Concurrent Diagnosis of Post-Gastric Bypass Hyperinsulinemic Hypoglycemia and Central Adrenal Insufficiency: A Case Report

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Case Presentation

A 65-year-old man with a history of Roux-en-Y gastric bypass (RYGB) in 2005 and chronic nutritional deficiencies presented following a near-syncopal episode. Over the preceding two months, he experienced progressive anorexia, decreased oral intake, and a 20-pound weight loss. Two days prior, he developed worsening fatigue and dyspnea. On the morning of presentation, while seated, he experienced acute diaphoresis, lightheadedness, and visual dimming. On arrival, he was hypotensive to 50/30 mmHg and hypoglycemic to 59 mg/dL. Recurrent hypoglycemia was noted during admission. A low morning cortisol with an inappropriately normal cosyntropin stimulation test confirmed secondary adrenal insufficiency, and oral hydrocortisone was initiated. Despite adequate glucocorticoid replacement, hypoglycemia persisted with a noted postprandial pattern. C-peptide was elevated (2.1 ng/mL), plasma insulin was elevated (8.3 uU/mL), proinsulin was non-suppressed (4.1 pmol/L), and beta-hydroxybutyrate was suppressed (0.1 mmol/L) during a hypoglycemic episode (blood glucose <55 mg/dL), confirming endogenous hyperinsulinism. Sulfonylurea screening was negative, narrowing the differential to insulinoma versus a diffuse beta cell process. Acarbose and a low-carbohydrate diet were started, resulting in the resolution of his hypoglycemic episodes. Given the postprandial pattern and RYGB history, he was tentatively diagnosed with post-gastric bypass hyperinsulinemic hypoglycemia. Discussions regarding further imaging and selective arterial calcium stimulation testing for definitive localization are ongoing.

Discussion

Hyperinsulinemic hypoglycemia is an important consideration in patients with recurrent hypoglycemia. The principal etiologies in adults with endogenous hyperinsulinism are insulinoma and diffuse islet cell dysfunction, including noninsulinoma pancreatogenous hypoglycemia syndrome (NIPHS) and post-gastric bypass hypoglycemia [1,2]. Although these entities share features such as postprandial hyperinsulinemic hypoglycemia and histologic findings of beta cell hypertrophy, often termed “nesidioblastosis,” they are considered distinct [2]. NIPHS occurs in patients without prior gastric bypass, whereas post-gastric bypass hypoglycemia occurs after RYGB or similar procedures that disrupt gastric emptying [2,3]. Notably, “nesidioblastosis” properly describes a histologic appearance and does not necessarily indicate islet dysfunction, as these changes have been observed in up to 36% of asymptomatic individuals at autopsy [4].

In contrast, insulinoma is a neoplastic beta cell proliferation forming a discrete tumor that autonomously secretes insulin, classically causing fasting hypoglycemia [5]. Most are benign and identifiable on imaging [5]. When imaging is negative, selective arterial calcium stimulation testing (SACST) can aid localization. In SACST, calcium gluconate is injected into arteries supplying different pancreatic regions while hepatic venous insulin is sampled; a twofold or greater rise in hepatic venous insulin constitutes a positive response [6]. In NIPHS and post-gastric bypass hypoglycemia, positive responses are typically observed after injection of multiple arteries, whereas insulinoma typically produces a focal response, though this distinction is not absolute [7].

First-line management of post-gastric bypass hypoglycemia consists of dietary modification and acarbose, which attenuate postprandial glycemic excursions [2,8]. Refractory cases may require diazoxide, octreotide, or graded pancreatectomy [1,8]. Insulinoma is typically treated with surgical resection [5].

This case highlights the importance of considering post-bariatric hyperinsulinemic hypoglycemia in RYGB patients and demonstrates the efficacy of conservative management as first-line therapy.

References

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