WW Med 2019 Vol:1 Issue:2 24-28

Worldwide Medicine

Case Report

Acute liver failure in a four-year-old child with relapse of hepatitis A

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Received: 1 April 2019 / Accepted: 9 April 2019

Abstract

Hepatitis A is one of the most common liver diseases among children worldwide and as such is the most common cause of acute liver failure (ALF) among them. Relapse may occur in 3 to 20% of the patients with acute hepatitis A, and in less than 0.5% of the cases it can cause a life-threatening complication such as acute hepatic failure. With this report, we describe a rare case of recurrence of hepatitis A in a four-year-old child with subsequent development of acute liver failure. A 35 days after treatment for acute hepatitis A and hospital discharge, the child was hospitalized again with nausea, persistent vomiting, darkening of the urine, and a prominent jaundice. Laboratory data were indicative for a recurrence of the disease. On the second day, the child became somnolent, falling asleep while eating, right-sided Babinski sign appeared. The treatment with ademetionine continued intravenously and to the therapy were added mannitol, cefoperazone, isogroup plasma infusion, vitamin K and oxygen. Within 5 days the signs of precoma gradually resolved, as the child recovered its activity and the neurological symptoms abated. Due to the lack of etiological treatment of hepatitis A and the severe course with high mortality rate of ALF, early detection of the onset of ALF and timely initiation of intensive care are needed. It must be considered the importance of hepatitis A vaccine as one of the most valuable preventive measures against possible development of ALF as a complication in the course of the disease.

Keywords: Acute liver failure; hepatitis A; hepatitis A vaccine

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DOI: 10.5455/ww.41595

Introduction

Acute liver failure (ALF) is a severe clinical syndrome associated with high mortality rate in the absence of timely treatment. The original term "fulminant hepatic failure", defined as "severe liver damage, reversible in nature and with an onset of hepatic encephalopathy within 8 weeks of the first symptoms and in absence of preexisting liver disease" is also relevant. More recent definitions point out various causes of the disease and therefore quantify the interval between the onset of symptoms and the development of encephalopathy. The interval often is shorter than eight weeks. The American Association for the Study of Liver Diseases (AASLD) pays particular attention to the prothrombin time and the mental changes in patients with acute viral hepatitis. AASLD determines ALF as a condition associated with the development of encephalopathy, together with laboratory data of coagulopathy and hepatic cytolysis without pre-existing liver disease.[1,2] The condition is rarely observed in the developed industrialized countries. Its etiology varies significantly. According to some authors, hepatitis B infection is the leading cause for ALF, followed by infections with hepatitis A or E and drug-induced ALF, most commonly by acetaminophen. In recent years it is observed a shift in the syndrome etiology. Reports of acute viral hepatitis A and B, causing ALF, declined significantly in North America and Western Europe, at the expense of toxic and autoimmune hepatitis, as well as those with unclear etiology. This is probably due to immunization programs and improved hygiene standards.[4] In the developing world, particularly the Asian countries and the Indian subcontinent, acute viral hepatitis A continues to be the leading cause of ALF.[3,5] Hepatitis A virus (HAV) causes diffuse inflammation of the liver. Most often, this is a self-limiting condition with asymptomatic or mild course that rarely becomes severe and never chronic. Relapse may occur in 3 to 20% of patients with acute hepatitis A.[6] Generally, in children and young people the symptoms are milder than in adults and the risk of developing ALF is low, about 0.1-0.4%. The virus is most often transmitted through contaminated food or water, and therefore the risk is highest in areas with low hygiene standards.[7]

Case Report

A 4-year-old girl was admitted to the Pediatric Clinic of Infectious Diseases with complaints of fatigue, loss of appetite and fever of five days duration. She has vomited twice in the last twelve hours. Contact with another child infected with hepatitis A virus was reported. Clinical examination found jaundice (++/4) and enlarged liver, about of 2 cm below the costal margin. Liver tests displayed the following: aspartate transaminase (AST), 860 U/L; alanine transaminase (ALT), 1,250 U/L; total bilirubin, 4.5 mg/dL; conjugated bilirubin, 7.9 mg/dL. The serologic test for anti-HAV-IgM was positive. The child was treated with glucose infusions and Silymarin, and discharged on day 6 after admission without jaundice and with transaminase levels as follows: AST 106 U/L, ALT 502 U/L. Her parents were advised to continue her treatment with Silymarin at home and to bring the child back for follow up exam after 30 days. After 35 days, the child appeared on a follow-up with a complaint of nausea, persistent vomiting, darkening of the urine, and a prominent jaundice. Laboratory data were indicative for a recurrence of the disease (Table 1). Markers for all types of viral hepatitis were tested, and only anti-HAV-IgM was positive. Abdominal ultrasonography showed mildly enlarged liver and spleen and a significant amount of ascites (Figure 1). The high levels of total bilirubin, transaminases and blood glucose, the values of prothrombin time and its international normalized ratio and the decreased protein synthesis, all pointed to the risk of developing ALF. Glucose infusions and oral treatment with ademetionine were initiated. On the second day, the child became somnolent, falling asleep while eating, right-sided Babinski sign appeared.

Table 1. Laboratory test results obtained during the second hospital admission of the patient.

