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Research Article

Asymptomatic Elevation of Liver Enzymes Associated with Levetiracetam: A Case Report

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Abstract

Levetiracetam is a new antiepileptic drug, increasingly used as monotherapy in partial seizures and proven to have great long-term efficacy with a low incidence of adverse events. Levetiracetam metabolism has been reported to be non-dependent on hepatic cytochrome P450 (CYP450) isoenzyme system. Therefore, it is often chosen as the drug of choice in patients with suspected or ongoing hepatic dysfunction. Recently, a few cases have been reported hepatic failure or transaminitis secondary to LEV in literature. We report a case of 6-year-old girl with epilepsy who developed asymptomatic elevation of liver enzymes with levetiracetam therapy.

Key Words: levetiracetam; hepatic failure or transaminitis; children

Introduction

Levetiracetam (LEV), a second-generation antiepileptic drug (AED) with a good efficacy and safety profile, is licensed as monotherapy for adults and children older than 16 years with focal seizures with or without secondary generalization. It exhibits good bioavailability, linear kinetics, and a rapid achievement of steady-state concentrations. Only 34% of the drug is metabolized and the remainder is renally excreted unchanged [1]. It owes its low risk of drug interactions to its minimal hepatic metabolism and effect on protein binding of other drugs [2].

Recently, a few cases have been reported hepatic failure or transaminitis secondary to LEV in literature [3-5]. In this case report, we report a case of 6-year-old girl with epilepsy who developed asymptomatic elevation of liver enzymes with LEV therapy.

Case Report

A 6-year-old girl presented with partial onset of secondarily generalized seizures. On history, the patient was followed by cerebral palsy and intellectual disability in another hospital. She was delivered in the 32th gestational week by normal delivery with 1800 grams of birth weight to consanguineous parents (first cousins).

On admission our hospital, her liver and renal function tests were normal. The patient was administered LEV monotherapy for seizures. LEV had started 10 mg/kg/day, but her seizures continued. The dose was increased to 20 mg/kg/day (400 mg/day), and her seizures were ceased. Serum aspartate aminotransferase (AST) and alanine aminotransferases (ALT) levels were normal limit in controls at the 1st, 2nd, and 4th months of the patient. But, at the 6th month of the control, serum ALT level was 308 U/L (normal: 5-40 U/L) and AST level was 278 U/L (normal: 0-35 U/L). In her laboratory examinations, hemoglobin, total bilirubin, total protein, albumin, ammonia, creatinine, urine routine and thyroid function tests were found to be in normal ranges. Serology for hepatitis A, hepatitis B, hepatitis C, human immune deficiency virus, rubella, cytomegalovirus, toxoplasma, herpes simplex virus type 1-2 and Ebstein-Barr virus were negative. Also, abdominal ultrasound procedures were normal. The patient had not been taking any other drugs apart from the LEV. There was no any recent change in his diet and no history of exposure to any toxic agent. Besides, she did not have any hepatic symptoms and infection signs.

We thought LEV may be the cause of increased ALT and AST and ceased LEV therapy. After stopping LEV, serum ALT and AST levels gradually decreased and returned into normal values. Liver biopsy was not performed.

Discussion

LEV is a widely used antiepileptic medication. The metabolism of it does not involve the hepatic cytochrome P450 (CYP) system, nor does it inhibit or induce hepatic enzymes and its metabolites are reported to be eliminated from systemic circulation via renal excretion [1,2]. Therefore, LEV is often chosen as the drug of choice in patients with suspected or ongoing hepatic dysfunction.

Prospective studies reported that chronic LEV therapy was not accompanied by significant elevations in serum aminotransferase levels and clinically apparent liver injury was not observed. Recently, there have been a few reports of a fatal fulminant hepatic failure and transaminitis with LEV in literature [3-5]. Gutiérrez-Grobe et al. [4] reported a case of acute liver failure with secondary multiorganic failure associated with LEV and lacosamide treatment in a patient with unknown etiology

seizures. Tan et al. [3] reported fulminant liver failure from LEV. Syed and Adams also reported on potential liver failure stemming from LEV use [5]. Sethi et al. [6] also reported on LEV-induced asymptomatic transaminitis. Similarly, we report on a 6-year-old girl with epilepsy who developed asymptomatic elevation of liver enzymes with LEV therapy which resolved following the discontinuation of LEV. The mechanism of LEV hepatotoxicity is unknown, but is likely to be hypersensitivity. LEV has minimal hepatic metabolism and does not affect CYP 450 isoenzyme activity.

Although, LEV has an extremely favorable side effect profile with few drug-drug interactions, low potential for hematological and hepatic toxicity, there have been a few reports LEV-induced hepatotoxicity. In conclusion, physicians prescribing LEV should go for careful liver function monitoring not only initially but throughout the time the drug is given.

Conflict of interest: The authors declare that there is no conflict of interests.

References

1. Patsalos PN. (2000). Pharmacokinetic profile of levetiracetam: toward ideal characteristics. *Pharmacol Ther*;85(2):77-85.
2. Radtke RA. (2001). Pharmacokinetics of levetiracetam. *Epilepsia*; suppl 4:24-27.
3. Tan TC, de Boer BW, Mitchell A, Delriviere L, Adams LA, Jeffrey GP, et al. (2008). Levetiracetam as a possible cause of fulminant liver failure. *Neurology*;71(9):685-686.
4. Gutierrez-Grobe Y, Bahena-Gonzalez JA, Herrera-Gomar M, Mendoza-Diaz P, Garcia-Lopez S, et al., (2013). Acute liver failure associated with levetiracetam and lacosamide combination treatment for unspecified epileptic disorder. *Case Rep Emerg Med*:634174.
5. Syed AA, Adams CD. (2012). Acute liver failure following levetiracetam therapy for seizure prophylaxis in traumatic brain injury. *Case Rep Clin Med*;1(2):41-44.
6. Sethi NK, Sethi PK, Torgovnick J, Arsura E, Cukierwar F. (2013). Asymptomatic elevation of liver enzymes due to levetiracetam: a case report. *Drug Metabola Drug Interact*;28(2):23-24.

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