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ABOUT COVER

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CASE REPORT

Demyelinating neuropathy in patients with hepatitis B virus: A case report

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Abstract

BACKGROUND

Hepatitis B rarely leads to demyelinating neuropathy, despite peripheral neuropathy being the first symptom of hepatitis B infection.

CASE SUMMARY

A 64-year-old man presented with sensorimotor symptoms in multiple peripheral nerves. Serological testing showed that these symptoms were due to hepatitis B. After undergoing treatment involving intravenous immunoglobulin and an antiviral agent, there was a notable improvement in his symptoms.

CONCLUSION

Although hepatitis B virus (HBV) infection is known to affect hepatocytes, it is crucial to recognize the range of additional manifestations linked to this infection. The connection between long-term HBV infection and demyelinating neuropathy has seldom been documented; hence, prompt diagnostic and treatment are essential. The patient's positive reaction to immunoglobulin seems to be associated with production of the antigen-antibody immune complex.

Key Words: Hepatitis B virus infection; Extrahepatic manifestations; Demyelinating neuropathy; Intravenous immunoglobulin; Electroneuromyography; Case report

Core Tip: We report an exceptional case of demyelinating neuropathy in an individual with hepatitis B virus (HBV) infection, emphasizing the importance for clinicians to consistently take this into account when making a diagnosis. The underlying disease process and mechanisms of peripheral neuropathy following HBV infection are still unknown. The patient's favorable reaction to immunoglobulin suggests the potential of long-term immune-induced neuropathy.

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INTRODUCTION

In 2015, the World Health Organization estimated that there were approximately 257 million individuals suffering from chronic hepatitis B (CHB), a liver disease characterized by long-term inflammation, due to continuous infection with the hepatitis B virus (HBV)[1]. The Western Pacific and African regions accounted for 68% of these individuals. Approximately 650000 individuals across the globe succumb annually due to CHB-related complications, including cirrhosis and hepatocellular carcinoma[2,3]. Currently, there are around 70 million individuals in China who have contracted HBV, and approximately 20 to 30 million of them are CHB patients[3], which poses a significant burden on both the families of these patients and the society as a whole. Extrahepatic manifestations, such as serum sickness-like syndrome, polyarthritis, polyarteritis nodosa (PAN), glomerulonephritis, cryoglobulinemia, and various neurological disorders, are occasionally observed in addition to the common manifestations of the affected liver, such as icteric hepatitis, ascites, cirrhosis, and liver cancer[4]. In this article, we describe an elderly gentleman who exhibited demyelinating neuropathy as the initial indication of HBV infection. Treatment with antiviral therapy and intravenous immunoglobulin (IVIG) led to a remarkable recovery. It is important to consider the potential for HBV infection in cases of demyelinating neuropathy, despite its low occurrence rate of 0.04%[5].

CASE PRESENTATION

Chief complaints

A previously healthy 64-year-old man was referred the Department of Neurology at our hospital after presenting with numbness and weakness five months previously.

History of present illness

The patient initially noticed numbness in the lower legs and on the ventral side of the fingertips that gradually progressed upwards to the wrists and ankles on both sides. In addition to numbness, he also experienced soreness and swelling of the muscles. He attended the local hospital for treatment and underwent cervical and lumbar magnetic resonance imaging, which revealed normal cervical and lumbar spine parameters. He was given methyl cobalamin tablets, but this treatment was ineffective. One month ago, he felt weak and was no longer able to walk independently. In the previous week, the patient began to experience nocturnal lower limb twitching, which occurred several times each night.

History of past illness

The patient had no previous medical history.

Personal and family history

The patient had no related family history.

Physical examination

During the neurological examination, it was observed that the proximal muscle strength was 4/5 and distal muscle strength was 4/5 in the limbs. Additionally, his muscle tone was low and the bilateral brachial biceps, triceps, knee, and Achilles tendon reflexes were all decreased. The distal limbs showed the greatest decrease in temperature and pain perception, and both lower limbs exhibited a negative Babinski sign. No evidence of altered consciousness, linguistic impairment, or cranial nerve paralysis was present.

