Managing complications I: leaks, strictures, emptying, reflux, chylothorax

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Abstract: Esophagectomy can be used to treat several esophageal diseases; it is most commonly used for treatment of esophageal cancer. Esophagectomy is a major procedure that may result in various complications. This article reviews only the important complications resulting from esophageal resection, which are anastomotic complications after esophageal reconstruction (leakage and stricture), delayed emptying or dumping syndrome, reflux, and chylothorax.

Keywords: Complications; leaks; strictures emptying; reflux; chylothorax

Introduction

Esophagectomy can be used to treat several esophageal diseases; it is most commonly used for treatment of esophageal cancer. Esophagectomy is a major procedure that may result in various complications. This article reviews only the important complications resulting from esophageal resection, which are anastomotic complications after esophageal reconstruction (leakage and stricture), delayed emptying or dumping syndrome, reflux, and chylothorax.

Anastomotic leaks

Overview

The incidence of anastomotic complications, clinical manifestations, and treatment are all related to the methods of esophageal resection and reconstruction; therefore, we first briefly introduce esophagectomy. The history of esophagectomy is less than 100 years, and esophageal reconstruction after resection was first documented in 1942 (1,2). To date, the common surgical approaches are the Ivor-Lewis esophagectomy (3); McKeown esophagectomy (4); Sweet procedure (single left-sided thoractomy), left thoracoabdominal approach, and transhiatal esophagectomy (THE) (5). The most common substitute organs are the stomach (>90% of the cases) (6), colon (when the stomach is resected or diseased), small intestine (microvascular surgery is required) (7), and pedicled skin-muscle flaps. Anastomosis methods include hand-sewn anastomoses (continuous and interrupted sutures, single or double layer sutures, absorbable or non-absorbable stitches), stapling (circular and linear), and combined hand-sewn and stapled anastomoses (8,9).

Since the advent of esophagectomy, anastomotic complications—especially for the prevention and treatment of anastomotic leaks—have been an important issue involving many factors. In general, thoracic surgeons should have the following three qualities in order to avoid fatal anastomotic leaks: "flexibility" between different surgical techniques; "knowledge" of the esophageal anatomy, physiology, and esophageal diseases; and "skill" in the esophageal surgical techniques. That is, the surgeon should have a personalized "flexible" selection of methods for esophageal resection and mode of reconstruction based on the characteristics of specific patients and tumors. Familiarity with every type and flexibility between different types of surgical approaches are important for good clinical

outcomes. Considering esophagectomy, no specific surgical approach is suitable for all patients at present. Experienced surgeons in large centers for esophagectomy play an important role in the success of the surgery. Mastering the knowledge about various complications is not only essential for the prevention of anastomotic complications, but it also plays a significant role in selecting the appropriate treatment. Recognizing early signs of complications and initiating appropriate treatments are important components in reducing the serious consequences of complications. Since the advent of esophagectomy 60 years ago, there has been a significant progress in the prevention and treatment of anastomotic leaks; however, continuous effort for long-term improvement is necessary. The emphasis on the details of the appropriate treatment has always been important among the various factors in the prevention and treatment of anastomotic leaks.

The importance of and susceptibility to esophagogastric anastomotic leaks

Esophageal reconstruction mostly involves the stomach (>90% of cases); therefore, we focus on esophagogastric anastomotic leaks. The esophagus is connected to the hypopharynx at the top, descends from the posterior mediastinum to the abdominal cavity, and connects to the stomach. It traverses three anatomical sites—the neck, thorax, and abdomen—and is mostly located within the thorax. Esophagogastric anastomotic leaks often involve pleural contamination, infection, and obstructions that affect the negative intrathoracic pressure, leading to respiratory problems and unstable hemodynamics, which are different from the characteristics of systemic inflammatory response syndrome caused by other gastrointestinal leaks. The clinical presentations are quite serious, and often fatal; therefore, esophagogastric anastomotic leaks are more important than other types of gastrointestinal leaks.

