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CASE REPORT

Postprandial hypoglycemia after ileocolic interposition and Billroth-II gastrojejunostomy: A case report

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Abstract

Postprandial reactive hypoglycemia, or late dumping syndrome, is a common but underrecognized complication from bypass surgery. We report an unusual case of postprandial reactive hypoglycemia in a patient with a severe esophageal stricture from corrosive agent ingestion who underwent ileocolic interposition and an antecolic Billroth-II gastrojejunostomy. A 22-year-old male patient with a one-year history of corrosive ingestion was referred to the hospital for a surgical correction of severe esophageal stricture. After the patient underwent ileocolic interposition and an antecolic Billroth-II gastrojejunostomy, he experienced multiple episodes of gastroesophageal refluxsymptoms during nasogastric feeding and had onset of hypoglycemic symptoms. His plasma glucose level was 59 mg/dL. After we had intraoperatively re-inserted a jejunostomy tube bypassing the ileocolic interposition, and reintroduced enteral nutrition, his hypoglycemic symptoms resolved. We performed a mixed meal tolerance test by nasogastric tube, but the results did not show postprandial hypoglycemia. Although the specific mechanism is unclear, this case suggests gastroesophageal reflux to the ileal interposition may have caused a state of exaggerated hyperinsulinemic response and rebound hypoglycemia. To the best of our knowledge, we are the first to report case of postprandial hypoglycemia after ileocolic interposition, which may have been caused by exaggerated hyperinsulinemic response due to gastroesophageal reflux to the ileal interposition. This syndrome should be considered in the patient who has had ileocolic interposition surgery and has developed postprandial hypoglycemia.

KEYWORDS

dumping syndrome, gastric bypass surgery, ileal interposition, mixed meal tolerance test, postprandial hypoglycemia, reactive hypoglycemia

1 | INTRODUCTION

Postprandial reactive hypoglycemia, or late dumping syndrome, is a common complication of esophageal, gastric, and bariatric surgery.[1] Prevalence varies depending on the type and extent of surgery,[2] ranging from 0.2% in Roux-en-Y gastric bypass (RYGB)[2] to 34.2% in bariatric surgery.[3] However, reports on the prevalence of hypoglycemia after other types of upper gastrointestinal surgery, including ileal interposition, duodenal switch, Nissen fundoplication, esophagectomy, and gastrectomy, are lacking.[3] Ileal interposition used to induce diabetic remission in obese and non-obese subjects may have short- and mid-term complications, including metabolic ketoacidosis, diarrhea, cholecystolithiasis, and bowel obstruction,[4] but no serious long-term surgical complications and severe hypoglycemia events have been reported.[5] Hypoglycemia-related symptoms are attributable to autonomic and neuroglycopenic symptoms.[6] Currently, no standard test or standard cut-off value for evaluating post-bypass hypoglycemia exists except for level 1 and level 2 hypoglycemia, defined as <70 mg/dL (3.9 mmol/L) and < 54 mg/dL (3 mmol/L), respectively, according to the American Diabetes Association guidelines.[7] Thus, we herein present the first case report of postprandial hypoglycemia in a patient with severe esophageal stricture from corrosive agent ingestion who underwent ileocolic interposition and an antecolic Billroth-II gastrojejunostomy with Braun jejunojejunostomy.

2 | CASE PRESENTATION

A 22-year-old Thai male with a 10-month history of corrosive ingestion was referred for surgical correction of severe esophageal stricture and treatment for severe malnutrition. Although we had been providing open-jejunostomy enteral nutrition for 8 months, the target calorie and protein intake were not achieved. He had fatigue and weight loss (from 50 kg to 38 kg). His appearance was cachexic with signs of volume depletion. His height and weight were 168 cm and 38 kg, respectively (body mass index 13.5 kg/m^2). Physical examination revealed severe glossitis, subcutaneous fat depletion, and muscle wasting. Transverse white bands were visible with sparse, dry hair, indicating chronic protein-calorie malnutrition. We made several attempts to perform endoscopic esophageal dilatation, but were unsuccessful. Thus, we performed cervical esophagostomy to prevent aspiration. We planned to perform esophageal reconstruction after improving his nutritional status, body weight, and serum albumin. Due to severe stricture and scar formation at the pylorus and antrum of the stomach, the right side of the colon and the terminal ileum were chosen as sites for an ileocolic conduit connecting the esophagus and stomach. We performed ileocolic interposition and an antecolic Billroth-II gastrojejunostomy with Braun jejunojejunostomy bypassing the pyloric obstruction and minimizing bile reflux gastritis (Figure 1). However, the patient could not take food orally, requiring enteral nutrition by jejunostomy tube. He underwent multiple operations during hospitalization because he developed a cologastric stricture, requiring endoscopic dilation of cologastric anastomosis. Several months after the surgery, medication feeding obstructed the jejunostomy tube. Therefore, we placed a nasogastric (NG) tube for enteral feeding. 2 h after NG feeding, the patient experienced gastroesophageal reflux symptoms with nausea, anxiety, irritability, and sweating, a syndrome suggestive of symptomatic hypoglycemia.

