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Case Report

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Postprandial Hyperinsulnemic Hypoglycemia Thirteen Years after Gastric Bypass Surgery

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ABSTRACT

As bariatric surgery for the treatment of obesity become popular, it is important to recognize its acute and chronic complicatons. Here, we report a case of a 39-year-old lady who presented to endocrine clinic with six months history of sever postprandial hypoglycemia thirteen years after Roux-en-Y-gastric bypass surgery. After confirmation of whipple's triad, she underwent provocative meal test which revealed hyperinsulinemic hypoglycemia. She responded well to medical nutrition therapy and Acarbose. Since most of the reported cases of post bariatric hypoglycemia presented 1-5 years after their surgery, we report this rare case who presented 13 years after her surgery.

Keywords: bariatric, postprandial hyperinsulinemic hypoglycema, PBH

INTRODUCTION

The prevalence of obesity has increased in the last thirty years. The complication of obesity carries substantial morbidity and mortality. This has led to increase in bariatric surgeries as one of the treatment modalities for obesity. Roux-en-Y-gastric bypass surgery (RYGB) is considered to be one of the preferred options. Bariatric surgeries result in permanent change of a patient's GIT anatomy, which result in acute and chronic complications. Postprandial hyperinsulinemic hypoglycemia is one of the common complications post RYGB and to a less extent post sleeve gastrectomy, fundoplication and rarely after adjustable gastric banding (1). After it was first reported in 2005, many cases had followed (2).

Due to the lack of a standardized definition for postbariatric hypoglycemia (PBH), in addition to the unidentified cases of asymptomatic hypoglycemia, the exact prevalence is unknown, with wide range differences between studies (3). It is probably <1% for sever hypoglycemia necessitating hospital admission and <10% for clinically significant hypoglycemia (4).

PBH has been reported to occur on a median of 40.6 months' post-surgery (3), but no upper limit of the number of years has been identified. In this case, we report the occurrence of PBH thirteen years after surgery.

THE CASE

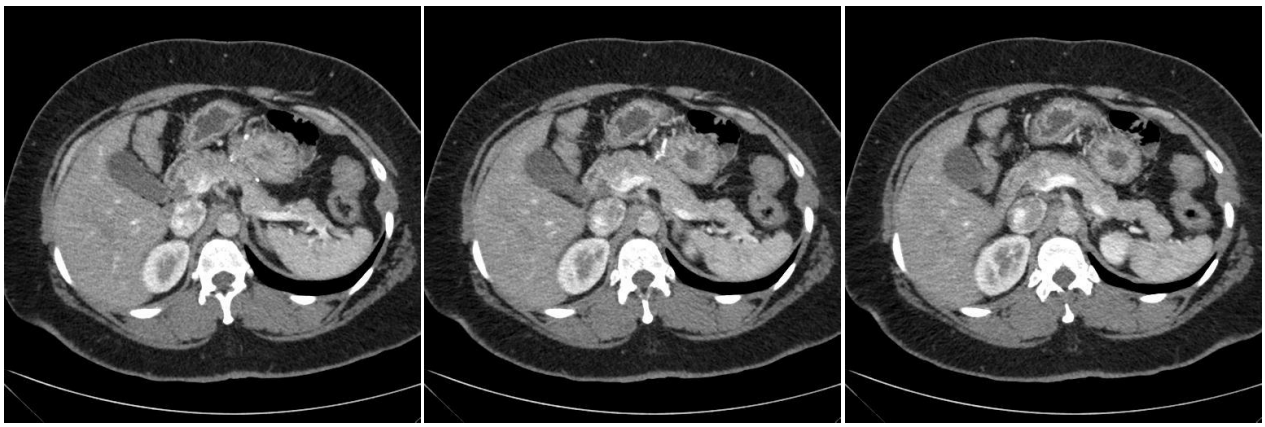
39-Year-old female, presented to Endocrinology clinic with postprandial hypoglycemia. She underwent laparoscopic Roux-en-Y gastric bypass (RYGB) surgery 13 years prior to presentation. There was no history of diabetes and there were no reported complications during surgery or in the immediate post- operative period. Post-surgery, she had significant weight loss. She was in a good health until approximately six months prior to her presentation, when she started to have recurrent episodes of palpitation, blurred vision, headaches, sweating and tremors. She also gave history of recurrent attacks of confusion with occasional episodes of loss of consciousness. These Symptoms occurred usually 2-4 hours after high carbohydrates meals. During these attacks, her glucometer blood glucose was low, up to 45 mg/dL. Her symptoms were relieved by eating food. There was no history of any fasting hypoglycemic episodes.

Patient reported weight gain in the last one year prior to her presentation (14 kg).

Her physical examination was unremarkable except for BMI 40 kg/m^2.

Her initial evaluation revealed normal complete blood count and basic metabolic panel including normal fasting blood sugar (4.7 mmol/L), hemoglobin A1C (5.1%) and thyroid function test.

