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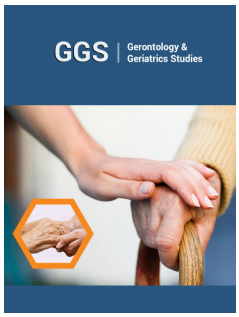
Short Communication

Esophagitis Secondary to Retrograde Bile Reflux: A Repercussion to Intestinal Surgery in Older Population

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Abstract

Our patient who had a past history of mesenteric abscess following which a small section of small bowel including terminal ileum and cecum. Since that time the patient has had intermittent problems with abdominal pain nausea and occasional vomiting. He presented to the ED with abdominal pain, nausea and vomiting. Work up including EGD had demonstrated the presence of erosive esophagitis secondary to bile reflux. Our clinical case represents the risk of bile reflux in patients with prior intestinal surgery due to alternation of gastric motility and emptying.

Oesophageal bile reflux alters the environment of oesophageal mucosa to alkaline PH. conjugated bile acids are toxic to the esophageal mucosa at acidic PH whereas unconjugated bile acids and trypsin heightened the oesophageal mutilation at neutral PH. Frequent pathologic findings found in patients with bile reflux include gastric mucosa erosions, foveolar hyperplasia, venous congestion, chronic inflammation, esophageal reflux and esophageal ulceration. Bile reflux induced esophagitis can provoke erosions, ulceration and in some cases intestinal metaplasia. Life style modifications, proton pump inhibitors, prokinetic agents and in refractory cases. surgery might be helpful. Longer duration of bile acid reflux has been associated with increased risk of Barrett esophagus and cancer Prompt recognition and treatment of this clinical phenomenon is essential to prevent future untoward consequences.

Keywords: Bile acid; GERD; Alkaline PH; Intestinal surgery; Esophageal erosions and Intestinal metaplasia

Clinical Case

64-year-old male with a past medical history of a necrotic bowel due to obstruction, and comes in to the ED for evaluation of intractable vomiting and abdominal pain. Patient reports that he had had 2 bites of food at lunch and immediately vomited the contents. Patient did not think anything of it and did not have any other episodes until late at night for dinner the same thing happened. Patient was able to tolerate liquids just fine. Next day on Thursday, patient had a few bites of yogurt and same thing happen, patient was unable to keep it down. This time the episode was associated with the pain. Again, he was able to hold down liquids but not solids. Friday the patient flew back to Georgia from Cleveland and had multiple vomitus episodes on the plane. Was unable to even keep down water at this time. Shortly after arriving in Georgia, he brought himself to Emergency department for further workup. His past history is pertinent for a mesenteric abscess in January of 2025 he had a 24cm section of small bowel removed and the terminal ileum and cecum. No obvious pathology to suggest Crohn's disease or records for the abscess. Since that time the patient has had intermittent problems with abdominal pain nausea and occasional vomiting but no colitis severe as this current episode.

He has recently been evaluated by Gastroenterologist with gastric emptying study small-bowel follow-through and CT scans no nose these notes are available at this time. Vitals on ED arrival include elevated blood pressure but otherwise unremarkable. The abdomen and pelvis CT showed distal esophageal wall thickening which can be related to esophagitis. CBC shows elevated blood count at 17.0. Routine chemistry shows lobar car at 15. Elevated ALT.

Lactic acid elevated at 3.5. During physical examination, patient reported to be filling much better since taking the antisemitic and pain medications. Patient had a vomit bag that had about 4 ounces of fluids that had come up about 30 minutes prior. Patient endorsed epigastric tenderness. Patient had normal breath sounds with good airway movement, normal cardiac auscultation. Abdominal sound positive for pain on palpation in epigastric region. Patient denied fevers, chills, cough, diarrhea, dysphagia, odynophagia.

