INTRODUCTION

We reported a case that typically mimics gastric cancer, though the root cause was a giant saccular aortic aneurysm, whose rupture led to the patient's death. Despite the rare occurrence of this manifestation of the aortic giant aneurysm, it should be considered in the differential diagnosis of gastric inlet obstructions.

Gastric inlet obstruction is a very rare manifestation of giant abdominal aortic aneurysm. Aortic aneurysms with transverse diameters greater than 10 cm are considered giant; moreover, pulsatile mass in the abdominal wall, as the most common symptom of aortic aneurysm, can cause extrinsic compression on the surrounding structures. Gastrointestinal compression rarely ensues following giant aneurysms and is seen as a duodenal obstruction with symptoms of vomiting, constipation, abdominal distention, and pain. Among upper gastrointestinal obstructions, gastric collapse and gastric inlet obstruction are unlikely manifestations and, to the best of our knowledge, has not been reported in the literature. This article reports a case with symptoms of 4 months of abdominal pain, constipation, and a weight loss of 25 kg. These symptoms in addition to an endoscopic examination reporting a fundal mass with suspicion of gastrointestinal stromal tumor (GIST) and hemoglobin of 7.8 were misleading to a gastric cancer diagnosis and made giant saccular aortic aneurysm as an implausible idea.

CASE REPORT

A 63-year-old male patient was admitted to the Gastrointestinal Department that the authors are affiliated to with a complaint of nausea, vomiting, constipation, and abdominal pain lasting for 4 months which was intensified since 2 weeks ago. Besides, 10 days before admission, the patient had been referred to an emergency department with nausea and vomiting that the symptoms were eliminated with symptom therapy.
and then was discharged. Thirty years ago, due to some wounds in distal of the lower extremities, he was examined and diagnosed with Buerger’s disease (thromboangitis obliterans); however, he did not quit smoking and continued to smoke heavily until 15 years ago. Moreover, no diabetic mellitus, hypertension, ischemic heart disease, and dyslipidemia were reported in his medical history; he had been abusing 5 methadone tablets per day and taking 2 clonazepam tablets per day since 20 years ago. He reported anorexia and a significant weight loss of 25 kg over the last 4 months. In this present admission, the patient refused to eat for fear of vomiting. Vomiting content was his consumed food; however, after stopping food receiving, it was just water and liquids. The patient reported gas passing though the last time of defecation was 5 days ago. He was conscious and dehydrated and had a cachectic appearance. Temporal muscle atrophy and a scaphoid abdomen were observed. Moreover, significant pulsation under the right last rib and epigastria was detected in both inspection and palpation, which is believed to be an aneurysm that needs an emergency investigation (Video S1). Vital signs included respiratory rate = 12 per minute, pulse rate = 102 per minute, blood pressure 90/70 mm Hg, O₂ saturation = 95%, and temperature = 36.8°C. In superficial palpation, the abdomen was soft and the patient had tenderness in the epigastric region and did not allow deep touching. Bowel sound was examined in 4 quadrants of the abdomen which was 3-5 sounds per minute.

Laboratory tests of the patient were as follows: WBC = 8.4 \((10^3/\mu L)\) (normal range 4-10), Hb = 7.8 (g/dL) (normal range 14-18), Hct = 25% (normal range 39-52), MCV = 63.9 (fl) (normal range 77-97), MCH = 19.9 (pg) (normal range 26-32), MCHC = 31.2 (g/dL) (normal range 32-36), Platelet = 296 (10³/µL) (normal range 140-440), RDW = 20.2% (high), PT = 15.2 seconds, PTT = 38 seconds, and INR = 1.43.

In the work-up documents conducted for the patient in another clinic before admission to our department, endoscopy reports showed that the upper 2/3 of the esophagus was normal; however, the lower 1/3 and lower esophageal sphincter were obstructed due to a fundal mass. The stomach was completely collapsed and was under the pressure of a huge submucosal tumor. The tumor was biopsied for pathological investigations and the report noted only mild chronic active gastritis, and there was no intestinal metaplasia and dysplasia or atrophy. Helicobacter pylori test was also negative. No tumor cells were reported.

Also, the report of endoscopic ultrasonography, purposed for ruling out GIST noted a large hypoechoic lesion at the fundus measured 72*63 mm originated from the muscularis mucosa. The lesion was heterogeneous with a necrotic area and regular border. Furthermore, no lymph node was detected around the lesion.

