

CLINICAL VIGNETTE

Gastroparesis: A Commonly Misdiagnosed Disease for Irritable Bowel Syndrome

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Case

A 28-year-old female with irritable bowel syndrome (IBS) presented to gastroenterology for a second opinion regarding her diagnosis. She states her symptoms of severe epigastric cramping began around age 12 years. Initially she was told she had “gallbladder disease”. At age 18 years old she underwent a HIDA scan which showed gallbladder dyskinesia and subsequently underwent cholecystectomy. When her abdominal cramping failed to improve post operatively she was given a diagnosis of IBS. She reports that eating fatty foods exacerbate her symptoms. She had tried multiple H2 blockers and proton pump inhibitors without any improvement. She tried dietary modification by becoming both vegetarian and gluten free for the past 3 years without much benefit. She felt she was really only able to tolerate fruit smoothies and this led to significant weight loss. In addition to her pain, she reported having a bowel movement every 3-4 days with 2-3 loose stools in a 24hr period. She reports her symptoms worsened as the day progresses. She had undergone prior endoscopies with her previous gastroenterologist which were normal.

After the 2nd opinion consultation she underwent repeat EGD and colonoscopy which were again normal. Stool studies were negative for infectious agents, serologies were negative for celiac’s disease and breath test was negative for small intestinal bacterial overgrowth (SIBO). She was empirically tried on dicyclomine and amitriptyline for treatment of IBS which also failed to significantly reduce her abdominal cramping. She was also given a trial of calcium channel blockers to empirically treat for sphincter of oddi dysfunction type 3, which did not result in any benefit either. Finally, a gastric emptying study was obtained 4 months after her initial consultation and it was positive for delayed gastric emptying. She was started on metoclopramide 10mg QID, 15 minutes before meals and before bedtime with significant improvement in epigastric cramping.

Discussion

Gastroparesis is a pathological disorder in which the stomach’s ability to empty solid food in the absence of anatomical obstruction, results in variety of symptoms ranging from: post prandial fullness, abdominal pain, nausea, vomiting, belching and most commonly bloating. Gastroparesis is diagnosed most commonly during upper endoscopy when there are retained food products in the stomach. However, the gold standard for diagnosis is a nuclear medicine gastric emptying study, gastric scintigraphy or breath testing. This disease is a result of neuro-

muscular dysfunction, which can be multifactorial. Dysfunction can due to derangements in extrinsic neural control, particularly vagal function, dysfunction of the intrinsic nerves and interstitial cells involved in the local control of gastrointestinal muscle function and the loss of function of smooth cells.¹

There are various causes of gastroparesis, however, most commonly it is idiopathic, making prevention and treatment difficult. More well understood, but less common etiologies are diabetic gastroparesis, iatrogenic (post surgical) and post infections etiologies. Diabetic gastroparesis accounts for 1/3 of all cases of gastroparesis, with 5-12% of all diabetics having symptoms of gastroparesis.² Salmonella gastroenteritis, herpes virus or Epstein-barr virus can cause acute dysautonomia that results in generalized motility disorder including gastroparesis.³ Currently it is hypothesized that gastroparesis affects 1.8% of the general population but only 0.2% have been formally diagnosed. Gastroparesis more commonly affects females but the exact reason remain elusive. Stomach motility is dependent on neuronal nitric oxide synthesis which is thought to be regulated by estrogen which may explain the predominance of females affected by this disease.⁴ Obesity is also considered to be a risk factor for gastroparesis, with 50% of patients with idiopathic gastroparesis being overweight or obese.⁵

Vagal innervation of the stomach by the vagus nerve is essential for accommodation of food. Antral contractions are controlled by extrinsic vagal innervation and intrinsic cholinergic neurons. Intrinsic inhibitory mechanisms, mediate pyloric relaxation and intragastric peristalsis. These nitrergic neurons cause relaxation of gastrointestinal muscle before a gastric contraction and inhibiting before an upstream contraction. Tachykinergic and cholinergic neurons are considered to be excitatory neurons which cause upstream contractions. The stimuli created by these different types of neurons are transmitted to the interstitial cells of Cajal, fibroblast like cells with pacemaker function and smooth muscle cells which results in a multicellular electrical syncytium ultimately resulting in coordinated contractions starting in the proximal stomach and moving to the pylorus. Analysis of various neurotransmitters, including protein gene product 9.5, vasoactive intestinal peptide (VIP), substance P, tyrosine hydroxylase, protein S100B, Kit, CD45, CD68 and neuronal nitric oxide synthase (nNOS), found only nNOS to be decreased in patients with idiopathic gastroparesis.⁶

Abdominal pain is the predominate symptom in 21% of patients with gastroparesis. Of this 21%, two thirds of them rate their pain as moderate to severe.⁷ Bloating is seen in ~41% of patients with gastroparesis. However neither of these symptoms correlate with the gastric emptying rate seen on gastric emptying studies.

In contrast IBS is a much more prevalent disease, affecting 10-12% adults in North America.⁸ The most common symptoms are recurrent abdominal pain, bloating, and changes in bowel habits, thus very similar to gastroparesis. Unlike gastroparesis however in which testing is highly recommended for making a diagnosis, IBS is not. "National guidelines for IBS management state that in a patient who has symptoms meeting the Rome IV criteria, with no alarm features, the physician should make a positive diagnosis of IBS without resorting to a battery of tests."⁹ This recommendation to avoid testing in patients with IBS symptoms can lead to misdiagnosis of gastroparesis as IBS.

Conclusion

Gastroparesis has many similar symptoms to IBS including chronic abdominal pain and bloating. When patients lack changes in bowel habits it can be difficult to differentiate between these two diagnoses. Thus while guidelines recommend history alone is needed in the majority of cases of IBS, gastric emptying studies clearly can provide benefit in differentiating between these conditions. Gastric emptying studies are non-invasive well tolerated tests that should be considered in patients with IBS symptoms in the absence of changes in bowel habits.

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