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Duodenal perforation following esophagogastroduodenoscopy (EGD) with cautery and epinephrine injection for peptic ulcer disease: An interesting case of nonoperative management in the medical intensive care unit (MICU)

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ABSTRACT

INTRODUCTION: The utilization of esophagogastroduodenoscopy (EGD) and related procedures continues to rise. Due to this increase in utilization is an inevitable rise in serious complications such as hemorrhage and perforation. One understudied and dreaded complication of EGD causing significant morbidity and mortality is duodenal perforation.

PRESENTATION OF CASE: We present the case of a 63-year-old male who presented to our institution's emergency room with dyspepsia, melanic stools, tachycardia, and hypotension. Initial laboratory evaluation was significant for severe anemia, lactic acidosis, and acute kidney injury, while CT scan of the abdomen pelvis did not suggest retroperitoneal hematoma or bowel perforation. An emergent EGD was performed which showed multiple bleeding duodenal ulcers that were cauterized and injected with epinephrine. Post-procedure the patient developed worsening abdominal pain, distension, diaphoresis, and tachypnea, requiring emergent intubation. A CT scan of the abdomen and pelvis with oral contrast confirmed pneumoperitoneum and duodenal perforation.

DISCUSSION: Due to the patient's hemodynamic instability and multiple comorbidities, he was treated non-operatively with strict bowel rest and intravenous antibiotics. The patient ultimately had a 19-day hospital course complicated by renal failure requiring hemodialysis and an ischemic limb necessitating above knee amputation.

CONCLUSION: This case describes an unsuccessful attempt at nonoperative management of duodenal perforation following EGD.

1. Introduction

As more innovations and indications for esophagogastroduodenoscopy (EGD) emerge, the utilization of EGD and related procedures will continue to rise. One inevitable consequence of this increased usage is an accompanied increase in iatrogenic complications. Major complications from EGD, although declining in their incidence due to the transition from rigid to flexible endoscopes, include perforation, hemorrhage, Mallory–Weiss tear, and infection/aspiration [1]. Based on a sample of 200,000 EGD examinations, which has since been repeated and validated, it was estimated that the overall complication rate of these procedures is 0.13%, with an associated mortality of 0.004% [1–3].

Although relatively rare, complications of EGD and their associated morbidity and mortality are not trivial. For example, Merchea et al. analyzed 217,507 EGD procedures and discovered perforations in 72 of them, with an associated morbidity and mortality of 40% and 17%, respectively [4]. Kavic et al. showed hemodynamically significant hemorrhage to occur in 0.15% of EGD procedures, with 13% of these hemorrhages requiring surgical intervention [1].

One dreaded complication of EGD that has attributed to substantial morbidity and mortality is perforation. Perforations following EGD most commonly affect the esophagus, but it is the perforations affecting the duodenum that lead to the highest rates of morbidity and mortality [4]. For example, EGD perforations typically occur in the duodenum in 32% of cases, but are responsible for 53.8% of perforation deaths, while perforations in the esophagus account for 51% of cases, but only 30.8% of perforation deaths [4]. Moreover, EGD perforations in the duodenum are more likely to cause acute

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Table 1
Laboratory and objective data during admission.

	12 h after admission	8 h post-EGD	6 days after admission
Hemoglobin	5.8 gL/dL		
Hematocrit	16.1 L%		
Heart rate		142 bpm	
Blood pressure		143/83 mm Hg	
Respiratory rate		48 breaths/min	
Oxygen saturation		92% on 100% NRB	
ABG		7.05/60./72.6/16.5	
Blood urea nitrogen			84 mg/dl
Creatinine			5.7 mg/dl
Bladder pressure			15 mm Hg

renal failure, enterocutaneous fistulas, and other morbid sequalae than perforations in other locations [4].