Laboratory examinations

Laboratory findings revealed normal routine blood counts, liver function, kidney function, and blood sugar. The level of Vitamin B12 was within the normal range. An increase of 4 mm/h in erythrocyte sedimentation rate was noted. There was no evidence of anti-nuclear, anti-DNA, anti-Ro antibody, anti-La antibody, or anti-neutrophil cytoplasmic antibodies

(ANCAs), which include proteinase 3 (PR3)-ANCAs and myeloperoxidase (MPO)-ANCAs, and there were no detectable serum cryoglobulins. Immunoelectrophoretic results were negative. Urine levels were as follows: κ light chain < 7.28 mg/L, λ light chain < 4.00 mg/L, and κ/λ light chain ratio 1.82. Hepatitis B surface antigen (HBsAg), hepatitis B core antibody, and hepatitis B e antibody were positive. Test results for hepatitis B surface antibody (HBsAb) and envelope antigen (HBeAg) were negative. Blood HBV DNA level was 6.14E+02 IU/mL. According to cerebrospinal fluid analysis, white blood cell concentration was 2×10^6/L, red blood cell concentration was 0×10^9/L, glucose concentration was 3.61 mmol/L, and protein concentration was 465.78 mg/L. The patient did not have antibodies against human immunodeficiency virus, rubella virus, *Campylobacter jejuni*, *Treponema pallidum*, Epstein-Barr virus, herpes simplex virus, and ganglioside (GD1a, GQ1b, GM1, and GM2).

Imaging examinations

The patient underwent brain and whole spine magnetic resonance imaging, and no abnormalities were found.

MULTIDISCIPLINARY EXPERT CONSULTATION

The patient was further evaluated *via* electroneuromyography, which revealed multiple peripheral neuropathies, suggesting the occurrence of motor and sensory demyelination (Table 1). We suggested that he undergo muscle biopsy for a clear diagnosis, but unfortunately, he refused.

FINAL DIAGNOSIS

Suspected demyelinating neuropathy related to HBV based on the above findings.

TREATMENT

Following the diagnosis of demyelinating neuropathy related to hepatitis B, our initial course of action was to administer lamivudine. To prevent persistence and replication of the virus, the IVIG dose of 400 mg/kg was given for 5 d, as corticosteroid treatment may encourage these processes.

OUTCOME AND FOLLOW-UP

The patient's weakness and numbness improved three weeks after treatment.

DISCUSSION

Around 20% of individuals infected with HBV display extrahepatic symptoms, with 5% experiencing neurological conditions such as peripheral neuropathy and myopathy[6]. In acute or chronic hepatitis, especially when peripheral nerve damage occurs in the advanced stage, after excluding other causes of peripheral nerve damage caused mainly by demyelination, the remaining conditions are classified as hepatic neuropathy. Chronic hepatitis is accompanied by peripheral neuropathy, and the main manifestations are abnormal sensation, weakness, significant decline or disappearance of the tendon reflex, and muscle atrophy. In addition, unilateral or bilateral nerves may be involved, and sphincter dysfunction and meningeal stimulation signs can also appear. There have been reports of different forms of peripheral neuropathy related to HBV, including Guillain-Barré syndrome; PAN; non-PAN vasculitis neuropathy; and chronic neuropathy syndromes, such as chronic polyneuropathy/polyradiculoneuropathy, mononeuritis multiplex, and chronic relapsing demyelinating polyneuropathy[6]. Distinct clinical and pathogenic variances exist among the various genotypes of HBV, potentially leading to diverse clinical presentations, treatment responses, and long-term prognoses based on the viral genotype/subtype. Clinical variations between subcategories and genetic types at different levels of extrahepatic manifestation are becoming more apparent, as indicated by growing evidence[7]. Guillain–Barré syndrome is the most common peripheral neuropathy associated with this viral disease[8,9]. Other polyneuropathies have been reported in CHB, although they are less common than Guillain–Barré syndrome. Multiple peripheral mononeuropathies are even rarer than previously mentioned[9]. Tsukada *et al*[10] reported the initial instance of demyelinating polyneuropathy with HBV in 1987. Subsequently, there have been occasional observations of individuals experiencing peripheral neuropathy, along with increased transaminase levels and liver enlargement. In 2017, Lupescu and Dulamea documented the case of a 57-year-old female who exhibited improvement in both clinical and electromyographic aspects of her demyelinating polyneuropathy following steroid, IVIG and antiviral treatments[11]. Demyelinating neuropathy is a prevalent characteristic often observed in various conditions including leprosy, diabetes mellitus, rheumatoid arthritis, systemic lupus erythematosus (also referred to as connective tissue disorders), sarcoidosis, primary systemic vasculitis, and paraneoplastic syndromes[12]. In our patient, chronic multiple demyelinating peripheral neuropathies were the first

