خزل المعدة
Gastroparesis

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INTRODUCTION and DEFINITION

- Normal gastrointestinal motor function is a complex series of events that requires coordination of the sympathetic and parasympathetic nervous systems, neurons and pacemaker cells (called interstitial cells of Cajal) within the stomach and intestine, and the smooth muscle cells of the gut. Abnormalities of this process can lead to a delay in gastric emptying (gastric stasis)
- Gastroparesis is a syndrome of objectively delayed gastric emptying in the absence of a mechanical obstruction and cardinal symptoms of nausea, vomiting, early satiety, bloating, and/or upper abdominal pain

ETIOLOGY

- **Idiopathic** Idiopathic gastroparesis may be the most common form of gastroparesis. It is estimated that no detectable primary underlying abnormality is found in approximately one-half of patients with delayed gastric emptying
- **Diabetes mellitus** Patients with diabetes mellitus have abnormalities at several levels in the process of gastric emptying, including abnormal postprandial proximal gastric accommodation and contraction, and difficulties with antral motor function. These abnormalities are primarily due to autonomic dysfunction or abnormal intrinsic nervous system).
- Hyperglycemia (blood glucose >200 mg/dL) may also contribute to delayed gastric emptying. Although acute hyperglycemia, associated with poorly controlled diabetes, typically has a reversible effect on gastric emptying, chronic hyperglycemia is associated with an increased risk of neuropathy.
- **Viral** rotavirus, cytomegalovirus, Epstein-Barr virus, and varicella-zoster virus
- **Medications** Several medications can delay gastric emptying.

(Narcotics, Alpha-2-adrenergic agonists, Tricyclic antidepressants, Calciumchannel blockers, Dopamine agonists, Octreotide ….)

ETIOLOGY

- **Postsurgical** — Previous gastric and thoracic surgery can result in gastric stasis due to intended or accidental injury to the vagus nerves (eg, with Billroth II gastrectomy, fundoplication, lung or heart transplantation)
- **Neurologic disease**
  ●Extrinsic neural control (eg, the vagus nerve and lower thoracic spinal sympathetic outflow) may be affected in disorders such as multiple sclerosis, brainstem stroke or tumor, diabetic or amyloid neuropathy, or primary dysautonomias.
- ●The myenteric plexus may be involved in a degenerative, diffuse neurologic disorder (eg, diabetes, AIDS, or parkinsonism). Focal loss of intrinsic inhibitory innervation of the pylorus is responsible for congenital pyloric stenosis. In Parkinson disease, enteric nervous system dysfunction has been reported to precede motor symptoms by years to decades
- ●Medications used to treat neurologic disease can also contribute to gastric stasis (eg, anticholinergics, dopaminergics).

ETIOLOGY

- **Autoimmune** — Delayed gastric emptying has been described in association with autoimmune gastrointestinal dysmotility . Autoimmune gastrointestinal dysmotility is a dysautonomia affecting the gastrointestinal tract that occurs idiopathically or in association with an anatomically remote neoplasm, most commonly small cell lung cancer. In addition to delayed gastric emptying, affected patients may have slow intestinal transit, slow colonic transit, and pelvic floor dyssynergia. The role of the autoimmune process in cases of acquired dysautonomia manifesting with symptoms that include gastroparesis is illustrated by the response to plasmapheresis treatment
- **Other** — Other causes of gastroparesis include mesenteric ischemia and diseases that result in infiltration or degeneration of the muscle layer of the stomach (eg, scleroderma). Patients with gastric or intestinal involvement due to scleroderma usually have clinically evident systemic disease involving the skin, lungs, and/or the esophagus

EVALUATION

- Gastroparesis should be suspected in patients with nausea, vomiting, early satiety, postprandial fullness, abdominal pain, or bloating. The goal of evaluation is to exclude a mechanical obstruction and establish the diagnosis of gastroparesis by an assessment of gastric motility. Evaluation should begin with a history and physical examination.
- **Exclude mechanical obstruction**
- **Assess gastric motility**
  - **Scintigraphic gastric emptying**
  - **Wireless motility capsule**
  - **^{13}C breath testing**

Treatment of gastroparesis

- **INITIAL MANAGEMENT**

1. **Dietary modification**
2. **Hydration and nutrition**
3. **Optimize glycemic control**

- **Pharmacologic therapy**

1. **Metoclopramide**
2. **Domperidone**
3. **Macrolide antibiotics**
4. **Antiemetics**

Treatment of gastroparesis

- **REFRACTORY SYMPTOMS** In patients with refractory symptoms of gastroparesis despite dietary modification, prokinetics, and antiemetics, it is important to re-evaluate compliance with dietary modification and pharmacotherapy and to provide nutritional support. We suggest placement of a jejunostomy and venting gastrostomy tube for enteral nutrition and decompression, respectively. We suggest parenteral nutrition only in patients who cannot tolerate enteral nutrition despite concomitant pharmacotherapy. We suggest gastric electrical stimulation only in patients with gastroparesis with intractable nausea and vomiting despite medical therapy for at least one year.
- **Decompression and feeding**
- **Surgery**
- **Other therapies (**Endoscopic placement of a transpyloric stent. endoscopic pyloromyotomy (gastric per-oral endoscopic myotomy) and laparoscopic pyloroplasty. However, randomized sham-controlled trials are required to establish the role of these therapeutic approaches in patients with gastroparesis. Intrapyloric injection of botulinum toxin is not recommended for patients with gastroparesis.

MANAGEMENT OF ACUTE EXACERBATIONS

- For acute exacerbations of delayed gastric emptying, we suggest erythromycin 3 mg/kg intravenously (IV) every eight hours. In patients without permanent enterostomy tubes, IV erythromycin relieves acute gastric stasis rapidly, obviating the need for nasogastric decompression. However, IV erythromycin is effective in relieving symptoms in approximately 40 percent of patients. We treat patients who fail IV erythromycin with subcutaneous metoclopramide (5 to 10 mg three times daily) after a 2 mg test dose.