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Case Report ***Open Access***

A Case Report on Drug-Induced Hepatic Failure in a Young Child Infected with Hav

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Abstract

Hepatitis A virus (HAV) is typical enterovirus that may cause liver damage. This infection may vary in intensity from a few weeks of moderate sickness to many months of severe illness. It may sometimes lead to liver failure and death, especially in individuals with a preexisting health condition or a compromised immune system. Hepatitis A infection is most common in children in developing countries. A very tiny percentage of those who have hepatitis A may die from fulminant hepatitis or extrahepatic manifestations. One of the main causes of liver damage is an adverse drug reaction, which may require withdrawing the offending medication, being hospitalized, or even undergoing liver transplantation. The most frequent cause of acute liver failure is, in fact, drug-induced hepatotoxicity. Acetaminophen is usually used in mild analgesic or antipyretic with in-dose limitations. Use of Acetaminophen during a prodromal period of Hepatitis A may induce acute liver failure from which mild ascites and pleural effusion may develop. There have been isolated cases of these pediatric Hepatitis A symptoms occurring in isolation, but it is highly unusual for them to coexist. In this article, a young boy with Hepatitis A infection is described who had both of these pleural effusions and ascites at the same time. Hepatitis A in children with drug-induced consequences such as liver failure is carefully treated. First-care clinicians must be aware of paracetamol-induced juvenile hepatitis extrahepatic symptoms. This article recommends before fever therapy cause of fever should be excluded.

Keywords: Hepatitis A virus; acetaminophen; paracetamol; young child

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Introduction

Human hepatitis A virus (HAV) member of the picornavirus family, enterically transmitted nonenveloped RNA virus, is a significant cause of acute viral hepatitis worldwide. HAV generally acquired by the fecal-oral route via either person-to-person contact or ingestion of contaminated food or water[1]. Infecting hepatic tissue with HAV causes hepatic injury and the development of symptoms[2]. According to WHO estimates, 7134 people died globally from hepatitis A in 2016 (accounting for 0.5 percent of the mortality due to viral hepatitis).

Globally, the incidence of acute hepatitis A is decreasing owing to increased hygiene and effectively inactivated vaccines[3]. Chronic infections are not characterized, and cytotoxic T cells are primarily responsible for viral clearance. In 6.4% to 8% of cases, the noteable manifestations are : pleural effusion, ascites, arthralgia, cutaneous vasculitis, cryoglobulinemia, hemophagocytic syndrome, acalculous cholecystitis, pancreatitis, aplastic anemia, Guillane-Barre syndrome, transverse myelitis, acute

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tubular necrosis, nephrotic syndrome and vasculitis[4]. In the majority of instances, the patient may remain asymptomatic through childhood, although a minority may develop prodromal symptoms such as nausea, vomiting, and fever with or without icterus, with a mortality rate ranging from 0.3% in children to 1.8% in adults[5]. The onset of symptoms typically occurs 15 to 50 days following exposure to the Hepatitis A virus[6].

Hepatotrophic virus mainly infects the hepatocytes[7]. In this prodromal stage, if a patient develops fever, Paracetamol, commonly known as Acetaminophen, is a drug which frequently used in fever symptoms. Tylenol and Panadol are common brand name. When used according to the instructions on the prescription, paracetamol may be a safe drug for fever reduction and pain relief. The World Health Organization (WHO) and certain country-specific recommendations suggest a paracetamol dosage range of 10-15 mg/kg (up to a maximum daily dose of 60 mg/kg) for infants and children (up to 12 or 18 years of age)[8]. A lethal overdose of paracetamol may cause liver damage and be deadly at extremely high doses. Chronic use of paracetamol may cause altered liver functions or liver failure[9]. Pediatric acute liver failure (PALF) can be defined as biochemical evidence of acute liver injury in a child with no known evidence of chronic liver disease along with at least one of the following, INR more than 1.5, not corrected with vitamin K supplementation, with encephalopathy, INR more than 2.0, not corrected with vitamin K supplementation, without encephalopathy[10].