Due to its relative infrequency, significant ambiguity exists surrounding the optimal management of duodenal perforations following EGD. Most investigators experienced with this complication agree that surgical and nonsurgical treatments have their own specific roles in individualized circumstances depending on patient symptomatology, stability, and comorbidities [2,4,5]. Consequent to the scarce investigations on the topic, most treatment recommendations regarding EGD duodenal perforation are extrapolated from experiences with perforations following colonoscopy, endoscopic retrograde cholangiopancreatography (ERCP), diverticulitis, peptic ulcer disease, and duodenal diverticulum [1,6–16].

We describe a case of nonoperative treatment for duodenal perforation following EGD that ended in mortality. To our knowledge, this is the first report of nonsurgical management of this complication in the medical intensive care setting.

2. Presentation of case

A 63-year-old white male with a past medical history of peripheral vascular disease (PVD) with claudication, hypertension, diabetes mellitus type-2, and chronic low back pain presented to the emergency room with dyspepsia and three days of melanic stools. The patient had been taking 800 mg of ibuprofen three times daily for two years for back pain. His physical exam was significant for sinus tachycardia (115 bpm), hypotension (90/60 mm Hg), left lower extremity weakness, pallor, and pulselessness, and diffuse skin pallor. Laboratory and microbiological evaluation was most notable for severe anemia (hemoglobin/hematocrit 8.1 gL/dL/22.5 L%; baseline hemoglobin/hematocrit 13.0 g/dL/40.0 L%), leukocytosis (white blood cell = 21,000 Hk/cmm; 38% bands), lactic acidosis (lactic acid = 6.2 mmol/L; bicarbonate = 17 mmol/L; anion gap = 19 mmol/L; arterial blood gas 7.36/23/72/13 on 3 L nasal cannula oxygen), acute renal failure (urea nitrogen/creatinine = 53 mg/dl/1.6 mg/dl), hyponatremia (sodium = 124 mmol/L), hypokalemia (potassium = 3.1 mmol/L), positive occult blood (fecal immunochemical test), positive blood cultures (4/4 bottles methicillin sensitive *Staphylococcus aureus*), and no coagulopathy or thrombocytopenia. His chest radiograph was unremarkable and electrocardiogram (ECG) showed sinus tachycardia without any ischemic changes. Computed tomography (CT) of the abdomen and pelvis with intravenous contrast was obtained which did not show signs of retroperitoneal hematoma or bowel perforation.

The patient was admitted to the Medical Intensive Care Unit (MICU) and standard management and resuscitation for upper gastrointestinal (GI) bleeding and acute renal failure was initiated with pantoprazole by continuous infusion, packed red blood cells, crystalloid, and gastroenterology consultation. He was presumed

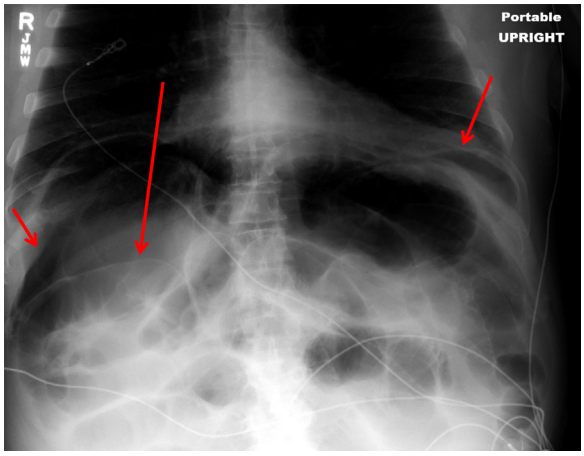

Fig. 1. Abdominal X-ray taken eight hours after EGD with cautery and epinephrine shows free air under the diaphragm. The arrows show rigler's sign and triangle sign. Of note, the triangle sign is the most specific albeit the least sensitive sign of free air.

to have bacteremia in the setting of leukocytosis and was empirically treated with vancomycin and piperacillin/tazobactam. For his ischemic left lower extremity, vascular surgery was consulted and recommended a CT angiogram of the affected limb which showed good distal runoff and opacification to the level of the ankle. Due to the patient's downward trending hemoglobin/hematocrit and worsening lactic acidosis "Table 1" despite adequate blood and crystalloid resuscitation, an emergent EGD was performed at bedside which showed multiple bleeding duodenal ulcers, the largest being 30 mm × 30 mm, covering 75% of the circumference of the duodenal bulb. The ulcers were cauterized and injected with epinephrine for hemostasis. The patient tolerated the procedure well and reported no new complaints immediately post-procedure.

