

Prolonged idiopathic gastric dilatation following revascularization for chronic mesenteric ischemia

Julia L. Gauci^a, Samantha Stoven^b, Lawrence Szarka^b, Konstantinos A. Papadakis^b

Malta Medical School, Mater Dei Hospital, Msida, Malta; Mayo Clinic, Rochester, Minnesota, USA

Abstract

A 71-year-old female presented with nausea, emesis, early satiety, and abdominal distension following revascularization for chronic mesenteric ischemia. Computed tomography angiogram showed gastric dilatation. Esophagogastroduodenoscopy, small bowel follow through, and paraneoplastic panel were negative. Gastric emptying was delayed. Despite conservative management, she required a percutaneous endoscopic jejunostomy. The development of a prolonged gastroparetic state has not been previously described.

Keywords Acute gastric dilatation, chronic mesenteric ischemia, revascularization, prolonged gastroparetic state

Ann Gastroenterol 2014; 27 (3): 273-275

Introduction

Acute gastric dilatation (AGD) is a rare condition the etiology of which is often multifactorial. AGD may develop after surgery and may be associated with neurologic or psychiatric diseases such as bulimia. The natural history of AGD is characterized by either complete resolution or progression to potentially fatal complications including gastric necrosis, perforation and sepsis if not promptly treated. AGD should be suspected in every postoperative patient who presents with abdominal distention and vomiting. We report a case of gastric dilatation following revascularization for chronic mesenteric ischemia leading to a prolonged gastroparetic state. The predisposing factors, clinical presentation, treatment and potential complications of AGD are discussed.

Case report

A 71-year-old woman with a past medical history significant for nicotine abuse, hyperlipidemia, and chronic mesenteric ischemia, who had undergone a superior

mesenteric artery (SMA) angioplasty and stenting, presented with a new onset postprandial abdominal pain suggestive of chronic mesenteric ischemia. Computed tomography (CT) angiography (CTA) demonstrated occlusion of the SMA stent, moderate stenosis of the inferior mesenteric artery, and a stable long segment occlusion of the celiac origin. She underwent a supraceliac aorta to hepatic artery bypass and SMA bypass for chronic mesenteric ischemia. On the sixth postoperative day she developed significant nausea and vomiting, requiring nasogastric (NG) decompression. CT scan showed gastric dilatation to the level of the mid-duodenum and a non-obstructed decompressed small bowel (Fig. 1); in comparison, a CT scan performed two months prior to surgery showed a normal-sized stomach. She was treated conservatively with NG decompression and bowel rest; at the time of the patient's dismissal approximately 2 weeks later, she was able to tolerate small amounts of oral food.

However, she was readmitted two weeks later with progressive nausea, non-bloody emesis, early satiety, anorexia, and abdominal distension.

On presentation, her vitals were normal, physical exam was notable for a prominent succussion splash and abdominal distension. Laboratory data showed a hemoglobin of 11.6 g/dL, white count of $6.8 \times 10^9/L$, creatinine of 0.7 mg/dL, sodium of 134 mmol/L, potassium of 4.6 mmol/L, glucose of 90 mg/dL, alkaline phosphatase of 91 IU/L, AST of 19 IU/L, total bilirubin of 0.3 mg/dL, direct bilirubin of 0.1 mg/dL, lactate of 1.1 mmol/L and a lipase of 15 U/L.

A repeat CTA scan revealed persistent gastric dilatation; the SMA and common hepatic artery grafts were patent (Fig. 2). Esophagogastroduodenoscopy (EGD) showed a normal esophagus, non-erosive gastropathy and a non-obstructing area of angulation in the third portion of the duodenum; a pediatric colonoscopy was easily passed through this area and advanced

^aMalta Medical School, Mater Dei Hospital, Msida, Malta (Julia L. Gauci); ^bDivision of Gastroenterology and Hepatology, Mayo Clinic, Rochester, Minnesota, USA (Samantha Stoven, Lawrence Szarka, Konstantinos A. Papadakis)

Conflict of Interest: None

Correspondence to: Dr Konstantinos A. Papadakis, Dept of Gastroenterology and Hepatology, Mayo Clinic, 200 First St. SW, Rochester, MN 55905, USA, Tel.: +507 293 2466, Fax: +507 255 6318, e-mail: Papadakis.Konstantinos@mayo.edu

Received 17 December 2013; accepted 28 January 2014



Figure 1 Coronal contrast-enhanced computed tomography scan of the abdomen and pelvis, performed in the first week following revascularization, demonstrating gastric dilatation to the level of the mid-duodenum and a non-obstructed decompressed small bowel



Figure 2 Coronal contrast-enhanced computed tomography scan of the abdomen and pelvis, performed three weeks after revascularization, demonstrating persistent gastric dilatation

to the proximal jejunum. A small bowel follow through using water-soluble contrast was normal and specifically did not show evidence for angulation or functional obstruction at the level of the third portion of the duodenum, as suggested by the EGD. Gastric emptying study was performed via ingestion of a radionuclide labelled meal (^{99m}Tc -sulfur colloid), percent

emptied from the stomach was assessed at 1, 2, and 4 h. The patient demonstrated normal gastric emptying at 1 and 2 h, but delayed emptying at 4 h (65% empty, normal was 81-100%). Work-up for paraneoplastic syndrome was negative; this test included the following antibodies: AChR Binding Ab, AChR Ganglionic Neuronal Ab, ANNA-1, ANNA-2, ANNA-3, AGNA-1, PCA-1, PCA-2, PCA-Tr, Amphiphysin Ab, CRMP-5 IgG, Striated Muscle Ab, N-Type Calcium Channel Ab, P/W-Type Calcium Channel Ab, and Neuronal (V-G) Potassium Channel Ab. She had no prior history of diabetes, psychiatric or neurologic disease.

