A 69-year-old woman with history of anorexia nervosa presented after a fall in the setting of weight loss, anorexia, fatigue, failure to thrive, severe malnutrition with a body mass index of 13.7 kg/m², significant anemia, and hypotension. In the few days before admission she fell several times due to generalized weakness. She reported not being able to consume food in the last couple of weeks other than liquids as she felt that solid foods were not passing through well — leading to postprandial fullness. She had lost 15 pounds in the last 3 months.

She had limited health care exposure and often declined preventative screening, thus had no known medical history. She took no chronic medications but reported occasionally using ibuprofen for joint pain later on in the hospitalization. She was an active smoker with a 51-pack-year history and consumed 2 alcoholic drinks daily. She had no history of acid reflux, no significant travel history, and no known exposure to Helicobacter pylori. Electrolyte evaluation was only significant for mild hypokalemia. Creatinine and lipase was found to be normal. She denied abdominal pain, nausea, or vomiting. Exam was significant for mild abdominal distension and succussion splash.

1. Based on this patient's initial presentation, which one of the following is a likely cause of her inability to tolerate significant oral intake and failure to thrive?
   a. Functional dyspepsia
   b. Pancreatitis
   c. Gastric outlet obstruction
   d. Gastroparesis
   e. Biliary colic

   Given her exam findings of abdominal distension and succussion splash, there is likely a structural etiology causing her symptoms. Functional dyspepsia would only be considered after a negative workup including normal esophagogastroduodenoscopy (EGD). She did not have any abdominal pain and had a normal lipase. According to the International Association of Pancreatology and the American Pancreatic Association, two of three criteria must be met for diagnosing acute pancreatitis: upper abdominal pain; lipase more than 3 times upper limit of normal; and imaging criteria with either computed tomography (CT), magnetic resonance imaging (MRI), or ultrasound.1 She already does not meet two of the above criteria and thus pancreatitis is unlikely to be contributing to her symptoms. Gastric outlet obstruction (GOO) is typically defined by epigastric abdominal pain and postprandial vomiting. Although she does not have pain or emesis, exam findings including succussion splash is highly suspicious for GOO and should be investigated further. Signs and symptoms of gastroparesis, which is a delay in gastric emptying, include nausea, early satiety, and vomiting of undigested food. There should be no evidence of GOO to consider this diagnosis. Finally, she may have gallstones but with lack of pain and nausea/vomiting, it is unlikely she has symptomatic gallstone disease. Given lack of medical history and severe cachexia, imaging was considered as there was concern for malignancy or GOO.

2. Which one of the following would be the next best appropriate diagnostic step in terms of radiologic imaging if suspecting gastric outlet obstruction?
   a. Obtain abdominal ultrasound
   b. Obtain CT of the abdomen
   c. Obtain abdominal MRI study
   d. Obtain abdominal x-ray
   e. Obtain inpatient positron-emission tomography scan

See end of article for correct answers to questions.
Abdominal ultrasound would minimize radiation exposure and is a reasonable starting point but would not give enough detail for diagnostic purposes. Given progressive weight loss and anorexia, further imaging is warranted and CT would provide high-resolution images of possible intra-abdominal pathologies leading to her symptoms. CT of the abdomen would be the next best appropriate diagnostic step in radiologic imaging for this patient. It can reveal gastric distention and sometimes reveal a cause for GOO. Abdominal MRI would minimize radiation and produce high-resolution images but is more expensive and takes longer to perform than CT. Abdominal x-ray would also be reasonable but again may not give enough diagnostic detail, especially if looking for malignancy. In patients with GOO, it can show an enlarged gastric bubble and a dilated proximal duodenum. If a calcified mass is seen in the right upper quadrant, this can suggest Bouveret syndrome, which is GOO secondary to gallstone impaction in the pylorus or proximal duodenum. A positron-emission tomography scan would be helpful as malignancy was suspected but is also less detailed and more expensive than CT and is less likely to be available as an inpatient option.

CT of her abdomen and pelvis was pursued but no diagnostic abnormalities were reported that could explain her anorexia. Because of her continued inability to tolerate oral intake, EGD was performed, which revealed antral deformity with contraction, scarring, and pinhole opening. The endoscope could not pass through to the duodenum, but mucosal biopsy samples were able to be obtained from the pyloric channel. A second endoscopy was performed under anesthesia support. Endoscopic ultrasound was used and core biopsy samples of the pyloric stricture were obtained. Mucosal biopsy samples of the pyloric channel stricture revealed reactive gastropathy and foveolar hyperplasia without inflammation. Core biopsy samples of the pyloric stricture were negative for malignancy and showed benign smooth muscle with mild chronic inflammation.

3. Given her history, which one of the following is likely to be a cause of this patient’s abnormal EGD and pathology findings?
   a. Peptic ulcer disease
   b. Ibuprofen use
   c. H. pylori infection
   d. Gastric hypersecretion
   e. Acetaminophen use

   EGD and pathology findings were consistent with pyloric stenosis. Secondary pyloric stenosis can be caused by inflammation in the gastric outlet, which could be caused by peptic ulcerations and nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen, which was used by the patient and likely the cause of her pyloric stenosis. H. pylori infection may also cause ulcerations in the stomach and first portion of the duodenum — increasing the risk for pyloric stenosis. Other conditions that increase acidity in the stomach, such as Zollinger-Ellison syndrome, may also lead to the development of pyloric stenosis. Acetaminophen use has not been associated with gastric ulcerations and would likely not be a cause of this patient’s pyloric stenosis.

