

Case Report

Concurrent hepatitis B and autoimmune hepatitis a rare presentation of acute liver failure: a case report

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ABSTRACT

Acute liver failure with concurrent Hepatitis b and autoimmune hepatitis is an extremely rare case. We report a 25 year female presenting to our emergency with fatigue and somnolence, distention of abdomen, jaundice, melena and increased hair loss leading to alopecia. Physical examination revealed jaundice, an enlarged liver, ascites and tenderness of upper right abdomen. Laboratory tests revealed an increased level, bilirubin, GGT, increased INR and prothrombin time with elevated IgG levels, and the presence of anti-smooth muscle antibodies, Anti-nuclear antibodies and HBV infection markers. The patient was diagnosed with liver failure resulting from chronic active hepatitis B with an autoimmune component. The treatment consisted of steroids, azathioprine, vitamin K, low-protein diet and lactulose enemas. After undergoing a molecular test (HBV DNA 3.23×10 IU/mL and HBeAg reactive), the treatment was modified by adding tenofovir disoproxil fumarate. After one month the patient was discharged in good clinical condition, with the recommendation of continued tenofovir disoproxil fumarate and prednisone. In subsequent follow-ups, no clinical deterioration or abnormal biochemical liver function test results were found.

Keywords: Acute liver failure, Autoimmune hepatitis, Chronic hepatitis B

INTRODUCTION

Infections with hepatitis B (HBV) viruses are major public health problems worldwide. In India, HBV prevalence in the general population is estimated at 4.26%, although clusters of higher HBV Prevalence have also been reported in the same area.¹

Clinical and laboratory features of HBV can sometimes be mistaken with those of autoimmune hepatitis (AIH), a disease characterized by increased immunoglobulins, circulating autoantibodies and a favourable response to immunosuppression. Indeed, coexistence of AIH with HBV should be considered especially in areas endemic for viral hepatitis, since viruses have long been associated with either the induction of autoimmune phenomena or

the development of overt autoimmune diseases. Non-organ specific autoantibodies (NOSA)

particularly antinuclear antibodies (ANA) and antismooth muscle antibodies (SMA) have been reported frequently in HBV- and HCV-infected patients.²⁻⁴ In most of these cases NOSA are detected in lower titers compared to those found in AIH patients, usually lack F-actin specificity of SMA and do not affect the treatment outcome, disease severity or progression of chronic viral liver diseases.²⁻⁶ Currently, there are scarce data to demonstrate the interaction between AIH and viral hepatitis. Therefore, in here we present our experience by reporting the patient characteristic, pattern of disease progression and outcome and also the difficulties in diagnosis and management of concurrent HBV and AIH.

CASE REPORT

A 25-year-old female was admitted to hospital because of fatigue and somnolence, which increased over a week, progressive distension of abdomen since last one month and loss of hairs since last fifteen days along with jaundice since then. Her past medical history included 1 hospitalization in early childhood and surgery for blunt trauma abdomen at the age of 17. Her general condition at admission was rated as sick as deep icterus along with ascites was present with history of melena since last seven days. Physical examination revealed jaundiced skin and sclera, enlarged liver, tenderness of upper right abdomen and distension with shifting dullness present. During hospitalization the patient complained of fatigue, skin pruritus, abdominal pain and headache. She also had melena 2 two episodes during her stay in hospital, somnolence and loss of appetite. Laboratory test results revealed elevated aminotransferases (ALT: 1115 U/L, AST: 647 U/L), as well as elevated bilirubin, with predominant conjugate bilirubin, (respectively 27.2 mg/dl and 16.43 mg/dl), GGTP (60 U/L) and increased prothrombin time of 56 sec and International Normalized Ratio of 5.3. Serum protein of 5.1g/dl and albumin of 2.5 g/dl, serologic tests revealed the presence of HBV infection markers: HBsAg, HBeAg, and HBV DNA load of 3.38×10^9 IU/dl. The HAV, HEV, HCV, EBV and CMV were excluded, as well as Wilson's disease and alpha-1 antitrypsin deficiency. On the pretext of history of hair loss an Anti-nuclear antibody was sent which was positive 3+ at 1:320 titer. Anti-smooth muscle antibody was positive 3+ at 1:640 titer and Immunoglobulin G level of 3012.3mg/dl.

