Clinical Presentation and Risk Factor in the Pathogenesis of Boerhaave Syndrome

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MEDICAL EXAMINER'S OFFICE

Background

- Rare, life-threatening condition caused by spontaneous, transmural esophageal rupture
- Risk factors are forceful emesis and drug abuse
- Presentation is often nonspecific with chest pain, emesis and dyspnea
- CT with contrast can be utilized for early recognition
- Surgical or endoscopic intervention are critical

Objectives

- Boerhaave syndrome remains under recognized in clinical practice
- Evaluate common diagnostic challenges associated with esophageal ruptures
- Provide education for timely intervention
- Discussion on how to improve future outcomes

History

- 24 year old male, found deceased at home on his bed
- Previous hx of cocaine use
- Drug paraphernalia found on scene
- No acute signs of trauma
- PMH of GERD, cyclic vomiting syndrome, liver problems and traumatic brain injuries

Autopsy Findings

- Full-thickness tear 3 x 1cm of the lower esophageal wall
- Multiple short mucoal partial-thickness tears adjacent to the rupture site
- Extensive green turbid fluid accumulation in the posterior mediastinum
- Cocaine metabolites on toxicology

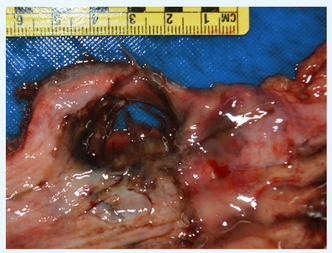

Figure 1

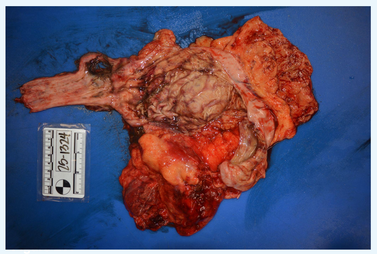

Figure 2

Fig. 1. The lower esophagus and stomach containing a 3 x 1 cm complete tear of the esophageal wall

Fig. 2 The lower esophagus contains a 3 x 1 cm complete tear of the esophageal wall on the posterior lateral aspect of the lower esophagus, about 5 cm above the gastroesophageal junction. In addition, there are multiple short mucosal tears adjacent to the ruptured site, averaging 1 cm in length. There is dark green discoloration of the lower esophageal mucosa.

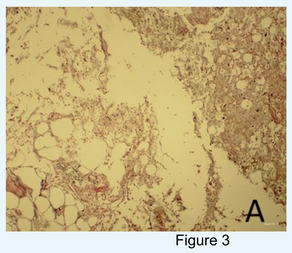

Figure 3

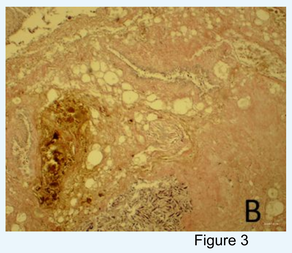

Figure 3

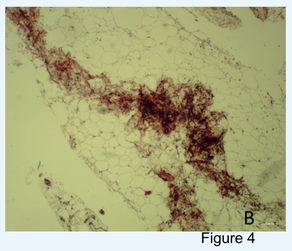

Figure 4

Fig. 3 A/B:Lower esophageal wall photomicrograph (4X) showing Necrosis involving mucosa, partial muscular layer, and periesophageal tissue; Prominent Intramural and periesophageal emphysema and edema, with focal acute inflammation. Fragments of gastric contents are seen

Fig. 4: Photomicrograph (4X) of the posterior mediastinum showing pieces of tissue with mild acute inflammation and necrosis. Soft tissue emphysema is seen. This is consistent with acute mediastinitis

Discussion

- Mortality rates up to 60% with intervention and 100% without timely intervention
- Most commonly in the left posterolateral aspect of the distal esophagus
- Mackler triad is the typical presentation: emesis, lower thoracic pain, and subcutaneous emphysema; only seen in 14% of patients
- CXR can show subcutaneous or mediastinal emphysema or mediastinal widening
- Most diagnosis are determined post-mortem
- Often symptoms mimic acute MI, tension pneumothorax, acute aortic dissection or pneumoperitoneum
- Early perforation is 12-24 hours, late perforation is >24 hours
- Primary surgical repair is the gold standard
- Data is limited on long term outcomes; indications that patients are at increased future risk of recurrent perforation

Conclusions

- Cyclic vomiting is most common cause and can occur in healthy patients
- More widespread use of Pittsburgh Score (PS) to determine mortality risk
- Future advancements of endoscopic vacuum therapy and endoscopic stenting therapy
- CT with water soluble contrast is the current gold standard
- EGD should not be first line due to increased risk of perforation

Acknowledgements

This project was supported by Pinal County Medical Examiner's Office, as well as A.T. Still University School of Osteopathic Medicine

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