Approximately eight hours post-procedure the patient developed worsening abdominal pain, distension, diaphoresis, and tachypnea. Physical examination revealed a temperature of 95.0 F, blood pressure 143/83, heart rate 142, respiratory rate 48, 92% oxygen saturation on non-rebreather mask (100% oxygen), and diffuse abdominal guarding, rebound, tenderness, and distension. An arterial blood gas showed significant mixed respiratory and metabolic acidosis (7.05/60.4/72.6/16.5). A STAT abdominal and chest radiograph was remarkable for pneumo-peritoneum consistent with bowel perforation "Figs. 1–5". The patient was emergently intubated and general surgery was consulted. A CT scan of the abdomen and pelvis with oral contrast was obtained which confirmed pneumo-peritoneum and duodenal perforation "Figs. 1–5". Although unclear whether the cause of the perforation was from the diagnostic or therapeutic portion of the EGD, the smaller size of the perforation suggests that it likely resulted from the therapeutic portion. Over the next hour the patient became increasingly hypotensive, requiring norepinephrine and vasopressin infusions. Due to the patient's hemodynamic instability and multiple comorbidities, general surgery recommended non-operative management with strict bowel rest, intravenous antibiotics, and intravenous fluconazole for enteric antifungal coverage.

The patient remained intubated on strict bowel rest and antibiotics for the following six days. His renal function and urine output progressively worsened, likely secondary to acute tubular necrosis from septic shock as well as from compartment syndrome as evidenced by bladder pressure measurements of greater than 15 mm Hg. Gradually, his oxygen and vasopressor requirements improved to allow for extubation on hospital day #7, but his metabolic derangements, worsening renal function, and compensatory severe

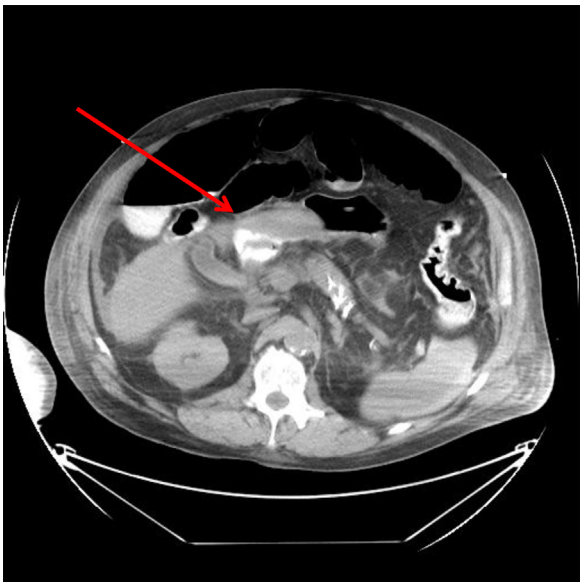

Fig. 2. Abdominal CT scan with oral contrast taken nine hours after EGD with cautery and epinephrine shows duodenal perforation with leaking oral contrast.

tachypnea lead to re-intubation twelve hours later. His acute renal failure and oliguria necessitated hemodialysis, which was initiated on hospital day #8. The patient remained intubated while having intermittent hemodialysis for several days subsequent to being re-intubated while continuing to receive broad spectrum antibiotics including micafungin while remaining in relatively stable condition and remained off vasopressor medications. His left lower extremity became cold and pulseless, raising concern for necrosis; however, vascular surgery recommended against surgical intervention given his tenuous status. On hospital day #15, the patient self-extubated and requested that he not be re-intubated in the future if necessary and that no aggressive measures be taken from that point onward. He continued to show evidence of compartment syndrome with bladder pressures consistently between 15 and 19 mm Hg and pain management was his greatest priority at that point. He and the family agreed to a palliative care approach and he subsequently expired on hospital day #19.