	1st day	Day 2	Day 6	Day 11	Day 16
AST U/I	289	223	75	56	48
ALT U/I	620	602	206	113	63
Total bilirubin d/L	20.4	27.7	24.5	16.9	15.7
Alcaline phosphatases U/I	890	-	795	211	129
Total white cells count /mm3	28.000	26.300	11.390	8.601	8.711
Protombine time %	17	22	38	84	88
INR	7.03	5.16	3.8	1.83	1.52
Total blood proteins g/dL	5.1	4.8	6.2	6.8	6.6
Albumin g/dL	1.8	1.6	2.4	3.5	3.2
Blood sugar mg/dL	141	136	102	105	98

The treatment with ademetionine continued intravenously and to the therapy were added mannitol, cefoperazone, isogroup plasma infusion, vitamin K and oxygen. Within 5 days the signs of precoma gradually resolved, as the child recovered its activity and the neurological symptoms abated. The cholestasis and jaundice persisted and oral ursodeoxycholic acid was added at the eighth day of treatment. Liver function recovered, cholestasis decreased, and the child was discharged on day 17 of her hospital stay. The three consecutive follow up exams at 3 weeks intervals did not reveal any impairment in the child's liver functions.

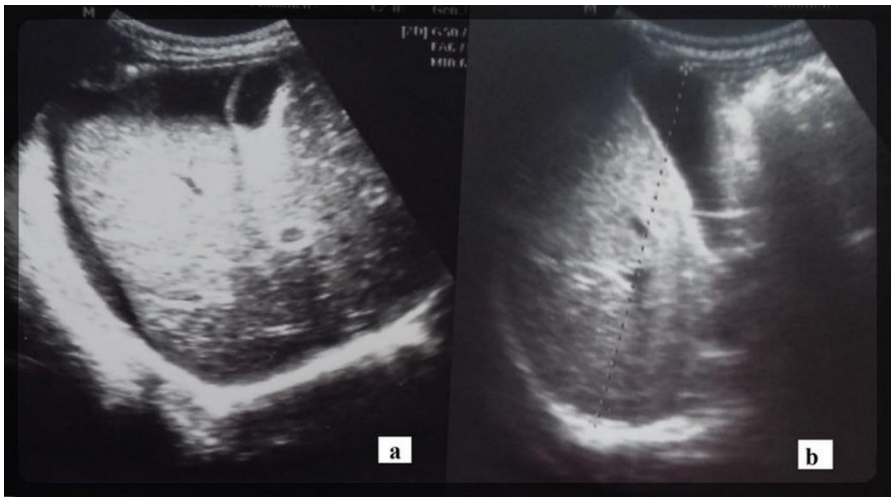

Figure 1. Abdominal ultrasonography: **a)** On day 1 of the hospital stay a large amount of perihepatic ascites is visible. **b)** On day 10 of the hospital stay the amount of ascites is markedly reduced.

Discussion

Acute liver failure is a rare but life-threatening critical illness. The clinical presentation usually includes signs of massive hepatic necrosis, jaundice, rapid transaminase elevations, impaired liver protein synthesis, coagulopathy and encephalopathy. After the onset of encephalopathy, hepatic coma often occurs, with multiorgan failure and death outcome in up to half of the cases. The most common criteria accepted by the clinicians as a sign of developing ALF are the elevated levels of ALT, blood glucose, INR and the presence of jaundice. The symptoms of encephalopathy may evolve to a hepatic coma. They are hardly recognizable in young children, with the most common in them being a weakening of the muscle strength and decreased movement activity. The children become sleepy during the day and spend most of it sleeping. Some authors also describe symptoms of agitation appearing mostly at night time.[8] The illness requires condition-specific supportive care in an intensive care unit, awaiting the liver to regain its functions. In children and young patients without pre-existing liver disease, the survival rate is higher. The main complications that can lead to a fatal outcome are severe cerebral edema, gastrointestinal bleeding and/or sepsis. In many cases, liver transplantation is the only option.[9]

As no etiological treatment is available the best way to reduce the incidence of hepatitis A-induced ALF remains the prevention of hepatitis A by vaccination. In Bulgaria, hepatitis A vaccination is recommended but it is not part of the country's mandatory immunization schedule. Because the cost of vaccination is at the expense of the patients, the vaccine is not very popular regardless the relatively high number of recorded cases of hepatitis A each year. In 2017 were reported 2,510 confirmed local cases in the country, mostly in children up to 14 years of age, and that among total of 25,156 cases for the whole European Union.[10] Most vulnerable to the disease are representatives of the Roma community in the country due to low social status, poor living conditions and insufficient health education.

Due to the lack of etiological treatment of hepatitis A and the severe course with high mortality rate of ALF, early detection of the onset of ALF and timely initiation of intensive care are needed. It must be considered the importance of hepatitis A vaccine as one of the most valuable preventive measures against possible development of ALF as a complication in the course of the disease.

Author contributions

MP, RH took care of the patient and made the literature research. VV, IK drafted the manuscript and supervised the manuscript. The final version has been read and approved by all authors.

Funding

There was no funding received for this paper.

Conflict of interest

All authors declare that they have no conflict of interest.

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