   With the gastric obstruction caused by pyloric stenosis, different management options were discussed while taking into account her significantly malnourished state.

4. Which one of the following is the most appropriate next step in management of this patient?
   a. Consult surgery for urgent myotomy
   b. Place a nasogastric tube for supplemental nutrition
   c. Place a nasojejunal tube for supplemental nutrition
   d. Initiate parenteral nutrition
   e. Endoscopic dilation of pyloric sphincter

   Given her severe malnutrition and anorexia, surgery would likely be deferred. A nasogastric tube would not be beneficial as the tube end would be pre-pyloric and with severe GOO, tube feedings can be difficult. It would be reasonable to place a nasojejunal tube for supplemental nutrition as
treatment options for her pyloric stenosis are considered; this was performed endoscopically in our patient. Parenteral nutrition could be considered if the patient is not able to tolerate the tube feedings but ideally feeding through the gastrointestinal tract would be preferred. Dilation of the pyloric sphincter could be considered as a treatment option, but first it would be important to start supplemental nutrition for this patient in case dilation is not able to be performed promptly.

To place a nasojejunal tube in the patient, a guidewire was inserted into the jejunum and a 12-French nasojejunal tube was advanced over the wire. Placement was confirmed with fluoroscopy. A pediatric scope was used to traverse the pyloric channel stricture. After plans for nutrition optimization with nasojejunal tube feedings were placed, treatment options for her pyloric stenosis were considered.

5. Which one of the following is the most preferred treatment of pyloric stenosis in this patient at this point?
   a. Gastrectomy
   b. Pyloromyotomy
   c. Pyloroplasty
   d. Endoscopic dilation
   e. Total parenteral nutrition

   At this point, the patient’s severely malnourished state made her a poor surgical candidate for surgical options including gastrectomy, pyloromyotomy, and pyloroplasty. Pyloromyotomy uses an incision in the wall of the pylorus and the lining of the pylorus bulges through the incision making an opening from the stomach to the duodenum; this procedure is used to treat hypertrophic pyloric stenosis in infancy. Pyloroplasty is a surgical procedure used to widen the pylorus so that gastric contents can empty in the duodenum and would be the most favorable treatment option if the patient was not so malnourished. The thickened pylorus muscle is cut so that it widens and is closed by a technique that keeps the pylorus open. Pyloroplasty has a more favorable risk profile and is often preferred over pyloromyotomy. One study showed that endoscopic dilation, removal of risk factors, and gastric acid suppression led to long-term remission with average follow-up being 43 months. It can be associated with high recurrence rate of stenosis and eventually some patients may need surgical intervention. Dilation was preferred for our patient as surgical options were limited due to her nutritional state. Total parenteral nutrition could be pursued if nasogastric or nasojejunal feeding were not possible before other management options.

   Because of the nature of her pin-hole-sized pyloric channel and malnourishment, fluoroscopy-assisted balloon dilation was used. After the procedure, she was instructed to avoid all NSAID use. We were able to use nasojejunal feedings and eventually transitioned her to oral nutrition; therefore, total parenteral nutrition was not pursued. At her 3-month follow-up visit, she was tolerating a general diet and was slowly gaining weight.

DISCUSSION

Pyloric stenosis is a benign disorder that results from hypertrophy of the circular muscle fibers of the pylorus sphincter. Acquired pyloric stenosis is uncommon and can be associated with malignancy, peptic or duodenal ulcer disease, bezoars, and intra-abdominal adhesions. Historically, pyloric stenosis was most commonly attributed to excess gastric acid, but with the advent of proton pump inhibitors, malignancy should be strongly suspected in adult pyloric stenosis. Our patient had no history of peptic ulcer disease, gastroesophageal reflux, and biopsy specimens were negative for H. pylori. Endoscopic biopsy samples also did not reveal malignancy. She did have a history of ibuprofen use, which would be consistent with her biopsy findings of reactive gastropathy and foveolar hyperplasia; these findings can also be seen in duodenogastric bile reflux. With prolonged use of NSAIDs, her pyloric stenosis likely formed a GOO over time leading to limited oral intake and resultant severe malnutrition.

Abdominal imaging can sometimes detect distal gastric thickening, but it is important to evaluate with endoscopy as biopsy samples...
are needed to rule out malignancy as well as detect *H. pylori* infection. Our patient’s history was fairly benign and was lacking history of gastroesophageal reflux disease, travel history outside of United States, and peptic ulcer disease. We were highly concerned for underlying malignant process and biopsy samples were essential in our evaluation. She initially had underreported her use of ibuprofen for pain and was elicited later in her course. The pathogenesis of pyloric stenosis in the setting of NSAID use was likely due to the caustic effect on these agents, leading to fibrosis.\(^{10}\)

There are multiple treatment options for adult-onset pyloric stenosis including endoscopic dilation, pyloromyotomy, pyloroplasty, or gastrectomy. Pyloroplasty performed laparoscopically has a favorable risk profile but is a more invasive option than endoscopic dilation.\(^5\) If patients are high-risk surgical candidates or prefer to attempt nonsurgical management first, then endoscopic dilations would be a preferred route of therapy.\(^5\) Unfortunately, the long-term success of endoscopic dilation has been reported to be poor but can be used while optimizing the patient for surgical options.\(^{12}\) It is very important to advise patients to avoid all use of NSAIDs and use proton pump inhibitor therapy if peptic ulcer disease is identified as a cause.

**Potential Competing Interests:** The authors report no competing interests.

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**REFERENCES**


**CORRECT ANSWERS:** 1. c. 2. b. 3. b. 4. c. 5. d