Introduction

Gastric outlet obstruction (GOO) is a clinical syndrome where the patient presents with progressive epigastric pain, postprandial nausea, vomiting, early satiety and distention due to gastric or duodenal mechanical obstruction. Gastric adenocarcinoma can rarely manifest as massive gastric distention due to partially obstructing mass or peptic stricture. Severe and fatal sequelae may develop, if early detection and appropriate intervention are delayed, such as gastric decompression, endoscopic evaluation and/or surgical resection. Herein, we present a case of a 60-year-old male who presented with progressive worsening of nonspecific symptoms over the 8-month period. He was found to have remarkable massive gastric distention on imaging which was chronic in etiology secondary to GOO due to metastatic signet-ring cell gastric adenocarcinoma.

Keywords: Gastric outlet obstruction, massive gastric distension, signet-ring cell gastric adenocarcinoma

Case History

A 60-year-old African-American male patient presented to the ED complaining of progressively worsening diffuse abdominal pain, intermittent nausea and vomiting, early satiety, and 25 lb weight loss over the last 8 months. He described normal bowel movements and passing gas. He denied any fever, hematemesis, or melena.

On examination: pulse 84 bpm, temp 36.6°C, BP 144/68, and RR 18 bpm. His BMI was 22 kg/m². He was alert and oriented with mild distress due to abdominal pain. His abdomen was mildly distended with tympanic percussion note, active bowel sounds, and positive succussion splash.

His lab results revealed WBC 4.7 k/mcl, Hgb 12.9 mg/dL, and normal basic metabolic panel. Computed tomography scans of the abdominal revealed markedly distended stomach with debris extending to the pelvis with a mass effect on surrounding abdominal structures [Figure 1].

Address for correspondence: Dr. Ali Zakaria,
16001 W Nine Mile Rd, Department of Internal Medicine,
Southfield, MI 48075, USA.
E-mail: alizakaria86@hotmail.com

How to cite this article: Zakaria A, Khan F, Ahmad S, Turk I, Levinson J. Massive gastric distention due to signet-ring cell gastric adenocarcinoma. J Family Med Prim Care 2020;9:2558-61.
He was treated conservatively with intravenous fluid, PPI, and nasogastric tube placement with 3 L of fluid removed over 24 h. EGD revealed ulcerated, partially obstructive pyloric malignant appearing lesion with a large amount of retained bezoar (food) [Figure 2]. The patient underwent distal subtotal gastrectomy with Roux-en-Y reconstruction and liver biopsy. Histopathology confirmed metastatic transmurally invasive poorly differentiated mucinous signet-ring cell adenocarcinoma (pT4bN3aM1) [Figure 3]. Immunohistochemistry was negative for HER2. The liver biopsy revealed micronodular cirrhosis grade II, stage IV.

He was discharged in a stable condition with outpatient follow up with oncology to discuss possible palliative chemotherapy versus comfort measures given his underlying cirrhosis and poor overall prognosis.

**Discussion**

GOO is a clinical syndrome where the patient presents with progressive epigastric pain, postprandial nausea, vomiting, early satiety and distention due to gastric or duodenal mechanical obstruction. It rarely progresses to massive gastric distention, which can be complicated with gastric wall ischemia, perforation, and death. Massive gastric distention was first described by Duplay in 1833, mostly as a postoperative complication.\[9\]

The underlying etiology of GOO has changed substantially over the last 5 decades. Benign etiologies (with peptic ulcer disease being the most common) accounted for more than 90% of the cases up until the late 1970s. Given the increased use of PPIs along with the decreased incidence of Helicobacter Pylori related ulcers, malignant etiologies have recently accounted for almost 80% of the cases.\[3-4\] Multiple gastrointestinal malignancies can present as GOO, with pancreatic adenocarcinoma with duodenal involvement and gastric adenocarcinoma being more common.

