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A case of recurrent Guillain-Barre syndrome with hepatitis B infection

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Abstract

A 38-year-old man was diagnosed as Guillain-Barré syndrome (GBS) and was initiated treatment with a conventional dose of intravenous immunoglobulin (IVIG). He recovered completely. However, one month later, he suffered from limb weakness once again. The alanine aminotransferase concentration was 11015 U/l and the aspartic acid transferase concentration was 427 U/l. Hepatitis B surface antigen (HBsAg), hepatitis Be antigen (HBeAg), and hepatitis B core antibody (HBcAb) were positive, quantitative PCR detected 7.69×10^6 copies/ml (<1.00×10^3) hepatic B virus (HBV) DNA. Electrophysiology studies showed peripheral axonal injury. He received conventional IVIG and liver protection treatment, in addition to treatment with interferon alfa-2b for 6 months. He recovered completely. Six months later, HBV DNA (<1.00×10^3) and HBsAg were negative; and which continued to date (for 5 years), he did not suffer from paralysis of the limbs. Therefore, more attention should be paid to controlling HBV replication in patients with GBS complicated with HBV infection who could relapse.

Keywords

Guillain–Barre syndrome; hepatitis B; electrophysiology

Introduction

Guillain-Barré Syndrome (GBS) is an acute peripheral polyneuropathy typically characterized by symmetrical muscle weakness and areflexia. GBS mostly has a monophasic course, but sometimes recurrent. Recurrent Guillain-Barré syndrome (RGBS) is defined as two or more episodes of acute inflammatory demyelinating polyneuropathy, each with an onset phase lasting for 4 weeks or less followed by complete or nearly-complete recovery [1]. We report a RGBS patient with hepatitis B infection.

Case Presentation

A 38-year-old man was admitted to hospital because of rapidly progressive paralysis of the limbs. There were no antecedent events such as Campylobacter jejuni and upper respiratory infections; however, the patient had a 10 year history of chronic hepatitis B. Seven days prior to admission, he noted bilateral lower extremity weakness, which gradually spread to the upper extremities. He could not walk steadily or hold an object steadily. On admission to our hospital, his strength in the upper and lower extremities demonstrating bilateral weakness was 4/5 proximally and 3/5 distally. Reflexes in the arms were hypoactive, deep tendon reflexes in the legs were absent, and toes on both feet were downgoing. He had normal pin-prick sensation bilaterally from his feet to his thighs and from his hands to his shoulders. Lasegue's sign was positive in both legs. Live function analysis showed the alanine aminotransferase concentration was 1241 U/l and the aspartic acid transferase concentration was 497 U/l. Hepatitis B surface antigen (HBsAg), hepatitis Be antigen (HBeAg), and hepatitis B core antibody (HBcAb) were positive, whereas hepatitis B surface antibody (HBsAb) and hepatitis Be antibody (HBeAb) were negative. Cerebrospinal fluid (CSF) analysis showed 113 mg/dl protein without cells. Nerve conduction studies of his bilateral median, ulnar, peroneal, and tibial nerves performed on the seventh day in hospital showed low compound muscle action potential amplitudes with nearly normal distal latencies and normal conduction velocities. Loss of F-waves ensued. No conduction blocks were evident. A conventional dose of intravenous immunoglobulin (IVIG), which was 0.4 g/kg/day for 5 days, was initiated. Liver protection treatment was also delivered. He recovered completely. Seventeen days after admission, he was discharged. However, 1 month later, he suffered from limb weakness once again. He did not have a respiratory infection or diarrhea. Neurological examination showed symmetrical muscle weakness; strength in the upper and lower extremities was 4/5 proximally and 3/5 distally. Tendon reflexes in the arms and legs were absent, Babinski's sign was negative, and there was no sensory impairment. CSF analysis showed 174 mg/dl protein with 3 leucocytes/mm3. The alanine aminotransferase concentration was 11015 U/l and the aspartic acid transferase concentration was 427 U/l. HBsAg, HBeAg, and HBcAb were positive, whereas HBsAb and HBeAb were negative. Quantitative PCR detected 7.69×10^6 copies/ml (<1.00×10^3) hepatic B virus (HBV) DNA. Electrophysiology studies showed peripheral axonal injury. He was diagnosed as RGBS. He received conventional IVIG and liver protection treatment, in addition to treatment with interferon alfa-2b for 6 months. Forty days after admission, he recovered completely. Six months later, HBV DNA (<1.00×10^3) and HBsAg were negative; and which continued to date (for 5 years), he did not suffer from paralysis of the limbs.

Discussion

GBS mostly has a monophasic course, and RGBS was reported in about 1–6% of all patients with GBS [2]. The etiology and pathogenesis of RGBS are unknown. In our case, the onset of GBS was accompanied by abnormal liver function and amplification of HBV DNA. Moreover, when HBV was removed, the patient did not relapse. Other studies also reported that RGBS was correlated with HBV [3–6]. These imply that HBV is involved in the pathogenesis of GBS. Molecular mimicry plays an important role in the pathogenesis of GBS. We presume that a common epitope is shared by HBV and myelin or neuraxon of peripheral nerves and an immune cross-reaction initiated by HBV may attack myelin or neuraxon of peripheral nerves. Otherwise,

hypothesize that HBsAg-mediated immune complex vasculitis and direct damage of the myelin sheath by HBV may be essential [3].

On the basis of our case, we propose that more attention should be paid to controlling HBV replication in patients with GBS complicated with HBV infection who could relapse.

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