Gastroenterology was consulted and they and they proceeded with performing esophageal gastric endoscopy which showed severe exudative esophagitis in the distal 2/3 of the esophagus with reflux of bile-stained fluid into the esophagus. Biopsies taken from the esophagus (Figure 1). Subsequently the patient is placed on Full liquid diet, Increase pantoprazole to 40mg b.i.d. and Carafate suspension 1g a.c. and HS. He reports a tendency to lose stools which is probably caused by combination of his small-bowel resection and cholecystectomy. He was on placed on Welchol 2 tablets b.i.d. oral next few days the patient gradually improved and his diet is advanced to liquid to solid diet. His biopsies are still pending and patient is fit to be discharged and can follow with outpatient gastroenterologist. The patient is currently stable and will be discharged today the discharge recommendations were clearly explained to the patient. Patient is advised to take pantoprazole and sucralfate daily and followed up with outpatient gastroenterologist for biopsy results. He was advised to return to the ED for severe or worsening abdominal pain, nausea, vomiting, blood in stool, persistent vomiting, or difficulty swallowing.

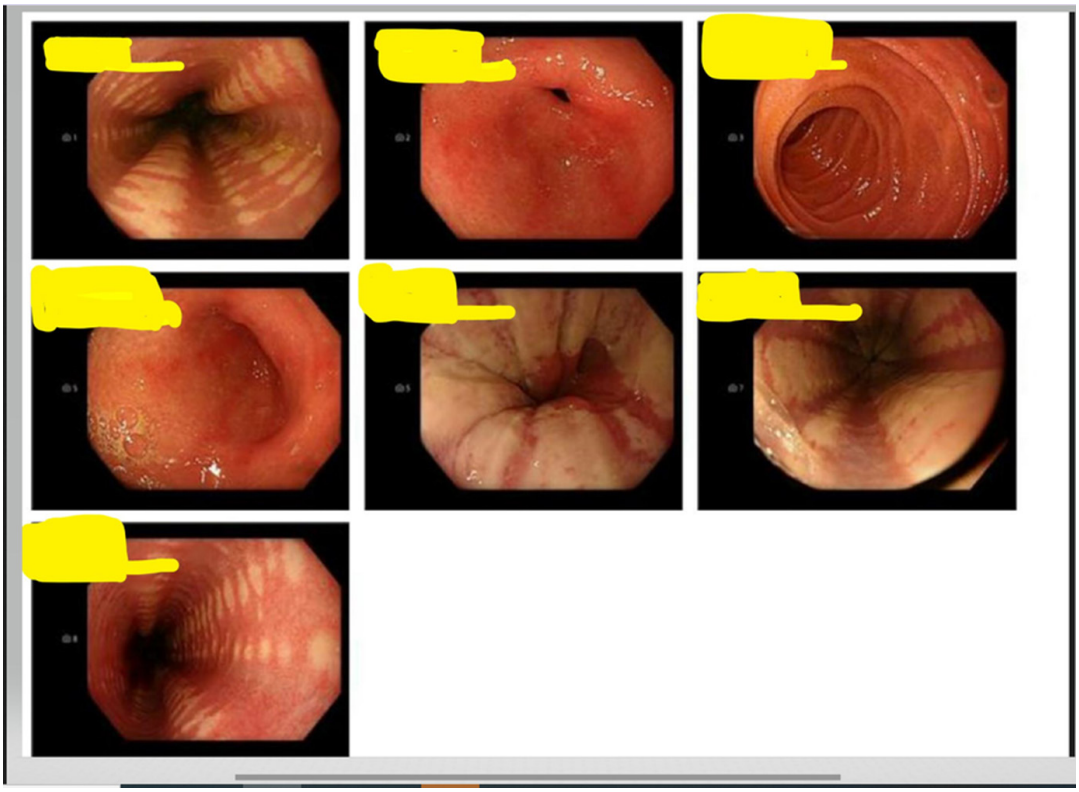


Figure 1

Discussion

Our clinical case represents a rare clinical case of bile reflux esophagitis in a patient who underwent prior intestinal surgery. Intestinal surgery might alter the dynamics of gastric motility and emptying, thus predisposing them to retrograde bile reflux induced oesophagitis. It is reported that most patients with gastric-esophageal reflux of bile acids have materializes in the setting of incompetence of the esophageal junction, disordered oesophageal clearance and disturbed gastroduodenal motility [1]. Previous studies have shown that bile acids and trypsin can degrade and injure the gastric mucosa [2]. Animal studies have demonstrated that trypsin perfusion can precipitate esophageal ulcerations and erosions in the absence of hydrogen ions [2]. It is documented that conjugated bile acids are toxic to the esophageal mucosa at acidic PH whereas unconjugated bile acids and trypsin heightened the esophageal mutilation at neutral PH [3].