Gastric adenocarcinoma is important to consider despite its rarity in the western countries likely due to the decreased prevalence of H. pylori.\[5-7\] Other less common malignancies include gastric lymphoma, neuroendocrine tumors, local extension of biliary malignancies, and duodenal adenocarcinomas. Benign etiologies include PUD, Crohn’s disease, pancreatitis, prolapsed gastric polyps, eating disorders,\[8\] gastric bezoar, gastric volvulus, Bouveret syndrome,\[9\] and superior mesenteric artery syndrome.\[10\]

The most common clinical features of GOO are epigastric pain, postprandial vomiting, distention, early satiety, and weight loss.\[11\] On rare occasions, patients may present with progressively worsening abdominal distention with massive gastric enlargement.\[12\] In cases of acute presentation, the patient can decompensate rapidly with gastric wall ischemia and necrosis, which may inevitably lead to perforation and peritonitis. This rarely occurs as the stomach is well protected due to significant collateral circulation. The main underlying pathophysiologic explanation is significant venous insufficiency with the point of tension needed to cause mucosal ischemia documented as 14 mmHg.\[1-3\]

The treating physicians should have a high index of suspicion of GOO when a patient presents with atypical clinical features. If symptoms are chronic in nature, early detection could be difficult as symptoms of weight loss, early satiety, and nausea might be nonspecific and can be explained by obstruction or gastric dysmotility.

Initial workup should include basic laboratory blood tests which can reflect dehydration and electrolyte disturbances due to prolonged vomiting.\[14\] It might also reveal low hemoglobin due to chronic blood loss. Imaging studies including plain abdominal X-ray, barium studies, and CT scan can reveal significant gastric enlargement, retained gastric content, air-fluid level, and possibly an underlying malignancy in the gastrointestinal tract.\[15-16\]
Zakaria, et al. : Massive gastric distension due to signet‑ring cell gastric adenocarcinoma

If the initial diagnostic work‑up reveals massive gastric distention, the patient should be treated with nothing by mouth, IVF hydration, electrolyte replacement, and NG‑tube decompression to decrease the risk of complication which includes: Gastric wall ischemia, necrosis, perforation, and aspiration. PPI should be used in all cases as it can decrease gastric inflammation and secretions; also as empiric treatment for possible underlying peptic ulcer disease. EGD should be performed to evaluate the underlying pathology. In cases of ulceration or the presence of a mass, biopsies could be performed to confirm underlying etiology. Poor sensitivity has been described with the use of routine biopsy techniques if the tumor is extraluminal or does not involve the mucosa.[3,17]

If gastric adenocarcinoma is confirmed as the cause, the patient should undergo proper staging which includes further imaging and complete oncology evaluation. The treatment should be individualized according to the stage of the disease, resectability or lack thereof, and overall performance status of the patient.[18] Treatment options include chemotherapy, palliative endoscopic stent placement and surgical resection or bypass. Endoscopic stent placement is associated with fewer complications, shorter hospital stay, lower costs, and quicker interval to oral intake, however, decreased food intake and stent complications may develop shortly after.[17‑19] Surgical resection with gastroenterostomy provides better technical success and longer survival.[19‑20] Despite successful curative resection, the 5‑year survival rate for gastric cancer is 31.5% thus long‑term survival remains poor.[20]

**Conclusion**

Gastric adenocarcinoma can rarely manifest as massive gastric distention due to partially obstructing mass or peptic stricture. Severe fatal sequelae can develop if early diagnosis and intervention with gastric decompression or resection are delayed. The treating physicians should have a high index of suspicion especially when the presentation is chronic in nature and accompanied by non‑specific signs and symptoms.

**Ethics statement**

The case report was performed in accordance with the ethical standards. The case was presented to the Ascension Providence Hospital Research Committee. The patient signed an informed consent form to share his images and other clinical information for publication. He understands that all efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

**Acknowledgment**

Authors would like to express their deepest appreciation to Dr. Juanita Evans for her valuable expertise in providing and reading the histopathology slides, and to Dr. Aruj Chawla for providing and interpreting the diagnostic images.

**Financial support and sponsorship**

Nil.

**Conflicts of interest**

There are no conflicts of interest.

