Management of Complications After Esophagectomy

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KEYWORDS

• Esophagectomy • Complications • Esophageal cancer

KEY POINTS

- Esophagectomy is a complex operation with many potential complications.
- Early recognition of complication is vital to patient survival.
- Minimally invasive approaches for managing complications are emerging.

INTRODUCTION

Surgical resection remains a mainstay in curative-intent treatment of esophageal cancer. Esophagectomy is a complex operation, however, with myriad potential complications. Moreover, esophagectomy itself is a heterogenous group of operations with different approaches and anastomoses, each with its own side-effect profile. In a recent analysis of the multinational and standardized online database by the Esophageal Complications Consensus Group (ECCG), the incidence of complications was 59% with severe complications, as defined by a Clavien-Dindo grade greater than IIIB occurring in 17.2%.[1,2] Clearly, esophagectomy remains fraught with complication, and management thereof is vital. Improved management of these postoperative complications, however, has led to improved survival rates over the past 30 years.[3] This article reviews common surgical complications and their management.

ANASTOMOTIC LEAK

The most pressing concern after esophagectomy is leak at the gastroesophageal anastomosis. Early identification of leaks provides the best opportunity to minimize morbidity and mortality from a historically mortal complication.[4]

Risk Factors

Usually, leaks arise as a result of ischemia at the anastomosis, preventing adequate healing. The gastroesophageal anastomosis is particularly susceptible because the gastric conduit relies on the right gastroepiploic vessel as the sole source of blood supply, and careful preservation of this vessel during creation of the gastric conduit is paramount. This vessel, however, does not reach high onto the fundus where the anastomosis is created, and thus anastomosis viability requires adequate submucosal microvasculature from the right gastroepiploic vessel to the tip of the conduit.[5] Consequently, where the anastomosis is situated affects the leak rate. Anastomoses in the neck generally are associated with a higher risk of leak owing to the longer gastric conduit required and the higher amount of tension. Randomized controlled trials by Chasseray and colleagues,[6] Walther and colleagues,[7] and Okuyama and colleagues[8] all trended toward higher leak rates in the neck. In a review of the Society of Thoracic Surgeons database, Kassis and colleagues[9] showed a cervical anastomotic leak rate of 12.3% versus 9.3% in the chest, which is comparable to the ECCG leak rate of 11.4% for all-comers.

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Thorac Surg Clin 30 (2020) 359–366
https://doi.org/10.1016/j.thorsurg.2020.04.002

Apart from where the anastomosis is located, how the anastomosis is formed also is diverse. Some surgeons prefer a hand-sewn anastomosis, others a stapled anastomosis either by circular stapler or by linear stapler. Blackmon and colleagues[10] retrospectively compared these 3 techniques and concluded that stapler techniques result in lower rates of stricture, but no difference in leak rates were identified. Wang and colleagues[11] subsequently performed a randomized controlled trial in 155 patients and demonstrated no difference in leak rates but a lower level of stricture using a linear stapler. Meta-analyses exploring this issue similar do not show any difference in leak rates between these methods, with the exception of perhaps more stricturing with circular stapler.[12,13] Overall, there is no apparent best technique for anastomosis construction.

In general, surgeons create a gastric conduit tube along the greater curve rather than utilize the whole stomach as a conduit. This is thought to help drainage of the conduit as well as to prevent thoracic stomach syndrome, where a distended stomach in the chest results in physiology similar to that of tension pneumothorax. The ideal width of this conduit, however, remains debated. Barbera and colleagues[14] showed that the narrower the conduit, the faster the emptying. Tubes that are narrower than 4 cm, however, are thought to affect the submucosal vessels and lead to conduit tip ischemia; however, this has not been demonstrated in randomized controlled trials. Tabira and colleagues[15] compared a subtotal stomach to a narrow gastric conduit, with 22 patients in each group, and Zhang and colleagues[16] compared a whole stomach conduit to a narrow gastric tube, with 52 patient in each group, and neither study showed a difference in leak rates but neither narrow tube was less than 4 cm. Other retrospective studies are conflicting in their conclusions.[17–19] Overall, conduit width down to approximately 4 cm does not seem to affect leak rate. Narrower or focally narrower conduits less than 4 cm may disturb the submucosal vessels and cause conduit tip ischemia but no formal study has shown this because likely no surgeon routinely uses conduits narrower than 4 cm.

