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An Unusual Case of Hepatitis A presenting with Prolonged Cholestatic Jaundice

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1. INTRODUCTION

Hepatitis A virus (HAV) is a non-enveloped, single-stranded RNA virus that primarily causes acute liver inflammation (1,2). Transmission typically occurs via the fecal-oral route, often through ingestion of contaminated food or water (2). Acute hepatitis A is usually a self-limiting illness, particularly in paediatric populations, with most children experiencing spontaneous resolution within two months (3). Symptoms range from mild, non-specific features such as malaise and anorexia to classical signs including jaundice, dark urine, and abdominal discomfort (4). In the majority of cases, the disease resolves without chronic sequelae, and lifelong immunity follows recovery.

Among the various clinical manifestations of hepatitis A, cholestatic hepatitis represents a rare and atypical form (5). It is characterized by prolonged jaundice, intense pruritus, elevated serum bile acids, and biochemical features consistent with intrahepatic cholestasis, but with minimal hepatocellular injury (6). This variant is more commonly observed in adults, especially older women, and is seldom reported in children. Although the clinical course of cholestatic hepatitis A may be more protracted, the overall prognosis remains favourable with supportive care.

Diagnosis of hepatitis A relies on clinical features supported by serological testing. The presence of immunoglobulin M (IgM) antibodies specific to HAV confirms acute infection (7). Recognition of cholestatic hepatitis as a possible presentation is essential, particularly when evaluating cases of persistent jaundice, to avoid unnecessary investigations and ensure appropriate management. This paper aims to highlight the clinical profile, diagnostic challenges, and outcomes of cholestatic hepatitis A, with an emphasis on its presentation in the paediatric population.

Case:

A 12-year-old girl, the first child of a non-consanguineous marriage with unremarkable birth and developmental history, presented with a one-month history of yellowish discoloration of the sclera, skin, and tongue, accompanied by severe pruritus (Figure 1). One week prior to the onset of these symptoms, she experienced fever, vomiting, and abdominal pain, which resolved spontaneously.

On General Examination:

General examination revealed marked jaundice and excoriation marks on both legs due to intense itching.

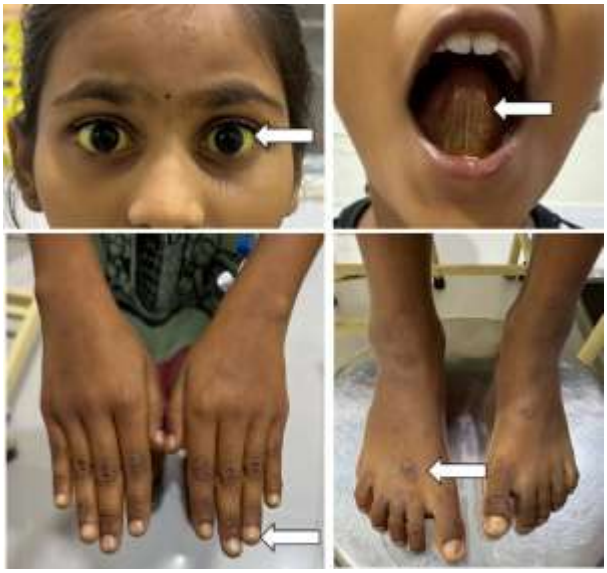

Figure 1: Clinical features of the patient

On abdominal examination:

- The liver was palpable 2 cm below the right costal margin with a span of 14 cm, smooth surface, firm consistency, and rounded margins. Grade 1 splenomegaly was present, but there were no signs of peritonitis or ascites. There were no clinical signs suggestive of hepatic encephalopathy or failure.
- Ophthalmologic evaluation ruled out the presence of Kayser-Fleischer rings.

Laboratory Investigations:

- (Table 1) revealed significantly elevated total bilirubin (TB) with a near-equal split between conjugated and unconjugated fractions.
- Liver enzymes were mildly elevated while alkaline phosphatase was markedly increased, consistent with a cholestatic pattern.
- Serum proteins and albumin were within acceptable limits.
- Coagulation profile showed a prolonged PT/INR while aPTT was normal.
- Serum ceruloplasmin was within the normal range
- Haemolytic work-up (HHH) was negative.
- This constellation of findings was suggestive of cholestatic hepatitis A, an atypical variant characterized by prolonged jaundice and pruritus in the absence of severe hepatocellular injury or failure.

