Octreotide to treat severe hypoglycaemia in Guillain-Barre syndrome

Sir,

The recovery course of patients with Guillain-Barre syndrome (GBS) can be complicated by non-neurological systemic perturbations. Autonomic dysfunction associated with GBS affects up to two-thirds of patients and can lead to a reduction in the counterregulatory catecholamine response. This increases the risk of severe hypoglycaemia, especially in diabetics.[1] Sudden, severe hypoglycaemia can damage both white and gray matter of the cerebrum resulting in encephalopathy.[2] Attention to glycaemic control is an extremely crucial component in the management of these patients. Due to associated autonomic instability, the warning signs of fatal hypoglycaemia may go unnoticed,[3] like the index case discussed herein. The patient was not a diabetic,

with no previous hypoglycaemic events. He had issues with gastric motility and positional circulatory changes suggestive of autonomic dysfunction. However, subsequent unexplained neurological deterioration with hypotension coerced us to probe into correctable life-threatening causes. Significant hypoglycaemia due to sepsis along with autonomic dysfunction were the possible causes. The patient was soon initiated on 6-hourly intravenous (IV) hydrocortisone 50 mg. Enteral tube feed was continued. His serum levels of insulin, C-peptide, cortisol, liver enzymes, procalcitonin, total leucocyte counts, and thyroxine levels were within normal limits. The hypotension responded to antibiotics and required a small dose of vasopressor infusion. In view of persistent hypoglycaemia with a blood glucose level of 50 mg/dl despite a dextrose rush, continuous IV 10% dextrose supplementation was initiated at 50 ml/hour and had to be continued for more than 48 hours. Intermittent normalisation of blood glucose levels could not be sustained in the absence of continued dextrose infusion. Other standard therapies to counter hypoglycaemia are steroids and IV glucagon (1 mg), which are proven effective in tiding over persistent hypoglycaemic crises. Non-availability of the same forced us to attempt administration of an alternate agent. Injection octreotide, a potent, long-acting synthetic somatostatin analog came to our rescue, which was administered as a subcutaneous (SC) injection of 100 μg once daily for three days. It is United States Food and Drug Administration approved for IV and SC injection for the treatment of vasoactive intestinal secreting tumors, metastatic carcinoid symptoms,[4] upper gastrointestinal bleeding and acromegaly.[5] Its off-label use has been described in drug-induced,[6] namely, fluoroquinolone, oral hypoglycaemic agent and insulin overdose-related hypoglycaemia. It suppresses the secretion of glucagon, insulin, growth hormone, gastrin and cholecystokinin. Earlier reports establish its role in refractory hypoglycaemia management when administered through the subcutaneous route at a dose of 50-100 μg at 6-12 hourly intervals. Single, double, or a maximum of three doses at 6 hourly intervals have been reported to be adequate to achieve euglycaemia across different case reports. Octreotide acts by inhibiting pancreatic insulin secretion thus preventing persistent hypoglycaemia. This three-day treatment allowed us to taper and totally wean off the patient from extraneous glucose administration and achieve a stable glucose level. This case report highlights the less commonly exploited therapeutic role of octreotide in the management of hypoglycaemia refractory to standard therapy.

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Conflicts of interest

There are no conflicts of interest.

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