Given that the stomach is devascularized and left to survive on the right gastroepiploic vessel at the time of conduit construction and anastomosis, a strategy to divide the vessels prior to conduit construction and anastomosis was devised as a form of ischemic preconditioning of the stomach. Two approaches have been described, one using arterial embolization and another using laparoscopic division of the vessels.[20,21] Arterial embolization showed less reduction in blood flow at the anastomotic site and lower leak rates but was complicated by splenic infarction and pancreatitis. Laparoscopic division allowed simultaneously for additional staging and lymph node dissection but meant that esophagectomy would need to occur in an operated field. Depending on the interval between laparoscopic intervention and esophagectomy, leak rates were lower or even higher, with 2 weeks the apparent minimum for benefit. A meta-analysis subsequently has been performed, demonstrating some minor reduction in leak rate from 14.1% to 8.8% but did not reach statistical significance.[22] Overall, there appears to be no strong benefit in small series and, given the logistical complexity and additional procedural risk, it does not appear that this approach is warranted.

Intraoperative vascular assessment is another strategy for assessment of the conduit. Use of intravenous indocyanine green dye and a near-infrared camera allows for real-time visualization of gastric conduit perfusion.[23] Small retrospective studies show this to be promising method to better situate the anastomosis.[24–26] A recent meta-analysis of the available literature supports the technique as being safe and as aiding in reducing leak; however, the quality of many of the articles was limited by small numbers and absence of a nonexposed cohort.[27] For now, it appears that use of intraoperative assessment may be helpful but further large-scale studies will be required to examine if anastomotic leak rates truly can be impacted.

Diagnosis

Early recognition of anastomotic leak is critical. In some cases, the leak will be apparent, that is, saliva or bile out a drain. In many cases, however, clinical presentation is subtle and any deviation from usual postoperative course should be cause for consideration of an anastomotic leak. Leaks can present as tachycardia, new atrial fibrillation, pain, respiratory compromise, and delirium, among others. It is important to never simply attribute something, such as atrial fibrillation or delirium, as a matter of postoperative course without consideration that a leak is the underlying cause. Anastomotic failures can be investigated in a variety of ways, each with its own pros and cons.[28] CT scan with oral contrast allows for identification of a leak by extraluminal contrast or air and also allows for assessment of collections and potential for drainage. Gastrografin with or without barium swallow is most sensitive but does not allow for much assessment of

surrounding structures. Endoscopic assessment allows for direct visualization of the defect and evaluation of the viability of the conduit and potentially allows for therapeutic intervention.

Management

Once a leak is identified, the extent of the leak needs to be assessed. Minor contained leaks, where a patient is clinically stable, can simply be observed for deterioration or treated with antibiotics, nasogastric decompression of the conduit, and distal enteral or parenteral nutrition. More significant leaks in the neck can be managed by opening the neck incision and placing drains to obtain source control. These drains can be shortened over the next few weeks as the defect heals. Major leaks in the chest are more challenging to deal with but follow the principles of source control. Collections need to be drained, the lung decorticated, and ongoing soilage of the chest controlled. In some cases, percutaneous drains are adequate to obtain source control. In more significant leaks, operative assessment may be required to assess whether the leak is from the anastomosis itself or along the gastric staple line. If the conduit is largely healthy, the defect can be reclosed and buttressed with vascularized tissue. The advent of minimally invasive approaches means that many muscle flaps remain available to the surgeon in the event of a leak, such as intercostal, serratus, or latissimus, and care during thoracotomy to protect potential flaps are needed.