Table 1 Nerve conduction study on admission

	Latency (ms)	Amplitude (mv)	Conduction velocity (m/s)
R-Ulnar nerve (m)			
Wrist – ADM	3.65	7.3	
Bl. elbow – wrist	9.38	6.4	33.5
Ab. elbow – Bl elbow	10.7	5.9	78
R-Median (m)			
Wrist – APB	3.92	5.2	
Elbow – Wrist	12	4.8	33.4
R-Tibial (m)			
Ankle – Abd hal	5.9	4.9	
Popliteal fossa – Ankle	17.1	3.0	32.9
R-Peroneal (m)			
Ankle – EDB	7.22	3.3	
Bl. Fib. head – Ankle	18.2	2.5	24.8
Ab. Fib. head - Bl. Fib. head	20.3	2.5	45.2
R-Ulnar nerve (s)			
Wrist – Digit V	2.15	4.7	48.8
Median (s)			
Wrist – Digit II	2.37	18.1	48.5
Superficial peroneal (s)			
Lower leg – Ankle	2.14	6.1	51.4
Sural (s)			
Mid lower leg – Lat alleolus	2.42	6.2	42.1

Ab: Above; Abd hal: Abductor halluces; ADM: Abductor digiti minimi; APB: Abductor pollicis brevis; Bl: Below; EDB: Extensor digitorum brevis; m: Motor study; s: Sensory study.

manifestation of hepatitis B. Hence, it is crucial to emphasize the significance of conducting a thorough assessment of the patient's epidemiological background, liver function, and serological examinations to detect potential HBV infection in individuals suffering from unidentified peripheral neuropathy. Moreover, the patient's liver function was normal, and the degree of neurological damage was inconsistent with the degree of liver function damage. None of the reported cases have exhibited disease exacerbation or severity of demyelinating neuropathy that correlated precisely with the HBV load [12]. The exact cause of demyelinating neuropathy in individuals with HBV infection remains unclear. Studies in this area suggest that the lesion results from the action of the virus itself on nerve fibers or perhaps from deposits of immune complexes on the vasa nervorum of the nerves, leading to vasculitis and consequent ischemia of the nerve fibers[13-15]. Replication of the virus or deposition of immune complexes directly injures the blood vessels, leading to vasculitis. Activation of the complement system initiates inflammation that later leads to endothelial damage. PAN, a condition that primarily affects medium-sized arteries and causes tissue death, is responsible for the majority of fatalities in patients. Significant evidence from the 1970s has demonstrated a robust correlation between the presence of HBV infection and the occurrence of PAN[16]. Testing positive for hepatitis B surface antigen (HBsAg+) can result in vasculitis, commonly appearing as PAN, which often occurs early in the progression of the illness and might be the initial sign of HBV infection. When linked with chronic active Hepatitis B, there is a reasonably prevalent association with demyelinating neuropathy and PAN. Our patient exhibited signs of chronic active Hepatitis B and multifocal sensorimotor mononeuropathy, a condition similar to vasculitis neuropathies such as PAN. The immediate effect of the virus on nerve fibers or the build-up of HBsAg and HBeAg immune complexes on the vasa nervorum may induce neuron damage related to the immune response, and could potentially lead to peripheral neuropathies[17]. Unfortunately, this patient refused muscle biopsy; however, the improvement in his symptoms following immunosuppressive therapy seems to suggest an immune-related pathogenesis. Furthermore, some studies suggest that antiviral medications such as interferon-α2b, lamivudine, or a combination of these substances exhibit efficacy in the treatment of HBV-related demyelinating neuropathy[18]. Scholars recently disclosed the effectiveness of brief corticosteroid treatment, succeeded by lamivudine and plasma exchange, for demyelinating neuropathy associated with hepatitis B[15,19]. In our study, we

successfully treated patients with immunoglobulin by inhibiting HBV replication with lamivudine without exacerbating liver dysfunction. This approach can help patients avoid the confusion of using corticosteroids in the acute stage of hepatitis, while immunomodulatory therapy should be administered to accumulate clinical experience.

CONCLUSION

Patients infected with HBV display demyelinating neuropathy as described in our patient. The fundamental disease process and functioning of peripheral neuropathy after HBV infection remain unclear. Additionally, the positive effect of immunoglobulin suggests the potential existence of chronic neuropathy triggered by the immune system.

FOOTNOTES

Author contributions: Yan XX managed the patient and wrote the manuscript; Huang J participated in data collection and revised the manuscript; Lin J was responsible for clinical management of the patient, and drafted and edited the manuscript; all the authors thoroughly reviewed and gave their approval for the final version of the manuscript.

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