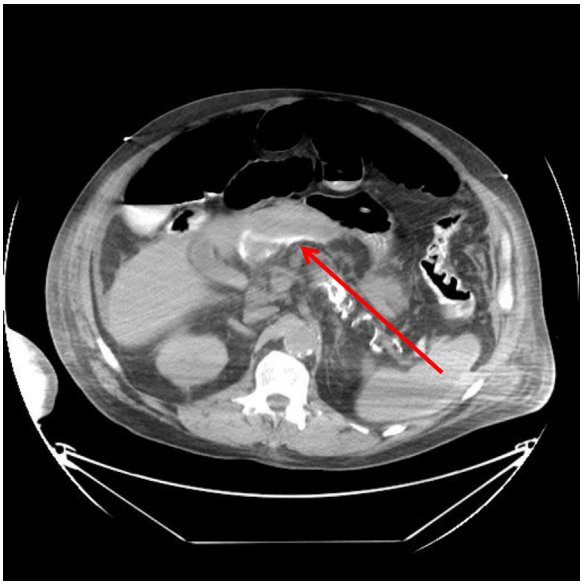

Fig. 3. Another abdominal CT image of the same CT scan with oral contrast taken nine hours after EGD with cautery and epinephrine shows duodenal perforation with leaking oral contrast.

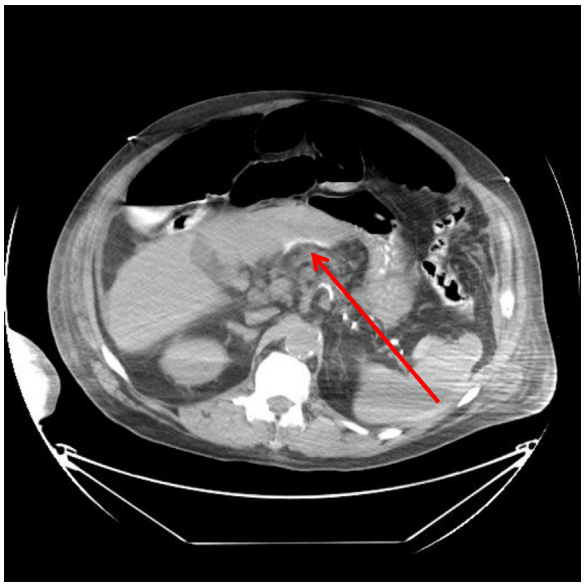

Fig. 4. Another abdominal CT image of the same CT scan with oral contrast taken nine hours after EGD with cautery and epinephrine shows duodenal perforation with leaking oral contrast.

3. Discussion

Perforations following EGD are rare, but when free wall perforation of the duodenum occurs (as opposed to perforation of the biliary tree or retroperitoneal portion of the duodenum, which are generally managed more conservatively as they tend to be smaller perforations), they tend to be associated with high morbidity and mortality, usually from multiorgan failure secondary to sepsis [4].

There are three major classifications of perforations during ERCP which use different variations based on perforation locations as well as whether perforation guidewire-related [17–19]. Since duodenal perforations following EGD occur in less than 0.1% of EGDs, there are currently no consensus guidelines on how to manage this complication. Despite the lack of guidelines, there is a consensus supporting surgical intervention for cases of severe duodenal perforation when it involves perforation of the lateral or free walls as these tend to lead to diffuse peritonitis or fluid collection in the retroperitoneum [20]. Furthermore, Turner et al. have

