Gastroparesis in the Hospital Setting

Definition and Epidemiology

Gastroparesis is a chronic condition characterized by delayed gastric emptying in the absence of mechanical obstruction, leading to symptoms such as nausea, vomiting, and early satiety. It significantly impacts quality of life and is a frequent cause of hospital admissions.

Prevalence: Affects ~4% of the U.S. population; higher in diabetic patients (5-12% of type 1 DM, 1-5% of type 2 DM). Women are disproportionately affected (4:1 female-to-male ratio).

Risk Factors: Diabetes mellitus (DM), post-viral syndromes, vagotomy, opioid use, connective tissue disorders.

Rare Demographics: Pediatric cases (often post-viral), amyloidosis, paraneoplastic syndromes.

Pathophysiology

Mechanisms: Gastroparesis results from impaired gastric motility due to dysfunction of the vagus nerve, interstitial cells of Cajal (ICC), or gastric smooth muscle. This disrupts coordinated peristalsis and pyloric relaxation, leading to stasis.

Effects: Delayed emptying causes gastric distension, increasing intragastric pressure, triggering nausea and vomiting. Chronic stasis promotes bacterial overgrowth and bezoar formation.

Molecular Pathways: In diabetic gastroparesis, hyperglycemia induces oxidative stress, reducing nitric oxide synthase (nNOS) in ICC, impairing gastric relaxation. Autoimmune damage (e.g., anti-GAD antibodies) may target enteric neurons. Loss of ICC (CD206+ macrophages) is a hallmark in idiopathic cases.

Key Pathway: Vagal/ICC dysfunction \rightarrow Impaired gastric peristalsis \rightarrow Stasis \rightarrow Symptom cascade (nausea, bloating, vomiting).

Causes

Category	Common Causes	Rare Causes	Notes
Diabetic	Type 1/2 DM, chronic hyperglycemia	Hypoglycemic agents (e.g., GLP-1 agonists)	30-50% of gastroparesis cases; HbA1c >8% increases risk
Idiopathic	Post-viral (norovirus, EBV)	Mitochondrial disorders, paraneoplastic	Often women, 20-40 y/ o; 35% of cases
Postsurgical	Vagotomy, fundoplication	Pancreaticoduodenectomy	Vagal nerve injury key mechanism
Medication- Induced	Opioids, anticholinergics	Cannabinoids, calcineurin inhibitors	Opioids slow motility via mu receptors
Systemic Diseases	Scleroderma, Parkinson's	Amyloidosis, Ehlers-Danlos syndrome	Scleroderma: Fibrosis of gastric wall
Neurological	Stroke, MS	Dysautonomia, Guillain-Barré	Central/peripheral nerve dysfunction

Clinical Presentation

- Symptoms:
 - Chronic nausea (90% of cases), vomiting (undigested food)
 - Early satiety, postprandial fullness, bloating
 - Epigastric pain (burning or cramping, 50% of cases)
 - Rare: Weight loss, anorexia (severe cases)
- Exam:
 - Abdominal distension, epigastric tenderness (non-specific)
 - Succussion splash (fluid in stomach >4h post-meal)
 - Rare: Cachexia, sclerodactyly (scleroderma), orthostasis (dysautonomia)
- Red Flags:
 - Unintentional weight loss >10%, hematemesis, dysphagia (suggest malignancy or obstruction)

Labs and Studies

- · Labs:
 - CMP: Hypokalemia (vomiting), elevated glucose (DM), hypoalbuminemia (malnutrition)
 - **CBC:** Anemia (chronic disease, B12 deficiency), leukocytosis (infection)
 - HbA1c: Assess glycemic control in DM (>8% worsens motility)
 - Advanced: Anti-GAD antibodies (autoimmune), serum porphyrins (porphyria mimic)

- Imaging:
 - Abdominal X-ray: Air-fluid levels, dilated stomach (rule out obstruction)
 - Upper Endoscopy (EGD): Rule out mechanical obstruction, bezoar, or gastritis
 - Gastric Emptying Scintigraphy: Gold standard; >10% retention at 4h confirms delayed emptying
 - Advanced: SmartPill (wireless motility capsule) measures gastric, small bowel, and colonic transit
 - Antroduodenal Manometry: Assesses pressure waves; distinguishes myopathic vs. neuropathic
- Other:
 - Breath Testing: Detects small intestinal bacterial overgrowth (SIBO) from stasis
 - Electrogastrography (EGG): Measures gastric slow-wave activity (research tool)

Diagnosis

- · Criteria:
 - Chronic nausea/vomiting, early satiety + delayed gastric emptying on scintigraphy, with no mechanical obstruction on EGD.
- Differential:
 - Functional dyspepsia, cyclic vomiting syndrome, rumination syndrome, gastric outlet obstruction, malignancy.
- Flowsheet:
- Step 1: History/Exam: Chronic nausea, fullness, DM history, or medication use
- Step 2: Labs: CMP, HbA1c to assess metabolic status; rule out electrolyte imbalance
- Step 3: EGD: Exclude obstruction, ulcers, or malignancy
- Step 4: Scintigraphy: Confirm delayed emptying (>10% at 4h)
- **Step 5:** Advanced Testing: Manometry (if neuropathy suspected), breath test (SIBO), or autoimmune panel (anti-GAD)

Treatment

- General Principles:
- Improve gastric motility, alleviate symptoms, correct nutritional deficiencies, and manage underlying causes.