Endoscopic stenting instead of operative repair of the leak more recently has been added to the management armamentarium. In this strategy, self-expanding fully covered metal stents are placed across the anastomotic defect to prevent further soilage of the thoracic cavity. It is important to remember that stenting is meant only to replace the operative closure of the leak. Drainage of existing collections for source control remains important. One major issue is that stents largely are designed to expand against strictures. In an esophagogastric anastomosis, the esophagus is of normal caliber but the conduit usually is larger. Thus, although the proximal flared portion of the stent may stay within the esophagus, the distal flared portion may not be large enough for the conduit, leading to inadequate sealing of the leak due to reflux of conduit contents behind the stent. Additional stents may be required to obtain a watertight seal for adequate source control. Another issue that can occur is the high propensity for stent migration because only the proximal flared portion can hold the fully covered stent in place. Thus, the largest fully covered double flared stent should be chosen to avoid this issue, and clips on the esophageal side may be required. Multiple series have been published demonstrating the success of such a strategy.[29,30] A large series by Plum and colleagues[31] of 70 patients over 10 years demonstrated a sealing success rate of 70%, with a median treatment time of 28 days. Complications occurred in approximately 30% of cases in the forms of stenosis, stent migration, persistence of leakage, and perforation/esophago-airway fistula. Survival was 87% in patients who received a stent. Factors predictive of success were not identified in this study; thus, whom to stent remains subject to clinical judgment.

Recently, an alternative to self-expanding metal stents in the form of endoscopic vacuum therapy has been developed for the treatment of leaks. Continuous negative pressure is applied to the defect and cavity via a sponge placed endoscopically and attached to a nasogastric tube. de Moura and colleagues[32] describe a few methods of creating such a device. This is changed every 3 days to 4 days until the cavity is closed by granulation. This avoids some of the difficulties with stenting, such as incomplete occlusion of the leak and stent migration, and has the additional benefit of actively promoting wound healing by drainage of infected fluid and induction of granulation tissue. It can be resource-intensive, however, requiring scheduled dressing changes in an endoscopic suite or operating room setting. In small cohorts, success in closing anastomotic leaks have been approximately 80% to 90%, with a stricture rate of approximately 5% to 10%.[33–35] Also, in small cohorts, vacuum therapy has been shown superior to stents.[36–38] Endoscopic vacuum therapy is a potential strategy to accelerate healing of leaks.

CONDUIT NECROSIS

The most devastating leak is that caused by conduit necrosis, where the blood supply to the conduit is inadvertently interrupted and the conduit no longer is viable. This usually is recognized by a critically ill patient in septic shock but occasionally can present with unexplained fever, tachycardia, and delirium similar to that of a leak. As with an anastomotic leak, high clinical suspicion is needed and mortality can reach 90% with this condition.[39] After urgent resuscitation and antibiotics, evaluation should proceed in the operating room. Diagnosis of the necrotic conduit should begin with endoscopy in the operating room. Resection of the conduit then should occur with formation of an end esophagostomy. The

longest possible length of residual esophagus should be preserved at this time to assist with future reconstruction and to better palliate the esophagostomy; an end esophagostomy situated on the chest can be hidden under clothes, and stoma appliances are secured more easily. A feeding enterotomy should be considered if not present and if the clinical status permits. After resolution of sepsis and optimization of nutrition, reconstruction can be performed. Conduit options after a necrotic gastric conduit include colon interposition and supercharged jejunal interposition in the substernal position.[40,41] In both cases, resection of the left hemimanubrium, clavicle, and first rib is needed to prevent obstruction and, when supercharging is required, to perform the vascular anastomoses.

