

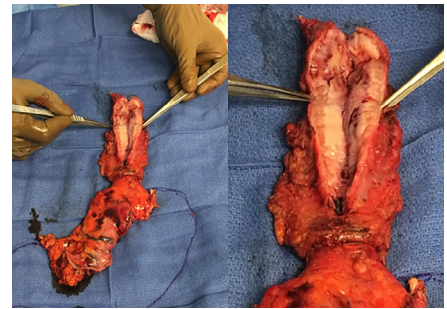
# Black and White Esophagus: Rare Presentations of Severe Esophageal Ischemia

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Benign esophageal strictures are typically the result of long-standing gastroesophageal reflux, and are usually treated with serial dilations and acid-suppressive therapy. Other causes of benign esophageal strictures include external beam radiation, caustic ingestions, prior surgery, and external compression from mediastinal fibrosis. We report 2 rare causes of ischemic esophageal structuring occurring after operations unrelated to the esophagus. The first is a patient who developed esophageal injury following radiofrequency ablation for atrial fibrillation. The direct thermal injury resulted in a “white esophagus” with a full-thickness, long-segmental stricture. The second patient presented with a “black esophagus” also known as acute necrotizing esophagitis. This occurred after an orthotopic liver transplant, which was complicated by multiple organ dysfunction secondary to hemorrhagic shock. In this report, we present 2 rare causes of esophageal stricturing that occurred after procedures not necessarily related to the esophagus itself. Early recognition and active management of these esophageal injuries may lead to better outcomes.

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**Keywords:** black esophagus, white esophagus, esophageal stricture, acute necrotizing esophagitis, esophageal ischemia



Gross specimen revealing a white, leathery esophagus.

### Central Message

We report 2 rare presentations of ischemic esophageal strictures that occurred after operations unrelated to the esophagus.

## BACKGROUND

Benign esophageal strictures are typically the result of long-standing gastroesophageal reflux, and are usually treated with serial dilations and acid-suppressive therapy.<sup>1</sup> They can also be caused by radiation, caustic ingestions, prior surgery, and external compression from mediastinal fibrosis. We report 2 rare presentations of ischemic esophageal strictures that occurred after operations unrelated to the esophagus.

### Case 1

A 67-year-old man with coronary artery disease, obesity, atrial fibrillation, and hypertension developed sudden onset of dysphagia to solids 1 week after undergoing coronary artery bypass and ablative maze procedure at another facility. The operative notes did not specify the energy device used for the ablation, but described

the placement of ablation lines along the posterior right atrial wall extending from the superior vena cava to the inferior vena cava. The left pulmonary veins were also ablated using an unspecified technique. A barium esophagram was performed and showed a long, very narrow string-like stricture extending from the carina to just above the gastroesophageal junction along with proximal esophageal dilation (Fig. 1). Following failure of conservative therapy, the patient was referred to us 9 months later for further management. Upper endoscopy revealed a 14-cm-long, severe, non-ulcerative stricture (Fig. 2) beginning at 28 cm from the incisors. This could be traversed only with a pediatric endoscope and showed white exudates in the affected area, but biopsies were negative for malignancy.

The patient underwent a minimally invasive Ivor Lewis esophagectomy with laparoscopic and right thoracoscopic approaches. The esophagus was thick, white, and leathery (Fig. 3), with dense fibrotic adhesions to the posterior airway and the pericardium. A high intrathoracic anastomosis was created at the level of the thoracic inlet using a 25 mm OrVil EEA stapler. He developed empyema secondary to severe postoperative atelectasis and right lower lobe pneumonia. No leak was seen on the postoperative esophagram. He improved rapidly after thoracoscopic washout and was discharged on a mechanical soft diet. Final pathology showed benign erosive esophagitis with dense polyclonal lymphoplasmacytic inflammatory infiltrate.