She was treated conservatively with NG decompression, bowel rest, and total parenteral nutrition. She was treated with several prokinetic agents for 2 weeks including metoclopramide 5 mg IV t.i.d. and erythromycin 3 mg/kg IV t.i.d. She was given the following antiemetics orally; ondansetron 4 mg rapid dissolve tablets every 4 h, mirtazapine 15 mg nightly, and promethazine 25 mg every 4 h. Despite these measures, she was unable to consume sufficient calories orally over the span of 6 days and therefore a nasojejunal (NJ) feeding tube trial was commenced.

She tolerated the NJ feeds as an outpatient and ultimately was transitioned to a venting percutaneous endoscopic gastrostomy with percutaneous endoscopic jejunostomy for enteral nutrition. Continued efforts to re-introduce oral feeds consistently resulted in nausea and vomiting.

Discussion

AGD is a rare event, associated with a number of diverse etiologies. The clinical presentation is characterized by profuse vomiting, which occurs in more than 90% of cases [1], abdominal pain and distension. An abdominal X-ray demonstrates an enlarged gastric shadow; however, the most accurate diagnostic test is the CT scan, which may also reveal potential complications.

The etiology of AGD can be broadly classified into mechanically obstructive processes and dysmotility. Obstructive processes include tumors [2], gastric volvulus [3], superior mesenteric artery syndrome [4] and iatrogenic causes such as Nissen fundoplication [1]. Dysmotility leading to AGD may result from diabetes mellitus [5], paraneoplastic phenomenon, or bulimia nervosa [6].

Postoperative gastric dilatation is a common consequence of postoperative ileus, and it resolves spontaneously [7]. Severe postoperative AGD on the other hand usually occurs in the setting of multiple risk factors such as an obstructive process in a patient with a pre-existing dysmotility disorder. Our patient was not at an increased risk for postoperative AGD since imaging studies did not reveal evidence of gastric or bowel obstruction and she did not have any risk factors for vagal dysfunction or gastroparesis. The pathogenesis of AGD in our patient is uncertain, but may involve ischemic injury during her vascular surgery with resulting impairment in gastric motility. The stomach is a highly vascular organ supplied by many collateral vessels originating from the celiac artery's three main

branches; namely the left gastric, splenic and common hepatic arteries each of which branch extensively and anastomose at various sites producing a rich vascular network. While our patient had a compromised vascular supply, as denoted by her need for a supraceliac aorta to hepatic artery bypass, her symptom prior to the surgery was postprandial pain preventing oral intake. Oral intake was not associated with or limited by nausea and vomiting. In addition, cross sectional imaging prior to the surgery demonstrated a normal gastric size. In a healthy individual, the stomach is supplied by dense vascular collaterals; we suspect in our patient that a combination of altered vascular perfusion, microvascular disease related to smoking, and relative intraoperative hypotension, may have resulted in gastric hypoperfusion with resultant ischemic damage to the gastric wall itself or its innervation, producing the impairment in gastric activity.

The treatment of AGD is nasal decompression via a NG tube and symptoms resolve rapidly. If untreated, the patient may deteriorate rapidly, requiring surgery. The importance of timely diagnosis and treatment of AGD is critical due to its potentially fatal complications; namely gastric necrosis, gastric pneumatosis [8,9], abdominal compartment syndrome [2], perforation and sepsis.

Our case is atypical in that the patient had a prolonged state of gastric dilatation, which did not spontaneously resolve following conservative management. Despite non resolution, she did not progress to the outlined complications, but rather to a prolonged gastroparetic state which makes it a unique case of AGD.

In conclusion, AGD is a rare condition that is usually characterized by either complete resolution or progression to potentially fatal complications. Our patient represents a case where her condition failed to resolve quickly and required

prolonged enteral nutrition support via a percutaneous jejunostomy tube. Unlike other cases, despite a prolonged state, she did not suffer any fatal complications. The etiology of AGD is not well understood, and is likely multifactorial. In this patient, multiple contributions to gastric vascular insufficiency may have played a role in producing impaired gastric motility.

References

1. Barker JA, Burnett H, Carlson GL. Gastric necrosis complicating acute gastric dilatation after Nissen fundoplication. *BMJ Case Rep* 2011;2011.
2. Cox A, Marks DJB. Acute gastric dilatation causing respiratory distress. *JRSM Short Rep* 2011;2:41.
3. Kusunoki M, Hatada T, Ikeuchi H, Okamoto T, Sakanoue Y, Utsunomiya J. Gastric volvulus complicating myotonic dystrophy. *Hepatogastroenterology* 1992;39:586-588.
4. Lim JE, Duke GL, Eachempati SR. Superior mesenteric artery syndrome presenting with acute massive gastric dilatation, gastric wall pneumatosis, and portal venous gas. *Surgery* 2003;134:840-843.
5. Nagai T, Yokoo M, Tomizawa T, Mori M. Acute gastric dilatation accompanied by diabetes mellitus. *Intern Med* 2001;40:320-323.
6. Tweed-Kent AM, Fagenholz PJ, Alam HB. Acute gastric dilatation in a patient with anorexia nervosa binge/purge subtype. *J Emerg Trauma Shock* 2010;3:403-405.
7. Lee LS, Lim NL. Severe acute gastric dilatation causing respiratory failure. *Singapore Med J* 2006;47:716-718.
8. Kim SJ, Cho H, Lee SW, Choi SH, Hong YS. Gastric pneumatosis and acute massive gastric dilatation without specific cause. *J Emerg Med* 2013;44:e111-e113.
9. Gonzales-Rozas M, Sanchez-Lite I, Romero-de-Diego A. An unusual cause of abdominal distention and acute dyspnea. *Gastroenterology* 2012;142:7-8.