

Gastric Conduit Necrosis following esophageal reconstruction

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Abstract— *The successful completion of esophageal reconstruction after esophagectomy is a great challenge in esophageal surgery. The gastric reconstruction is widely employed in benign and malignant esophageal disease. Compared to other digestive organs, stomach has a good blood supply and gastric procedure requires a single reconstructive anastomosis. The gastric tube is a durable graft, retaining its size, shape, and function over time. Therefore the stomach has become the most commonly adopted graft to replace the resected esophagus. The viability and function of the graft are the most two important factors affecting postoperative surgical outcome and functional results. Gastric graft necrosis is the most disastrous complication that can lead to leakage, sepsis, multi system failure and death. Therefore, knowledge of risk factors, diagnosis, management, and prevention of graft necrosis is key to understand and to successfully perform an esophageal reconstructive surgery.*

Keywords— *Esophageal Reconstruction, Gastric Graft, Necrosis.*

I. INTRODUCTION

The selection and the preparation of the digestive graft and completion of the esophageal anastomosis constitute the greatest challenge during esophageal reconstruction after esophagectomy or bypass. Stomach is the first digestive organ used as an esophageal substitute after esophagectomy. The blood perfusion of the graft is essentially assured by the right gastroepiploic artery arcade. The viability and function of the graft are the most two important factors affecting postoperative surgical outcome and functional results. Gastric graft necrosis is the most disastrous complication that can lead to leakage, sepsis, multi system failure and death. Therefore, knowledge of risk factors, diagnosis, management, and prevention of graft necrosis is key to understand and to successfully perform an esophageal reconstructive surgery.

Stomach graft features

The gastric reconstruction is widely employed after oesophagectomy and the stomach has become the most commonly adopted graft to replace the resected esophagus. Firstly, the stomach has a blood supply from vessels that are of good size and demonstrate little anatomic variability. In other hand, the stomach is very easy to mobilize and gastric procedure requires less time to be achieved with the need of an only single reconstructive anastomosis (esophagogastric). Properly mobilized, the stomach can be used for both partial and total esophageal reconstruction. It is a durable graft, retaining its size, shape, and function over time. Gastric reconstruction is now accepted and widely used as a reconstructive surgical procedure for benign and malignant esophageal disease. However stomach has the disadvantages of long term gastroesophageal reflux which can lead to complications such esophageal ulceration and anastomotic stenosis [1]. In order to minimize the postoperative reflux and regurgitation, it is highly recommended to perform either a high thoracic or cervical anastomosis in benign conditions. During massive caustic injury, the stomach is often injured which compromises its use as an esophageal substitute.

Surgical technique of graft creation

The technique of creation of the gastric tube for esophageal replacement is closely linked to the subsequent development of ischemia and necrosis. So there are several factors to be considered when mobilizing and creating the future gastric tube [2]:

- a) Maintaining an adequate blood supply to the future graft particularly the cranial part (fundal tip)
- b) Achieving an adequate surgical margin if the resection is performed for carcinoma
- c) Preserving the fundus so that the stomach maintains its full length
- d) Creating a gastric tube of an adequate shape, diameter and length that will empty and function as an esophageal substitute and be easily pulled up through either the upper mediastina space or substernal space when cervical anastomosis is planned. However and as generally agreed,

