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Gastric and intestinal dysmotility syndromes

Disorders of motility of the stomach and small intestine are common, and patients often present with a variety of nonspecific symptoms, including nausea, abdominal pain, bloating, and vomiting. The challenge for the internist is determine the nature and extent of motility dysfunction.

GASTRIC DYSMOTILITY

Rule out mechanical obstruction before considering a diagnosis of gastroparesis

Gastric motor dysfunction comprises two main disorders: accelerated and delayed gastric emptying.

Accelerated gastric emptying is seen almost without exception after gastric surgery, specifically vagotomy and drainage procedures that result in dumping. Typical symptoms occur after meals and include nausea, bloating, pain, diarrhea, and vasomotor symptoms such as syncope, flushing, and palpitations.

Most office-based internists are more likely to encounter delayed gastric emptying (also known as gastroparesis). Typical symptoms of gastroparesis include early satiety, nausea and vomiting (mostly postprandial), abdominal pain, and weight loss. Individually, none of these symptoms indicates delayed gastric emptying, but when they occur together, the likelihood of gastroparesis is strong.

However, before one considers a diagnosis of gastroparesis, mechanical obstruction must be ruled out. Benign or malignant pancreatic disease or mesenteric ischemia should be suspected if weight loss and pain are the predominant symptoms.

Causes of gastroparesis

Gastroparesis can be a feature of many disorders and syndromes, such as collagen vascular diseases and metabolic and endocrine disorders, and an effect of medications such as anticholinergics or narcotics. However, most cases encountered in clinical practice are caused by vagotomy, diabetes, or unknown factors.

Vagotomy. Postsurgical gastroparesis occurs less frequently now that antibiotic treatment for *Helicobacter pylori* infection and potent acid-suppressing medicines have replaced surgery for treating peptic ulcer. Most patients who undergo a vagotomy and a pyloroplasty or gastroenterostomy experience little alteration in gastric emptying of solids, although the initial phase of liquid emptying can be accelerated, resulting in dumping. Fewer than 3% of postgastrectomy patients experience clinically significant gastroparesis.

Diabetic gastroparesis. Altered gastrointestinal function is common among patients with longstanding type I diabetes mellitus, particularly those with complications such as retinopathy, peripheral neuropathy, or nephropathy. Delayed emptying of solids is common in diabetic patients and is frequently asymptomatic.

Among symptomatic patients, gastroparesis frequently runs a fluctuating course, with episodes of pronounced symptoms interspersed with relatively symptom-free intervals. Detecting gastroparesis is especially important in patients with diabetes because it interferes with nutrient delivery to the small intestine and can result in fluctuating blood glucose levels.

Gastric motor abnormalities in diabetes include loss or disruption of antral migrating motor complex (MMC) activity, fasting and postprandial antral hypomotility, and abnormalities of the proximal small intestine. The etiology of gut dysmotility in diabetes is not clear, but evidence exists for autonomic nervous system and enteric nervous system dysfunction, in addition to the metabolic effects of hyperglycemia.

Idiopathic gastroparesis. Many patients with gastroparesis have no primary abnormality. Most are young women. Some of these patients report having a viral-like illness before the onset of gastric symptoms. Specific viral infections, such as cytomegalovirus and herpes simplex, are known to cause gastroparesis in immunocompromised patients; the gastroparesis resolves quickly following antiviral therapy.

Diagnosis

Gastroparesis in diabetic patients interferes with nutrient delivery and can result in fluctuating blood glucose levels

The most important diagnostic step for a patient who presents with nausea, vomiting, bloating, or postprandial abdominal pain is to rule out mechanical obstruction, either of the stomach or the small intestine. After mechanical obstruction has been ruled out, gastric motor function is best evaluated by a radioscintigraphic solid-emptying study. This test, in which the patient consumes a meal that contains a radioisotope, allows for a simple, sensitive, quantitative, noninvasive measure of solid-phase emptying.

TREATMENT OF GASTRIC DYSMOTILITY

Dietary changes

If radioscintigraphy reveals delayed gastric emptying, the initial phase of treatment should be directed toward dietary changes.

Small, frequent meals, preferably in liquid form, are encouraged because gastric emptying is volume-dependent, and liquids empty faster than solids.

A low-fat diet is helpful because fat is the most potent inhibitor of gastric emptying.

A low-residue diet is also important because the disturbed motor activity prevents nondigestable food from emptying, and can result in bezoar formation.

Pharmacologic therapy

Available agents that stimulate gastric emptying are not ideal, because they have an inconsistent effect on the motor function of the stomach. The ideal prokinetic agent for gastroparesis would target the defined pathophysiology, enhance stomach function (rather than contractions), and cause few side effects.

Cisapride is the most commonly used prokinetic agent because of its good safety profile. It is usually given at 10 mg four times a day at least 30 minutes before meals and at bedtime. Cisapride has a very good safety profile and minimal side effects.

Metoclopramide, a dopamine agonist, has efficacy equal to that of cisapride. However, up to 20% of patients have side effects, the most troubling of which are related to extrapyramidal manifestations and mood changes.

Erythromycin is not particularly effective as a prokinetic agent when given in oral form.

Cholinergic agents are rarely used because of their high incidence of side effects.

If tolerance develops to one of these prokinetic agents, I would increase the dose or add another prokinetic agent.

Surgical therapy

If dietary and pharmacologic methods fail in patients with medical causes of gastroparesis, a feeding jejunostomy may be attempted.

Subtotal gastrectomy and Roux-en-Y gastrojejunostomy can be helpful in severe postsurgical cases of gastroparesis, but are not effective in cases caused by medical diseases.

Gastric pacing, which requires the placement of stimulating electrodes on the stomach, is experimental and its value has yet to be established.

INTESTINAL DYSMOTILITY

Small-bowel motor activity and its control mechanisms are quite similar to those of the antrum of the stomach. Normal small bowel motility serves to mix nutrients with digestive

enzymes, allows sufficient time for absorption, and propels undigestible parts of the chyme distally to the colon. Symptoms of intestinal dysmotility can be quite similar to those of gastroparesis, though abdominal distention and changes in bowel habits indicate small-bowel disease. The disease is typically chronic.

Diagnosis

There is no adequate functional study of the small bowel that is analogous to solid phase gastric emptying studies of the stomach. A diagnosis of intestinal dysmotility must rely on symptoms, plain abdominal radiographs, barium studies, and intestinal manometry. As in gastric dysmotility, mechanical obstruction should be ruled out in the evaluation of patients with symptoms of intestinal dysmotility.

Flat abdominal radiographs are not sensitive enough to distinguish obstruction from pseudo-obstruction, and contrast studies may be needed to exclude mechanical obstruction. If pseudo-obstruction is suspected, I would proceed to manometry.

Manometry. Certain abnormal patterns on abdominal manometry (eg, lack of response to food) are consistent with visceral neuropathy. Manometry has particular value in patients with unexplained symptoms and a negative work-up. In such cases, normal findings on manometry help to exclude dysmotility.

Treatment

Treatment of intestinal dysmotility includes a low-fat diet, antibiotics (cycled to prevent bacterial overgrowth), and prokinetic agents. In general, prokinetic agents are less effective in treating intestinal dysmotility than in gastroparesis.

In refractory or severe, intermittent intestinal dysmotility, a venting jejunostomy is helpful. Every attempt should be made to use the small bowel by infusion of liquid formulas including elemental preparations. Total parenteral nutrition may be required with bowel failure. Intestinal transplantation and pacing are still experimental. ■

SUGGESTED READING

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