3 | INVESTIGATIONS AND TREATMENT

Laboratories showed the plasma glucose level was 59 mg/dL while symptomatic. His baseline random plasma glucose levels were mostly 100–120 mg/dL. His hypoglycemic symptoms improved promptly after administering an intravenous glucose infusion. His plasma glucose level rose

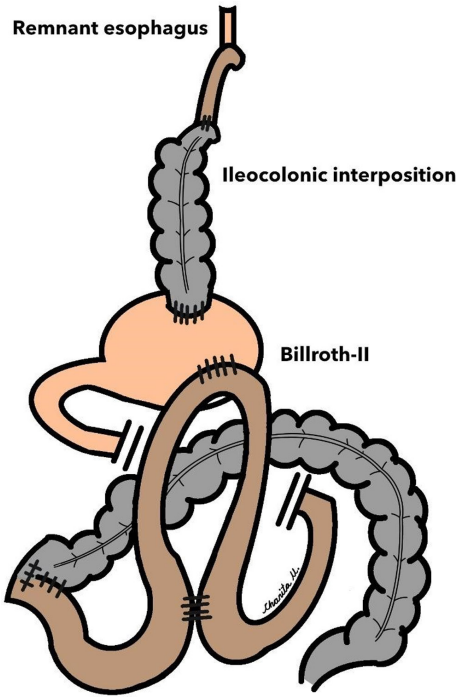

FIGURE 1 Exploratory laparotomy with partial gastrectomy, gastrojejunostomy anastomosis (Billroth II), intrathoracic esophageal resection, and ileocolonic interposition.

to 188 mg/dL. Postprandial hypoglycemia after NG feeding was diagnosed using all three criteria in Whipple's triad. The patient experienced multiple episodes of food regurgitation and postprandial hypoglycemia after NG feeding. Thus, he needed an intravenous glucose infusion at a rate of 10 g/h. However, we did not measure insulin and c-peptide levels during the postprandial hypoglycemic episodes.

We modified the patient's enteral feeding by prescribing a low glycemic index and low carbohydrate enteral formula. Feeding was divided into six meals per day to decrease the caloric and carbohydrate intake in each meal. Also, we switched the mode of feeding from bolus to continuous feeding. The patient's hypoglycemic symptoms improved after our nutrition intervention, and we weaned him off intravenous glucose infusion successfully.

4 | OUTCOME AND FOLLOW-UP

After that, we performed endoscopic dilatation of cologastric anastomosis to correct the food reflux, and re-inserted the jejunostomy feeding tube intraoperatively. The hypoglycemia and acid reflux symptoms resolved completely after switching the route of feeding from NG to jejunostomy tube to bypass the ileocolic conduit. To attempt to confirm the hypoglycemic episode, we performed the mixed meal tolerance test (MMTT) since we planned to remove the jejunostomy tube feeding and initiate an oral diet after improving his nutrition status. An NG tube was inserted intraoperatively to the remnant of the stomach. We speculated an oral diet or NG tube feeding to the remnant of the stomach might have aggravated food reflux to ileocolic conduit and precipitate postprandial hypoglycemia. Thus, we performed MMTT using meals suspected of causing his hypoglycemic symptoms by NG tube feeding (Table 1). Nevertheless, we could not demonstrate the postprandial hypoglycemia. The hypoglycemia may have resolved after the endoscopic dilatation of cologastric anastomosis had corrected food regurgitation. Thus, the diet may not have reached the ileocolic conduit, and may not have stimulated an incretin response from ileocolic conduit. Several months later, we weaned the patient of enteral feeding, and he could tolerate oral intake without developing postprandial hypoglycemia.

