Esophagopulmonary Fistula and Left Lung Abscess After Transoral Incisionless Fundoplication

Jessica M. Titus, MD, David P. Mason, MD, Daniel P. Raymond, MD, Thomas W. Rice, MD, and Sudish C. Murthy, MD, PhD

Heart and Vascular Institute, Department of Thoracic and Cardiovascular Surgery and Vascular Surgery, Cleveland Clinic, Cleveland, Ohio

Endoscopic treatment of gastroesophageal reflux disease (GERD) is emerging as an alternative to open, laparoscopic, or robotic antireflux surgical procedures. Early transoral devices failed because of poor efficacy and serious adverse events. In recent trials the EsophyX device has demonstrated acceptable safety and a reasonable efficacy profile. We present the case of a woman who experienced a distal esophageal perforation and esophagopulmonary fistula after treatment for GERD with the EsophyX device. Although considered minimally invasive, endoscopic procedures for GERD treatment can have significant deleterious consequences, and early recognition of these complications is vital to limit associated morbidity.


Over the past decade, endoscopic therapies have been developed for treating gastroesophageal reflux disease (GERD) [1]. Early therapies approved by the US Food and Drug Administration were radiofrequency ablation and injection of the lower esophageal sphincter (LES) [1, 2]. Both were ultimately abandoned because of limited efficacy, serious adverse events, or both [1, 3, 4]. Endoscopic suturing/plication devices have more recently emerged as treatment alternatives. Modern trials have shown reasonable palliation of symptoms at 2-year follow-up [1, 4, 5]. The EsophyX device (EndoGastric Solutions, Inc, Redmond, WA) is the most popular of these newer devices. It uses suction tissue molds and endoscopically placed “H-fasteners” to reduce small hiatal hernias and restore the sphincteric mechanism at the gastroesophageal junction (GEJ) [4, 6]. Adverse events are uncommon and have been limited to cervical esophageal perforation from device insertion and hematemesis requiring transfusion [6, 7]. We present a case of unrecognized distal esophageal perforation from the EsophyX device that eventually manifested as a left lung abscess and sepsis.

A 42-year-old woman presented to an outside institution with intractable reflux. Upper endoscopy showed a small, reducible hiatal hernia with no evidence of esophagitis or Barrett esophagus. She subsequently underwent endoscopic reduction of the hiatal hernia and transoral incisionless fundoplication with use of the EsophyX device. The operative report stated that a 3-cm 270° fundoplication was performed. She was admitted for overnight observation. The next morning she was noted to have a cough, low-grade fever, and weakness. A chest radiogram showed left posterior basilar infiltrate and a small pleural effusion presumed to be a left lower lobe aspiration pneumonia. Intravenous antibiotics were begun, and she was discharged 3 days later, receiving a full liquid diet and oral ciprofloxacin. She had multiple readmissions over the next 3 weeks for similar signs and symptoms with worsening severity until an esophagram and computed tomography scan of the chest demonstrated distal esophageal perforation with transmediastinal fistulous connection to a large cavitary left lung lesion (Fig 1A). Blood cultures were positive for methicillin-sensitive Staphylococcus aureus, and she was transferred to our hospital for treatment.

Upon arrival at our institution the patient was in early septic shock. Her symptoms included shortness of breath and productive cough. Esophagram confirmed the leak at 5 cm above the GEJ. A transthoracic echocardiogram was negative for endocarditis.

After she was resuscitated, she was taken to the operating room for endoscopy and exploration. Esophagogastroduodenoscopy showed an H-fastener in an area of inflamed distal esophagus at the level of the suspected leak (Fig 1B). No clear perforation was encountered. Inspection of the hiatus on retroflexion showed multiple H-fasteners in place, with the fundoplication no longer intact (Fig 1C). The left side of the chest was explored through a seventh-interspace thoracotomy. Dense inflammatory adhesions from the left lower lobe to the diaphragm were taken down. A fibrosed fistulous tract was encountered and appeared to have obliterated. Insufflation through an endoscope confirmed fistula closure. Wide debridement of the fistulous phlegmon was performed. The distal end of the fistula terminated in the left lower lobe and led to a large parenchymal abscess cavity (Fig 1D). The cavity was drained, debrided, and marsupialized (Fig 1E). Four percutaneous drains were left within the thorax.

The patient’s postoperative course was remarkable for acute kidney injury. An esophagram on postoperative day 4 showed no leak, and she underwent transition to oral antibiotics along with diet advancement. She was discharged on postoperative day 8 with a 4-week course of oral fluconazole, amoxicillin-clavulanate, and doxycycline. At 1-month follow-up, she was tolerating soft foods with no further shortness of breath or cough, and, surprisingly, no complaints of reflux.

Comment

To our knowledge, distal esophageal perforation from deployment of the EsophyX device has not been reported. Upon review of complications of its use, esophageal perforation has occurred proximally in the hypo-
pharynx, relating to insertion rather than deployment of the device [7, 8]. We suspect that the mechanism of injury was an H-fastener placed too far proximally that had pulled through, lacerating the distal esophagus. H-fastener remnants were found in the fundus of the stomach approximately 2 to 3 cm distal to the majority of others. We also suspect that in attempting to reduce the hiatal hernia, too much tissue was suctioned into the device and was subsequently plicated. This errant fastener created a full-thickness linear laceration of the distal esophagus with subsequent fistula to the left lung.