**References**

1. Todd SR, Marshall GT, Tyroch AH. Acute gastric dilatation revisited. Am Surg 2000;66:709‑10.
2. Johnson CD. Gastric outlet obstruction malignant until proved otherwise. Am J Gastroenterol 1995;90:1740.
3. Shone DN, Nikoomanesh P, Smith‑Meek MM, Bender JS. Malignancy is the most common cause of gastric outlet obstruction in the era of H2 blockers. Am J Gastroenterol 1995;90:1769‑70.
4. Goldstein H, Boyle JD. The saline load test—a bedside evaluation of gastric retention. Gastroenterology 1965;49:375‑80.
5. Tendler DA. Malignant gastric outlet obstruction: Bridging another divide. Am J Gastroenterol 2002;97:4‑6.
6. Samad A, Khanzada TW, Shoukat I. Gastric outlet obstruction: Change in etiology. Pak J Surg 2007;23:29‑32.
7. Hall R, Royston C, Bardhan KD. The scars of time: The disappearance of peptic ulcer‑related pyloric stenosis through the 20th century. J R Coll Physicians Edinb 2014;44:201‑8.
8. Zvizdic Z, Jonuzi A, Djuran A, Vranic S. Gastric necrosis and perforation following massive gastric dilatation in an

**Figure 3:** Histopathology reveal poorly differentiated mucinous signet‑ring cell (black arrows) adenocarcinoma. Hematoxylin and eosin staining x40 (a), x100 (b), x200 (c and d)
adolescent girl: A rare cause of acute abdomen. Front Surg 2019;6:3.
9. Cox A, Marks DJB. Acute gastric dilatation causing respiratory distress. JRSM Short Rep 2011;2:41.
10. Feng YM, Wan D, Guo R. Case report of gastric distension due to superior mesenteric artery syndrome mimicking hollow viscus perforation: Considerations in critical care ultrasound. Medicine (Baltimore) 2018;97:e10757.
11. Green ST, Drury JK, McCallion J, Erwin L. Carcinoid tumour presenting as recurrent gastric outlet obstruction: A case of long-term survival. Scott Med J 1987;32:54-5.
12. Sejal J, Pranavi AR, Mohsina S, Sureshkumar S, Naik D, Kate V. Massive gastric dilatation in outlet obstruction - is it always benign? Int J Adv Med Health Res 2019;6:74-6.
13. Arie E, Uri G, Bickel A. Acute gastric dilatation, necrosis and perforation complicating restrictive-type anorexia nervosa. J Gastrointest Surg 2008;12:985-7.
14. Osmund WE, Copeland J. Gastric dilatation as a cause of respiratory distress. Can Fam Physician 2010;56:151-2.
15. Dicken BJ, Bigam DL, Cass C, Mackey JR, Joy AA, Hamilton SM. Gastric adenocarcinoma: Review and considerations for future directions. Ann Surg 2005;241:27-39.
16. Jeurnink SM, Steyerberg EW, Vleggaar FP, van Eijck CH, van Hooft JE, Schwartz MP, et al. Predictors of survival in patients with malignant gastric outlet obstruction: A patient-oriented decision approach for palliative treatment. Dig Liver Dis 2011;43:548-52.
17. Johnson CD, Ellis H. Gastric outlet obstruction now predicts malignancy. Br J Surg 1990;77:1023-4.
18. Rudolph HU, Post S, Schlüter M, Seitz U, Soehendra N, Kähler G. Malignant gastroduodenal obstruction: Retrospective comparison of endoscopic and surgical palliative therapy. Scand J Gastroenterol 2011;46:583-90.
19. Khashab M, Alawad AS, Shin EJ, Kim K, Bourdel N, Singh VK, et al. Enteral stenting versus gastrojejunostomy for palliation of malignant gastric outlet obstruction. Surg Endosc 2013;27:2068-75.
20. Kang HW, Kim SG. Upper gastrointestinal stent insertion in malignant and benign disorders. Clin Endosc 2015;48:187-93.
21. SEER Cancer Stat Facts: Stomach Cancer. Bethesda, MD: Available from: https://seer.cancer.gov/statfacts/html/stomach.html. [Last accessed on 2019 Oct].