moderated [Table 1]. He had no biochemical evidence of hepatic biosynthetic defect with normal serum albumin levels, prothrombin time, and blood ammonia levels.

Ultrasonography showed hypoechoic liver and normal intra- and extrahepatic bile ducts. For cholestasis, in addition to UDCA, he was treated with cholestyramine (4 g BD) and rifampicin (10 mg/kg/day OD) for 12 days, which failed to elicit a clinical response. Investigations were done to detect other causes of prolonged hepatic manifestations: tests for detection of antibodies against HBV, HCV, and nuclear, smooth muscle, mitochondrial, and liver-kidney antigens were negative. The serum ceruloplasmin was 0.4 OD (normal: 0.2–0.5 OD). Oral prednisolone was then started with 40 mg/day on 55th day of illness for recalcitrant pruritis in the absence of any other chronic cholestatic liver disease. Within a week, there was a dramatic reduction in pruritus, jaundice started regressing, and hepatic tenderness subsided with the biochemical resolution of cholestasis [Figure 1 and Table 1]. The prednisolone dose was tapered to 30 mg/day after 6 weeks when the serum bilirubin levels halved, after which the dose was tapered by 10 mg every 2 weeks and steroids were withdrawn after 110 days. At follow-up, 4 months after the steroid therapy, the patient continues to be asymptomatic and has not shown any biochemical or clinical evidence of relapse.

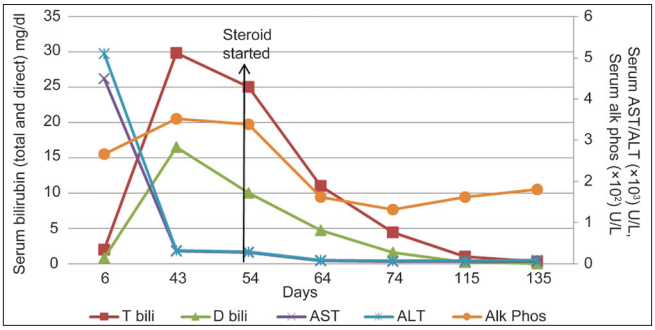

Figure 1: Serial biochemistry of patient. T bili: total bilirubin, D bili: direct bilirubin, AST: aspartate transaminase, ALT: alanine transaminase, Alk Phos: alkaline phosphatase

Prolonged Cholestasis Following Hepatitis A virus Infection: Revisiting the Role of Steroids

Sir,

A 12-year-old boy presented with complaints of progressively increasing cholestatic jaundice and intense pruritis that disturbed sleep for 6 weeks preceded by fever for 5 days. The patient was diagnosed to have hepatitis A virus (HAV) infection on the basis of raised liver enzymes and positive HAV immunoglobulin M test [Table 1] and was treated with ursodeoxycholic acid (UDCA, 30 mg/kg/day) without any success. There was no history of drug ingestion, blood transfusions, or similar episodes. He had a soft and tender liver, palpable 2cm below the right costal margin (liver span: 7cm). At presentation, the serum bilirubin level had skyrocketed to 29.9 mg/dl while serum aspartate transaminase and alanine transaminase levels had

Table 1: Results of serial biochemical tests

Investigation	Reference range	Day of illness								
		7	43*	55†	64	74	96	115	135	150
Total serum bilirubin (mg/dl)	Up to 1	2.0	29.9	25	11	4.4	1.5	1.0	0.3	0.3
Direct serum bilirubin (mg/dl)	Up to 0.3	0.8	16.5	10	4.7	1.6	0.5	0.2	–	–
Serum AST (U/l)	5-40	4500	301	270	68	51	–	58	52	37
Serum ALT (U/l)	5-40	5100	318	295	69	74	–	71	88	28
Serum protein (g/dl)	6-7.5	6.3	6.8	6.8	7.5	7.1	7.4	7.1	7.2	7.2
Serum albumin (g/dl)	3-4.5	3.6	3.1	3.5	3.8	3.9	4.2	4.0	4.1	4.2
Serum alkaline phosphatase (U/l)	0-280	266	352	338	162	132	164	162	180	130

AST: aspartate transaminase, ALT: alanine transaminase; *Day of presentation at our institution; †Day of initiation of steroid therapy

The presence of intense pruritus, elevated serum bilirubin for over 12 weeks of illness despite normalization of hepatic transaminases clinched the diagnosis of prolonged cholestatic jaundice (PCJ) following HAV infection in this child. This phenomenon is exceedingly rare in children.[1,2] It is defined as a peak serum bilirubin of more than 10 mg/dl (with direct bilirubin higher than 50% of the total bilirubin),[2] and hyperbilirubinemia or jaundice lasting for more than 12 weeks in the absence of hemolysis and renal failure with the alanine transaminase level below 500 U/l.[3-5] Till date, in children, only two reports of successful treatment of HAV-associated PCJ with corticosteroids are available in English literature. This communication intends to provide a more detailed description of therapy and response that was lacking in earlier reports.[6-8] Corticosteroids alleviate cholestasis by stimulating the alternate efflux pathway for bile salts and by anti-inflammatory action, while UDCA stimulates both normal and alternate efflux pathway and even stimulates glucocorticoid receptor, underscoring the need for their combined use for maximum response.[9,10]

It is hoped that this case would remind pediatricians that, though rare, PCJ can occur in children with HAV infection and hence extensive investigational workup, especially liver biopsy, may not be warranted. And if the child continues to have disabling symptoms even after an adequate trial of choleretic agents, oral prednisolone therapy can be tried under close supervision. This communication, being a report of a single case (without control), cannot be considered as the ultimate proof of effectiveness of corticosteroids. It is hoped that this report will spur clinicians to publish their experiences and also use various study designs to prove the efficacy of combination therapy (UDCA and corticosteroids).

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