- Supportive Care:
 - Dietary Modification: Small, low-fat, low-fiber meals (4-6/day); liquid diet if severe
 - Hydration: IV fluids (NS with KCl for hypokalemia)
 - Glycemic Control: Insulin optimization (DM patients, target HbA1c
 <7%)
- · Specific Therapies:
 - Prokinetics: Metoclopramide 5-10 mg TID (FDA black box for tardive dyskinesia), domperidone 10 mg TID (off-label, QT risk)
 - Antiemetics: Ondansetron 4-8 mg IV/PO q8h, prochlorperazine 10 mg q6h
 - Pain Management: Tramadol 50 mg q6h (avoid opioids), gabapentin
 300 mg TID (neuropathic pain)
- Advanced:
 - Erythromycin 125 mg TID (short-term, tachyphylaxis), prucalopride 2 mg daily (off-label)
- Interventional:
 - Botulinum toxin A pyloric injection (200 units, controversial efficacy),
 gastric electrical stimulation (Enterra device)
- Surgical:
 - Gastrojejunostomy or subtotal gastrectomy (refractory, rare)
- Rare Causes:
 - Immunosuppression (e.g., tacrolimus for amyloidosis), levodopa adjustment (Parkinson's)
- Monitoring:
 - Daily symptom assessment (nausea, vomiting frequency)
 - Weekly weight, CMP to monitor electrolytes, nutrition
 - Scintigraphy follow-up (3-6 months) to assess treatment response

Complications

- Acute:
 - **Dehydration:** Hypokalemia, metabolic alkalosis from vomiting
 - Aspiration Pneumonia: Vomiting increases risk, especially in elderly
 - **SIBO:** Stasis promotes bacterial overgrowth, causing diarrhea
- · Long-Term:
 - Malnutrition: Weight loss, vitamin deficiencies (B12, iron)
 - Bezoar Formation: Undigested food forms mass, risking obstruction
 - Rare: Esophageal candidiasis (chronic vomiting), gastric volvulus

Clinical Scenarios

Case 1: Diabetic Gastroparesis

- Presentation: 50 y/o F with type 1 DM, HbA1c 9.2%, presents with nausea, vomiting undigested food, and bloating for 3 months. Vitals: BP 130/80, HR 88, RR 16. Exam: Epigastric tenderness, no distension.
- Labs/Studies: CMP: K 3.2 mEq/L, glucose 250 mg/dL. EGD: Normal.
 Scintigraphy: 20% retention at 4h.
- Interpretation: Diabetic gastroparesis, moderate severity.
- Management: Metoclopramide 5 mg TID, ondansetron 4 mg q8h, insulin optimization. Dietary consult for small, low-fat meals. Monitor CMP daily. Discharge day 3 with GI follow-up.

Case 2: Idiopathic Gastroparesis (Refractory)

- Presentation: 35 y/o F with 2 years of nausea, early satiety, and weight loss (15% body weight). Prior metoclopramide failed. Vitals: BP 110/70, HR 92, RR 14. Exam: Cachexia, succussion splash.
- Labs/Studies: **Scintigraphy:** 30% retention at 4h. Manometry: Neuropathic pattern. Anti-GAD positive.
- Interpretation: Idiopathic gastroparesis, possible autoimmune etiology.
- Management: Domperidone 10 mg TID, trial of IV erythromycin 125 mg TID, TPN for malnutrition. Refer for gastric stimulator evaluation. Monitor weight weekly.

Case 3: Scleroderma-Related Gastroparesis (Rare)

- Presentation: 45 y/o F with scleroderma, presents with vomiting, bloating, and Raynaud's phenomenon. Vitals: BP 100/60, HR 96, RR 18. Exam: Sclerodactyly, abdominal distension.
- Labs/Studies: **Scintigraphy:** 25% retention at 4h. EGD: Normal. Breath test: SIBO positive.
- Interpretation: Gastroparesis secondary to scleroderma, complicated by SIBO.
- Management: Metoclopramide 10 mg TID, rifaximin 550 mg TID for SIBO,
 PPI for GERD. Rheumatology consult for immunosuppression. Liquid diet,
 monitor for aspiration.

Expert Tips

· Check for SIBO in refractory cases; rifaximin can improve symptoms

- Use low-dose metoclopramide (5 mg TID) to minimize tardive dyskinesia risk;
 monitor for EPS
- Consider autoimmune etiology (anti-GAD, ANA) in idiopathic cases, especially young women
- Avoid chronic erythromycin (tachyphylaxis after 4 weeks); switch to azithromycin if needed
- Trial G-POEM (gastric peroral endoscopic myotomy) in centers with expertise for refractory cases
- **Pitfall:** Misdiagnosing as functional dyspepsia; scintigraphy is key to confirm delayed emptying
- Advanced: Use EGG to guide stimulator placement; research miRNA profiles for personalized therapy

Key Pearls

Scintigraphy (>10% retention at 4h) confirms gastroparesis; EGD rules out obstruction

Diabetic gastroparesis requires tight glycemic control to improve motility

Metoclopramide is first-line but carries neurological risks; domperidone is alternative

SIBO and bezoars are common complications; screen with breath testing, EGD

Rare causes like amyloidosis or mitochondrial disorders require multidisciplinary care

References

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