

CASE REPORT

HEPATITIS A VIRAL INFECTION TRIGGERS AUTOIMMUNE HEPATITIS IN A PATIENT: A CASE REPORT

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ABSTRACT: Hepatitis A virus is an infectious agent known to trigger autoimmune hepatitis (AIH). We present a case in a 40years old woman with Autoimmune hepatitis who presented 4 months after viral hepatitis A infection. Diagnosis of hepatitis A virus was attributed on viral serological tests and autoimmune hepatitis (AIH) in accordance with international autoimmune hepatitis group system.^[1] She is in remission with steroid therapy. The case we present is unusual with paucity observed in the world literature.

KEYWORDS: Acute hepatitis A, Autoimmune hepatitis.

CASE REPORT: The patient was 40years old woman with jaundice as the chief complaint. History of present illness. The patient had been seeing a local doctor for fever since one month, with no sign of hepatic pathology. Starting 1 week back she began vomiting and revisited the local doctor. Jaundice was noted and the patient was referred to our hospital for thorough testing. Physical findings on admission.

The patient was 150cms tall and weighed 56kgs. Although yellowing of the bulbar conjunctiva was noted, no other abnormalities were identified. Test findings on admission (Table 1) Aspartate aminotransferase (AST), 337 IU/L; alanine aminotransferase (ALT), 331 IU/L; total bilirubin (TB), 15.3 mg/dL; prothrombin time INR 1.8; hepatitis B surface antigen (HBsAg) negative; hepatitis C virus (HCV) RNA negative and immunoglobulin (Ig) M HA antibody positive (titer, 1.2 cut of index). Acute HA was therefore diagnosed she was treated symptomatically and was discharged 10 days later.

She was lost to follow up and returned 4 months later again with same complaints. Furthermore, as levels of ANA, DsDNA, anti SMA and IgG (>3000 mg/dL) were elevated, AIH was suspected. MRCP showed no hepatic atrophy, but thickening of the gallbladder wall was apparent, suggesting acute hepatitis.

LAB PARAMETERS	RESULTS
HB/HCT/PLT/RBC	10.2/36.7/9.41L /4.71 L
NA/K/CL	136/3.8/99
Bilirubin Total/Direct	15.3/10.1
AST/ALT	337/331
IgM HAV	POSITIVE
HBsAg/HCV	NEGATIVE
BSL	109

TABLE 1: LAB DATA ON ADMISSION

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LAB PARAMETERS	RESULTS
HB/HCT/PLT/RBC	9.2/36.7/7.41L /4.42 L
NA/K/CL	134/4/92
Bilirubin Total/Direct	5.3/4.1
AST/ALT	776/456
ANA ,DsDNA ,anti SMA	POSITIVE
IgG	(>3000 mg/dL)
ANTI LKM1	NEGATIVE

TABLE 2: LAB DATA AFTER 4 MONTHS

Clinical Course: As the patients most of the autoimmune markers were positive she was asked to undergo liver biopsy. However patients PT INR was persistently above 1.5 despite of medical treatment and FFP transfusion.

Although AST and ALT levels started to improve, PT INR deteriorated. To avoid onset of fulminant hepatitis, steroid pulse therapy was performed for 3days, followed by oral steroid therapy. Levels of AST, ALT, PT INR improved. At first, 50mg/d of oral steroid (prednisolone) was administered, with dose reduced by 10 mg/d every week. Levels of AST, ALT and PT INR gradually improved and the patient was discharged while receiving 20 mg/d of steroid. After discharge, dose of oral steroid was reduced by 5mg/d every week.

The patient is currently undergoing treatment in an outpatient basis with oral steroid therapy 10 mg/d, and no further elevation of AST or ALT levels has been reported.

DISCUSSION: Hepatitis A virus (HAV) is an enteric transmitting virus which is the most common cause of acute viral hepatitis worldwide. Hepatitis A virus is also incriminated as a triggering cause for autoimmune hepatitis.^[1-5] In most of these cases, AIH did not occur at the same time as acute hepatitis, with AIH often occurring several months after the onset of acute HA.

The present patient did not display AIH at the time of acute hepatitis. However, since levels of ANA and IgG were high, excessive immune reactions were present. We suspect that these excessive immune reactions may lead to onset of AIH. No studies have investigated AIH during the early stages of acute hepatitis, and excessive immune reactions during acute HA have not been documented.

While immune reactions are not often assessed in acute HA, as was the case with the present patient, excessive immune reactions may occur during acute HA. AIH is a rare disease with unknown etiology which causes chronic necroinflammatory changes in the liver. Hepatitis C has been associated with AIH more than any other hepatitis viruses.^[6]

Viral proteins of viruses may be similar to the amino acid chain of different auto antigens in the liver. Cross immune reactions may also be responsible for the damage in the liver tissue. AIH appears to be induced by antibody dependent cell mediated cytotoxicity which involves both antibody mediated and cellular immunity against specific liver antigens as hepatocyte membranes.^[7] There are several genetic factors including human lymphocyte antigen (HLA) types which are related to AIH such as DR3, DR4.^[8]

Hepatitis A as a triggering factor was considered in the case as Hepatitis IgM HAV was positive & Hepatitis B surface antigen, HCV antibodies were negative in the case. Liver histopathology

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was not done in view of deteriorating PT INR and a possible risk of fulminant hepatitis. ANA, ASMA, AMA, DsDna were positive and anti LKM1 antibodies were negative.

Many autoimmune conditions like thyroiditis, ulcerative colitis, celiac sprue, mixed connective tissue disorder, interstitial lung disease, Autoimmune haemolytic anemia are associated with AIH.^[9]

In the present case there have not been enough observed reasons to speculate any other assumptive etiological clue. HAV infection is probably one which is well documented in the case out of several unknown triggers that may induce autoimmune hepatitis. It is for the said reason vaccination of HAV is recommended worldwide for all patients with chronic liver disease including hepatitis C patients to prevent decomposition due to HAV super infections.^[10,11]

However Bary & Smith 2007 described the first ever case of AIH in association with vaccination against HAV after 10 days post vaccination.^[12] There is paucity in literature of HAV with AIH. In our case, there are strong evidences favoring Hepatitis A virus (HAV) as a triggering factor leading to AIH.

Herein, we presented the case of a patient with AIH that appears to have been triggered by acute HA. Given the existence of such patients and the need for careful follow-up of patients with acute HA, the clinical features of AIH patients after acute hepatitis need to be investigated.

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