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Case Report

Laparoscopic Spleen-Preserving Distal Pancreatectomy as Treatment for Nesidioblastosis after Gastric Bypass Surgery

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Nesidioblastosis is characterized by hyperfunction of pancreatic islets caused by hypertrophic beta cells. Postprandial symptoms of hypoglycemia are the clinical presentation of the disease. A female patient with diabetes mellitus who underwent a Roux-en-Y gastric bypass began to present postprandial symptoms of hypoglycemia. There was no radiologic (MRI) evidence of insulinoma. Selective arterial calcium-stimulation test identified hyperfunction only in the splenic artery. Laparoscopic spleen-preserving distal pancreatectomy was performed. The patient has been entirely free of any postprandial symptoms for 10 months after the partial pancreatectomy.

Key words: Nesidioblastosis, gastric bypass surgery, hypoglycemia, laparoscopic distal pancreatectomy

Introduction

Nesidioblastosis is characterized by hyperfunction of pancreatic islets caused by hypertrophic beta cells. Some patients who have undergone gastric bypass surgery have postprandial symptoms that have been ascribed to rapid emptying of gastric contents.[2] This phenomenon, referred to as the dumping syndrome, is characterized by vasomotor symptoms of diaphoresis, weakness, dizziness, and flushing, but not neuroglycopenia.[3] We are reporting a patient whose clinical presentation typified that of the non-insulinoma pancreatogenous hypoglycemia syndrome (postprandial neuroglycopenic hyperinsulinemic hypoglycemia and pancreatic nesidioblastosis).[4] There is some speculation that hyperfunction of pancreatic islets did not lead to obesity but that beta-cell trophic factors may have increased as a result of gastric bypass. We report a case of laparoscopic spleen-preserving distal pancreatectomy for nesidioblastosis after gastric bypass surgery.

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Case Report

A.O.C., a 34-year-old woman who had undergone a Roux-en-Y gastric-bypass (RYGBP) 18 months previously, presented with postprandial hypoglycemia 12 months after the surgery. High blood pressure had been diagnosed at the age of 21 and diabetes mellitus during bariatric preevaluation.

Laboratory Analysis

The patient presented with glycemia of 46 mg/dl, insulin of 22 μU/ml and C-peptide of 5.4 nanog/ml, which confirmed the symptoms of hypoglycemia.

Selective arterial calcium-stimulation tests were[5] performed as a measure of the basal insulin level in the right hepatic vein in response to the sequential injection of calcium into the splenic, superior mesenteric, and gastroduodenal arteries. The double

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gradient detected in the hepatic vein after sequential calcium injection in those arteries was considered to indicate hyperfunction of the beta cells in the vascular distribution of the artery studied, which was identified in the angiographic findings.[5] In general, the body and the tail of the pancreas were within the splenic-artery distribution, and the head and the uncinate process of the pancreas were within the gastroduodenal artery and superior mesenteric artery distribution.

Selective arterial calcium-stimulation test identified hyperfunction only in the splenic artery. This finding demonstrated hyperfunction only in the distal pancreas (Table 1).

Laparoscopic Operative Procedure

A 45° laparoscope was introduced through a Hasson's trocar inserted at the umbilicus by the open method, and 5 trocars were placed.

A window was created in the gastrocolic ligament as the surgeon retracted the greater curvature of the stomach, using the Harmonic Ace® (Ethicon, Cincinnati, OH) to dissect the ligament. The stomach and colon were retracted, and the body and tail of the pancreas were freed from the dorsal attachments of the stomach up to the splenic hilum, which allowed visualization of the body and the tail of the pancreas. The inferior border of the pancreas was mobilized by opening the fatty tissue.

The splenic artery was dissected and separated from the upper border of the pancreas. The distal pancreas was retracted both anteriorly and inferiorly to expose and bind the transverse branches of the splenic artery and vein to preserve the spleen. Several transverse branches of the splenic vein were carefully bound with titanium clips, and mobilization of the body and tail was successfully achieved.

Table 1. Selective arterial calcium-stimulation test

Arteries	Basal	After Calcium-stimulation		
		30+	60+	120+
Splenic*	10	19	13	10
Gastroduodenal*	22	16	09	17
Superior mesenteric*	07	05	06	09

*insulin level in μU/ml; +time in seconds.

The pancreas was transversely transected with the Harmonic Ace® at the level of the superior mesenteric artery. The proximal pancreas was sutured, and biologic glue was applied.