Compared with other digestive organs, the esophagus does not have a serosa and mainly comprises longitudinal muscle; therefore, it has insufficient suture strength and demonstrates poor healing. The blood supply to the esophagus is segmental with communicating branches that extend over long distances, which can easily lead to ischemic necrosis around the esophageal anastomotic site. The anastomosis is directly located in or in close proximity to the thorax that has a negative pressure, which causes gastric fluid to be easily drawn through anastomotic sutures or staples lines, resulting in leaks after surgery. When

the stomach is elevated from the abdominal region to the thoracic cavity, the blood supply to the stomach depends completely on the right gastroepiploic artery; this easily leads to ischemia of the stomach around the anastomosis after surgery. The aforementioned reasons can lead to the incidence of anastomotic leaks after surgery.

Diagnosis of anastomotic leaks

For a long time, the definition of anastomotic leaks has not been very clear. The definition of anastomotic leaks included the outflow of gastric fluid from chest tubes, pleural or mediastinal infections, or outflow of saliva from the neck, which are obvious "leaks", as well as leaks without the presence of any clinical symptoms but with only occult leaks observed with esophagography (10,11). The concept of anastomotic leaks has become clearer after Lerut graded it in 2002 (12). However, we believe that conduit necrosis (Lerut Grade IV) should not be classified as an anastomotic leak, but should be considered a separate terminology because this condition, in order to save lives, requires emergency debridement, exteriorization of the cervical esophagus, and repositioning of the residual vital gastric conduit back to the peritoneal cavity. After the condition improves, colon interposition may be performed via retrosternal approach. Owing to the different definitions and grading systems, the comparability between studies on anastomotic leaks is poor. Therefore, clear diagnostic criteria are the bases for the comparisons between studies, as well as the guidelines for treatments. Obvious clinical presentations, such as gastric fluid drawn from the thorax or saliva drawn from the neck, can help to provide a clear diagnosis and treatment. In addition, esophageal imaging by using orally administered Gastrografin is the gold standard for the diagnosis of anastomotic leaks.

Prevention of anastomotic leaks

The prevention and treatment of anastomotic leaks are closely related, where prevention is more important than cure. The esophagus does not have a serosa, is mainly composed of longitudinal muscle, and has a segmental blood supply from a few anastomotic branches. Therefore, both hand-sewn and staple sutures require gentle maneuvers during operation to avoid tears and damage. It is easier for anastomosis when the length of the free esophagus is more; however, a longer isolated esophagus is more prone to ischemia, and therefore, the good blood supply and the

ease of operation need to be balanced. The gastric conduit is supplied blood only via the right gastroepiploic artery, which provides blood to the 60% of the stomach that is proximal to the pyloric end, whereas the remaining 40% of the stomach that is distal to the pyloric end depends on the supply from the submucosal network of small vessels. The stomach is tailored along the greater curvature, forming a 4 cm-wide neo-esophagus, therefore, the gastric conduit should be extended to obtain a "tubular shape stomach", and the distal fundus that has poor circulation should be removed, which allows the anastomosis to be moved closer to the start of the right gastroepiploic artery.

The more area that is responsible for acid secretion is removed, the lower risk of anastomotic leakage will be encountered. The degree of pathophysiologic changes in consequence of intrathoracic anastomotic leaks is positively correlated with the amount of gastric fluid entering the thorax through anastomosis. The "tubular shape stomach" has only half of the gastric tissue, and the rest has been removed; therefore, it has less area for secreting acid. The decrease in the secretion of acidic gastric fluid is another factor that may reduce the extent of clinical damage after the occurrence of anastomotic leaks.

A "tubular shape stomach" results in the same length of both lesser curvature and greater curvature of the reconstructed stomach; this allows the positioning of the anastomosis and pylorus in a straight line, which solves the anatomical and mechanical problems of gastric retention and emptying, and thus also reduces the risk of anastomotic leaks.

Our usual practice is to simultaneously elevate the gastric conduit and the drainage tube from the abdominal cavity to the location of the anastomosis. The drainage tube starts from the abdominal wall to fully achieve mediastinal drainage and reduce the risk of infection caused by mediastinal exudate; this positioning helps to decrease the risk of anastomotic leaks from the thoracic stomach.

