

The Effect of Levetiracetam on Insulin/Glucose Level

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Altered patterns of hormonal secretion due to the direct action of epileptic discharges have been observed in humans and animals, but it is difficult to elucidate the underlying cause of untoward effects of antiepileptic medications that can be multifactorial in epileptic patients. Monotherapy should be administered to avoid the possible occurrence of adverse events and complications.^[1-5] Studies have clearly demonstrated the impact of antiepileptic drugs on hormonal status, including effects on fertility, sexual function, bone structure, and thyroid hormone function.

Levetiracetam (LEV) is distinct from other antiepileptics, although its effect on cyp450 enzyme induction remains unclear. LEV specifically binds to synaptic vesicle protein (SV2A), which is commonly found in the central nervous system and endocrine tissues.^[6] It was observed that LEV induces the secretion of testosterone and estrogen without stimulating the release of gonadotropin from ovarian follicular cells. This observation suggests that endocrine functions are affected by LEV, particularly in women of childbearing age.^[7]

Previous studies have shown that thyroid functions are not significantly influenced by LEV in children and adults.^[8] In case-control studies, valproic acid was shown to lower blood glucose concentration independent of weight gain and hyperinsulinemia.^[9] There are no studies in the literature on the impact of LEV on serum insulin and/or glucose levels. A 20-year-old female patient presented at our outpatient

clinic with facial numbness, involuntary contractions in the right arm, and subsequent loss of consciousness. She was diagnosed with complex partial epilepsy, and her routine follow-up EEG examination was normal. LEV treatment was initiated at a dose of 2000 mg/day. She had type 1 DM and was receiving basal+bolus insulin therapy with no history of a hypoglycemic episode. Both basal and bolus insulin requirements were reduced after LEV treatment. There was no change in the patient's diet.

During follow-up, the patient experienced hypoglycemic episodes 2 hours after taking her LEV dose. Previously, she was being treated with 5 units of bolus insulin homologue +0.6 units/kg of basal insulin via subcutaneous infusion. Following initiation of LEV therapy, she no longer required basal insulin but continued taking homologue insulin at the same dose.

Compared to a dose of 2000 mg/day, the number of hypoglycemic episodes was reduced after reducing her LEV dose to 1000 mg/day. Currently, the patient is receiving a 750-mg daily dose of LEV without any hypoglycemic episodes. Based on this case, we consider that further studies are needed to demonstrate the effects of LEV on insulin and glucose metabolism and to investigate its role in DM treatment from a novel perspective.

Disclosures

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