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Successful treatment of refractory functional dyspepsia with osteopathic manipulative treatment

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Functional gastrointestinal disorders (FGIDs) describe patients who present with symptoms that arise from the gastrointestinal (GI) tract yet have failed to be explained by a standard GI work-up. Somatic findings at midthoracic levels correspond to localized visceral dysfunction such as decreased peristalsis and vasoconstriction. The somatovisceral concept suggests that osteopathic manipulative treatment (OMT) provides relief by normalizing the autonomic tone. There are few reports in the literature that provide examples of successful treatment of FGID by OMT. This case describes a patient with postprandial nausea and vomiting who was successfully treated with OMT.

Case presentation

A 37-year-old female patient presented to her family physician with a four-year history of intermittent postprandial nausea and vomiting, which the patient described as "regurgitation." These symptoms were not related to a particular food, time of day, or the onset of any new medications. She reported abdominal pain only at the time of vomiting and she denied hematemesis, dysphagia, diarrhea, constipation, hematochezia, melena, or weight loss. Upon review of systems, she denied fever, flank pain, vaginal discharge, pelvic pain, dysuria, hematuria, urinary hesitancy, or incontinence. She had been seen four years previously by a gastroenterologist and had an esophagogastroduodenoscopy, upper gastrointestinal (GI) series, and trial of several medications including H-2 blockers, proton-pump inhibitors, and metaclopromide. At that time she was given a diagnosis of gastroesophageal reflux disease (GERD) and denied seeking additional testing or treatment for her GI symptoms from other providers in the intervening years.

Other than GERD, her past medical history included hypertension, hypothyroidism, ovarian cysts, major depressive disorder, transient ischemic attack, and endometriosis for which hysterectomy was performed. She was a G2P2 with a history of two normal spontaneous vaginal deliveries.

Her medications were sertraline, levothyroxine, metoprolol, amlodipine, and pantoprazole. She denied taking any over-the-counter medications or supplements.

Her relevant family history revealed her father had died at age 74 from a cancer in the small intestine of which she did not know further details. Her maternal uncle had liver cancer of which she could also not provide further details. Her social history was negative for tobacco, alcohol, and recreational drug use.

On presentation, her blood pressure was 126/94 and pulse was 84 beats per minute. Physical examination showed a healthy-appearing female weighing 165 pounds. Her abdomen was soft, nontender, nondistended, and with normoactive bowel sounds. Her osteopathic examination revealed tissue congestion over the superior

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mesenteric ganglion, a flexed T5 rotated right and sidebent right, and a group curve at T7-9 flexed rotated right and sidebent left. The remainder of her physical examination was normal.

Diagnosis and topic discussion

Given this patient's prior diagnosis of GERD by her gastroenterologist after extensive diagnostic studies, and the chronic nature of symptoms over four years without worrisome progression or serious signs such as weight loss, and coupled with the current history and physical examination, our differential diagnosis included GERD, gastroparesis, bulimia nervosa, food allergy, somato-visceral and viscero-somatic reflex, and medication side effects. She was given a working diagnosis of postprandial nausea and vomiting with somatic dysfunction of the thoracic spine.

Given that the patient had been worked up in the past by gastroenterology, wide-ranging workup was not pursued. However, further investigation by her primary care provider for dyspepsia/postprandial nausea and vomiting at this presentation would have included further questioning about over the counter medications including: NSAIDs, Aspirin, and less common causes such as Garlic, Gingko, Saw Palmetto, Feverfew, Iron, and Chaste tree berry. Further labwork to be considered would include TSH/Free T4 if the patient would have exhibited symptoms of dyspepsia along with symptoms of thyroid disease and/or fasting blood glucose/HbA1C if the patient had not been screened or had exhibited symptoms of diabetes.

Additionally, one may consider imaging studies including barium esophagography with small bowel follow-through, ultrasound to investigate the liver, gallbladder, and pancreas, and direct visualization of her upper GI tract with esophagogastroduodenoscopy.

One special consideration for evaluation for dyspepsia is H. Pylori testing by stool antigen or 13C-urea testing. The utility of H. Pylori testing in patients with dyspepsia without known PUD is debatable and beyond the scope of this paper– though, the decision could be made to perform this test given clinical presentation.

Functional gastrointestinal disorders (FGIDs) are a group of abnormalities that afflict patients with symptoms that are assumed to arise from the gastrointestinal tract yet have failed to be explained by a standard work-up of endoscopy, laboratory studies, and radiological evaluation.[1] The Rome Foundation process has sought to classify FGIDs since 1990 and has evolved its classifications in three separate consensuses: ROME I, ROME II, and, most recently, ROME III. The Rome I consensus divided FGIDs into six divisions based on the anatomic region in which the symptoms are thought to originate.[2] Functional dyspepsia (FD) is the disorder thought to encompass the gastroduodenum. The ROME III consensus categorized FD into two major presentations: (a) Postprandial distress syndrome (PDS), which is characterized by early satiety, postprandial fullness, and nausea (and/or vomiting); and (b) epigastric pain syndrome (EPS), characterized by meal-independent epigastric pain and burning.[2] The difficulty with the overlap of GERD and EPS, with its frequent simultaneous presentation in patients with FD, has been recognized by ROME III; however, uniform guidelines for classification and characterization have not been established.[3] Nonetheless, this probably causes more of an academic problem for research stratification and less of an impedance for patient care. This patient appeared to have PDS given her four-year history of postprandial nausea and vomiting without a specific organic, systemic, or metabolic explanation despite prior workup by a gastroenterologist.

