Calzonic Gastritis: A Case Report of Gastric Ischemia Secondary to Large Volume Food Ingestion

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Abstract

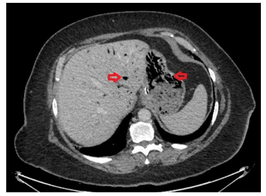
- Acute gastric ischemia is a rare and often fatal condition occurring predominantly in patients with substantial vascular comorbidities or with significant cardiovascular dysfunction. The stomach's robust vascularization and extensive collateralization is protective against acute ischemic events in otherwise healthy patients. We present a case of acute gastric ischemia secondary to massive food ingestion leading to pressure induced ischemia of the stomach. We discuss presentation, cross sectional imaging findings, endoscopic and histologic findings associated with this condition, and the successful non-operative management of the patient.

Presentation

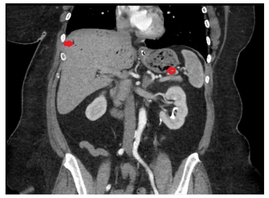
- We present a case of a 52-year-old-female with past medical history significant for hypertension and morbid obesity with body mass index (BMI) of 45 who presented to the emergency department with severe epigastric abdominal pain, nausea, and vomiting, after consuming a large calzone. She subsequently had multiple episodes of non-bloody vomiting and watery diarrhea. The symptoms persisted over the next 12 hours, leading her to seek evaluation in the ED.

Initial Workup

- Labs without significant abnormalities
- Computerized tomography (CT) scan showed evidence of emphysematous gastritis, and portal venous gas prompting surgical consultation



Axial CT image showing portal venous gas in liver (arrow on image left) and gastric pneumatosis (arrow on image right)



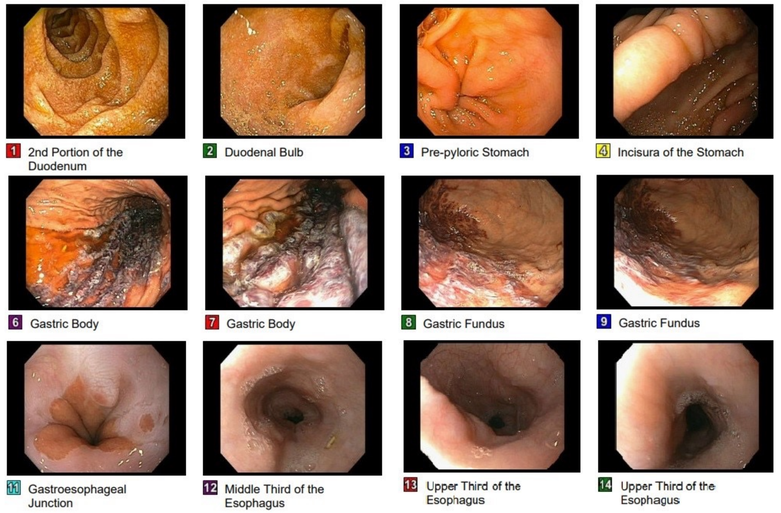
Coronal CT scan again demonstrating portal venous gas out to the periphery- of the liver (arrow on image left) and gastric pneumatosis (arrow on image right)



Mid sagittal CT scan showing patent celiac and SMA (lower arrow) and again demonstrated portal venous gas in liver (upper arrow

Further Workup

- CT angiography was performed demonstrating patent mesenteric vasculature and specifically patent gastric vascular pedicles.
- Endoscopic examination of the stomach revealed necrotic mucosa which was biopsied.
- Final histologic examination revealed mucosal erosion, prominent hemorrhage in lamina propria, and focal loss of glands consistent with ischemic gastritis.



Images from Esophagogastroduodenoscopy.: The top row shows a normal-appearing duodenum, prepyloric, and incisura of the stomach. The middle row demonstrates linear ulceration and necrosis of the gastric fundus and body. The bottom row shows a normal-appearing esophagus and gastroesophageal junction.

Treatment

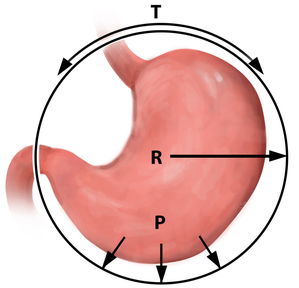
- The patient was treated in the hospital ward with nil per os (NPO), proton pump inhibitor (PPI) and prophylactic antibiotics. The patient tolerated slow advancement of diet and is currently on a regular diet with repeat imaging showing resolution of her gastritis.

Discussion

- The stomach has a rich vascular supply, making gastric ischemia a rarely reported phenomenon.
- Most cases of gastric ischemia involve predisposing factors such as atherosclerosis, vasculitis, gastric volvulus, psychiatric dispositions, DIC, small bowel obstruction, recent surgery, or sepsis.
- Reported cases of gastric ischemia often have high morbidity/mortality and are associated with shock, sepsis, volvulus, embolism, vasculitis, DIC, or corrosive ingestion. These cases often require extensive medical support, surgery, and/or prolonged hospital stay.
- **Rarely, as with this patient, acute gastric ischemia develops in otherwise healthy individuals, who have undergone massive food intake leading to pressure-induced ischemia and necrosis**

LaPlace's Law
- Wall Tension = Pressure x Radius
- Wall Stress = Wall Tension / (2 x Wall Thickness)

- In the setting of acute and massive distension of the stomach, the increased wall stress in compliant areas likely decreased perfusion to mucosal tissues, ultimately resulting in localized ischemia



Summary

- This case illustrates first that significant gastric dilatation in the setting of food consumption, even with intact gastric blood supply, can lead to gastric ischemia. Second, that this gastric ischemia can be managed expectantly with bowel rest, proton pump inhibitors, and prophylactic antibiosis. This management has led to no long-term sequelae from her ischemic episode.

References

1. Sharma A, Mukewar S, Chari ST, Wong Kee Song LM. Clinical features and outcomes of gastric ischemia. Dig Dis Sci 2017;62: 3550-3556. DOI: 10.1007/s10620-017-4807-4.
2. Tang SJ, Daram SR, Wu R, Bhaijee F. Pathogenesis, diagnosis, and management of gastric ischemia. Clin Gastroenterol Hepatol 2014; 12:246-52.e1. DOI: 10.1016/j.cgh.2013.07.025.
3. Slam, K.D., Calkins, S., Cason, F. LaPlace's law revisited: Cecal perforation as an unusual presentation of pancreatic carcinoma. *World J Surg Onc. 2007, 5: 14,* doi: 10.1186/1477-7819-5-14.