However, to our knowledge, there has only been one documented instance of a child developing ascites and pleural effusion as a result of taking paracetamol for a relatively short period. Here, we present a 4 year old boy with hepatitis A infection who developed ascites & pleural effusion together after taking a short course of paracetamol.

Case Presentation

A 4 year old boy was admitted to the Paediatric Gastroenterology department of Bangabandhu Sheikh Mujib Medical University with a history of 1 month of yellowish discoloration of eyes, sclera and urine with abdominal distension for the same duration Before that, he had a history of fever for 2 days which was intermittent in nature. The highest recorded

Table 1: Classification of dermatophytes according to morphology[3,22]

Name of Investigation	Results	Normal Value (Child)
Haematological investigations		
Haemoglobin	11.7 g/dL	11 - 13.7 g/dl (2-6 Years)
ESR	15 mm in 1st hour	00 -10(M) in 1st hour
Total count		
WBC	16.5X109/L	4.5 to 11.0X109/L (2 years to adult)
Red Blood Cells	3.98X1012 /L	4.5 to 5.5X1012/L
Platelet count	200X109/L	150 to 450X109/L
Peripheral blood film	Microcytic hypochromic anaemia with neutrophilic leukocytosis	
Liver Function Test		
Serum Billirubin		
Bilirubin (D)	14.56 mg/ dl	< 0.3 mg/dl
Bilirubin (T)	18.9 mg/ dl	0.2 to 1.2 mg/dl
Total Protein	68 gm/dl	52 to 87 gm/dl
Albumin	32 gm/dl	32 to 48 gm/dl
Alanine aminotransferase	185 U/L	10 to 49 U/L
Prothrombin time		
Control	12.00 Seconds	
Patient	15.00 Seconds	12.00 – 16.00 Seconds
Index	80.00%	
Ratio	1.25	
INR	1.26	
Viral markers		
Anti HAV IgM	Positive	
HBsAg	Negative	
Anti HCV	Negative	
Anti HEV IgM	Negative	
Radiology and Imaging		
Ultrasound of whole Abdomen	Suggestive of acute hepatitis with moderate ascites with hepatomegaly	
Chest X-ray	Mild pleural effusion on the right lung	

temperature was 101°F. The fever subsided by taking paracetamol. According to the statement of his mother, he had a history of taking Tab. Paracetamol 500 mg 6 hourly for 2 days. After that suddenly he developed yellowish discoloration of the eyes, sclera and urine with abdominal distension. Jaundice was progressive in nature. During jaundice he had no fever but jaundice was associated with nausea and vomiting. Vomitus was profuse in proportion to the quantity of food consumed but was not associated with bile or blood. He is the third child of his parents. During this time, his other family members were living a healthy life. On general examination, the patient was ill-looking, below average nutrition and , pulse 100/m, Blood Pressure 70/40 mm of Hg, Respiratory Rate 32/min, exhibited jaundice with no anaemia, cyanosis, edema, clubbing, koilonychia, leukonychia, edema, JVP not raised, no lymphadenopathy and thyromegaly. On systemic examination lip, gums, and teeth were normal but dorsum surface of the tongue was yellowish. On Abdominal examination, the umbilicus was centered and inverted. There was hepatomegaly with a liver span of 15 cm with sharp margin, a smooth surface and firm consistency accompanied by mild tenderness and fluid shift was present. There was no palpable spleen and ballotable kidney. No hepatic bruit or rub was present. On Respiratory system examination movement was slightly restricted on the lower part of right side of the chest, trachea centrally placed, Apex beat in left 4th intercostal space in the midclavicular line. Vocal fremitus was reduced in the right lower chest up to the 6th intercostal space with slightly restricted chest expansion over the right lower chest. There was stony dullness in the right lower chest up to the 6th intercostal space. Breath sound and vocal resonance were diminished on the right lower chest.