A Penrose drain was inserted and positioned in the region of the pancreatic transection. The specimen was then extracted (Figure 1) by means of an endoscopic bag through a minimally enlarged umbilical incision, and all fascial incisions were closed. Postoperative course was uneventful, and the patient was discharged in 6 days.

Pathological Findings

Hypertrophic beta cells (Figure 2) and conductuloinsular complexes[6] were found on histologic analysis, which confirmed the nesidioblastosis diagnosis.

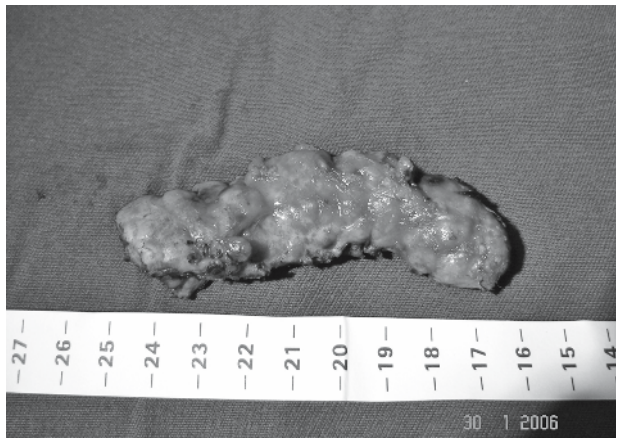


Figure 1. Surgical specimen – body and tail of pancreas.

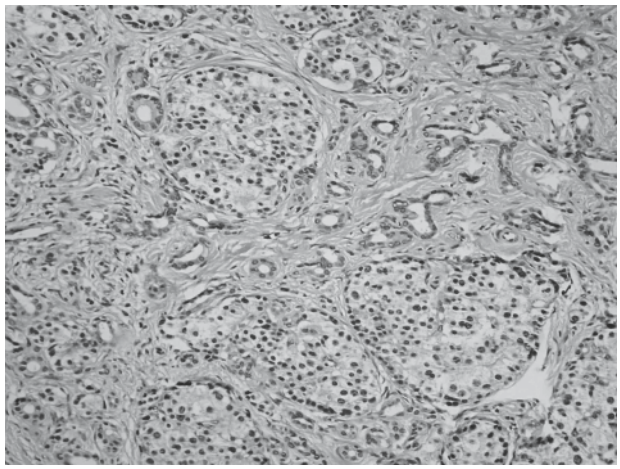


Figure 2. Pathological analysis showed hypertrophic beta cells (H & E, medium power).

Follow-up

The patient has been entirely free of any postprandial symptoms for 10 months after the partial pancreatectomy. The last laboratory tests demonstrated glycemia of 83 mg/dl, insulin of 6.5 μU/ml, and C-peptide of 1.5 nanog/ml.

Discussion

Persons with the dumping syndrome as a result of previous gastric surgery[4] have been reported to have increased levels of glucagon-like peptide-1,[7,8] possibly owing to the rapid presentation of nutrients (a stimulus for the secretion of this peptide) to the distal ileum, the site of L cells which are the source of glucagon-like peptide-1. Glucagon-like peptide-1 increases beta-cell mass in rodents through neogenesis and proliferation[9,10] and decreases apoptosis of islets in humans.

The diagnosis suspicious of pancreatic hyperfunction after the RYGBP surgery was made in a patient with postprandial symptoms of hypoglycemia, a decrease in level of glycemia, an increase of level of insulin, a positive selective arterial calcium-stimulation test[4] and an absence of radiologic (MRI) evidence of insulinoma.

The distal pancreatectomy or reversal of the gastric bypass are the options of treatment.

We performed laparoscopic pancreatectomy for treatment of nesidioblastosis, with an excellent result similar to the other six patients already described[4] who underwent open surgery.

Our patient underwent a laparoscopic distal pancreatectomy because there were strong clinical and laboratory indications of hyperfunction of the pancreas. Besides, she already had diabetes, which reinforces the suspicion of hypertrophy of beta cells and reduces the chances of an insulinoma as the cause.

Therefore, a choice has to be made: either reverse the gastric bypass and continue with previous problems like obesity and diabetes, or choose the distal pancreatectomy to treat nesidioblastosis and avoid having diabetes and obesity again. The medical team and the patient chose the pancreatectomy, which was successful. To reinforce this decision, we require more research about this cause, which is justified by the increase of gastric bypass surgery[11] and consequently the future understanding of nesidioblastosis.

Conclusion

In selected cases, when there is strong diagnostic suspicion of hypertrophic beta cells, laparoscopic pancreatectomy is an excellent option for treatment of nesidioblastosis.

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