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Telbivudine-Related Myopathy: A Case Report

Telbivudin İlişkili Miyopati: Bir Olgu Sunumu

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ABSTRACT

Telbivudine has potent antiviral activity against hepatitis B virus. Although there are several reports concerning the safety profile of telbivudine, most adverse events are described as mild and transient in nature. In this paper, we report a case of reversible telbivudine-induced myopathy. To detect this adverse event, monitoring of serum creatine kinase level and recognition of myopathic signs and symptoms are necessary.
Keywords: Telbivudine, myophathy, case report

ÖZ

Telbivudin hepatit B virüsüne karşı güçlü bir antiviral etkinliğe sahiptir. Telbivudinin güvenlik profili ile ilgili birçok rapor olmasına rağmen, çoğu yan etkinin ılımlı ve geçici özellikleri tanımlanmıştır. Bu yazıda telbivudine bağlı geri dönüşümlü bir miyopati olgusu bildirilmiştir. Bu yan etkiyi saptamak için, serum kreatin kinaz düzeyinin izlenmesi ve miyopatik belirti ve bulguların tanınması gereklidir.
Anahtar Kelimeler: Telbivudin, miyopati, olgu sunumu

Introduction

Hepatitis B is a viral infection that affects the liver and can cause chronic liver disease. An estimated 240 million people have chronic hepatitis B (CHB) infection. More than 780.000 people die every year due to cirrhosis and liver cancer associated with CHB infection (1). Nucleos(t)ide analogues have an important role in the treatment of CHB. Telbivudine is a new nucleoside analogue (2,3). Transient creatine kinase (CK) elevation and rarely myopathy have been reported in a few number of patients who were treated with telbivudine (3). However, we observed proximal myopathy and elevated serum CK level, which thought to be associated with telbivudine in a CHB patient. To our knowledge, this is the first reported case of telbivudine-related proximal myopathy confirmed by electromyography in Turkey.

Case

A 30-year-old man was admitted to our outpatient clinic. He was diagnosed with CHB 10 years ago. Three months before being admitted to our clinic, his serum viral DNA level, serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels were 213.200 IU/mL, 231 (normal: Up to 41 U/L) and 115 IU/mL (normal: Up to 41 U/L), respectively. Liver needle biopsy was performed. Knodell histology activity index and İshak fibrosis score were 4/18 and 3/6, respectively. Telbivudine treatment (600 mg once daily) was started and the serum viral DNA level gradually decreased to 25 IU/mL after three months. However, he had pain in his arms and legs. Neurological examination was normal. His family history was negative for neuromuscular disorder. He had not taken any other medications that would be regarded as a cause of his symptoms while taking telbivudine. Laboratory examinations were as the following: AST: 25 IU/L, ALT: 24IU/L, total bilirubin: 0.9 mg/dL, blood urea nitrogen (BUN): 31 mg/dL, and serum creatinine: 0.9 mg/dL. His serum CK level was elevated from 187 to 234 IU/L (normal range: 0-200 IU/L). At the 19 month of telbivudine treatment, he had fatigue and could not lift his arms and legs. The serum CK level was 1068 IU/L. Electromyography (EMG) was performed and showed frequent positive sharp waves with myogenic motor unit action potentials in the iliopsoas muscles and a few positive sharp waves in the deltoid muscle. EMG findings showed proximal myopathy. Muscle biopsy was planned for confirmation, but the patient refused muscle biopsy. The antiviral agent was changed to 100 mg

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lamivudine once. He revisited our clinic one month after telbivudine withdrawal and his clinical symptoms improved. The serum CK level was also decreased to 157 IU/L.

Discussion

Telbivudine is a nucleoside analogue which is used in the treatment of CHB (4,5). Nucleoside analogues inhibit polymerase-gamma, which is responsible for mitochondrial DNA replication. This mechanism is thought to be associated with myopathy and lactic acidosis (5). Wang et al. (4) have reported the results of 655 patients treated with telbivudin and they observed myophaty and myositis in 0.6% of patients. Serum CK elevation was developed in 15.9% of patients over 4 years. They reported that serum CK elevation was transient and improved after discontinuing telbivudine treatment (4). Zou et al. (6) have reported an incidence of serum CK elevations and myopathy during telbivudine treatment of 84.3% and 5%, respectively. CK elevations were observed more frequently in men than in women, in patients aged ≤45 years and those with negative hepatitis B e antigen. They have reported that CK elevations healed spontaneously without discontinuing telbivudine in most of patients.

In the literature, there are a few case reports of telbivudine-induced myopathy (3,6). To our knowledge, this is the first report in Turkey regarding telbivudine-related myopathy confirmed by EMG. We could not confirm telbivudine-related myopathy by muscle biopsy. Our patient was not treated with interferon or other nucleoside analogues previously. He had not taken any other medications that would be regarded as a cause of his symptoms. Myopathy occurred after taking telbivudine and improved after discontinuing the treatment.

Conclusion

We thought further closer monitoring is necessary for the evaluation of CK elevation or myopathy in patients treated with telbivudine. In this way, telbivudine can be used safely and effectively in clinical practice.

Ethics

Ethics Committee Approval: The study were approved by the İzmır Bozyaka Training and Research Hospital of Local Ethics Committee, Informed Consent: Consent form was filled out by all participants.

Peer-review: External and Internal peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: Bengisu Ay, Concept: Şebnem Çalık, Design: Banu Karaca, Data Collection or Processing: Erman Özdemir, Analysis or Interpretation: Nalan Ünel, Literature Search: Banu Karaca, Writing: Bengisu Ay, Şebnem Çalık.

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