the creation of an optimal graft is the most important factor. On the other hand, there is not agreement on the specific technique to be used to create an optimal gastric graft. Overwhelming evidence indicates that the right gastroepiploic arteriovenous arcade is sufficient to permit gastric mobilization without ischemic complications [3]. The right gastric artery and vein are small and are divided routinely allowing a complete Kocher maneuver to lengthen the gastric graft maximally. As reported by Liebermann-Meffert and colleagues [4], the contribution of the right gastric artery is negligible and a viable greater curvature-based gastric tubes can be based solely on the right gastroepiploic artery and vein. Furthermore, it is well documented that 60% of the blood supply to these gastric tubes comes directly from tributaries of the right gastroepiploic artery, 20% comes through collaterals of the left gastroepiploic artery, and 20% is supplied to the fundal tip through mucosal and submucosal microvascular collaterals [4]. Controversially, other authors emphasize the importance of the lesser curvature vessels in the blood supply to the fundus. So the fundus is supplied mainly through intramural vascular anastomotic system along the lesser curvature rather than through the greater curvature segment. So the tip end of the conduit where the arcades end and the short arteries begin, is the most vulnerable region for ischemia and necrosis because blood supply to this cranial part of graft is based lenoly lonely on the intragastric collateral flow and microvascula perfusion. Lindecken and Vogel [5] demonstrated the significant contribution of the lesser curvature vascular arcade to the total gastric blood supply and therefore recommended the preservation of this arcade. Likewise, Collard et al. [6] recommended the preservation of the lesser curvature blood vessels by using the whole stomach for reconstruction. Blood supply to the tip of the gastric tube has been identified as a main issue in esophageal reconstruction. The preservation of the blood supply at the lesser curvature results in a better perfusion at the tip of the fundus rotation gastroplasty (FRG) compared to the conventional Kirschner-Akiyama gastric tube [7,8]. An increase in tube length of sup to 30% adds to the improved arterial blood perfusion of the FRG [7,8]. The length of esophageal reconstruction is a paramount parameter and thus the length of the gastric graft is a risk factor in esophageal reconstructive surgery. The fundus rotation gastroplasty (FRG) is associated with increased length tube and improved blood supply to the cranial part of gastric graft [7]. Therefore this reconstruction procedure was preferentially applied in patients with a cervical or high

intrathoracic esophagogastric anastomosis. There is disagreement as to the optimal width of the gastric tube. Collard and colleagues [9] have advocated a conservative approach to gastric tailoring to optimize submucosal blood flow to the fundal tip. Pierie and colleagues [10] have demonstrated that the too narrow gastric tube results clearly in fundal tip necrosis. As suggested by authors [4,10], the ideal width of gastric graft is 4 to 5cm of diameter. A gastric tube of that diameter fulfils all of theoretical factors needed to achieve the ideal gastric graft. The posterior mediastinum is the shortest route and offers the most natural alignment of the gastric graft and thus is the preferred route among intrapleural and substernal routes [11]. With the substernal route, the manubrium and left clavicular head can compress the gastric graft, compromising blood circulation leading to anastomotic leakage and graft necrosis. So authors advocated to enlarge the thoracic inlet by removing the left half of manubrium and internal third of left clavicle to ensure there is no compression on the graft at the cervical level [12-16].

Incidence of conduit necrosis

The ischemia of gastric graft seems to be largely arterial and a separate clinical entity of ischemia caused by venous obstruction has not been described. The clinical range of gastric graft ischemia is broad and includes subclinical cases that resolve without need to re-intervention, ischemic-related anastomotic leak or stricture, and frank graft necrosis. Regardless of proximal site of esophageal anastomosis (intrathoracic or cervical), there is no difference graft ischemia rate in short versus long-segment grafting. So the length of gastric graft has not an independent effect on the ischemia graft rate. As tested by a new available methods, a properly mobilized gastric graft, even one that seems to be healthy and viable, has a reduced blood flow compared to pre-mobilized stomach. This effect is more pronounced at the fundal tip, then less so in a graded fashion back toward the pylorus. This decrease in gastric graft blood flow is transient and is not apparently important.