She underwent inpatient modified supervised fasting test for 36 hours which did not result in any hypoglycemic attacks. Furthermore, she underwent provocative mixed meal testing which resulted in hypoglycemic symptoms after 3 hours of starting the test, her laboratory values revealed venous blood sugar of 40 mg/dL, elevated insulin (185.8 pmol/L. Reference range: 17.8 - 173) and C-peptide (1.92 pmol/L. Reference range: 0.37 - 1.47), a negative oral hypoglycemic agent screen and normal cortisol level. Abdominal CT with contrast was done with no evidence of insulinoma (**Figure 1**).



(a) (b) (c)

Figure 1. Patients abdominal CT with contrast showing no evidence of insulinoma

Patient was managed medically with Acarbose tablets which were increased gradually for which she responded to, with no further episodes of hypoglycemia. She reported no adverse effects from Acarbose.

DISCUSSION

PBH has been recognized as a serious and possibly life-threatening complication of bariatric surgeries. Its pathophysiology is debatable and not fully understood, but several theories has been reported. The main contributing factors are rapid delivery of food into the intestine due to altered gut anatomy post-surgery. This will result in postprandial hyperglycemia and hyperinsulinemia due to increase beta cells sensitivity and increased secretion of GLP-1 with possible direct nutrient effects and neural factors (5). All these changes will result in hypoglycemia. Decreased glucagon secretion may be one of the contributing factors for PBH (6).

The diagnosis of PBH can be difficult since it overlaps with dumping syndrome which could present with the same postprandial autonomic symptoms especially in the first 6-12 months post bariatric surgery (7). Some researchers suggest strict diagnostic criteria in order to diagnose PBH. These include the presence of neuroglycopenic symptoms that occur 1-3 hours post meals, with no fasting hypoglycemia, blood glucose less than 54 mg/ dl, and improvement of hypoglycemic symptoms by eating carbohydrates (COH). These symptoms occur 6-12 months post bariatric surgery (1,8).

If the patient fulfils these criteria, no further workup is needed for diagnosis. On the other hand, if the patient presented with symptomatic fasting or activity related hypoglycemia in the first 6 months post bariatric surgery, provocative testing and imaging studies are needed to rule out insulinoma and other causes of hypoglycemia (8).

Although our patient's presentation was typical for PBH, she presented after 13 years of her RYGB which is considered to be a long duration that was not reported in the literature. Because of that, we chose to investigate her further to rule out insulinoma and other hypoglycemic disorders.

Dietary modification is the key management with good response in mild to moderate cases. Patients are instructed to consume small meals with high fibers and protein. Patients should also avoid food with high glycemic index and drinking water with meals (9). It is not recommended to avoid COH completely.

PBH cannot be treated the same way as hypoglycemia due to other causes. If hypoglycemia is severe, it can be treated by ingestion of 15 g of COH or glucagon injection, taking into consideration that simple COH may result in rebound hypoglycemia. The best way to treat hypoglycemia is by ingestion of food containing a mixture of simple COH, fat, and protein.

Continuous glucose monitoring (CGM) is of great benefit as it allows patients with severe hypoglycemia or hypoglycemia unawareness to monitor their blood glucose closely and mange hypoglycemia when necessary (9).

If dietary modification failed to resolve the hypoglycemia, alpha-glucosidase Inhibitor such as Acarbose can be used. It decreases the secreted insulin through inhibition of carbohydrates absorption which consequently lead to prevention of hypoglycemia. However, the use of Acarbose is limited due its gastrointestinal side effects. This can be minimized by gradually increasing the dose (10).

Further testing is needed if the hypoglycemia is refractory to dietary modification and acarbose. These tests include a provocation meal test in which the patient ingest the same food that cause them hypoglycemia at home as we did for our patient (9).

If there is no complete response to dietary modification and Acarbose, additional pharmacotherapy may be considered. These include Diazoxide (11), Somatostatin analogs (12), Calcium channel blockers (13) and GLP-1 antagonists (14).

Rarely, patients who underwent bariatric surgery may present with hypoglycemia due to insulinoma. In sever PBH cases which persists despite the previous dietary and pharmacotherapy options, this possibility should be sought and investigated with prolonged fasting tests and subsequent imaging studies if positive (9).

For patients who fail to respond to the previous measures, G-tube insertion and feeding into the bypassed portion of the stomach has been successful in reducing hypoglycemic episodes. The patient should abstain from taking any oral carbohydrates. The formula and rates of the G-tube feeding can be customized to the patient preference (15).

One study with short follow up period showed improvement in the postprandial hypoglycemia in patients

who underwent restoration of their gastric restriction by placement of a silastic ring or an adjustable gastric band. However, gastrointestinal adverse effects of these procedures have been observed (16).

Persistent sever hypoglycemia with neuroglycopenic symptoms might necessitate reversal of the gastric bypass surgery. It has proven its effectiveness in many patients (17), however, unfortunately this was not the ultimate solution for all patients.

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