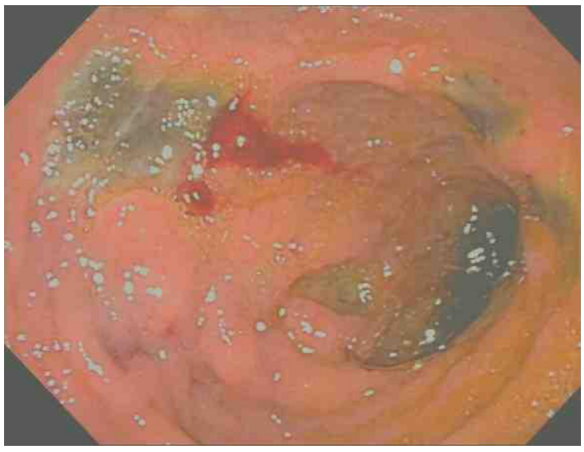

Fig. 5. Endoscopy image showing bleeding ulcer in duodenum.

compiled case reports and case series describing the management of type 1 (lateral/medial duodenal wall perforation) and type 2 (periampullary perforations related to sphincterotomy) duodenal injuries caused by endoscopic procedures and primary surgical repair was overwhelmingly performed over more conservative strategies [21].

Some groups of authors have provided general principles to aid in improving outcomes in the setting of perforation. For example, Baron et al. emphasize that the presence of free air does not automatically require surgery but that free oral contrast material extravasation should lead to prompt intervention via endoscopic closure or surgery [2,4,5]. Others have suggested considering endoscopic repair and conservative management for duodenal perforations that lead to localized peritonitis as evidenced by contained fluid collections on CT with oral contrast [20]. In our patient, CT with oral contrast was consistent with diffuse peritonitis which would prompt immediate surgical management in most patients. Unfortunately, his other comorbidities precluded him from taking such an approach.

Endoscopic management of EGD perforations includes the use of clips, stents and suturing devices and have been gaining popularity within the advanced endoscopy community. Various techniques including through-the-scope (TTS) and over-the-scope (OTS) clips have been used increasingly; however, closing larger perforations remain a challenge with clipping. While no comparison studies exist to evaluate TTS versus OTS clips, the current evidence from animal studies suggests that the OTS technique is superior to TTS [5]. Furthermore, in experienced operators, using an OTS approach in clipping can circumvent the need for surgery in 90% of cases [5]. With regards to our patient, his duodenal ulcer was approximated to being 30 mm in diameter, and the subsequent perforation was suspected to be large given his accelerated decline into septic shock, and evidence of diffuse peritonitis on CT, therefore an endoscopic approach to closure was not felt to have been of significant benefit. Endoscopically-placed stent and sutures are alternatives to clipping; however, device availability and operator preference and experience make these techniques less frequently used. For duodenal perforations, the only stents available are for esophageal interventions and this poses a significant technical challenge [5]. Baron et al. also clearly suggest that patients with hemodynamic intability, severe sepsis or septic shock generally require laparotomy with modified Gram patch procedures, while for cases of duodenal perforation leading to retroperitoneal fluid collection without peritonitis, interventional radiology-guided percutaneous drainage is recommended [5]. In our case, a major complicating factor was the patient's severe peripheral artery disease which led to necrosis of his left lower extremity after being in septic shock requiring vasopressors. Even once his septic shock resolved, he had persisting evidence of compartment syndrome, which prevented him from having vascular surgery. Ironically, his peritonitis and critical limb ischemia precluded surgical repair of each other.

4. Conclusion

The case we present here is novel and unique in that it describes the medical management of an uncommon and devastating complication of a procedure that is frequently performed. It also illustrates that, when feasible, the optimal treatment for this complication is likely surgical intervention. Hence, outcomes for duodenal perforation following EGD appear to be improved when select patients with duodenal free wall perforation leading to peritonitis and hemodynamic instability are taken for laparotomy and surgical repair.

Conflict if interest

None of the authors have any conflicts of interest to declare.

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Ethical approval

Ethics approval was not required for this case report.

Author contribution

Jason Chertoff MD, MPH – study concept or design, data collection, data analysis or interpretation, writing the paper.

Vikas Khullar MD – study concept or design, and writing the paper.

Lucas Burke MD – study concept or design, and writing the paper.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Guarantor

Jason Chertoff MD, MPH.

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