CONDUIT-AIRWAY FISTULA

Another devastating complication that can occur in the setting of an anastomotic leak is a fistula between the anastomosis and the airway.[42] In the neck, the trachea is involved, and, in the chest, the trachea, carina, or either main bronchus could be involved. Airway injury during over-dissection may predispose a patient to such a fistula, but an undrained leak causing inflammation and digestion by gastric juices still may create an airway fistula. Stents used to treat leaks have eroded into the airway but may be a result of inadequate drainage rather than the expansile force of the stent.

Endoscopic approaches to repair these fistulae include the use of fibrin glue and Vicryl plugs, but, in 2 patients with airway-conduit fistulae, only 1 healed with this approach.[43] Clips to reapproximate such fistulae also have been attempted but, apart from a case report of a conduit to bronchial fistula, no large series of success have been reported.[44,45]

Another endoscopic strategy is the use of stents, esophageal, airway, or both to cover the defect. In a series of 6 patients by Schweigert and colleagues[46] with conduit-airway fistula, 2 underwent esophageal stenting, 1 underwent airway stenting, and 1 had both esophageal and airway stenting. Three of the 4 cases achieved definitive closure of the fistula after 6 weeks. The remainder of the patients were unable to undergo stenting due to an ischemic conduit and instead had resection of the conduit, repair and coverage of the airway defect with vascularized tissue, and end esophagostomy. Unfortunately, all died as a result of septic shock.

Endoscopic treatments appear to defy the surgical principles of fistula repair, that is, division of fistula, repair of both defects, and interposition of vascularized tissue. Small series, however, have demonstrated success in selected cases without conduit necrosis, likely reflecting the acute nature of the inflammatory process and lack of fistula epithelialization. Decision making during this complication is challenging. Source control is paramount, because ongoing soilage of the lungs and mediastinum propagates sepsis and obviates any chance for survival. Surgical source control by repairing the fistula during septic shock, however, is risky. Balakrishnan and colleagues[47] reviewed a single-center series of surgical management of conduit-airway fistula in 11 patients. Of the 3 deaths, 2 were performed in the early postoperative period (<2 weeks). In cases of conduit necrosis, the decision is clear that the dead viscera must be resected. In cases of leak, however, if rapid source control can be achieved with a stent, this may allow the patient to resolve the sepsis before definitive treatment. Moreover, as evidenced by some small series, closure of the fistula can sometimes occur simply with stenting.[48]

CHYLOTHORAX

Owing to the course of the thoracic duct close to the esophagus, inadvertent thoracic duct injury occurs approximately 4% of the time according to the ECCG, leading to chylothorax.[1] Although chyle generally is milky in color, the lack of fat intake in the early postoperative period can result in a clear liquid. Thus, chyle leak should be considered when large volumes of fluid are being drained in the early postoperative period. A pleural fluid triglyceride level greater than 110 mg/dL is chemical confirmation of the chylothorax.

Conservative management generally is attempted first in postoperative chylothorax. Principles include drainage to re-expand the lung and to assess daily volume of leakage, nutritional support, and medication to reduce chyle flow. In the postoperative esophagectomy population, feeding in the postoperative period involves feeding jejunostomy or total parenteral nutrition. Jejunal feeds should be switched to a high-protein, low-fat formulation with medium-chain triglycerides.[48] Avoidance of long-chain triglycerides avoids their breakdown into monoglycerides and free fatty acids that are carried as chylomicrons in chyle. Octreotide can be attempted to decrease the flow of chyle by decreasing foregut secretions.

For esophagectomy, early reintervention for thoracic duct ligation should be considered because it is most likely that the thoracic duct itself was injured rather than any peripheral branch and unlikely to resolve. In addition, the increased use of induction radiation reduces the ability of the lymphatic network to heal. Lagarde and colleagues[49] used a cutoff of 2 L after 2 days to indicate

surgery, and Reisenauer and colleagues[50] recommend surgery for an output of greater than 1.1 L over 24 hours at any point. Thus, if drainage does not fall below 1 L/24 hours within 1 day to 2 days of initiating conservative management, surgical intervention should be considered. The thoracic duct can carry up to 4 L of lymphocyte, lipid, and protein-rich chyle daily; thus, delay in sealing the chyle leak can result in malnutrition and immunodeficiency and affect healing of the anastomosis and delay postoperative recovery.