After examination and history taking, a surgery consultation was requested. In order to an exact investigation of the tumor according to documents and the examination, the surgeons requested a thoracoabdominal computed tomography (CT) scan and the patient was queued for surgery. However, surprisingly, proximal aneurysmal dilation of the abdominal aorta with an irregular border and maximal cross-sectional diameter of 62 millimeters, anteroposterior diameter of 54 millimeters, and length of 80 millimeters were observed in the computed tomography scan that was a broad hypodense collection with maximal thickness of 75 millimeters around the aneurysm, suggesting hematoma and clot. The aforementioned findings are either indicative of saccular giant aneurysm with a large mural thrombus in the eccentric sac, or a focal mycotic aneurysm with laceration and formation of tangential hematoma which had imposed compression and complete collapse of the stomach and lower esophageal sphincter obstruction. Moreover, it had a compressing effect on the superior mesenteric artery and celiac trunk (body), though the perfusion of these vessels was vividly observable (Figure 1).

After the investigation of the CT scan, the patient was transferred to the intensive care unit. Unfortunately, 8 hours after the admission—before any treating measures and

**FIGURE 1** A (Axial view) and B (Coronal view): computed tomography with IV and oral contrast: Axial and coronal view of saccular abdominal aortic aneurysm with eccentric sac containing hematomas that compress the stomach (black arrows)
transferring to the operation room—the patient's heart rhythm became asystole and the patient underwent an advanced cardiopulmonary resuscitation (CPR). After 2 hours of CPR, the patient expired. The cause of death was reported as the rupture of the aneurysm.

3 | DISCUSSION

Giant aneurysm of the aorta is referred to an aneurysm with a transverse diameter greater than 10 cm. The prevalence of giant aneurysms in the aorta is unknown exactly; on the other hand, they are not generally prevalent in the general population. The etiology of the giant aneurysm is not yet fully understood; however, several reports in the literature have suggested that atherosclerotic vascular diseases, Marfan syndrome, giant cell arthritis, tuberculosis, syphilis, and HIV-associated vasculitis are some important risk factors for giant aneurysms.

Aneurysms can be completely asymptomatic or can only occur as a pulsatile mass in the abdomen. However, giant aneurysms present a different challenge. The huge size of aneurysms can cause nonspecific symptoms which result from the extrinsic compression on the vessels and surrounding structures. Furthermore, the possibility of complications such as rupture and dissection of the aneurysm is directly related to the size of the aneurysm. As in aneurysms with transverse diameters of 6 cm or more, there is a 14% possibility of aneurysm rupture. This probability reaches 30-50% in aneurysms with diameters of 8 cm or more.

The upper gastrointestinal obstructions are among the symptoms caused by compressive effects of the giant aneurysm on the surrounding structures; they are in the form of duodenal obstruction with typical gastric outlet obstruction symptoms such as recurrent vomiting, abdominal distention, obstipation, abdominal pain, weight loss, and electrolyte disturbance. The prevalence of duodenal obstruction caused by aortic aneurysm is not high which was indicated in several publications.

Computed tomography scan has the specificity and sensitivity necessary for detecting aortic aneurysms and is sufficient for an accurate and complete diagnosis.

Treatment approach to these aneurysms is open surgery or endovascular aortic repair. However, not every aneurysm is an indication for the treatment, and symptomatic aneurysms or aneurysms larger than 5 or 5.5 cm are candidates for surgical removal. Small aneurysms can be followed; however, if rapidly progressing and developing to symptomatic, they need to be treated.

Note that these patients differ from duodenal obstruction patients in terms of the type of vomit. Patients with duodenal obstruction generally vomit digested food, whereas patients with symptoms of the gastric inlet obstruction usually have regurgitation and vomiting undigested food.

The correct approach after examining the patient and sonography would be conducting a CT scan. It seems that making a decision only based on the patient's paraclinical test without careful examination had led the previous physicians to perform endoscopy and biopsy, which are serious risks for an aneurysm. This case is a true instance of the fact that still in the 21st century, careful examination of the patient is the best way to reach a diagnosis and choose the right approach.

This case is very rare in terms of its manifestation, and this article is the first to note its large size compared to other reported cases.

4 | CONCLUSION

In this article, a case is reported that typically mimic gastric cancer, though the root cause was a giant saccular aortic aneurysm whose rupture resulted in the death of the patient. Therefore, despite the rare occurrence of this manifestation of the aortic giant aneurysm, it should be considered in the differential diagnosis of gastric inlet obstructions.

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CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

FM and FJ: initiated the preparation of this manuscript. All authors were involved in searching in the literature, the clinical management of the patient and contributed to revising the manuscript for important intellectual content. All authors read and approved the final manuscript.

ETHICAL APPROVAL

Institutional review board approval for case report is not required at our institution. To keeping ethical principles, name of the patient was not pointed in the paper and the right of the subject was protected.

INFORMED CONSENT

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

DATA AVAILABILITY STATEMENT

Data in the current study are available from the corresponding author on reasonable request.
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SUPPORTING INFORMATION
Additional supporting information may be found online in the Supporting Information section.

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