After surgery for esophageal cancer, jejunostomy is performed or a nasoduodenal feeding tube is routinely place to ensure enteral nutrition. Studies have shown that small intestinal function recovers within 12 hours post-operation; therefore, enteral nutrition administration may start within 24 hours post-operation. The advantages of early enteral nutrition are as follows: promote the recovery of gastrointestinal function and downward movement of intestinal contents, protect the mucosal barrier, prevent the translocation of bacteria, balance metabolic stress, and promote anastomotic healing.

Gentle maneuvers should be performed always when the stomach is being mobilized, and excessive kneading of the fundus should be avoided to prevent direct damage and venous thrombosis. Pulling up the gastric conduit should be carefully performed while paying careful attention to the direction of movement for preventing intrathoracic gastric volvulus or twisting of the blood vessels. The presence of tightness in the thoracic inlet should be assessed. If it is very tight, the sternum ligaments should be cut, and the clavicular head or sternoclavicular joint should be resected when necessary.

Although it is debatable, some surgeons insist on routinely performing pyloroplasty or pyloric myotomy. Of course, the patient's comorbidities such as diabetes mellitus, malnutrition, and atherosclerosis should also be noted before the operation. Additionally, strengthening perioperative management and performing early postoperative lung expansion, as well as preventing hypoxemia and hypotension, are all important measures to reduce the risk of anastomotic leak.

Treatment for anastomotic leaks

The aforementioned preventive measures can largely reduce the incidence of severe anastomotic leaks. No special treatments are necessary for occult anastomotic leaks that occur in patients who do not show any clinical symptoms and that are only discovered by using upper gastrointestinal contrast radiography; these occult anastomotic leaks can be cured by delaying the time to oral food intake. For small leaks in stable patients, food and water intake should be restricted, and enteral or/and parenteral nutrition support should be strengthened. In addition, if there are signs of infection, a broad-spectrum antibiotic therapy should be administered, as well as somatostatin treatment to reduce the secretion of gastric acid, and proton pump inhibitors to reduce the production of gastric acid. When thoracic fistula occurs and the encapsulated fluid exhibits empyema around the intrathoracic anastomosis, adequate drainage can be achieved by using a CT-guided thoracocentesis.

For treating abscesses formed around the wound in the neck, adequate drainage can be achieved by opening the incision, if necessary, at the bedside. Further treatment measures depend on the location of the anastomosis and the surrounding fluid. For example, patients with severe intrathoracic anastomotic leakage (Lerut Grade III) and gastric tube necrosis (Lerut Grade IV) have perioperative mortality rates of approximately 60% and 90%,

respectively; patients with gastric tube necrosis often require a second surgery for removing the necrotic tissues and retracting the distal stomach back to the abdominal cavity after excluding the proximal esophagus to the neck. Adequate surgical drainage from the mediastinum and sincere collaborations in the multidisciplinary supported treatment post-operation are important for success. Crestanello *et al.* (13) reported the experience of early surgical intervention for postoperative anastomotic leaks after treatment for esophageal cancer at the Mayo Clinic for a span of ten years. Approximately 70% of the patients who required a second surgical treatment underwent direct fistula repair, whereas the remaining patients underwent traditional esophageal diversion. The mortality rate for the patients who underwent direct fistula repair was approximately 15%.

Anastomotic stricture

Anastomotic stricture causes dysphagia; however, postoperative dysphagia may not necessarily result from anastomotic stricture. Theoretically, dysphagia can be classified into anastomotic stricture-induced dysphagia and functional dysphagia. In addition, anastomotic stricture can be classified into scar contracture and anastomotic leakage-induced stricture. Functional dysphagia may be explained as described below. Firstly, subtotal esophageal resection, especially cervical anastomosis where the remnant esophagus is extremely short at the location of the esophagogastric anastomosis, can result in insufficient muscle strength during swallowing. Neck incisions can cause damage to the muscles in the neck, which leads to weakening of the accessory swallowing muscles. Age and malnutrition can lead to weakness of muscles in the tongue. Denervation and devascularization of the substituted esophagus can cause delayed gastric emptying. All of the above can lead to functional dysphagia.