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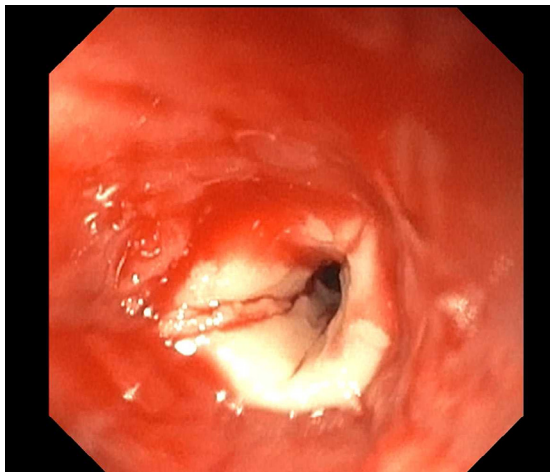
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**Figure 1.** Preoperative barium esophagram demonstrating a narrow stricture from the carina to the gastroesophageal junction with proximal esophageal dilation.

**Case 2**

A 51-year-old woman underwent orthotopic liver transplantation for alcoholic cirrhosis. Ten days later, she underwent an exploratory laparotomy for postoperative bleeding and severe hypotension that resulted from a lacerated phrenic artery. She developed multiple organ dysfunction secondary to hemorrhagic shock, requiring tracheostomy and hemodialysis. She developed dysphagia to solids and liquids except her own saliva. An upper endoscopy revealed a “black esophagus” with severe stricturing (Fig. 4) that was seen as a “string sign” on barium esophagram (Fig. 5). Because of severe de-



**Figure 2.** Upper endoscopy revealing severe non-ulcerative stricture. (Color version of figure is available online.)

conditioning and malnourishment, she was considered a poor candidate for esophagectomy with immediate reconstruction, and was managed with serial dilations and esophageal stenting. Three months later, she presented with respiratory distress, requiring re-intubation. On bronchoscopy, the esophageal stent was seen to have eroded into the posterior tracheal wall, causing a 1 cm × 1 cm perforation, located 3 cm above the carina. An emergent esophagectomy was performed through a right posterolateral thoracotomy to remove the stent and repair the tracheoesophageal fistula.

Primary closure of the tracheoesophageal fistula was not possible because of its size and severe inflammation. Hence, a composite patch repair of the fistula was performed using human cadaveric dermis (AlloDerm) and a pedicled intercostal muscle flap. A silicone tracheal Y-stent (Dynamic) was also placed to protect the repair from positive pressure ventilation. Cervical esophagostomy and redo tracheostomy were also performed. The stent was removed 4 weeks later and the fistula was noted to have completely healed. Final pathology of the esophagus revealed necrotic debris and inflammatory cells with no viable tissue.

**DISCUSSION**

Esophageal injury following radiofrequency catheter ablation for atrial fibrillation is a rare occurrence and possibly occurs from transmural transmission of thermal energy during ablation of the posterior wall of the left atrium. Although left atrial-esophageal fistulas have been reported following non-cut-and-sew maze procedures, we were unable to find any previous reports of esophageal stricture as a complication after maze ablation in literature.<sup>2</sup> Doll et al reported that 1% of patients had an esophageal perforation after ablation in a series of 387 patients who underwent intraoperative radiofrequency ablation for atrial fibrillation.<sup>3</sup> These patients presented with sudden neurologic symptoms from esophago-atrial air embolization within 9 days from their operation, and 3 required extensive esophageal resection. In comparison with those patients, our first patient had a unique presentation with sudden onset of dysphagia in the first week following his surgery, which progressed over months. We did not recommend an esophageal stent or dilation because of the full-thickness, long-segment stricture and concerns of perforation. The gross and microscopic pathology appeared consistent with a chronically ischemic esophagus. We hypothesized that this was likely due to direct thermal injury from the ablation leading to segmental devascularization of the esophagus.

The black appearance of the esophagus in our second patient was strikingly different from that of the first patient. “Black esophagus,” also known as acute necrotizing esophagitis, was first described by Goldenberg et al in 1990<sup>4</sup> as a form of ischemia-induced

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