TÜBİTAK

CASE REPORT

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Guillain-Barré Syndrome Due to Hepatitis A

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Abstract: Guillain-Barré syndrome (GBS) is reported frequently following hepatotrophic viruses, but its occurrence due to the hepatitis A virus has been rarely reported to date. We present a case of serologically positive acute hepatitis A infection associated with GBS. There was an increased protein level in the cerebrospinal fluid and prolonged nerve conduction, with minimal axonal damage findings in electromyography.

Key Words: Guillain-Barré syndrome, acute hepatitis A, electromyography

Hepatit A'ya Bağlı Guillain-Barre Sendromu

Özet: Guillain-Barré sendromu (GBS) hepatotrofik virüsleri takiben sık rapor edilmiştir, ancak hepatit A virüsüne bağlı olanı nadirdir. Biz serolojik olarak pozitif akut hepatit A enfeksiyonu ve GBS olan bir hastayı sunuyoruz. Serebrospinal sıvıda artmış protein düzeyleri, uzamış sinir yanıtı ve elektromiyografide minimal aksonal hasar bulguları mevcuttu.

Anahtar Sözcükler: Guillain-Barré sendromu, akut hepatit A, elektromiyografi

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Introduction

Guillain-Barré syndrome (GBS) is an inflammatory postinfectious disease. It is a life-threatening disease, characterized by polyneuritis associated with symmetrically ascending motor weakness (1-8). Neurological symptoms appear 1-3 weeks after a viral infection or immunization (3). It has been shown that viral agents such as the Epstein-Barr virus, HIV and the cytomegalovirus are responsible for the etiopathogenesis of GBS in the majority of cases (8). However, to date, reports of GBS caused by hepatitis A have been exceedingly rare. We present a case of GBS due to hepatitis A.

Case report

A 22-year-old man presented with progressive ascending weakness and distal paresthesia in all extremities. His history revealed a serologically positive acute hepatitis A infection 2 weeks previously. His neurological complaints started 10 days after the viral infection. His initial neurological complaint was paresthesia in the distal region of both legs; 1 day later weakness appeared in the distal muscles, which progressed to quadriparesis in 3 days. On physical examination his blood pressure was 120/80 mmHg, heart rate 82/min rhythmic, temperature 36.5 °C, and there was slight icterus over the body, but there was no organomegaly, tender liver, respiratory insufficiency or circulatory abnormalities. On neurological examination generalized weakness in all extremities was observed, along with diffuse areflexia and glove and stocking type hypoesthesia. Mental status and sphincter functions were normal. Plantar reflexes were flexor.

A routine blood test showed normal renal function, electrolytes, glucose and hematological values. Serum level of alanine aminotransferase (ALT) was 181 IU/l (normal < 40 IU/l), serum aspartate aminotransferase (AST) 116 IU/l (normal < 40 IU/l) and total bilirubin 1.6 mg/dl (normal < 1 mg/dl). Serum IgM antibodies against hepatitis

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A were positive. Cerebrospinal fluid protein level was 66 mg/dl (normal: 15-45 mg/dl) with no cells. Nerve conduction velocities in peripheral nerves were prolonged with slightly decreased axonal amplitudes in electroneuromyography (ENMG).

Intravenous Ig G (IVIG) therapy was planned, but it was not started because of financial factors. Prednisolone 750 mg/day intravenous was given for 5 days as well as supporting therapy. Motor weakness recovered totally and paresthesia was partially improved within a month.

Discussion

GBS is the most common cause of generalized paralysis. Although its etiology is unclear, the majority of GBS cases develop following a viral infection (9-11). Hepatitis A is regarded as the most benign form of viral hepatitis. Additionally, despite the fact that hepatitis A is the most frequent type of hepatitis disease, GBS due to hepatitis A and C is seldom reported. Since hepatitis infection is seen more frequently in young people, GBS is seen also in young adults and almost all cases were reported during the acute period of hepatitis (3,8).

The etiogenesis of GBS with hepatitis is unknown, but it is considered that the direct cytotoxicity of the virus or immune-mediated damage and molecular resemblance between hepatitis A and myelin structures of peripheral nerves may be responsible for this entity (3,8,9).

General therapy methods for GBS can be also used for GBS patients with hepatitis (6). However, liver enzymes should be continuously monitored, especially after medication is started. Immunoglobulins should be the first choice in the treatment of this disease, because of the lower risk for hepatic side effects and because it is the most effective agent. However, steroid and plasmapheresis may be also tried as an alternative therapy (6). The prognosis is good in GBS due to hepatitis A and almost all patients are cured, as was our patient.

In conclusion, although only a few cases of GBS due to hepatitis A have been reported to date, this complication should be kept in mind as hepatitis A is not always benign.

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