Gastroduodeno-oesophageal reflux alters the environment of esophageal mucosa from neutral PH to alkaline PH. Accordingly, the greater time esophageal mucosa dwells through alkaline PH, there is higher chances of esophageal damage, erosions and ulcerations. This opens the door for measuring the bile acid concentration in the esophageal mucosa with 24h ambulatory bile-reflux monitoring for assessing the degree of esophageal mucosal damage [4]. Most of the reports of barret esophagus reported thus far implicated a

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combination of reflux including acid and bile, without specifying the individual role of bile in instigating the oesphageal pathology [5,6]. However, they report that most of the bile acid reflux to the esophagus transpires within the PH range of 4-7 [5,6].

Regardless of the underlying pathological abnormality, synergism of acid and bile acid reflux to the esophageal junction brings to light Barrett esophagus [7-9]. In line with these findings, combined PH and bile monitoring was increases the sensitivity (56-79%) and accuracy (69-83%) in detecting bile reflux esophagitis [10]. In children with gastroesophageal reflux disease, combined acid and bile acid reflux is found in 11% and 70% of the children with mild and severe esophagitis respectively [10]. Clinical studies have demonstrated that low level expression of P53 and Ki-67 genes partly from chronic inflammation, indicating a low risk of cancerous transformation in bile acid reflux patients [11]. Some of the risk factors that are proposed to increase the risk of bile acid reflux include gastrectomy, cholecystectomy, pyloric hypotension, cholelithiasis, pancreatitis, duodenal inflammation, sphincter of Oddi dysfunction, therapeutic biliary procedures, gall bladder dysfunction and choledochoduodenostomy [12]. Studies showed that patients with bile acid reflux esophagitis are more likely to be older, and bound to have gastric mucosal edema along with decreased incidence of *helicobacter pylori* positivity [12].

Frequent pathologic findings found in patients with bile reflux include gastric mucosa erosions, foveolar hyperplasia, venous congestion, chronic inflammation, esophageal reflux and esophageal ulceration [12,13]. However, in subsets of patients, intestinal metaplasia has been noticed in patients with higher amounts of biliary acid exposure [14]. Electron microscopy changes in bile reflux esophagitis include disruption of cell membranes, dilated intercellular space, blunted villi and reduced mucosal lining [15-17]. Bile acid reflux can be diagnosed by bilitec monitoring system where the color of the oesophageal refluxate can be assessed by using photo-colorimetric device [18]. Bile acid reflux can be reduced by lifestyle modifications include weight loss, avoidance of late-night meals, and smoking cessation. Proton pump inhibitors (PPI) can be efficacious in lessening the reflux of acid as well as bile into the esophagus [18]. Prokinetic agents (Metoclopramide) which increase the gastric motility and gastric emptying as well as agents which reduce the relaxation of esophageal dysfunction can be helpful in those resistant to PPI [18]. In medically refractors, surgical consultation for antireflux surgery can be resorted. It is important is distinguish between acid and bile acid reflux because the presence of higher and longer duration of bile acid reflux has been associated with increased risk of Barrett esophagus and cancer [18].

Conclusion

Older patients are more like to develop reflux esophagitis particularly those with incompetent esophageal sphincter, delayed esophageal clearance and impaired gastro-duodenal motility. As esophageal mucosa is exposed to bile for the PH changes from neutral to alkaline environment, thus increasing the risk of deleterious consequences. Both conjugated and unconjugated bile acids are toxic to the esophageal mucosa, thus resulting in erosions, edema, ulcerations and rarely intestinal metaplasia and cancer. Differentiating between bile and acid in the refluxate is important as former is more deleterious if present for longer duration. Life style choices, drugs and surgery in resistant cases might afford symptomatic relief and preventing long-term esophageal complications in the future.

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