As thought, Gastric graft ischemia increases the risk of both anastomotic leak and stricture. The increase in anastomotic leak and stricture rates of cervical anastomosis seems to support this association. Many factors contribute to the occurrence of leak and stricture. However the principal factor is the quality of blood supply to the cranial part of the graft (fundal tip). When pulling up of the gastric graft to the neck, the greater is the negative effect of gravity and compression on blood flow into and through the

stomach graft. The cervical leak rate rises dramatically when using the substernal route [17]. As demonstrated by authors, the substernal route is the longest route with potential risk of graft compression at the level of thoracic inlet and authors suggested to enlarge the thoracic inlet during substernal esophageal reconstruction [12-16]. As previously reported by authors, postoperative anastomotic dilatation was needed in 26 % of patients undergoing esophagectomy with cervical anastomosis [17]. In contrast, intrathoracic esophageal anastomotic strictures are uncommon. As reported by Orringer and colleagues in a large series of esophageal reconstruction using gastric tube, the rate of graft ischemia was 2.6% [18]. External compression of the right gastroepiploic arcade secondary to a tight diaphragmatic hiatus was the cause of necrosis in only one case. The authors concluded that the graft necrosis resulted as consequences of stomach mobilization and graft preparation. This series remains one of benchmark studies on transhiatal esophagectomy with gastric tube reconstruction. Peracchia and colleagues reported a rate of 3 (1.2%) of graft necrosis [19] and patients who developed necrosis had a peptic ulcer disease diagnosed preoperatively. The microcirculation of the stomach wall may be altered in the presence of peptic ulcer and authors suggested to find an alternative option in patient who had a peptic ulcer disease. Annettoni and colleagues reported a necrosis rate of (0.7%) in a large series of 850 gastric reconstruction after transhiatal esophagectomy for both malignant and benign conditions [20]. The necrosis was located to the gastric graft tip in six patients. Hypovolemic hypotension with subsequent tissue hypoperfusion was observed in three of patients who had a graft necrosis. Furthermore the routine use of a tacking suture to suspend the graft to the prevertebral fascia was identified by authors to be another cause of graft ischemia. Based on these results, the authors have stopped to suspend the graft to prévertébral fascia. Davis and colleagues [21] reported in a large series of 959 gastric reconstructions (gastric tube) a graft necrosis rate of 0.5% (5 patients). As reported by Schuchert and colleagues in a series of 222 patients [22], the incidence of graft necrosis was 3.2% and the authors observed that the incidence increased with the use of a narrow gastric tube. Comparing between gastric pull-up and colon reconstruction, Briel and colleagues [23] reported a prevalence of gastric graft ischemia of 10.4% which was significantly higher than that reported by others authors. Based on previous reports, the risk of gastric graft necrosis is low and the reported incidence varied from 0.5% to

10.4% [18-24]. The incidence of ischemic complications associated with the use of a gastric conduit is summarized in **Table 1**.

Risk factors

Identified risk factors for gastric graft necrosis include using improper technique in the creation of the gastric tube, postoperative hypotension, low perioperative cardiac output, underlying gastric ulcer disease, diabetes, chronic obstructive pulmonary disease, neoadjuvant therapy, twist of the graft when it is pulled up through the posterior mediastinum or substernal space, and a tight, restrictive hiatus. Most risk factors can be avoided by using careful technique to mobilize, create, and handle the gastric tube. Avoiding to use tacking stitch to anchor the fundal tip to the prevertebral fascia during transhiatal esophagectomy as recommended by Annettoni and colleagues [20]