Surgical ligation of the duct requires rethoracotomy or thoracoscopy. To facilitate identification of the duct, a bolus of high-fat material, such as cream or oil, or through the jejunostomy tube no longer than 30 minutes to 60 minutes prior to operation can be helpful to make the chyle appear more opaque. Thoracic duct embolization is increasingly available as a nonsurgical option. Percutaneous embolization was successful in approximately of 80% of cases in small series.[51,52] Thoracic duct embolization was shown to be a useful technique after failed surgical thoracic duct ligation and identified aberrant duct anatomy, collaterals, or incomplete ligation causing failure.[53] In small series, lymphangiography alone also was shown to be successful.[54–56] With increasing experience, this may become the first line of treatment, but the complication profile remains to be fully defined. For now, in an otherwise fit patient, surgical treatment seems to be the best first option, but institutional expertise guides treatment.

Given the low risk of thoracic duct clipping, routine prophylactic duct ligation at the time of surgery may prevent the development of chylothorax. Guo and colleagues[57] performed a retrospective cohort study and showed that routine ligation of the duct reduced chylothorax rates from 10% to 1.5% in approximately 70 patients per group, and Dougenis and colleagues[58] reduced the rate from 9% to 2.1%. The approximately 10% rate is higher, however, than in other reported series. Lin and colleagues[59] compared routine ligation to selective ligation based on a bolus of olive oil given orally preoperatively and showed a reduction of chyle leak from 10% in the routinely ligated group to 0% in the selective group. The 10% leak rate in the routine ligation group seems high in contrast to the other studies. Reisenauer and colleagues[61] showed no difference (4% to 4%) but only a small number of patients underwent prophylactic ligation.

RECURRENT LARYNGEAL NERVE INJURY

The incidence of recurrent laryngeal nerve injury is 3.5%, according to the ECCG.[1] Due to its anatomic location, it is most at risk with a cervical anastomosis and with a 3-field lymph node dissection. Injury to the nerve can occur as a result of traction, overdissection, or transection. Although many injuries result in only transient dysfunction, the dysfunction is during the immediate postoperative period, when risk is highest. To prevent injury, Orringer and colleagues[60] recommend the "compulsive avoidance" of using metal instruments in the neck and suggest finger retraction and dissection of the esophagus as ideal. The symptoms of recurrent laryngeal nerve injury are due to lateral displacement of the affected vocal cord. Hoarseness is the primary symptom, but the voice also can be affected by ineffectively cleared secretions pooling in the pyriform sinus. This difficulty in clearing secretions also predisposes to aspiration of food and saliva, leading to pneumonia. Finally, the inability to close the glottis disrupts the Valsalva mechanism and makes straining difficult.

Upon discovery of a hoarse voice postoperatively, speech and language pathology should be involved to evaluate swallowing as soon as a patient is cleared for diet from the esophagectomy perspective. Maneuvers, such as supraglottic swallowing, may help minimize aspiration during swallowing.[62] Treatment of the dysfunction requires the assistance of otolaryngology. Due to the often transient nature of the nerve dysfunction, the first intervention usually is an injection augmentation of the affected cord to move it into the midline.[63] This injection is temporary but mitigates dysphonia and dysphagia in the short term to await return of function of the recurrent laryngeal nerve. After 6 months, if there is no recovery, medialization laryngoplasty can be performed for more permanent medialization of the cord.[64]

SUMMARY

Esophagectomy, in all its varieties, remains a complex operation with high potential for postoperative complications. Understanding the causes of complications allows for both prevention and salvage of the patient when it does occur.

DISCLOSURE

The author has nothing to disclose.

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