The incidence and prevalence of FD is difficult to accurately describe because of the nature and overlap of the symptoms. However, it has been reported that as many as 20% to 30% of people have chronic or recurrent dyspepsia and the annual incidence of first-time sufferers is about 1%; more than 50% of these patients lack an organic case despite diagnostic testing.[3]

The parasympathetic nerve supply to the upper gastrointestinal tract is from the anterior and posterior vagal trunks derived from the vagus nerve (CN X). Increased parasympathetic activity results in increased hydrochloric acid production in the stomach and a higher rate of peristalsis.[4] The sympathetic nerve supply originates from the T5-T9 segments of the spinal cord, forming the greater splanchnic nerve with synapsis in the celiac plexus. Increased sympathetic activity results in decreased peristalsis and vasoconstriction.[4]

The anatomy and physiology of the assumed origin of the symptoms in a patient with FD becomes of value when considering the somato-visceral connection. Since the early work of Louisa Burns, DO, the somato-visceral concept has been widely accepted and used in osteopathic medicine,[5-9] suggesting that somatic dysfunction in T5-T9 could result in corresponding changes in acid production, gut motility, and vascular tone (and consequently oxygen/nutrient delivery). A review of the literature does not reveal any specific studies or case reports on the use of OMT in the treatment of patients with FGIDs. There are, however, case reports on the successful adjuvant use of OMT in the treatment of peptic ulcer disease (PUD)[10] and GERD.[11] Both the report on PUD, by Morris and Dickey, and the report on GERD, by Branyon, suggest that the mechanism by which OMT provides relief is the effect the manipulation has on normalizing the autonomic tone, which is consistent with the historical understanding of the somato-visceral concept.[12] They also provide thorough discussions on the osteopathic literature supporting this mechanism.

In this case, the chief complaint was postprandial nausea and vomiting, which differs from the most common presentation of either GERD or PUD. Recent studies have indicated another possible mechanism of OMT on

Figure 1 Physician demonstrates the OMT technique on a simulated patient.
Seated muscle energy technique for treating thoracic non-neutral somatic dysfunction.[14]
Diagnosis: Flexed T5 rotated right, sidebent right.
Procedure: The patient is seated on the examination table. The physician stands beside the patient, with right thumb on the right transverse process of T5 and left hand on the patient's right shoulder. The physician localizes to the restricted barrier by extending, rotating, and sidebending the patient to the left down to the T5 level. The patient is instructed to return to a natural seated position (flex, rotate, and sidebend to the right) against the physician's isometric counterforce. The isometric contraction is held for 3 to 5 seconds. The patient is told to relax and the position is set up again and repeated two more times. The segment is then reexamined to monitor for improvement.

decreasing nausea and vomiting—the generation of cannabimetic effects.[13] The endogenous cannabinoid system consists of neuroreceptors and ligands and its effects include reducing anxiety, regulating satiety, increasing a sense of well-being, and decreasing nausea and vomiting.[13] Our patient did indeed have somatic dysfunction at the corresponding segments of the origin of the sympathetic nervous system innervation to the upper GI tract, supporting the viscero-somatic concept, but in light of recent research, multiple mechanisms are possible. Further study into the mechanism and use of OMT as adjuvant treatment in patients with disorders of the upper GI tract is needed.

Patient course

At the time of initial visit for this symptom presentation, the patient was treated with soft tissue stretching and kneading to the thoracic and lumbar region. Her thoracic findings were treated with muscle energy technique in the seated position (Figure 1) and high-velocity, low-amplitude (HVLA) technique in the supine position. The patient tolerated the treatment without difficulty.

At the completion of the visit the patient left with a prescription for metoclopramide 2.5 mg with meals with instructions to follow up in one month. She was instructed that if she did not show improvement with treatment of her somatic dysfunction, further work-up for her postprandial nausea would be pursued.

The patient did not follow-up until six months later and stated that she had 90% improvement in her postprandial nausea and vomiting after her last office visit and wanted to know if further manipulation would help the remaining 10% of her symptoms. She reported that she enjoyed eating again and was not afraid to go out to restaurants anymore. She also said she thought she had more energy. She had taken the metoclopramide for a few days but stopped because it did not seem to make any difference in her symptoms. The patient attributed her improvement solely to the OMT. She denied seeing any other providers, taking any other medications, or making any dietary changes that could have otherwise explained her improvement. At this follow-up office visit, her musculoskeletal examination findings revealed T8-10 flexed sidebent left and rotated right, T3 flexed sidebent right and rotated left, and congestion over the celiac ganglion. OMT performed included soft tissue (ST) to thoracic spine, HVLA to T9, and HVLA to T3. The patient was told to discontinue the pantoprazole in the coming weeks and follow up as needed. The patient has been seen periodically for her other chronic medical issues and has had no reported recurrence of her previous postprandial nausea and vomiting in the following year.

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