Test	Value	Normal Range (if known)
Total Bilirubin (TB)	41.2 mg/dL	0.3 – 1.2 mg/dL
Conjugated Bilirubin (CB)	21.1 mg/dL	< 0.3 mg/dL
Unconjugated Bilirubin (UB)	20.1 mg/dL	—

Total Proteins	6.3 g/dL	6.0 – 8.3 g/dL
Albumin	3.2 g/dL	3.5 – 5.0 g/dL
A:G Ratio	1.03	~1.0 – 2.0
SGOT (AST)	47 U/L	10 – 40 U/L
SGPT (ALT)	28 U/L	7 – 56 U/L
ALP	337 U/L	44 – 147 U/L
aPTT	34 sec	25 – 35 sec
PT/INR	23.6 sec	PT: ~11–13.5 sec / INR: ~1.0
Serum Ceruloplasmin	60 mcg/dL	42 – 90 mcg/dL
Haemoglobin (Hb)	12.3 g/dL	12 – 16 g/dL (female)
Total Leukocyte Count (TLC)	14,600 /µL	4,000 – 11,000 /µL
HHH	Negative	—

Table 1: Investigations on admission.

Imaging:

Ultrasound revealed mild hepatomegaly with a liver span of 14.5 cm, along with borderline splenomegaly. Additionally, there were signs of gallbladder wall edema, which may reflect associated inflammatory or cholestatic changes. The remainder of the abdominal structures appeared within normal limits.

Management:

- The patient was initiated on ursodeoxycholic acid (UDCA) at a dose of 15 mg/kg/day orally, administered in two divided doses.
- Given persistent symptoms, rifampicin was added at a dose of 10 mg/kg/day orally in two divided doses.

At the time of discharge:

- The clinical condition of the patient showed gradual improvement.
- Investigations revealed a decline in total bilirubin levels, with both conjugated fractions decreasing.
- Liver enzyme levels showed mild improvement, while alkaline phosphatase (ALP) remained elevated, indicating ongoing but resolving cholestasis.
- Serum protein and albumin levels improved, suggesting recovering hepatic synthetic function.
- Coagulation profile showed a decrease in PT/INR and aPTT, reflecting stabilization of clotting parameters.
- Haemoglobin was stable at 12.6 g/dL. These trends supported the clinical diagnosis of resolving cholestatic hepatitis A with favourable progression under conservative management.

Test	At Admission	Follow-up	Change
Total Bilirubin (TB)	41.2 mg/dL	26.2 mg/dL	↓ Decreased
Conjugated Bilirubin (CB)	21.1 mg/dL	13.6 mg/dL	↓ Decreased
Unconjugated Bilirubin (UB)	20.1 mg/dL	12.6 mg/dL	↓ Decreased
SGOT (AST)	47 U/L	40 U/L	↓ Decreased slightly
SGPT (ALT)	28 U/L	24 U/L	↓ Decreased slightly

ALP	337 U/L	220 U/L	↓ Decreased
aPTT	34 sec	23 sec	↓ Improved
PT/INR	23.6 sec	19.1 sec	↓ Improved
Total Protein	6.3 g/dL	7.0 g/dL	↑ Increased
Albumin	3.2 g/dL	3.8 g/dL	↑ Increased
Haemoglobin (Hb)	12.3 g/dL	12.6 g/dL	↑ Slightly increased

Table 2: Comparison of investigation at admission and at the discharge

2. DISCUSSION

Infection by the hepatitis A virus (HAV) is predominantly asymptomatic in children and is typically self-limiting in approximately 90% of cases (2). Symptomatic manifestations occur in only a small proportion, estimated between 4–16% (8). Among these, prolonged cholestatic hepatitis A is an uncommon presentation, especially in the paediatric population. This variant is characterized by persistent jaundice, pruritus, and sometimes low-grade fever lasting for more than eight weeks, with bilirubin levels often exceeding 10 mg/dL (9).

- It is important to exclude other hepatotropic viral infections, pre-existing liver disorders, and hepatotoxic drug exposure in such cases.
- Imaging, particularly abdominal ultrasound, should show a normal biliary tree, helping to confirm intrahepatic rather than extrahepatic cholestasis.

Pathogenesis:

The pathogenesis of intrahepatic cholestasis in the context of acute viral hepatitis remains multifactorial (10).

