mild. However, in individuals with underlying red blood cell enzymatic defects such as glucose-6-phosphate dehydrogenase (G6PD) deficiency, haemolysis may be severe.
6. Methemoglobinemia is an uncommon but important cause of hypoxia resulting from impaired oxygen delivery due to oxidation of haemoglobin.
7. We report a rare case of acute hepatitis A complicated by haemolysis and methemoglobinemia in a patient with previously undiagnosed G6PD deficiency, presenting as a saturation gap.