Esophagopulmonary fistula (EPF) is a rare complication of iatrogenic esophageal perforation. It is more commonly neoplastic in origin, inasmuch as EPFs are often described in the broader context of the esophagorespiratory fistulas resulting from esophageal and pulmonary malignancies. Esophagorespiratory fistulas can involve any area of the respiratory system and complicate 1% to 22% of esophageal cancers, with up to 10% of these heralding the cancer [8]. Treatment of EPFs with self-expanding, covered metallic stents has been attempted, with palliation of symptoms in as many as 86% of patients [8]. In this patient, temporary esophageal stenting was briefly considered as an alternative if control of the perforation could not be achieved surgically. However, given the presence of the fistula and abscess, this possibility was quickly abandoned.

In our case, it appears that the perforation was present shortly after the index procedure, because both the symptom complex and the radiograms were highly suggestive. Given the delay in diagnosis, it was quite fortunate that an EPF developed instead of free perforation with mediastinitis. Expectoration of the abscess contents presumably transitioned the process into a more chronic, slowly evolving disease. This was tolerated for almost a month by this young, previously healthy patient before sepsis became manifest.

No doubt a more thorough investigation with esophagram, computed tomographic scan, or both of her early postprocedure problem would have identified it much sooner and eliminated multiple hospital readmissions. Recognition before formation of lung abscess might also have permitted more conservative therapy, such as esophageal stenting and antibiotics, to treat the condition and preclude the need for thoracotomy.

**Conclusion**

Lung abscess from EPF secondary to esophageal perforation is a rare and life-threatening complication of endoscopic fundoplication. Early diagnosis is critical, and unexpected early chest pain, cough, or fever should
prompt an aggressive diagnostic work-up. Transoral treatment of GERD in combination with a large hiatal hernia component should be avoided.

References

Ventricular Assist Device for Failing Systemic Ventricle in an Adult With Prior Mustard Procedure
Robert C. Neely, MD, Robert Patrick Davis, PhD, Elizabeth H. Stephens, MD, PhD, Hiroo Takayama, MD, PhD, Zain Khalpey, MD, PhD, Jonathan Ginz, MD, Sun Hi Lee, MD, and Jonathan Chen, MD
Division of Cardiothoracic Surgery, Department of Surgery, Columbia University Medical Center, New York, New York; Division of Cardiothoracic Surgery, Department of Surgery, The University of Arizona Medical Center, Tucson, Arizona

The Mustard procedure is a palliative surgical procedure used to repair complete transposition of the great arteries. Cardiac transplantation remains the only definitive therapy for patients who develop heart failure after a Mustard procedure. However, pulmonary hypertension represents a major hemodynamic contraindication. The use of a ventricular assist device as destination therapy has not yet been established after a Mustard procedure. Here, we present the case of a 41-year-old patient who presented with systemic right ventricular failure following Mustard procedure complicated by pulmonary hypertension. The patient received a HeartMate II (Thoratec, Pleasanton, CA) ventricular assist device as a bridge to decision.

© 2013 by The Society of Thoracic Surgeons

The Mustard procedure represents an early effort at palliative surgical correction of dextro-transposition of the great arteries (d-TGA); however, it is rarely performed today. Current practice guidelines advise surgical correction through an arterial switch, thereby reestablishing the morphologic left ventricle (LV) as the systemic ventricle. In the Mustard procedure, a pericardial intraventricular baffle is created to redirect ventricular inflow. The Senning procedure, the other common surgical option prior to the arterial switch, employs the atrial septum and wall to create the baffle. In both instances, the right ventricle (RV) continues to function as the systemic ventricle, while the LV continues to provide pulmonary circulation [1]. Pulmonary vascular disease is a well-documented complication in the absence of d-TGA repair [2]. Although repair offers patients improved survival and quality of life, right ventricular failure occurs in 8% to 44% of patients [3].

Pulmonary hypertension is a major hemodynamic contraindication to cardiac transplant [4]. Thus, patients who present with congestive heart failure and concomitant pulmonary hypertension after Mustard procedure often are ineligible for a ventricular assist device (VAD) as a bridge to transplant. However, the natural history of pulmonary hypertension in adults with congenital heart disease undergoing mechanical support is not yet fully understood. Therefore, the use of a VAD as a bridge to decision—rather than a priori destination therapy—should be considered a viable therapeutic option.

Our patient is a 41-year-old man with history of d-TGA who underwent a prior Mustard procedure. The patient subsequently developed systemic RV failure and progressive pulmonary hypertension as an adult. He presented with worsening shortness of breath, and a transthoracic echocardiogram revealed a severely hypertrophied, dilated RV with an ejection fraction of 10% to 15%. Right-side heart catheterization revealed elevated pulmonary artery pressures (79/34 mm Hg) and elevated pulmonary vascular resistance (>17 Wood units). The patient was not eligible for cardiac transplant secondary to severe pulmonary hypertension, but qualified for a VAD with pulmonary vasodilator therapy.

The patient was taken to the operating room for placement of a HeartMate II VAD (Thoratec, Pleasanton, CA). Transesophageal echocardiogram confirmed an atrial baffle and dilated RV. Effort was made to locate the moderator band, which was ultimately not identified. Preoperative central venous pressure was 27 mm Hg. The patient was administered heparin and placed on cardio-

Accepted for publication Dec 13, 2012.

Address correspondence to Dr Neely, Milstein Hospital Bldg, Rm 7GN-435, Division of Cardiothoracic Surgery, Department of Surgery, Columbia University Medical Center, New York, NY 10032-1317; e-mail: rcn2101@ columbia.edu.

© 2013 by The Society of Thoracic Surgeons
Published by Elsevier Inc