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the immediate postoperative period and a late form occurring from weeks to years following surgery. The incidence of gastric atony varies with the indication for surgery and type of procedure initially performed. In patients operated on for gastric outlet obstruction, for example, the incidence of postsurgical gastroparesis is as high as 27%–50%. Gastroparesis is seen in 1.4% of patients following vagotomy and drainage, in 2.4%–9% of patients after vagotomy and antrectomy, and in 3% of patients following subtotal gastrectomy without vagotomy. Overall, the incidence of postsurgical gastroparesis is 2%–3% [1].

In patients who have previously undergone Roux-en-Y reconstruction, gastroparesis or stasis in the Roux limb are termed the Roux syndrome. The incidence of this syndrome has been reported to be as high as 25%–30%. The incidence in patients who undergo Roux-en-Y diversion as a means of treating alkaline reflux gastritis is 10%–50%.

Patients with postsurgical stasis syndromes present with postprandial epigastric fullness or bloating, early satiety, epigastric pain, nausea, and vomiting of incompletely digested food. Some patients develop bezoars. Patients may attempt to compensate for their condition by consuming only soft, semisolid foods or liquids. Weight loss and nutritional deficiencies often result.

The diagnosis of postsurgical stasis syndromes is one of exclusion, as symptoms mirror those of other postgastrectomy syndromes. These include mechanical problems such as stomal obstruction from edema, kinking, or stricture, recurrent ulcer, and gastric stump carcinoma. In addition, symptoms may be confused with those of afferent or efferent limb syndrome or postoperative small bowel obstruction. Functional problems, such as gastroparesis related to diabetes mellitus, may be confused with a postoperative complication. These conditions must be excluded before instituting therapy for presumed postsurgical stasis. When surgery is necessary, a near-total gastrectomy is indicated with good long-term results [71,72].

Afferent limb syndrome

The afferent limb syndrome occurs in rare patients following Billroth II gastrojejunostomy. In this condition, the limb of duodenum and jejunum responsible for proximal intestinal, biliary, and pancreatic drainage becomes partially or completely obstructed proximal to the gastric anastomosis. The cause of this obstruction is mechanical and is usually related to excessive length. When the limb is too long, internal small bowel herniation, kinking from redundancy or adhesions, loop volvulus, and intussusception can occur. In addition, obstruction at the gastrojejunostomy from recurrent ulceration, stricture formation, and carcinoma may cause the afferent limb syndrome.

Two forms of afferent limb syndrome have been described: acute and chronic. Acute afferent limb syndrome occurs in the early postoperative period, usually within the first week. Obstruction of the afferent limb leads to accumulation of intestinal, pancreatic, and biliary secretions within the proximal jejunal lumen. As lumenal pressure increases, venous pressures are quickly exceeded, resulting in ischemia and pressure necrosis of the intestinal mucosa. Obstruction of pancreatic and biliary outflow may arise if lumenal pressures are sufficiently elevated. Disruption of the duodenal stump may result.

The symptoms of acute afferent limb syndrome are abrupt in onset. Severe epigastric pain, nausea, nonbilious vomiting, tachycardia, and fever are uniformly present. Abdominal tenderness and fullness are usually present. If uncorrected, shock may ensue. Elevated serum amylase and liver function tests often confuse the clinician into believing postoperative pancreatitis or biliary pathology is the cause of the patient's deterioration. This leads to delay in appropriate surgical management. The diagnosis is confirmed by ultrasound or computed tomography scan, which reveals a fluid-filled mass in the epigastrium. Acute afferent limb syndrome is a form of closed loop obstruction and, as such, is a surgical emergency. Mortality rates associated with acute afferent limb syndrome approach 50% [1].

Chronic afferent limb syndrome may occur at any point in time after the initial surgery. It results from intermittent, partial mechanical obstruction of the afferent limb. Patients typically present with postprandial epigastric discomfort, pain, and fullness. This results from the rapid accumulation of pancreatic, biliary, and duodenojejunal secretions within the limb in response to a meal. As intralumenal pressure exceeds the resistance to outflow caused by the obstruction, the patient experiences explosive bilious vomiting, usually void of foodstuff. This emesis relieves the patient of symptoms. These last two features distinguish chronic afferent limb syndrome from alkaline reflux gastritis, in which emesis usually contains undigested food and does not relieve the discomfort. A minority of patients develop bacterial overgrowth in the partially obstructed afferent limb, resulting in a blind loop syndrome. Plain abdominal radiographs are usually nondiagnostic. Ultrasound or computed tomography is the test of choice. Endoscopy should be performed to exclude the presence of alkaline gastritis, recurrent ulcer, anastomotic stricture, and anastomotic carcinoma.

Patients with afferent limb syndrome require remedial surgery. In the average patient, this is best accomplished via conversion to a Roux-en-Y gastrojejunostomy as seen in Figure 44.11. Alternatively, a Braun enteroenterostomy between the afferent and efferent limbs is effective in decompressing the obstructed afferent limb. In severely ill patients with acute obstruction, the afferent limb and duodenal stump must be examined for viability. In the rare patient with necrosis of the afferent limb, resection of the devitalized tissue, reconstruction of the afferent limb, bypass of the obstruction, and drainage of the duodenal stump is indicated. If extensive necrosis exists, pancreaticoduodenectomy may