Hypoglycemia Induced by Pregabalin

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Sir,

70 year old female with type-2 diabetes and hypertension of 15 years duration, came with the complaints of burning, tingling, numbness and pin-prickling sensation over both the legs and feet for over 3 months. On examination, her BP was 140/90. Neurological examination revealed peripheral neuropathy affecting both the lower limbs. Other systems were normal. Lab data revealed HbA1C 7.2%, creatinine 0.9 mg/dL. Lipids were as follows: Cholesterol 120 mg/dL, Triglyceride 90 mg/dL, LDL 80 mg/dL, HDL 38 mg/dL. Urine examination was normal. Patient was taking telmisartan 40 mg, glycalazide 40 mg twice a day and for neuropathy, she was prescribed Pregabalin 75 mg 3 times a day.

After one week, patient came with history of recurrent hypoglycemia with symptoms of sweating, increased appetite and tremors which improved with intake of one glass of juice/hypotab (contains 5 gm of glucose). On two occasions during the episode, blood glucose estimation was done and results were 64 mg/dL and 59 mg/dL respectively.

Reduction in the dosage of Pregabalin to one capsule at night improved her condition. Since then, for the last two months, patient had no episode of hypoglycemia.

Her diet was found to be same throughout the period of observation. There was no change in antidiabetic therapy. Her renal function was normal. She was taking 1600 calorie diet. There was no apparent weight loss. There was no clinical evidence of addison’s disease.

On search of literature, it was found that gabapentin or pregabalin can cause hypoglycemia. They increase insulin release by enhancing voltage dependent Ca2+ channels as an agonist on the GABA receptor. The autocrine signalling of insulin release thus involves GABA release, GABAA receptor activation followed by membrane depolarization leading to opening of voltage dependent calcium channels. The Ca2+ influx stimulates release of insulin. Thus, a possible effect of gabapentin/ pregabalin on pancreatic beta-cells involves either GABA receptors or voltage dependent Ca2+ channels.

References