A Rare Case of Phlegmonous Gastritis in a Previously Healthy Male.

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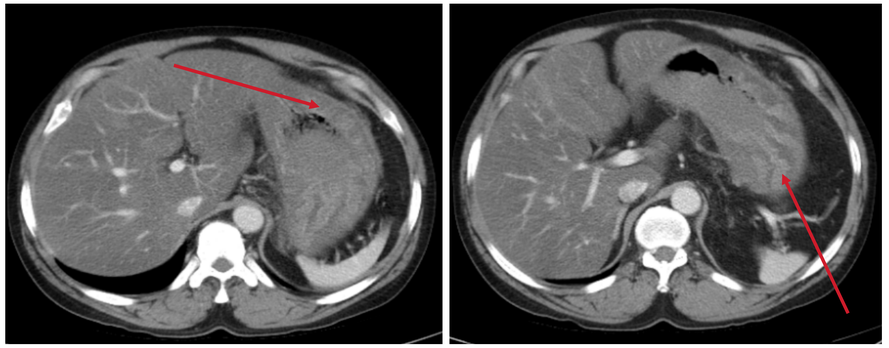
INTRODUCTION

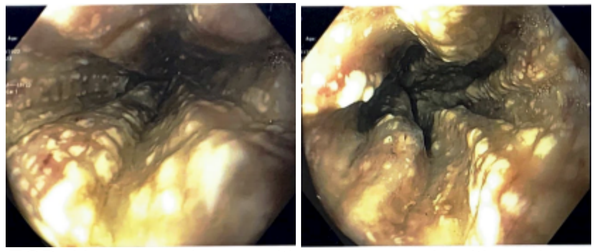
- Phlegmonous gastritis (PG) is a rare and often fatal disease characterized by severe bacterial invasion of the gastric wall.[1]
- Due to its rarity and subtle clinical manifestation, early diagnosis is difficult, causing delays in identification and treatment leading to increased mortality.[2]
- We present a case of a previously healthy male who developed PG and responded well to antibiotics.

CASE PRESENTATION

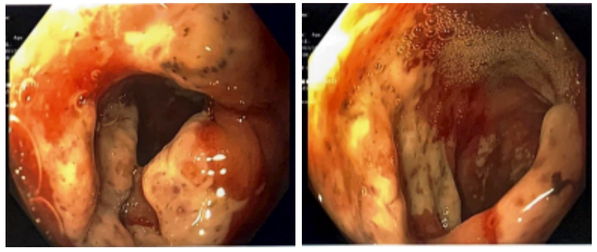
- A 55-year-old male with history of hypertension and hyperlipidemia presented to the ED with odynophagia, intractable nausea and coffee ground emesis for one day.
- Denied alcohol, tobacco or drug use.
- Negative family history.
- **Physical exam** positive for diffuse abdominal tenderness to palpation.
- Hemodynamically stable on arrival. Afebrile.
- **Initial labs:** WBC 19,500/mcL, Hgb 17g/dL, ESR 46. Blood cultures negative.
- **Images:** CT abdomen/pelvis showed thickening of the gastric wall with prominent gastric folds.
- Placed on Vancomycin, piperacillin/tazobactam and intravenous pantoprazole.
- **EGD**: severe esophagitis, gastritis and duodenitis with areas of micro-abscesses.
- **Biopsies:** patchy areas of necrosis with areas of fibrin thrombi and eosinophils, which prompted a vasculitis workup.



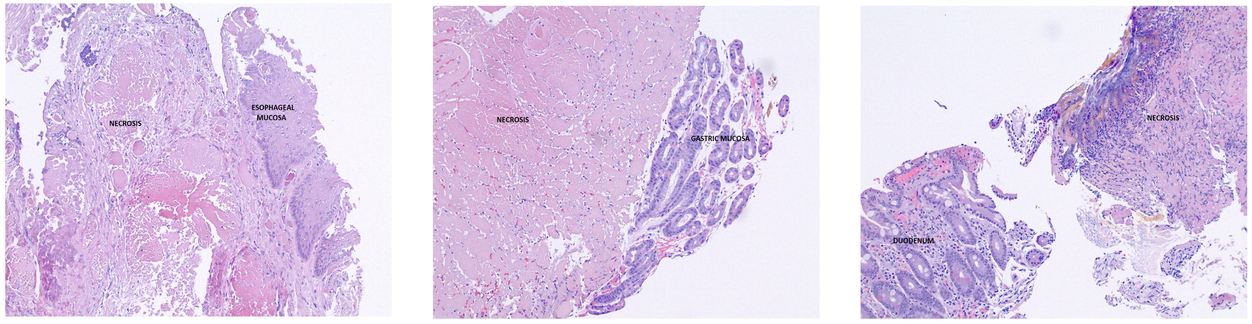
CT abdomen/pelvis: Thickening of the gastric wall with prominent gastric folds.



EGD: Upper esophagus with severe esophagitis and micro-abscesses.



EGD: Severe ulcerative duodenitis.



Esophageal, gastric and duodenal biopsy, respectively, showing abrupt transition from normal tissue to necrosis.

CASE CONT.

- Given short course of prednisone however had no improvement.
- **Additional labs**: HIV, syphilis, hepatitis panel negative. Anti-CCP, ANA, c-ANCA, p-ANCA, Scl-70 Ab, anti-centromere Ab, anti-RNA polymerase III AB and complement C3 and C4 were negative.
- Was not able to tolerate oral intake requiring total parenteral nutrition for 36 days.
- Eventually was able to tolerate oral intake and was discharged home tolerating a soft diet.

DISCUSSION

- PG is caused by bacterial invasion of the gastric submucosa with possible further invasion into the mucosa and serous membranes due to a compromised mucosal wall as in gastritis, or via lymphatic spread from intra-abdominal infection or bacteremia.
- Pre-existing chronic alcohol use, immunocompromised states, and malignancy have been implicated as associated factors.[1]
- Antibiotics have led to a drastic decline in mortality from as high as 92% before antibiotics to roughly 40% at present day. If antibiotics fail, surgery may be considered.[1]
- There have only been a few reports of PG in previously healthy individuals without comorbidities as in this case.

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