Diagnosis and management

The clinical presentation of graft necrosis depends on the necrosis extent and the site location of the esophageal anastomosis. Graft necrosis is suspected in patients who have high spiking fevers, leucocytosis, unexplained acidosis, respiratory failure and especially associated with continued "coffee ground" nasogastric tube drainage. CT of the neck and chest shows leakage however clear radiographic findings of necrosis are less specific. The contrast esophagogram demonstrates the leakage but not necrosis. Endoscopic exam (esophagoscopy) demonstrates ischemic changes and the extent of necrosis, but this exam has a risk to cause or extend a leak in these patients. In case of cervical anastomosis location, the cranial part of gastric graft can be explored and assessed directly by visual inspection at the same time cervical drainage is performed. Located graft necrosis at the tip of conduit can be revealed by early leakage after initial surgery. So necrosis should be evoked in patient whose anastomotic leakage is well drained (especially cervical leaks) but who continues to be febrile and toxic for longer than 24 hours. The management of necrosis depends on the severity of clinical pattern and location of esophageal anastomosis. Mild cases of gastric tube necrosis manifesting as an anastomotic leak can be managed conservatively with drainage in select cases, especially in the cervical location. As a conservative attitude, Ichikura and colleagues [25] reported three cases of gastric graft necrosis whose diagnostic was confirmed endoscopically. The ischemia was managed conservatively by inserting a rubber t-tube into the esophagogastric anastomosis. The rubber tube was placed to continuous suction in order to decrease salivary drainage and to prevent

stricture after wound healing. After 3 to 4 weeks, the t-tube was removed and replaced by plastic esophageal prosthesis to prevent salivary leakage and anastomotic stricture. The prosthesis was removed and aside from anastomotic dilatation, oral feeding was resumed in all three patients. Fullthickness necrosis is a disastrous complication associated with high mortality rate in absence of early diagnosis and adequate management. Reported mortality rate of this complication is as high as 90%. The treatment in such situation consists of surgical exploration, take-down of the gastric pull-up, resection of the necrotic part, cervical esophageal diversion, and placement of a feeding jejunostomy. After recovery, patients are evaluated for later, staged reconstruction using either colon or jejunum according to the reconstruction distance.

II. PREVENTION

Once ischemia occurred, it is not reversible. Clearly the best way is the prevention of graft ischemia. Patients who are planned for esophagectomy should be evaluated before surgery in order to optimize their cardiorespiratory status, nutritional state and blood counts. Operatively, optimizing a gastric graft creation by mobilizing the stomach carefully using adequately the surgical technique, maintaining perioperatively proper hemodynamics, blood counts, and oxygenation are the paramount parameters to be taken into consideration to minimize the risk of graft ischemia. On other hand, some authors advocated methods to delay the gastric graft preparation in order to improve graft blood supply. One method advocates a two-stage esophageal reconstruction surgery. The first surgery consists of removing the diseased esophagus, and cervical esophageal diversion and the gastric graft is prepared but left in abdomen cavity. The second step of surgery is to pull up the gastric graft for reconstructive anastomosis. Urschel [26] proposed, at the same time as pre-resective laparoscopic staging, to devascularize partially and laparoscopically the gastric fundus in situ. After a wait of 1 to 4 weeks, the esophageal resection and reconstruction is performed using the delayed gastric graft. This technique demonstrated an improvement of esophagogastric anastomotic healing in animal model however this favorable outcome is still unknown in humans. Other authors proposed to augment the blood flow and improve venous drainage by performing microvascular anastomosis. Sekido and colleagues [27] performed a supercharge (microvascular anastomosis) of the gastric graft during esophageal reconstruction. The procedure was performed in cases in which poor arterial inflow or venous drainage was noted intraoperatively. Graft necrosis occurred in two

patients. One patient underwent elongated gastric tube transposition with venous augmentation only by anastomosing the splenic vein to the internal jugular vein. In the second patient, the left gastroepiploic vein of graft was anastomosed to the internal jugular vein without arterial augmentation. In this study, only 3 of the 82 patients had either arterial or venous augmentation separately and the authors conclude that one artery and one vein should be anastomosed to recipient vessels to improve graft outcome.

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Tabl.1: Gastric graft necrosis

Series	Patients	Deaths (%)	Leak (%)	Ischemia (%)
Orringer et al [18]	1085	4	13	2.6
Peracchia et al [19]	242	0.8	5.8	1.2
Davis et al [21]	959	10.6	3.9	0.5
Schuchert et al [22]	222	1.4	-	3.2
Briel et al [23]	230	3.5	14.3	10.4
Moorehead and Wong [24]	760	3.8	-	1.0