The use of endoscopy and upper gastrointestinal radiography at this point will not reveal any objective evidence of anastomotic stricture. Results of our prospective database survey showed that >10% of the patients exhibited various degrees of dysphagia within one year after the surgery; however, examination findings confirming the presence of anastomotic stricture that requiring dilatation only accounted for <1% (unpublished data). Dysphagia in the rest of the patients either reduced or disappeared over time. Several studies have suggested that anastomotic stricture spontaneously disappears in a few years without

the need for dilatation (14). Early benign stricture can be classified into scar contracture and fistula induced stricture. The former can be easily cured by dilatation, whereas dilatation for the latter in serious conditions is sometime counterproductive. The stricture is often exacerbated by dilatation related injuries. Therefore, careful acquisition of the patient's medical information, especially through UGI endoscopy and radiography, is required to distinguish between functional dysphagia, scar contracture, and a true case of fistula induced stricture. The treatment for functional stricture should mainly focus on enhancing enteral nutrition and swallowing training, and not on rash offer a dilatation.

Early anastomotic stricture after the operation is mostly benign, whereas attention should be paid to late anastomotic stricture, which may be related to tumor recurrence. Endoscopy and PET/CT examinations are effective approaches to identify the benign and malignant stricture. The esophagus is a muscular tube with elastic walls. In the resting state, the esophageal lumen is in a collapsed state, whereas during the swallowing process, the muscles of the esophagus relax to accommodate the food bolus (anterior-posterior diameter, up to 2 cm; lateral diameter, up to 3 cm) (15). However, esophagogastric anastomotic healing is a scar-healing process resulting in an inelastic anastomosis and a fixed-size scar around the anastomosis; the size of the anastomosis varies depending on the diameter of the stapler, the anastomotic method, and an individual's degree of scar retraction, but remains fixed and inelastic. Therefore, patients who consume food that forms a bolus exceeding the diameter of the anastomosis or who do not have the strength to swallow may exhibit symptoms of dysphagia. Thus, either hand-sewn anastomosis (continuous or interrupted, single or multiple layers), staple anastomosis (circular or linear), or combined hand-sewn and staple anastomosis—either end-to-end, end-to-side, or side-to-side anastomosis—may cause dysphagia or even true anastomotic stricture owing to anastomotic scar formation, which requires detailed examination to distinguish between them.

Most anastomotic stricture results from anastomotic leaks; therefore, they have similar factors which cause. At present, the clinical definition and classification of anastomotic stricture are not clear. Additionally, a diagnosis of anastomotic stricture is often based on the symptoms of subjective dysphagia; therefore, the reported incidence rates greatly vary between 10-40% (9). However, the majority of scholars believe that the incidence rate for anastomotic

stricture is high, which renders it as one of the most common complications after esophagectomy.

Delayed gastric emptying, dumping syndrome, and reflux

Long-term survival, operative mortality rate, and complication incidence are generally quality indicators for esophgectomy. In recent years, postoperative long-term quality of life has received increasing attention, and has become an important component in assessing esophagectomy. The stomach is the first choice substitution of esophagus for most surgeons. However, compared to the normal stomach, the stomach elevated into the thorax induces many anatomical and physiological changes, which results in a series of clinical manifestations. Meanwhile, the shape and size of the remodeled stomach, its pulling up pathway, and its status in the thorax greatly affect the incidence and severity of the symptoms, which manifests as abnormal motility of the thoracic stomach as well as common pathophysiological changes including delayed gastric emptying, dumping syndrome, and reflux.

Anatomical and physiological bases for motility defects of the substituted esophagus

Blood supply to the stomach is provided by six vessels, which are the left gastric, right gastric, left gastroepiploic, right gastroepiploic, short gastric, and posterior gastric arteries. These blood vessels not only provide nutrients to the stomach but also fix the stomach in the upper abdomen, in order to allow the stomach to perform its normal function. However, the free thoracic stomach has only the right gastroepiploic artery for blood supply (15).

