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ACUTE HEPATITIS A WITH UNRECOGNIZED G6PD DEFICIENCY LEADING TO ACUTE LIVER FAILURE IN CHILDREN: A CASE SERIES OF 3 SUCCESSFULLY MANAGED CASES

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ABSTRACT

Background: Hepatitis A virus (HAV) infection is usually self-limiting in children but may take a severe course when complicated by glucose-6-phosphate dehydrogenase (G6PD) deficiency, particularly when undiagnosed. **Objective:** To describe clinical presentation, management, and outcomes of three children with acute hepatitis A and previously unrecognized G6PD deficiency presenting as acute liver failure. **Methods:** Three male children aged 4, 7, and 11 years, none previously diagnosed with G6PD deficiency, presented over one year with acute liver failure. HAV infection was confirmed by ELISA in all cases. **Results:** All patients presented with vomiting, abdominal pain, jaundice, and dark urine. Common signs included pallor, jaundice, and encephalopathy. One patient presented with hypoxemia and severe anemia (hemoglobin <5 g/dL). All had bilirubin >20 mg/dL with cholestasis and liver enzymes elevated more than 25 times the upper limit of normal. LDH was >1000 IU/L in all cases. All required mechanical ventilation for hepatic encephalopathy. The highest INR was 4.8. All patients were successfully managed with supportive care and discharged in stable condition. **Conclusion:** Acute hepatitis A in children with previously unrecognized G6PD deficiency can lead to severe acute liver failure with significant hemolysis and cholestasis. Early recognition and supportive care can ensure favorable outcomes.

KEYWORDS: Hepatitis A, G6pd deficiency, Children.

INTRODUCTION

Hepatitis A virus (HAV) is generally a benign illness in children, but when complicated by glucose-6-phosphate dehydrogenase (G6PD) deficiency, the clinical course can worsen due to oxidative stress-induced hemolysis and hepatic injury. G6PD deficiency often remains undiagnosed until exposure to infections or oxidative stress.[1] Data on HAV infection presenting with unrecognized G6PD deficiency and leading to acute liver failure (ALF) in children are limited. This report describes three such cases successfully managed in a tertiary care setting.

CASE SERIES

Clinical Presentation

Three previously healthy male children aged 4, 7, and 11 years presented between the 5th and 8th day of illness. None had been previously diagnosed with G6PD deficiency.

Common presenting symptoms included: Vomiting, Abdominal pain, yellowish discoloration of eyes (jaundice) and Dark-colored urine.

Common clinical signs observed: Pallor Jaundice and encephalopathy (Grade II–III).One patient presented with hypoxemia at admission and severe anemia (hemoglobin <5 g/dL), requiring immediate blood transfusion. All tested positive for hepatitis A virus via ELISA.

G6PD enzyme assay showed deficient levels during hospitalization. Total bilirubin was >20 mg/dL in all patients, predominantly unconjugated, with cholestasis. Liver enzymes were >25 times the upper limit of normal in all three cases. Lactate dehydrogenase levels (LDH) were Elevated (>1000 IU/L) in all cases. Coagulopathy was severe with INR Ranging from 4.5 to 4.8.

Management

All patients developed hepatic encephalopathy requiring mechanical ventilation. Management included Intravenous fluids, mechanical ventilation, packed red cells, N-acetyl cysteine, and vitamin K.

Outcomes

All patients showed gradual clinical improvement. Liver enzymes, bilirubin, and INR normalized within six weeks post-discharge. All three were successfully discharged in stable condition.

DISCUSSION

This case series highlights that unrecognized G6PD deficiency can worsen the course of acute hepatitis A, leading to hemolysis, cholestasis, and severe hepatic dysfunction. Markedly elevated bilirubin levels and liver enzymes, combined with coagulopathy and encephalopathy, indicate severe disease requiring intensive care.

Hypoxemia and severe anemia in one patient further emphasize the variability and severity of clinical presentations. The need for mechanical ventilation underscores the importance of critical care support in such cases.

Early suspicion, prompt diagnosis of G6PD deficiency, and aggressive supportive management including ventilation and transfusion where necessary, are key to favorable outcomes.

CONCLUSION

Acute hepatitis A complicated by previously unrecognized G6PD deficiency can lead to life-threatening acute liver failure in children, with profound hemolysis, cholestasis, and hepatic encephalopathy. However, full recovery is achievable with timely diagnosis and supportive intensive care.

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