Hepatitis A in children at Amirkola Children's Hospital, Northern Iran; experience for a decade

Letter to Editor

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Dear Editor,

Hepatitis A virus (HAV) is a single-stranded RNA virus belonging to the Picornaviridae family that is the most common form of acute viral hepatitis worldwide with around 1.5 million clinical cases each year, but the rate is apparently ten folds higher because of underreporting [1, 2]. The clinical spectrum of HAV infection ranges from asymptomatic to sever hepatitis A [3]. Severe hepatic damage can lead to acute liver failure (fulminant hepatitis) and death in some subjects, which is strongly dependent on the age of the patients [4, 5].

In the last two decades, improved sanitary condition and hygiene practices among developing countries have led to reduced transmission of HAV, and this viral infection has shifted from childhood to adolescence with more severe and even life-threatening course [1, 6, 7]. Here, we aimed to report clinical and serological features of all cases of hepatitis A, hospitalized at Amirkola Children's Hospital (North of Iran) from February 2009 to July 2019.

During the 10-year period, 16 children aged 1-13 years old (7±3 years) were admitted with diagnosis of hepatitis A. Among them, 12 (75%) were male and 4 (25%) lived in rural areas. Glucose-6-phosphate dehydrogenase (G6PD) deficiency was observed in 31% [5] cases. History of contact with a person infected with hepatitis A and recent travelling were 31% [5] and 37% [6], respectively. In a study, HAV infection was more common in males than females [8], and another one found seroprevalence of HAV which was significantly lower in urban than rural populations (73.3% vs. 82.2%) [9].

More common symptoms were jaundice 93% (15), fever 81% (13), dark urine 81% (13), pale stools 81% (13), nausea and vomiting 62% (10), abdominal discomfort 62% (10) and less common symptoms included loss of appetite 25% (4), diarrhea 18% (3) and cough 6% (3). Mean± standard deviation of laboratory tests in study group revealed total bilirubin of 10±5 mg/dl, direct bilirubin 7.08±4.09 mg/dl, total protein 13±19 g/dl, albumin 5±7 g/dl, alkaline phosphatase of 886±409 IU/l, serum aspartate aminotransferase of 1268 ± 997 IU/l, alanine aminotransferase of 1546 ± 991 IU/l, prothrombin time of 13±2 second (s) and partial thromboplastin time of 43±12 s.

Asymptomatic HAV infection has been observed in about 70% of children < 6 years old, in contrast, symptomatic infection is usually associated with jaundice and remarkably high levels of serum aminotransferases [3]. Fever, malaise, nausea, vomiting, abdominal discomfort, dark urine, and jaundice are typical symptoms of acute hepatitis A and myalgia, pruritus, diarrhea, arthralgia, and skin rash are less common symptoms. Biochemical tests have shown elevated total bilirubin (mean peak 7 mg/dl), alkaline phosphatase (mean peak 319 IU/l), serum aspartate aminotransferase of 1,754 IU/l, and alanine aminotransferase of 1,952 IU/l [3].

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Among 16 children, fulminant hepatitis A occurred in one boy 5.5 years old with the history of febrile seizure and without the history of any chronic liver disease so that liver transplant was done for him, but post-transplant lymphoproliferative disorder (PTLD) developed and led to his death. In 0.01% of acute hepatitis A cases, fulminate hepatitis can occur with an estimated mortality rate of 60–80% [10].

Although hepatitis A infection is usually asymptomatic in children, acute liver failure due to the hepatitis A may occur in patients with preexisting chronic liver disease [2]. A study from Latin America indicated that the mean age of population with acute liver failure (ALF) was 5 years and 43% of them had acute HAV infection. The HAV was the main etiology of ALF in these patients [11] and other study from Argentina stated that 61% of children with ALF had hepatitis A infection [12]. A study of north of Iran mentioned that the anti-HAV antibody seroprevalence declined among children <15 years with and without chronic liver disease; so, it is important to employ preventative strategies against HAV in chronic liver diseases [2]. This study demonstrated that though during 10-year period, fulminate hepatitis occurred just for one child infected with hepatitis A, but this infection might lead to liver failure even in children without preexisting chronic liver disease; therefore, the preventative strategies against HAV should be planned in children population. So, universal vaccination plan for all children above one year old and health tips observation are recommended.

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