observer variability in defining metaplasia of this length, current guidelines recommend against biopsy in this setting. Individual cases in practice however have demonstrated reasons to question this paradigm.

CASE DESCRIPTION/METHODS: A 42 year old white man with obesity and 10 years of reflux symptoms was diagnosed with a GE junction nodule on index endoscopy. Cold forceps biopsy of the nodule revealed high grade dysplasia, prompting referral to the BE Treatment Center at Thomas Jefferson University Hospital (TJUH). His repeat EGD revealed salmon-colored mucosa suggestive of intestinal metaplasia in an area less than 1 cm in length (Z-line at 39 cm). An 8 mm nodule was identified in the area of abnormal mucosa and removed by endoscopic mucosal resection. Pathology of the nodule revealed a T1a moderately differentiated intramucosal adenocarcinoma arising in BE. All margins were negative for invasive carcinoma, dysplasia, and intestinal metaplasia. The patient underwent endoscopic eradication therapy (EET) with radiofrequency ablation at the GE junction and in subsequent endoscopy achieved complete eradication of intestinal metaplasia (CE-IM) and dysplasia (CE-D).

DISCUSSION: Although current guidelines advise against biopsying, observer variability in Z line given its status as a relatively low risk lesion, data used to generate many of these risk predictions for EAC exclude visible lesions in their studies. Therefore in practice there may be under-evaluation of SIM-EGJ, potentially missing advanced dysplastic or cancerous disease. This case illustrates that the consideration BE risk factors and all endoscopic findings, such as nodules, need to be taken together when assessing abnormal mucosa at the GE junction, even with variability of the Z line.

INTRODUCTION: Acute esophageal necrosis (AEN), also known as “black esophagus” or “acute necrotizing esophagitis”, is a rare entity thought to be caused by a known ischemic insult from a low perfusion state. Etiologies include multi-organ dysfunction, hypoperfusion, vasculopathy, sepsis, among others. Sunitinib, a chemotherapeutic agent for renal cell cancer and gastrointestinal stromal tumors, has common gastrointestinal (GI) side effects of diarrhea and vomiting but severe GI side effects are rare. We present a unique case of Sunitinib-induced AEN.

CASE DESCRIPTION/METHODS: A 72-year-old man presented to the hospital for a two days history of epigastric abdominal pain, bilious vomiting, and multiple episodes of non-bloody diarrhea. His medical history was significant for metastatic papillary renal cell carcinoma on Sunitinib. On presentation, his blood pressure was 74/52 mm Hg and heart rate 107 beats per minute. He exhibited generalized abdominal tenderness without rebound or guarding. His hemoglobin was 15 g/dL. He was admitted to the ICU for hypovolemic shock thought to be secondary to severe gastroenteritis requiring the use of vasopressors. He later developed ongoing melena and his hemoglobin decreased to 10.8 g/dL requiring urgent Endoscopy. The examination revealed circumferential, black, necrotic mucosa in the lower one-half of the esophagus with a sharp demarcation of healthy mucosa at the level of gastroesophageal junction without any active bleeding. Findings were consistent with AEN.

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The patient remained hypotensive despite aggressive resuscitation and the use of vasopressors. Unfortunately, the patient died several days later.

**DISCUSSION:** AEN is characterized by the classic endoscopic image of diffuse, circumferential, black-appearing necrotic mucosa. The exact etiology leading to the hypoperfusion state in our case is unclear and could be attributed to Sunitinib. The most common adverse GI reactions to Sunitinib include diarrhea (34%), nausea (48%), and vomiting (24%) which were intractable in the above case. This medication has also been linked to severe GI hemorrhage. Sunitinib inhibits platelet-derived growth factors and vascular endothelial growth factors which play a major role in angiogenesis and apoptosis. We believe that by inhibiting these cellular processes, the patient was predisposed to development of AEN which was potentiated by the patient’s hypovolemic state.

