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Case Report

Cholestatic Hepatitis A in a Jordanian Child

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Abstract

Hepatitis A infection represents the most common type of acute infectious hepatitis worldwide. Although hepatitis A is a self-limiting non-chronic infection with no specific treatment, rare complications occur. Fulminant hepatitis is the most serious. In adults and older children, persistence of hepatitis might be secondary to development of cholestatic hepatitis. Cholestatic hepatitis A presents with persistence of jaundice and intractable itching and might be mistaken for obstructive jaundice. Treatment includes ursodeoxycholic acid, rifampin and sometimes steroids. We here report a case of cholestatic hepatitis A in a 10-year-old girl, diagnosed after confirmed hepatitis A persistence, while excluding other causes of cholestasis and as made based on history of ill contacts with people having symptoms suggestive of hepatitis A, in addition to resolution of symptoms with the use of Ursodeoxycholic acid.

Keywords: *Hepatitis; Cholestasis; Hepatitis A Virus; Ursodeoxycholic Acid*

Introduction

Hepatitis A is the most common cause of infectious hepatitis worldwide. Hepatitis A is RNA virus that is highly contagious. Hepatitis A is usually transmitted through the fecal-oral route or consumption of contaminated food or water. Improvement of sanitation and personal hygiene reduced the rate of infection [1].

The clinical presentation ranges from asymptomatic in younger age-groups to having classical symptoms of abdominal pain, nausea, vomiting, poor appetite and jaundice. Hepatitis A is a self-limited disease that does not result in chronic infection. Unusual complications include fulminant, cholestatic, relapsing and autoimmune hepatitis [1]. The cholestatic variant of hepatitis A is rare in children. It shows persistent jaundice, chemical evidence of intrahepatic cholestasis and absence of substantial hepatocellular disease [2].

Purpose of the Study

The purpose of this report is to present a cholestatic type of hepatitis A in a young child, who improved on Ursodeoxycholic treatment.

Case Presentation

Two months prior to presentation, a 10-year-old girl developed abdominal pain, nausea and vomiting. Two weeks ago developed progressive scleral jaundice. Her urine darkened and the stool became pale. Her symptoms were accompanied by severe pruritus as well.

She had no change in the level of consciousness, behavior or bleeding from any sites. She had no history of blood transfusion, dental procedures or any drug ingestion prior to her symptoms. She is vaccinated for Hepatitis B according to the national program. The family denied any chronic liver disease. Six cousins and neighbors developed jaundice at same time that resolved spontaneously. As her symptoms persist; she sought medical help where a liver panel requested and showed direct hyperbilirubinemia (Total/Direct bilirubin 343.6/294.6 umol/L) with elevated transaminases; AST: 47.1 U/L and ALT: 100.6 U/L and assured her readings will improve over time.

Her jaundice progressed, her itching became so bothering and developed pyrexia and the patient was referred to our surgical department to role out cholecystitis/cholangitis process.

On admission, she looked uncomfortable. Her vital signs were stable except for being febrile. Her growth parameters were at 50th centile and 80th centile for weight and height respectively. She was jaundiced with no stigmata of chronic liver dysfunction. Her abdominal exam revealed a soft and lax abdomen with enlarged liver with smooth non-tender edge. Her liver span was 15 cm. No splenomegaly appreciated. The rest of her exam was normal. Urgent abdominal ultrasound showed no gallbladder or common bile duct stones. No wall thickening or pericholecystic fluid collection. The liver and spleen looked normal with no evidence of intra or extrahepatic biliary dilatation.

A new liver panel at our facility showed; ALT, 83.7U/L, AST, 53.9 U/L, ALK.Phos. 372U/L0 and a GGT 18U/L- Bilirubin T/D: 361/356.4 umol/L. Her clotting profile was normal. Her complete blood count was normal. No evidence of hemolysis, infection or long-standing chronic liver dysfunction. Bile salts chromatography is not available at our facility (Table 1).

