

# Massive gastric distension due to signet-ring cell gastric adenocarcinoma

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### ABSTRACT

Chronic massive gastric distention is a rare condition that can occur due to an underlying obstruction or dysmotility. Gastric outlet obstruction (GOO) is often the culprit that can manifest as the result of the luminal, mural, or extrinsic compression. 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

### 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 fatal sequelae can develop if early detection and intervention with gastric decompression or resection are delayed. The treating physicians should have a high index of suspicion for the above, especially in chronic cases with nonspecific symptoms on presentation.

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### 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/m<sup>2</sup>. 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 succession 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].

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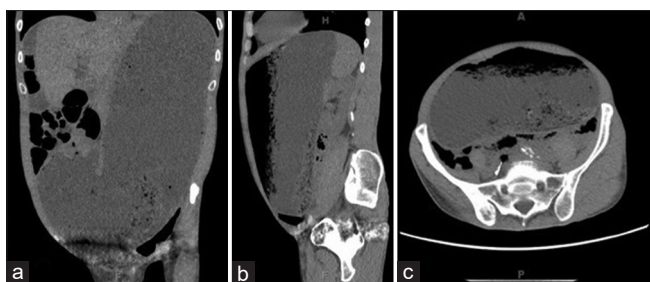
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**Figure 1:** (a) Computed tomography (CT) scan of the abdomen and pelvis (coronal) reveals markedly distended stomach with ingested debris extending to the pelvis with a mass effect on surrounding abdominal structures. (b) CT scan of the abdomen and pelvis (sagittal) reveals markedly distended stomach with ingested debris extending to the pelvis with a mass effect on surrounding abdominal structures. (c) CT scan of the abdomen and pelvis (axial) reveals markedly distended stomach with ingested debris extending to the pelvis with a mass effect on surrounding abdominal structures

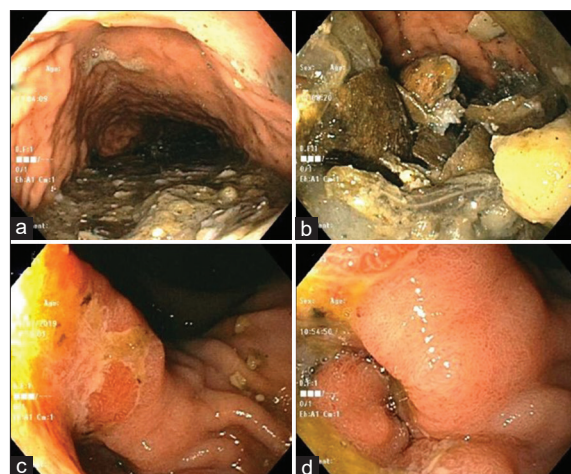
He was treated conservatively with intravenous fluid, PPI, and nasogastric tube placement with 3 L of fluid removed over 24 h. EGD revealed ulcerated, partially obstructive pyloric malignant appearing lesion with a large amount of retained bezoar (food) [Figure 2]. The patient underwent distal subtotal 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.<sup>[1]</sup>

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.<sup>[2-4]</sup> Multiple gastrointestinal malignancies can present as GOO, with pancreatic adenocarcinoma with duodenal involvement and gastric adenocarcinoma being more common.



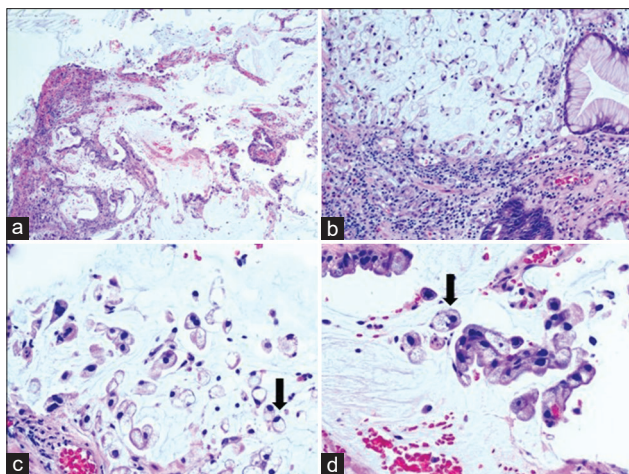
**Figure 2:** Esophagogastroduodenoscopy (EGD) reveals large amount of retained bezoar/food (a and b) and ulcerated; partially obstructive pyloric malignant-looking lesion (c and d)

Gastric adenocarcinoma is important to consider despite its rarity in the western countries likely due to the decreased prevalence of *H. pylori*.<sup>[5-7]</sup> 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,<sup>[8]</sup> gastric bezoar, gastric volvulus, Bouveret syndrome,<sup>[9]</sup> and superior mesenteric artery syndrome.<sup>[10]</sup>

The most common clinical features of GOO are epigastric pain, postprandial vomiting, distention, early satiety, and weight loss.<sup>[11]</sup> On rare occasions, patients may present with progressively worsening abdominal distention with massive gastric enlargement.<sup>[12]</sup> 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.<sup>[1,13]</sup>

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.<sup>[14]</sup> 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.<sup>[15,16]</sup>



**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)

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.<sup>[3,17]</sup>

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.<sup>[18]</sup> 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.<sup>[17-19]</sup> Surgical resection with gastroenterostomy provides better technical success and longer survival.<sup>[19,20]</sup> Despite successful curative resection, the 5-year survival rate for gastric cancer is 31.5% thus long-term survival remains poor.<sup>[21]</sup>

## 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.

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## Conflicts of interest

There are no conflicts of interest.

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