**Black Esophagus: A Case of Acute Esophageal Necrosis**

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**INTRODUCTION:** Acute esophageal necrosis (AEN) is a rare clinical entity characterized by diffuse, circumferential, black-appearing distal esophageal mucosa on EGD typically limited to the gastroesophageal junction. In this case, we discuss AEN in a chronically ill female precipitated by an acute low perfusion state in the setting of her preexisting vasculopathy.

The patient initially presented to our institution for symptoms of nausea, vomiting, and melena. She was found to have a systolic blood pressure in the 80s. Physical exam was notable for pallor and delayed capillary refill time. Labs revealed a hematocrit of 18.8 g/dL, white blood count of 17 uL, procalcitonin of 1.8 ng/mL, creatinine of 1.2 mg/dL, INR of 2.7, and albumin of 2.5 g/dL. CT of the chest and abdomen revealed a right lower lobe lung consolidation and a very decompressed IVC concerning for a low volume state. Patient was adequately fluid resuscitated, kept NPO and was placed on a pantoprazole (PPI) infusion. An EGD was performed which revealed diffuse severe mucosal changes characterized by ulceration with necrotizing black mucosa in the lower third of the esophagus, and non-blending, crinkled and superficial duodenal ulcers. One week later, a repeat EGD demonstrated significant esophageal mucosal healing with esophagitis and distal esophageal ulcerations. Esophageal biopsies revealed demal submucosa, marked acute inflammation with negative HSV, CMV and fungal cultures. Given the significant improvement in symptoms and findings, her diet was advanced. She was discharged on oral PPI and sarecarel with a plan for repeat EGD in three months.

**CASE DESCRIPTION/METHODS:** A 76-year-old female with past medical history of severe coronary artery disease, high-grade distal aortic stenosis, gastroesophageal reflux disease, history of ischemic colitis presents with a two-day history of melena. Patient initially presented at an outside hospital for symptoms of anemia and was transfused packed red blood cells. She was transferred to our institution and was found to have a systolic blood pressure in the 80s. Physical exam was notable for pallor and delayed capillary refill time. Labs revealed a hematocrit of 18.8 g/dL, white blood count of 17 uL, procalcitonin of 1.8 ng/mL, and albumin of 2.5 g/dL. CT of the chest and abdomen revealed a right lower lobe lung consolidation and a very decompressed IVC concerning for a low volume state. Patient was adequately fluid resuscitated, kept NPO and was placed on a pantoprazole (PPI) infusion. An EGD was performed which revealed diffuse severe mucosal changes characterized by ulceration with necrotizing black mucosa in the lower third of the esophagus, and non-blending, crinkled and superficial duodenal ulcers. One week later, a repeat EGD demonstrated significant esophageal mucosal healing with esophagitis and distal esophageal ulcerations. Esophageal biopsies revealed demal submucosa, marked acute inflammation with negative HSV, CMV and fungal cultures. Given the significant improvement in symptoms and findings, her diet was advanced. She was discharged on oral PPI and sarecarel with a plan for repeat EGD in three months.

**Dysphagia Lusoria Associated With Imperforate Anus**

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**INTRODUCTION:** Dysphagia lusoria is a rare intrathoracic vascular abnormality that results in the compression of the esophagus, causing dysphagia. The most common cause of dysphagia lusoria is an aberrant right subclavian artery of the aortic arch, though nearly all major arteries within the thorax have been reported to cause cause this condition.

**CASE DESCRIPTION/METHODS:** A 68-year-old male with a history of developmental delay and imperforate anus s/p colostomy and colostomy revision presented with decreased appetite, decreased bowel movements, and inability to pass solids or liquids. Past medical history was notable for GERD and prior small bowel obstructions. Abdominal imaging of the chest showed tortuosity of the thoracic aorta. Fluoroscopic swallow showed absent bolus transit through the esophagus. CT angiogram showed an anomalous right subclavian artery of the aortic arch trapping the proximal esophagus anteriorly and posteriorly, resulting in a narrowed channel with a normal appearing distal esophagus (Figure 1). CTA chest confirmed compression of the esophagus by the anomalous aortic arch (Figure 2). Multiple treatment options were considered.