	At referral hospital	At presentation	At discharge
ALT (IU/L)	100.6	83.7	39.7
AST (IU/L)	47.1	53.9	49.1
Bil T (mmol/l)	343.6	361.3	344.9
Bil D (mmol/l)	24.6	356.4	279.7
Alk. Phosphatase (IU/L)		372	331
Albumin(g/dL)		40.2	36.9
GGT(IU/L)		18	11

Table 1: *Liver panel.*

The patient underwent Magnetic Resonance Cholangiopancreatography (MRCP); confirmed biliary tree patency with no cholelithiasis.

Further work up included: immune markers and immunoglobulin, ceruloplasmin and hepatitis markers (Table 2). The only positive finding was HAV IgM. The diagnosis of cholestatic hepatitis A was suspected, the patient started on Ursodeoxycholic acid at a dose of 20

Infectious	
HAV IgM	Positive
HAV IgG	Negative
HBsAg	Negative
HBsAb	6
HCV AB	Negative
Immunological	
Total IgG	Normal for age
ANA	Negative
ASMA	Negative
AMA	Negative
Metabolic	
Ceruloplasmin	Normal
Clotting profile	
PT	13.4
INR	0.99
PTT	30.1
Radiological	
Liver ultrasound	No gallbladder stones, no obstruction
MRCP	Normal study

Table 2: *Other liver work-up.*

mg/kg/day. On follow up, her liver enzymes and bilirubin levels dropped and her itching improved dramatically. 8 weeks later phone follow up with the parents; the family reported complete resolution of the child symptoms (jaundice, abdominal pain and itching), and were not keen to do any further blood work-up to confirm normalization of her bilirubin and liver enzymes.

Discussion

Our patient was diagnosed with prolonged cholestatic hepatitis A, after confirming HAV IgM positivity while rest of work up was negative. On the same time, her bilirubin total and direct where elevated, but her transaminases were just mildly elevated.

Prolonged cholestatic Hepatitis A is extremely rare in young children. Usually presents with persistent jaundice, hepatitis symptoms and intractable pruritus [1]. It affects adults much more than young children [2]. Our patient showed persistence of her symptoms, while her peers who developed hepatitis picture resolved on the first place.

Other differential diagnosis for persistence of hepatitis symptoms might be secondary to the development of new pathology. Hepatitis A infection is capable to induce autoimmune hepatitis [3]. In our patient; Cholestatic Autoimmune hepatitis was excluded by negative markers and normal cholangiogram. On the same time radiological evaluation confirmed no obstruction.

The pathophysiological bases of prolonged cholestatic hepatitis in HAV infection is not well-understood [4]. Effect of the virus genotype were raised. HAV 1a, 1B were blamed to be responsible for such protracted course [5]. Unfortunately, this was not investigated in our patient as such testing is not available at our facility.

Host factors as carrying a pro-cholestatic genetic susceptibility were identified before as a contributing factor. Krawczyk et al reported an adult with prolonged normal GGT cholestasis triggered by hepatitis A, found to have two pro-cholestatic polymorphism. They concluded that hepatitis A, in genetically susceptible individuals could lead to sever cholestasis although of being asymptomatic previously [6]. Interestingly, in our patient her GGT was not elevated all through the course of the illness. The family denied any family history of cholestasis or prolonged hepatitis phase in other family members. This is not enough to refute or confirm this theory, but such advanced investigative tools are not available at our facility.

Ursodeoxycholic acid is a secondary bile acid. It interferes with the enterohepatic circulation reducing bile salt reabsorption of bile salts; in addition, it increases the hydrophilicity of the bile enhancing its excretion. Previous reports reported minimal improvement of itching and level of bilirubin with the use of ursodeoxycholic acid and recommended coupling it with steroids, bile salts adsorbents, enzyme inducers, like rifampicin and others [2,7,8]. In our patient; her symptoms improved with the use of URSO for eight weeks without the need of any other choleretic/anti-inflammatory agents.

Conclusion

Hepatitis A is the most viral hepatitis worldwide. It is a non-chronic self-limiting disease with no specific treatment. Persistence of cholestasis might be secondary to development of cholestasis even in young children. Ursodeoxycholic acid has a positive effect on the cholestatic symptoms and liver chemistry.

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