5 | DISCUSSION

We have reported a case of postprandial hypoglycemia in a patient with severe esophageal stricture who had undergone ileocolic interposition and an antecolic Billroth-II gastrojejunostomy with Braun jejunojejunostomy. The patient experienced symptomatic hypoglycemia 2 h after enteral feeding by NG tube into the remnant of his stomach. Hypoglycemia developed after he had experienced a burning sensation in his chest, suggesting food reflux. The hypoglycemia may have been caused by the enteral feeding by NG tube, precipitating food reflux to the ileocolic conduit, stimulating a postprandial GLP-1 response. This may have caused an exaggerated hyperinsulinemia, which then caused rebound hypoglycemia. All symptoms resolved after switching the route of feeding from NG to jejunostomy tube to bypass the ileocolic conduit. Thus, the postprandial hypoglycemia could not be explained by the Billroth-II operation.

TABLE 1 Laboratory values from the mixed meal tolerance test.

Time	Capillary glucose (mg/dL)	Venous plasma glucose (mg/dL)	Insulin (IU/mL)	C-peptide (ng/mL)	Beta-OH-butyrate (mmol/L)
Baseline after fasting for 8 h	86	81	5.0	1.25	0.22
During feeding at 2 h	165				
The end of feeding at 4 h	107				
Minutes after feeding					
30	105	97	9.8	6.64	<0.1
60	79	71	4.7	3.250	<0.1
90	80	73	3.5	2.21	<0.1
120	83	73	2.6	1.67	<0.1
150	86	80	3.5	1.49	<0.1
180	87	83	2.1	1.28	<0.1

Note: The mixed meal tolerance test consisted of suspected meals fed by nasogastric tube to the remnant of the stomach with fasting and 30-min interval blood draws to obtain glucose, insulin, c-peptide, and beta-OH-butyrate levels.

Postprandial reactive hypoglycemia, or late dumping syndrome, is a common complication of esophageal and gastric surgery as well as bariatric surgery.[8] These procedures change the gastric anatomy and cause rapid delivery of nutrients into the distal small bowel, particularly at the ileum. Rapid carbohydrate absorption induces a higher peak response in glucose, stimulating an incretin-driven hyperinsulinemic response, causing subsequent hypoglycemia, and is related to late dumping syndrome. An exaggerated GLP-1 response is the key mediator of the hyperinsulinemic and postprandial hypoglycemia.[9–14]

GLP-1 is an incretin hormone produced in the L-cell of the distal intestine, particularly in the ileum and colon. It is secreted after a meal and stimulates postprandial insulin secretion.[15,16] Bariatric surgery, particularly RYGB, is the procedure most associated with post-bariatric surgery hypoglycemia.[8] Postprandial GLP-1 secretion increases by as much as 10-fold in patients undergoing RYGB comparing with non-surgical obese patients,[17] and is significantly higher in patients with post-bariatric surgery hypoglycemia.[12] The pathogenesis of this condition has not been fully elucidated. Nevertheless, an exaggerated postprandial GLP-1 response is a critical mediator for post-bariatric surgery hypoglycemia.

Ileal interposition, during which a length of the ileum is moved proximally and positioned in the jejunum, alone or in combination with sleeve gastrectomy, is used to treat type 2 diabetes in obese and non-obese patients.[18,19] The procedure exposes the interposed ileal segment to ingested nutrients, causing increase in plasma incretin, particularly GLP-1, stimulating insulin secretion.[20] Surgery improves glucose tolerance and insulin sensitivity,[21] and delays the onset of diabetes.[20]

To the best of our knowledge, this is the first ever reported case of postprandial hypoglycemia after ileocolic interposition. Although we could not confirm the hypoglycemia by MMTT, and the specific mechanism is unclear, we hypothesize food reflux to the ileocolic conduit may have caused an exaggerated hyperinsulinemic response and rebound hypoglycemia.

