

Endoscopic Management of Anastomotic Esophageal Strictures Secondary to Esophageal Atresia

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KEYWORDS

- Esophageal atresia Tracheoesophageal fistula Esophageal stricture
- Esophageal dilation Esophageal stenting Intralesional steroid injection
- Endoscopic incisional therapy Mitomycin C

KEY POINTS

- Esophageal dilation with balloon or savory dilators are equally safe and effective for the treatment of anastomotic strictures associated with esophageal atresia.
- Dilations should be performed every 2 weeks for 2 to 3 months before deeming a stricture refractory.
- Intralesional steroid therapy should be strongly considered as first-line therapy for refractory strictures.
- Other adjunct therapies, such as Mitomycin C, endoscopic incisional therapy, and esophageal stent placement, have some reported benefits; however, they also have more inherent risk.
- Nissen fundoplication should be considered if gastroesophageal reflux is suspected to be contributing to recurrent structuring.

INTRODUCTION

Esophageal atresia (EA) with or without tracheoesophageal fistula (TEF) is the most common congenital anomaly of the esophagus.¹ The overall incidence of EA/TEF ranges from 1 in every 2500 to 4500 live births. The first successful EA/TEF repair was performed by Dr Cameron Height in 1941. The technical goal of the surgery is to first divide the TEF very close to the trachea and then ligate it with nonabsorbable

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Gastrointest Endoscopy Clin N Am 26 (2016) 201–219 http://dx.doi.org/10.1016/j.giec.2015.09.002 1052-5157/16/\$ – see front matter © 2016 Elsevier Inc. All rights reserved.

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Disclosure Statement: The author has nothing to disclose.

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sutures. Following TEF ligation, the EA is repaired through the creation of an anastomosis from the proximal esophageal pouch and the distal esophageal segment. The anastomosis is usually achieved in end-to-end fashion.^{2,3}

Survival rates for patients with EA with or without TEF have improved greatly over the past 2 decades with technical advances in surgery as well as with critical care medicine. The most recent survival rates have ranged from 91% to 97%.^{4–6} The survival rates for infants born full-term with no associated congenital anomalies have been of reported to approach 100%.^{6,7} Nevertheless, despite high survival rates, patients with EA may deal with significant postoperative morbidity. **Box 1** lists the common morbidities associated with EA postsurgical repair both in the immediate postoperative period, as well as those that can occur later in a patient's life. In this review, we focus on the endoscopic management of esophageal strictures as the most common morbidity associated with EA repair.

ESOPHAGEAL STRICTURE

Pathophysiology and Incidence

The normal process of wound healing after creation of the esophageal anastomosis involves the creation of scar tissue. During the tissue remodeling phase of wound healing, fibroblasts promote wound contraction. Tissue contraction of open wounds is beneficial to close the injury; however, wound contraction in the setting of a circular end-to-end anastomosis creates narrowing. Therefore, it is quite natural to see a degree of narrowing at the site of the esophageal anastomosis after EA repair.

The reported incidence of anastomotic stricture after EA repair has varied in case series from as low as 9% to as high as 80%.^{3,8–15} There are several factors implicated in the pathogenesis of anastomotic stricture. These include creation of the esophageal anastomosis under excessive tension, ischemia at the ends of the esophageal pouches, creation of the anastomosis with 2 suture layers, use of silk suture material, anastomotic leak, esophageal gap length greater than 4 cm (long gap EA), and post-operative gastroesophageal reflux.¹⁶

Esophageal Stricture Symptoms and Definitions

When a swallowed food bolus becomes too large to pass through the narrowed portion of the esophagus, symptoms of dysphagia will occur. Typical symptoms of

Box 1 Common morbidities associated with postsurgical esophageal atresia repair
Esophageal stricture
Esophageal leak or perforation
Anastomosis dehiscence
Recurrent tracheoesophageal fistula
Gastroesophageal reflux disease
Dysphagia
Esophageal dysmotility
Aspiration
Esophagitis
Barrett esophagus
Esophageal cancer

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