Esophageal resection typically involves the complete removal of the vagus nerve for malignant lesions; therefore, innervation of the thoracic stomach solely depends on autonomic innervation by the gastric myenteric plexus, which greatly affects the regular relaxation function of the pyloric sphincter In addition since the stomach is elevated from the positively pressured abdomen to the negatively pressured thorax, which can further impede gastric emptying. During gastric mobilization and pulling up, the angle of His disappears and the spring action of the diaphragm on the lower esophagus is lost, which causes the loss of the anti-reflux mechanism at the gastroesophageal junction. Thus, compared with normal esophagus, the emptying and acid clearance rates after esophagectomy are impaired. Owing to the extremely shortened esophagus that remains, the weakened swallowing ability, and the loss of the anti-reflux mechanism, the acid clearance capacity of the remnant esophagus is reduced.

Resection of the fundus results in changes in the receptive expansion and volume of the stomach when shaping the new esophagus; the pressure inside the thoracic stomach is very easily affected by the amount of stomach content.

Gastric secretion changes affect pyloric opening and closure as well as gastric emptying when shaping the new esophagus.

As mentioned above, changes in the physiological function ultimately lead to abnormal motility of the substituted esophagus. The clinical presentations are delayed emptying, dumping syndrome, and reflux, of which delayed emptying and reflux are most prevalent (16-18).

Prevention and treatment

Delayed gastric emptying

Delayed gastric emptying is the most common problem in patients with motility dysfunction of the thoracic stomach, with an incidence of 50% after esophagectomy reported in the literature. The basis of gastric emptying is that the pylorus opens to empty food when the pressure in the stomach exceeds pyloric pressure. Reduced stomach volume, weakened receptive expansion, and vagotomy cause pylorus dysfunction, which leads to delayed emptying that manifests as early satiety or vomiting. Results of earlier studies suggest that the thoracic stomach does not exhibit contractility (16,17), and the food passes relying solely on gravity. However, recent studies have revealed that the myenteric plexus and the remnant of the vagus nerve in the antrum of the lesser curvature can gradually become the center of gastric motility (18). Through muscle and topical hormone coordination, the contractility of the thoracic stomach can be restored to some extent. Therefore, some people do not favor excessive removal of the tissues near the antrum of the lesser curvature, and hope that the remnant nerve can exert its function. When early emptying is delayed, enteral nutrition supply should be administered, and most patients recover over time.

Pyloric drainage procedures such as myotomy, pyloroplasty, or pyloric balloon dilatation can be performed during the resection. This operation is inspired by vagotomy for ulcers; however, esophagectomy is different, which leads to controversy. However, some surgeons would

argue that these pyloric drainage procedures may pose the risks of fistula, bile reflux and dumping syndrome. However, it is currently believed that regardless of the pyloric draining procedure, pyloric balloon angioplasty is still effective once delayed emptying occurs.

The whole stomach pulled up as a neo-esophagus includes three types, i.e., full stomach, subtotal stomach, and narrow gastric tubes. Each has its own advantages and disadvantages. As of now, narrow gastric tubes have demonstrated more advantages. The use of the whole stomach as a substitute helps to recover gastric motility; however, because of large gastric volume and receptive expansion, the pressure inside the stomach does not easily reach above that of the pylorus, which results in gastric dilatation, as well as retention, reflux, and delayed emptying. In addition, owing to the excessive length and lack of innervation, the low-tension greater curvature of the stomach is prone to gastroptosis, causing the pyloric opening to be higher than the lowest point of the stomach. The presence of the pyloric opening at a higher point or even the presence of an acute angle between the axis of the stomach and the pylorus, both lead to delayed thoracic stomach emptying. The shaping of a narrow gastric tube results in the lesser and greater curvatures to be of the same length, the anastomosis and pylorus to be positioned along a straight line, and limited gastric expansion; all of these can cause an increase in the pressure in the stomach, which is easier than that of the pylorus, thus triggering gastric emptying. The pathways for gastric pulling up include those along the posterior mediastinum, retrosternal as well as anterior sterna subcutaneously. Studies have shown that the pathway along the posterior mediastinum has a minimal impact on emptying. Meanwhile, compared to narrow gastric tubes, whole stomach pulling up can easily cause axial torsion or even folds, resulting in delayed emptying. Strengthening postoperative management and keeping the stomach empty will benefit the positioning of the tubular stomach at the posterior mediastinum, thus ensuring good emptying.