- It may be mediated by cellular or humoral immune mechanisms that interfere with hepatocyte function or bile canaliculi, leading to impaired bile flow (6).
- Another proposed mechanism is the obstruction of bile flow secondary to periportal spotty necrosis, which disrupts normal bile excretion (11).

Given the overlap with metabolic liver disorders, such as Wilson's disease, it is essential to perform a comprehensive evaluation including 24-hour urinary copper estimation, serum ceruloplasmin levels, and ophthalmologic examination for the presence of a Kayser-Fleischer ring to rule out this differential (12).

Management:

The management of cholestatic hepatitis A involves primarily supportive and symptomatic care.

Ursodeoxycholic acid (UDCA) plays a crucial therapeutic role by enhancing bile secretion through both primary and alternative pathways and exerting anti-inflammatory effects via glucocorticoid receptor stimulation.

- It has a cytoprotective effect by replacing toxic bile acids in the bile acid pool with a less toxic, more hydrophilic bile acid. This reduces hepatocyte and cholangiocyte injury, which helps preserve liver function and bilirubin clearance (13).
- UDCA increases bile secretion, helping to flush out conjugated bilirubin more efficiently into bile and out through the intestines and also stimulates bile salt export pump (BSEP) and MRP2, aiding excretion. (13).
- UDCA also has immunomodulatory effects and reduces inflammation in the bile ducts (important in autoimmune cholestatic diseases like PBC). Decreases cytokine-mediated damage that would otherwise impair bilirubin processing and excretion.

It also improves hepatocellular transporter function by upregulating transporters like MRP2, which are involved in excreting conjugated bilirubin into bile.

(13).

- Additionally, rifampicin is used as a cholestasis-modulating agent through enzyme induction, particularly of cytochrome P450 enzymes (especially CYP3A4) which can reduce bilirubin levels and alleviate pruritus (14). Also Enhances conjugation of bilirubin by acting on UGT1A1, which is responsible for glucuronidation of unconjugated

bilirubin, making it water-soluble (conjugated form) so it can be excreted via bile. The third mechanism is via Increased hepatic clearance by increasing the expression of MRP2, which transports conjugated bilirubin from hepatocytes into bile for excretion.

- In the present case, a combination of symptomatic treatment, UDCA, rifampicin, and a high-protein diet resulted in clinical and biochemical improvement, underscoring the effectiveness of conservative management in paediatric cholestatic hepatitis A. In addition to our case, several reports from the literature have highlighted the atypical presentation of prolonged cholestatic jaundice following acute hepatitis A virus (HAV) infection, a phenomenon more commonly seen in adults but occasionally reported in paediatric populations as well (9,15–17). These cases provide insight into the clinical variability, diagnostic challenges, and therapeutic responses observed in such patients. Alebaji et al. reported a 14-year-old girl with persistent jaundice post-HAV infection failed to improve with supportive care alone. A liver biopsy revealed marked cholestasis, and with conservative management, she eventually recovered without the need for immunosuppressants. Another case reported by Jayappa et al., described the successful use of corticosteroids in a patient who had not responded adequately to ursodeoxycholic acid (UDCA), leading to a significant reduction in serum bilirubin and pruritus (18). These examples underscore the importance of considering second-line therapies in cases refractory to initial treatment.

Gordon et al., in a case series of six patients with cholestatic hepatitis A also noted similar features that include high serum bilirubin (>10 mg/dL), persistent pruritus, and a protracted clinical course lasting more than 12 weeks. All patients recovered completely without long-term complications, highlighting the self-limiting nature of the condition in most cases. However, not all cases are benign (19).

Together, these cases illustrate the diverse spectrum of cholestatic hepatitis A and reinforce the need for a tailored approach to management. In paediatric cases, as in our patient, thorough evaluation including exclusion of Wilson disease and structural biliary abnormalities—is crucial. Supportive treatment with agents such as UDCA and rifampicin, along with nutritional support, can be effective in promoting resolution and improving quality of life during the prolonged illness phase.

3. CONCLUSION

This case highlights a rare presentation of prolonged cholestatic hepatitis A in a paediatric patient. Comprehensive evaluation is important in patients with persistent jaundice following hepatitis A infection. Early recognition and diagnosis by clinical, biochemical, and radiological investigations play a critical role in timely management. The symptomatic treatment with ursodeoxycholic acid, and rifampicin, along with nutritional support resulted in favourable clinical and biochemical recovery. Timely intervention can significantly improve outcomes and prevent complications.

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