The patient was diagnosed with liver failure resulting from Acute on Chronic hepatitis B with autoimmune hepatitis component. The treatment consisted of steroids, diuretics (spironolactone) vitamin K, low-protein diet, lactulose enemas and Fresh frozen plasma transfusion. Patient was given tenofovir disoproxil fumarate for hepatitis. In the first days of hospitalization, despite the above therapy, increasing levels of liver damage markers were observed. From the second week of therapy, a gradual clinical improvement was observed, with concomitant decrease of aminotransferase and bilirubin levels and decrease of prothrombin time. On d 30, the HBV viral load had decreased to 1.1×10^2 IU/ml, ALT levels had decreased to 48 U/L, prothrombin time decreased to 21.0 sec and ascites decreased too.

The patient was discharged in good clinical condition with the recommendation of continued tenofovir disoproxil fumarate, prednisone and liver supportives. Systematic follow up in the Gastromedicine Clinic. After another month of treatment ALT levels normalized. Tapered dose of Steroid with continued antiviral treatment was advised. At this time a liver biopsy was performed which showed plasma cell infiltrates at the portal triad along with non-specific inflammation of surrounding parenchyma with trabecular thickening, loss

of continuity of the basal lamina at $\frac{1}{4}$ of its circumference due to inflammatory infiltrate, expanded portal spaces, and multiple focal necrosis of lobules with periportal necrosis. Histological diagnosis of chronic hepatitis B was established, with Grade 1 inflammatory activity, and Grade 2 fibrosis according to modified Scheuer's scale. Patient is still in regular close follow up with improving Liver function tests and good general condition.

DISCUSSION

Authors have described a patient with liver failure in the course of chronic hepatitis B with autoimmune component. Such patients with exacerbation of hepatitis B and concurrent AIH are very rare and a diagnostic dilemma along with treatment difficulty is noted in such kind of patients. By definition, the diagnosis of autoimmune hepatitis is based on typical criteria and the exclusion of other causes of liver disease, including viral infection. AIH classification is virtually impossible in view of the criteria employed, such as alcohol intake, or the presence of viral infections (EBV, CMV, HHV-6), which may initiate autoimmune processes or autoantibody production.^{7,8} Using this classification we would not have been able to diagnose AIH in our patient because of the presence of HBV infection and elevated Immunoglobulin G. ANA and ASMA are known to be present in 20% to 40% patients with HBV infection, but there are no reports of the raised IgG level in patients with HBV infection.⁷

Viral infections may induce autoimmune processes. Tabak et al described AIH in the course of prolonged viral hepatitis A Michalska et al reported the emergence of autoimmune processes within 5 to 18 years from the diagnosis of chronic hepatitis B in 5 HBe-negative patients.^{9,10}

In our patient, the dominant role of HBV infection as a cause of both autoimmune processes and liver failure, was confirmed not only histologically, but also clinically - by rapid improvement after the HBV-specific antiviral treatment with tenofovir disoproxil fumarate was started, as well as by sustained normalization of liver function tests despite tapering of steroids for autoimmune hepatitis. The reason for the use of this specific nucleoside analogue was the patient's clinical characteristics - features of liver failure and presence of autoimmune processes. It was also justified by a high genetic barrier of tenofovir disoproxil fumarate combined with immunosuppressive agents proved effective in the treatment of liver failure in the course of HBV infection with concurrent autoimmune process.

CONCLUSION

Viral hepatitis infections concomitant with AIH are often very difficult to be recognized given the heterogeneity of liver diseases, the absence of specific markers for the diagnosis. High clinical suspicion of concurrent AIH

should be raised in HBV when an otherwise unexplained increase of transaminases and IgG levels are present along with other NOSA positive. A combined disciplinary approach to manage both AIH and acute hepatitis early in the disease course is imperative for favorable outcome.

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