Other Forms of Gastroparesis

Postsurgical, Parkinson, Other Neurologic Diseases, Connective Tissue Disorders

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KEYWORDS

• Gastroparesis • Parkinson disease • Multiple sclerosis • Motor neuron disease • Neurologic • Post-surgical • Fundoplication • Scleroderma

KEY POINTS

- Fundoplication, bariatric procedures, and pancreatic surgeries are nowadays the surgical approaches most commonly complicated by gastroparesis.
- Virtually any neurologic disorder may be complicated by gastroparesis, and its development may affect nutrition and drug availability.
- Gastroparesis is a common feature of gastrointestinal involvement in scleroderma and other connective tissue disorders.

POSTSURGICAL GASTROPARESIS

Although acute gastroparesis may be a component of the ileus syndrome that can complicate many surgical procedures and of the acute pseudo-obstruction syndrome that may accompany severe sepsis and multiorgan failure, this review focuses on chronic manifestations of gastric dysmotility.[1] In contrast to chronic gastroparesis, whose pathophysiology is often poorly understood, inflammatory processes seem fundamental to the inhibition of motility in the acute form.

Gastroparesis and other disorders of gastric sensorimotor function may complicate specific surgical procedures.[2] In the important Olmstead County study of the community prevalence of gastroparesis, 7.2% of all cases of definite gastroparesis were related to prior gastrectomy or fundoplication.[3] Rates of postsurgical gastroparesis vary widely depending on many factors, including the site and nature of the surgical procedure. For example, in their comprehensive review, Dong and colleagues[4] noted

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that rates ranged from 0.4% to 5% following gastrectomy, from 20% to 50% after pylorus-preserving pancreaticoduodenectomy, and from 50% to 70% after cryoablation therapy for pancreatic cancer.

Vagotomy

Although vagotomy is infrequently performed nowadays in the management of acid-peptic disease, inadvertent vagal injury may complicate other interventions, rendering an understanding of the complex effects of vagotomy on gastric motor function still relevant. Receptive relaxation, a vagally mediated reflex, is impaired. As a consequence, the early phase of liquid emptying is accelerated. This acceleration causes rapid emptying of hyperosmolar solutions into the proximal small intestine and may result in the early dumping syndrome. By contrast, and as a consequence of impaired antropyloric function, the later phases of liquid and solid emptying are prolonged by vagotomy. Other motor effects of vagotomy include an impairment of the motor response to feeding (which contributes to the pathophysiologic mechanisms of postvagotomy diarrhea) and a suppression of the antral component of the migrating motor complex. The latter phenomenon is particularly prevalent among individuals who have symptomatic postvagotomy gastroparesis.

The now standard addition of a drainage procedure, such as a pyloroplasty or gastroenterostomy, has tended to negate the effects of vagotomy alone. In most patients, the net result of the combined procedure is little alteration in the gastric emptying of liquids or solids. Thus, prolonged postoperative gastroparesis (ie, lasting longer than 3–4 weeks) is, in fact, rare (<2.5% of patients after either vagotomy and pyloroplasty or vagotomy and antrectomy).[5] Significant postoperative gastroparesis may occur, however, in patients who have a prior history of prolonged gastric outlet obstruction. In this circumstance, normal gastric emptying may not return for several weeks.

Longitudinal studies suggest that vagotomy-related gastroparesis tends to resolve over time, with one study suggesting gastric emptying rates in those who had undergone either a truncal or a highly selective vagotomy being similar by 12 months after the procedure.[6]

Persisting postsurgical gastric motor dysfunction often presents a formidable management challenge. Therapeutic responses to prokinetic agents have proved particularly disappointing in this group. In these resistant cases, a completion gastrectomy may be the best alternative. It should be noted, however, that in one large series this approach was deemed successful in only 43% of patients.[7]

Gastrectomy

Antral resection by removing the antral mill renders the stomach incontinent to solids and leads to accelerated emptying, and symptomatic “dumping” may occur in up to 50% of patients after Billroth I or II gastrectomy.[8] Late dumping symptoms occur 90 to 120 minutes after a meal and are a consequence of reactive hypoglycemia. The accommodation reflex is impaired among symptomatic patients.[9] By contrast, delayed gastric emptying sometimes occurs after a Billroth II gastrectomy as a result of a large atonic gastric remnant.[8] Meng and colleagues[10] reported a 6.9% frequency of gastroparesis among 563 patients who underwent radical gastrectomy for gastric cancer in their unit in Shanghai, China. Preoperative gastric outlet obstruction and the performance of a Billroth II anastomosis were the principal risk factors for the occurrence of gastroparesis. Of note, they documented a similar rate of gastroparesis (3.7%) among a smaller group of patients who underwent a laparoscopic gastrectomy.[10] Laparoscopy-assisted, pylorus-preserving gastrectomy represents a