A CASE OF ACUTE NON-A, NON-B SPORADIC HEPATITIS WITH EVOLUTION OF LIVER CIRRHOSIS ON SERIAL HISTOLOGIC FOLLOW-UP

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Summary

Progression of acute non-A, non-B (NANB) posttransfusion hepatitis to liver cirrhosis has been well recognized as in hepatitis B infection, whereas no progression of acute NANB sporadic hepatitis to liver cirrhosis has yet been documented.

We reported a 29-year-old male with prolonged transaminase elevations in whom acute NANB sporadic hepatitis progressed to histologically confirmed cirrhosis during follow-up of about 3 years.

It is suggested that some of the cryptogenic cirrhosis of non-B type may develop from acute NANB sporadic hepatitis and long-term observation is also needed in patients with acute hepatitis of this category.

Key Words: acute non-A, non-B (NANB) sporadic hepatitis, histologic follow-up, cirrhosis of the liver.

Introduction

By application of specific serologic and virologic markers for hepatitis A virus (HAV) and hepatitis B virus (HBV), another type of hepatitis, non-A, non-B (NANB) hepatitis, has been noted in which no markers of HAV or HBV infection can be detected. The frequency of NANB hepatitis varies according to the method of selection of the patients, the geographic area and also to the serologic techniques used for diagnosis. Nearly 90% of acute posttransfusion hepatitis and 50% of acute sporadic hepatitis are caused by NANB virus(es) in our series¹.

Recently several reports have pointed out that progression of acute NANB posttransfusion hepatitis to chronic liver disease including liver cirrhosis is frequently encountered either on biochemical or on histologic follow-up studies²⁻⁶. On the other hand, long-term studies on prognosis of acute NANB sporadic hepatitis are rare and this type of hepatitis is thought to bear a better prognosis⁴. No report so far appeared in which development of liver cirrhosis after acute NANB sporadic hepatitis is histologically proven.

We report here a case in whom acute NANB sporadic hepatitis progressed to liver cirrhosis confirmed by serial liver biopsies during the fol-
low-up of about 3 years.

Case Report

A 29-year-old male, non-medical hospital employee, was admitted to our clinic on June 26, 1978, because of nausea, anorexia, and general malaise with high fever (38.0–39.0°C). There was no past history of jaundice, alcohol abuse, known exposure to hepatotoxic drugs, transfusion of blood or blood products, illicit self-injection, other needle puncture exposure, or homosexuality and no family history of liver disease. He was known to have had normal biochemical liver function tests 2 years before.

On physical examination the patient was slightly jaundiced, but had no hepatosplenomegaly. Liver function tests at that time included: SGOT 450 KU, SGPT 550 KU, alkaline phosphatase 17.7 KA, total bilirubin 2.3 mg/dl, ZTT 3.5, TTT 0.8. His subjective symptoms improved without any specific treatment, but the levels of serum transaminase have been fluctuated (Fig. 1). The percutaneous liver biopsy, taken 4 months after admission, showed features of acute hepatitis (Fig. 2-A). All markers for current and past infection of HBV—hepatitis B surface antigen (HBsAg) by reversed passive hemagglutination, antibody to HBsAg by passive hemagglutination and antibody to hepatitis B core antigen by immune-adherence hemagglutination—were negative. Hepatitis A antibody of the IgM class was not detected by radioimmunoassay. There was no serological evidence of infection with cytomegalovirus by complement fixation method and with EBstein-Barr virus by fluorescence antibody method between paired sera in the early and somewhat later stage. A diagnosis of acute NANB sporadic hepatitis was established.

After the first biopsy the patient has been examined regularly at intervals of 2 or 4 weeks in the outpatient clinic. In February 1979 he was admitted again to the hospital because of...