Delayed gastric emptying might be treated with the appropriate medication. Currently used drugs for gastric motility include metoclopramide, cisapride, bethanechol, and domperidone, all of which have been shown to alleviate symptoms of delayed gastric emptying. However, they cause significant adverse effects. Erythromycin is a motilin receptor agonist, which induces migrating motor complexes by stimulating the motilin receptors enriched in the gastric antrum and duodenal smooth muscle, thus promoting pyloric motility and gastric emptying. In addition, erythromycin is more suitable for the treatment of endogenous paralysis or paralysis caused by vagotomy, with rare or minimal adverse effects. Erythromycin is routinely used after esophageal resection to improve gastric emptying.

Dumping syndrome

Dumping syndrome is a common clinical complication of abnormal motility of the thoracic stomach. However, only 5% of patients show symptoms with moderate severity, and 1% show very severe symptoms. The causes of dumping syndrome are the same as that of delayed gastric emptying; the causes include devascularization, denervation, abnormal pyloric function, and decreased gastric capacity, all of which results in rapid emptying. It is currently believed that rapid entry of hypertonic food into the intestine results in the movement of parenteral fluid into the intestine. However, the detailed mechanism is not fully understood, but may be related to gastrointestinal hormones. The clinical manifestations are gastrointestinal symptoms (diarrhea, bloating, etc.) and/or hypovolemic symptoms.

Most symptoms of dumping syndrome can be alleviated by modifying eating habits and styles; these modifications include eating multiple small meals (at least 6 meals/24 h), avoiding drinking more fluid immediately after a meal, avoiding eating foods containing monosaccharides (sugar, cookies, sweets), replacing foods containing monosaccharides with those containing polysaccharide (such as fruits, pasta, potatoes and other grains), avoiding dairy products, and appropriately increasing the proportions of fat and protein. In severe cases, drugs including propranolol, verapamil, prednisolone, methysergide maleate, acarbose, and octreotide can be administered. Avoiding damage to the vagus nerve has been proposed as a preventive measure; however, it is only possible during the treatment for early-stage cancer of the gastroesophageal junction or Barrett esophagus, and is not suitable during surgery for locoregional advanced squamous cell carcinoma.

Reflux

Damage to the anti-reflux mechanism at the gastroesophageal junction, diaphragm, and angle of His is the cause of reflux. In addition, partial localization of the stomach in the positively pressured abdominal cavity, delayed gastric emptying, and denervation can all aggravate reflux. Clinical manifestations include bile-and gastric acid-induced laryngitis, vomiting, repeated coughing, pneumonia, and inability to lie in a supine position. The affecting

factors include low anastomosis (below the aortic arch), substitution of the whole stomach, and partial localization of the stomach in the abdomen. Preventive methods that help alleviate reflux include sufficient mobilization of the stomach (allowing it to be localized in the thorax during pulling up), placement of the anastomosis at a high position, adequate shaping of the tubular stomach, resection of most of the area secreting acid, performing operations that enhance emptying, changing eating habits (including having multiple small meals), and avoiding lying in the supine position immediately after meals and walking. Many researchers have performed experimental operations on patients with anastomosis to imitate anti-reflux function, such as fundoplication; however, the expected results have not been achieved, and therefore, these operations have not been widely accepted.

Chylothorax after surgery for esophageal cancer

Chylothorax after surgery for esophageal cancer is a rare complication with an incidence rate of approximately 2.7-3.8% (19,20). However, improper handling can lead to fatality, which is caused by the damage to thoracic ducts and/or its branches. Chylothorax is defined as the leakage of lymphatic fluid (containing lymphocytes, immunoglobulins, and various biological enzymes) that is enriched with chylomicrons and lipids (including lipid-soluble vitamins, chylomicrons, and triglycerides) into the thorax (21). Chyle does not contain fibrinogen; therefore, unlike blood, the damaged thoracic tube cannot heal on its own. Persistent loss of chyle causes decrease in the lymphocyte numbers, nutritional deficiencies, reduced immunity, and eventually leads to systemic infections. If large amounts of chyle leakage (daily chylous drainage >1,000 mL) persists, a decrease in the lymphocyte number should be expected.