Initial investigations revealed hemoglobin 11.7 g/dL, total leukocyte count 16.5 X 10^9/L, platelet count 200 X 109/L and peripheral blood film showed microcytic hypochromic anemia with neutrophilic leukocytosis. He also had conjugated hyperbilirubinemia with a total bilirubin of 18.9 mg/ dl, alanine aminotransferase of 185 U/L, the prothrombin time is 15.00 seconds and INR 1.26. Ultrasound examination revealed suggestive acute hepatitis with moderate ascites & hepatomegaly. There was a mild pleural effusion on the right lung on the chest X-ray, but he had no complaints about it, therefore it was an incidental finding. Anti HAV-IgM antibody serology test indicated a positive etiological finding. Hepatitis B, C, and E serology tests all were negative. After admission, the child was treated with 7 days of injectable antibiotics to prevent secondary bacterial infection. He had no bleeding manifestation with INR 1.26. Intravenous vitamin K injections were used to treat this, and it gradually improved by the fourth week of sickness. Throughout the illness, the child was hemodynamically steady. He didn't have any signs of encephalopathy. He was treated cautiously without any intrusive procedures. The ascites and pleural effusion had cleared on repeat ultrasonography and chest X-ray by the third week of sickness, and the biochemical markers had steadily improved by the fourth week.

Discussion

The hepatitis A virus infection, which causes hepatitis A, causes liver inflammation (HAV). When an uninfected (and unvaccinated) individual consumes food or water that has been tainted by an infected person's feces, the virus is most commonly disseminated. Oral-anal sex, contaminated food or water, poor sanitation, poor personal hygiene, and these factors are all strongly associated to the disease[11]. Acute hepatitis A infection in children is generally mild, and extrahepatic consequences, such as those shown in this case, are very uncommon[12]. The most prevalent cause of acute liver damage is drug-induced liver injury (DILI). Usually the diagnosis of DILI is determined by determining a temporal relationship between medication exposure and the onset of liver disease symptoms and indications[13].

Paracetamol, known as acetaminophen, is a common antipyretic that has long been recognized to induce liver damage when consumed over therapeutic doses. Hepatotoxicity followed by paracetamol overdose, whether intentional or not, is the most frequent cause of DILI[9]. Acetaminophen is either glucuronylated or sulfa-conjugated to compounds that are excreted in the urine. A fraction of the drug is metabolized by certain the cytochrome P450 superfamily of enzymes to a toxic intermediate metabolite (N-acetyl-p-benzo-quinone imine, NAPQI) that can interact with intracellular proteins and induce hepatocyte death[13]. Liver damage is a complication of hepatitis infection, which can be aggravated by acetaminophen. Ascites is occurred due to portal hypertension, in which portal vein pressure is raised, and low albumin levels. Diseases which cause severe liver damage can lead to ascites. However, the exact process of pleural effusion remains uncertain. But transport of fluid from diaphragmatic lymphatics, leaking from a diaphragmatic defect, or immune complex deposition

are all possible causes of pleural effusion[4].

This case demonstrates how hepatitis A infection can be complicated by extrahepatic signs due to the use of paracetamol during the prodromal stages of the disease, as well as a success story of conservative treatmen.

Conclusion

Hepatitis A in children with drug-induced consequences, such as acute liver failure with extrahepatic symptoms, is handled with caution. This case report is significant for first care practice, and clinicians at this level must be aware of extrahepatic symptoms of paracetamol-induced juvenile hepatitis. The purpose of this article is to emphasize the random use of paracetamol after the onset of fever owing to any underlying reason. During fever therapy, underlying numerous reasons should be considered.

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Conflict Of Interest

The authors have no conflicts of interest to disclose.

Authors' contributions

Ferdous J were involved in the conceptualization of the study, data collection, and preparing the initial manuscript draft. Hossain MI was involved in the literature review, revising, and preparing a final manuscript draft. Ferdous J were involved in the literature review, revising, and editing of the manuscript. Both authors accepted and approved the final version of the manuscript.

Data Availability

The data used to support the findings of this study are included within the article.

Consent

Written informed consent for publication of the patient's clinical details and images was obtained from the patient's legal gurdians prior to submission. A copy of the signed consent form is available for review by the editor of the journal.

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