TEACHING FILES (GRAND ROUNDS)

IS IT CHRONIC HEPATITIS A INFECTION?

Lavina Desai¹, Ira Shah² ¹Seth G S Medical College, Mumbai, India, and ²Pediatric Infectious Diseases, Levioza Health Care, Mumbai, India.

ARTICLE HISTORY

Received 30 August 2017 Accepted 1 July 2018

Clinical Problem

A 5 year old boy presented in August 2015 with jaundice and decreased appetite for 2 months. He had been investigated by his pediatrician and found to have hepatitis. In view of non-improvement, he was referred to us for further management. On examination, he had hepatomegaly. Other systems were normal. Ultrasonography (USG) abdomen revealed hepatomegaly with normal echotexture of the liver. His serial liver function tests are depicted in Table 1. HbsAg, Hepatitis C antibody, Hepatitis A IgM, Hepatitis E IgM, ANA, anti-smooth muscle antibody, anti-liver kidney microsomal antibody were negative. Serum ceruloplasmin levels were normal. He tested positive for hepatitis A IgG antibody in August 2015.

Can this be chronic hepatitis A infection?

Discussion

Hepatitis A virus (HAV) infection leads to an initial anicteric phase with non-specific symptoms such as low grade fever, nausea, vomiting, reduced appetite, and abdominal pain lasting for about 7 days. This is followed by the icteric phase which commences with dark urine secondary to bile excretion, pale stools. Jaundice is seen in 10% of children less than 6 years and 40% of children between 6 and 14 years of age. Symptoms can last upto several weeks with a mean of 4 weeks, and may be directly correlated to viral load.^{1,2} Patients with acute liver injury due to viral hepatitis have moderate elevation of liver enzymes (5-10 times the upper limit of normal) at 200 IU/L for AST (91% sensitivity, 95% specificity) and 300 IU/L for ALT(sensitivity 96% and specificity 94%) which begins to peak before jaundice appears and have a gradual decrease over 5-20 weeks.^{3,4} Immunoglobulins to HAV (IgM anti- HAV antibodies) are first detected within 5-20 days after exposure and are diagnostic of acute infection. IgM anti-HAV antibodies peaks during acute or convalescent phase, remains positive for 4-6 months and represent recent or current infection.^{1,5} However persistence of HAV IgM far beyond the acute illness and immediate convalescent period has been

CONTACT Lavina Desai Email: lavinadesai16@gmail.com Address for Correspondence: Lavina Desai, Seth G S Medical College, Mumbai India.

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reported. A study revealed some patients became seronegative early in the course while others (13.5%) remained seropositive for greater than 200 days and this variability should be considered when IgM is used as serological marker of recent infection.⁶ IgG antibodies become detectable in the serum shortly after appearance on anti-IgM antibodies and represent past infection or immunity.¹

About 10% of patients may present with relapse of symptoms during the six months after acute illness, clinical relapse may last for less than 3 weeks and biochemical relapse may last as long as 12 months . The cause of this is unknown and no risk factors have been identified, but the pathogenesis of the relapse involves and interaction between the persistent viral infection and immune mechanisms responding to the antigenic stimuli. It is characterized by an apparent clinical recovery followed by an increase in aminotransferase levels (may exceed 1000 IU/L) after acute infection and initial near normalization of serum levels. Serum Anti-HAV IgM antibodies is detectable throughout the course of the disease and HAV is recovered from the stool of relapse patients and hence they can be a source of infection.^{7,8} Relapses and prolonged cholestasis are unusual manifestations of Hepatitis A, and even in these circumstances, recovery is the rule and chronic hepatitis is not seen.⁹

Since in this child anti-HAV IgM was negative, it is unlikely to be chronic HAV infection. Presence of anti-HAV IgG antibodies is suggestive of a past infection.

Compliance with Ethical Standards Funding: None

Conflict of Interest: None

LFTs	May 2015	June 2015	July 2015	Aug 2015	Sep 2015	Oct 2015	Nov 2015
Billirubin (direct) (mg/dl)	3.6(3.4)	0.9(0.5)	2.58 (1.32)	1.08	0.6	0.49	0.44
SGOT/SGPT (IU/L)	2081/24 86	301/486	-/2105	802/101	80/79	136/191	64/52
Total protein/Albu- min (gm/dl)				9.0/4.3	7.7/4.4	6.6/3.7	6.8/4.0
Alkaline phospha- tase (IU/L)			388	291	253	219	237
				149			

Table 1: Serial Liver function tests

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