Diagnosing a case of chylothorax requires a combination of clinical presentations and the results of laboratory tests and imaging examinations. The clinical presentations include early, postoperative, and unexplained drainage of large amounts of a brownish or beige fluid into the thorax; rapid emergence of contralateral pleural effusion; or drainage of a white milky fluid in the pleura of patients with restored enteral nutrition (22). Drainage of a fluid with triglyceride concentrations >110 mg/dL into the thorax can be diagnosed as a case of chylothorax. In addition, the lymphocyte concentration in the chyle is significantly higher than that in the peripheral blood (the percentage of lymphocytes amongst total white blood cells usually exceeds

90%) (23). Lymphaticangiography can help to diagnose thoracic duct injury and its severity (24), with a diagnostic accuracy of up to 81% (25).

Conservative treatment is often used as the first-line treatment for chylothorax, although the success rate is relatively low. Adequate surgical drainage and promoting lung re-expansion can help in the adhesion and final closure of the trauma in the thoracic duct. Nutritional modification may improve the resolution of chyolothorax: medium-chain fatty acids may be administered, or enteral nutrition may be temporarily replaced with parenteral nutrition support. Somatostatin inhibits the secretion of intestinal fluid and the activity of various enzymes; therefore, it can reduce chylous drainage to some extent (26). Etilefrine is a sympathomimetic drug, which helps to treat postoperative chylous leakage by stimulating smooth muscle contraction in the thoracic duct (27). After conservative treatment, if the daily volume of chest drainage is <200-300 mL, then it suggests that the condition has been effectively controlled, and normal enteral nutrition supply or a high-fat diet can be considered. If no further increase in the daily volume of chest drainage (<450 mL) is observed, closed thoracic drainage may be stopped (23).

If chylothorax persists after conservative treatment, a secondary surgery is required; these include video-assisted thoracoscopic surgery and traditional thoracotomy, as well as laparotomy and horizontal ligation of the thoracic duct at the crura of the diaphragm. The principles for the secondary surgery are as follows: (I) confirm the location of the damage to the thoracic tube and ligated; (II) use methods similar to pleurodesis to cause the lung to adhere to the parietal pleura for obliterating residual pleural space; and (III) address the problems of co-existing morbidities, such as empyema and anastomotic leakage (25). Tube feeding of high-fat enteral nutrients 30 minutes before surgery can help to determine the sites of intraoperative injury in the thoracic duct. Treatments for damage include ligation with a vascular clip, or use of ultrasonic scalpel and ligation after solidification, suture with hemostatic gauze, ligation with fibrin glue, use of tetracycline or talc pleurodesis, and postoperative radiotherapy. The thoracic duct is one of the main branches of the lymphatic system. It originates in the abdominal cavity (behind the second and third lumbar vertebrae) and the cisterna chyli (between the aorta and right crura of the diaphragm), travels along the right side of the vertebral body, enters the thorax through the aortic hiatus in the diaphragm, moves horizontally to the left at the fifth thoracic vertebra, continues up to

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the neck, and finally reaches the junction where the left internal jugular vein and subclavian vein converge. Clinical data show that the aforementioned route occurs only in 55% of cases, whereas other abnormal routes occur in the remaining 45%.

Summary

Since the beginning, esophageal resection and reconstruction has always been a thoracic surgery with complex operations and various postoperative complications. Mastering the causes of postoperative complications and principles for treatment is the cornerstone for improving the efficacy of surgery for esophageal cancer. These concepts should not be limited to thoracic surgeons, but should include anesthetists, intensive care physicians, and nurses who are also involved in the treatment. The goals of prolonging lives and improving the postoperative quality of life cannot be reached without sincere cooperation and elimination of the various associated dangers.

Acknowledgements

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