Our patient had the typical manifestations of hypoglycemia with a nadir glucose level of 59 mg/dL 2 h postprandially. Late dumping syndrome is more accurately classified by hypoglycemic symptoms rather than nadir glucose level during oral glucose tolerance test (OGTT).[17] Symptoms of postprandial reactive hypoglycemia, or late dumping syndrome, usually occur 1–3 h postprandially.[1] Hypoglycemia is usually defined by Whipple's triad,[22] consisting of[1] symptomatic hypoglycemia,[2] documented low plasma glucose, and[2] resolution of clinical signs of hypoglycemia after the plasma glucose is normalized. Hypoglycemic symptoms are non-specific, and are classified as autonomic (i.e., sweating, hunger, palpitation, anxiety, and tremors) and neuroglycopenic (i.e., fainting, weakness, blurred vision, dizziness, confusion, and cognitive impairment). Autonomic symptoms occur if the plasma glucose level drops below 3.3 mmol/L, or 60 mg/dL, and neuroglycopenic symptoms predominate when glucose level drops below 2.8 mmol/L, or 55 mg/dL.[6] Although, no consensus on the definitive cut-off value for postprandial hypoglycemia in patients after gastric surgery or post-bariatric surgery exists, the use of a glucose level below 3.3 mmol/L, or 60 mg/dL, has been suggested as a sensitive cut-off value for the diagnosis of postprandial hypoglycemia during MMTT or OGTT in both pediatric and adult patients.[23–25] By continuous glucose monitoring (CGM) of postprandial hypoglycemia, a cut-off value >3.9 mmol/L, or > 70 mg/dL, was proposed.[26] MMTT is the gold standard provocation test to detect postprandial hypoglycemia. The other test is the OGTT. Both have shown a high positive result in post-bypass hypoglycemia, but there is a weak correlation between laboratory results and the occurrence of the symptoms, and positive tests may be detected in healthy volunteers.[25,27] CGM measures the glucose level to detect postprandial hypoglycemia, but the variability in glucose level increases after gastric bypass.[25] The incidence of hypoglycemia regardless of late dumping syndrome symptoms are 75% and 29% for CGM and MMTT, respectively.[28]

Management of postprandial reactive hypoglycemia is challenging. Dietary modification is the initial and usually-effective approach. The mainstay of treatment is avoidance of a high glycemic index diet and simple sugars. Dietary modification includes splitting meals into multiple small, frequent meals (five to six meals per day), increasing the dietary soluble fiber intake to help increase intestinal transit times, delay glucose absorption, and ameliorate postprandial hypoglycemia. The rate of switching from feeding by bolus to continuous feeding also causes a lower peak glucose and lessens the hyperinsulinemic response. Alpha-glucosidase inhibitors, acarbose, miglitol, and voglibose are all oral hypoglycemic agents decreasing carbohydrate absorption by pancreatic alpha-amylase inhibition. They can decrease the postprandial glucose surge, and prevent postprandial reactive hypoglycemia. Calcium channel blockers, diazoxide, and somatostatin analogues have also been used to treat postprandial hypoglycemia.[1,8] The surgical options for mechanism correction and insulin reduction are RYGB reversal and distal pancreatectomy, but their benefits remain controversial. According to the current body of evidence, pancreatectomy is not recommended in the setting.[8,29] Our patient's hypoglycemic

symptoms improved after the nutrition intervention, and we weaned him off intravenous glucose infusion successfully.

6 | CONCLUSION

To the best of our knowledge, this is the first ever reported case of postprandial hypoglycemia after ileocolic interposition. Although we could not confirm the episodes of hypoglycemia by MMTT or clearly demonstrate the specific mechanism, we suggest gastroesophageal reflux to the ileocolic conduit may have caused an exaggerated hyperinsulinemic response and rebound hypoglycemia. This syndrome should be considered in the patient who has had ileocolic interposition surgery and who has suspected postprandial hypoglycemia.

AUTHOR CONTRIBUTIONS

Chanita Unhapipatpong: Conceptualization; data curation; formal analysis; investigation; methodology; project administration; resources; software; validation; writing – original draft; writing – review and editing. **Pitichote Hiranyatheb:** Conceptualization; formal analysis; resources; supervision; validation; writing – review and editing. **Pariya phanachet:** Supervision; writing – review and editing. **Daruneewan Warodomwichit:** Conceptualization; methodology; supervision; writing – review and editing. **Chutintorn Sriphrapradang:** Investigation; methodology; supervision; writing – review and editing. **Prapimporn Chattranukulchai Shantavasinkul:** Conceptualization; data curation; formal analysis; investigation; methodology; project administration; supervision; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors have no conflicts of interest to declare.

DATA AVAILABILITY STATEMENT

All data used is in the manuscript.

ETHICS STATEMENT

This case report was approved by the Human Research Ethics Committee, Faculty of Medicine Ramathibodi Hospital, Mahidol University (code, MURA2019/1011).

CONSENT

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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