Controlled Esophagocutaneous Fistula with a T-Tube Drainage, as a Method of Control of Late Diagnosed Perforation of Acute Oesophageal Ulcer

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Summary

Oesophageal ulcers are a rare pathological finding and usually they are not reason for consideration. The complications they can cause are strictures of the oesophagus as a result of callous chronic ulcer, acute haemorrhage from an oesophageal ulcer or chronic bleeding with development of post-haemorrhagic anaemia, formation of an oesophageal-bronchial fistula, malignant degeneration and perforation with development of mediastinitis and empyema. They occur most frequently in the lower third of the oesophagus, where it is located on the left of the spinal column, just before the card oesophageal junction. Oesophageal ulcers with a higher location to the border between the lower and the middle third of the oesophagus, have also been reported. These ulcers often occur along with a duodenal ulcer and duodenal gastroesophageal reflux. Due to late acceptance of the diagnosis, in most cases after development of mediastinitis and empyema, the treatment is very difficult and with high death rate and incidence rate. The surgical method of treatment with T-tube drainage for release of the oesophageal lumen and the adequate drainage of the mediastinum and the pleural cavity become more and more common in comparison to the conventional methods with removal of the oesophagus and creation of esophagogastrostomy, used widely in the past.

Keywords: Perforated ulcer; Reflux; T-tube catheter

Introduction

Oesophageal ulcer is a type of peptic ulcer, most commonly located in the lower third of the oesophagus. Oesophageal ulcers usually occur as a result of an infection with bacteria, called Helicobacter pylori. Bacteria damage the mucosa of the oesophagus and as a result, it becomes susceptible to damage from hydrochloric acid and pepsin at regurgitation of gastric contents. The disease maybe caused by erosion, due to regurgitation of hydrochloric acid from the stomach with GERD (gastroesophageal reflux disease). The mixed reflux of gastric and duodenal contents is more harmful to the oesophagus than the regurgitation of only gastric contents. There have also been reports that other infection from mycoses and viruses can also lead to oesophageal ulcers. The use of nonsteroidal anti-inflammatory drugs can also be a reason for the occurrence of oesophageal ulcer. The most common symptom of the oesophageal ulcer is burning pain behind the sternum with irradiation to the back. Other symptoms like: acids, due to acidic reflux, vomiting, pain when swallowing, frequent difficulties when swallowing, sour taste in the mouth, and different combinations from the above. The cases of asymptomatic course are not rare. Predisposing factors may be smoking, excessive alcohol and spicy foods use, and also genetic factors related to the type of the vegetative nervous system.

Clinical Case

It is a matter of a 68-year-old patient in severe overall condition, admitted urgently, with complaints from three days. He was admitted at another medical facility for twenty-four hours with suspicion for bronchoesophageal fistula and perforation of a hollow abdominal organ as a result of incarcerated diaphragmatic...
hernia. In the medical history, the patient reports of sudden acute pain in the left chest half, coughing and dyspnoea. According to him, he choked while eating. His temperature rose to 38.5°C. Nausea and vomiting appeared, and the cough was with the release of profuse mucous phlegm. The following is established from the objective status: skin-sweaty, pale; pulse - tachycardia rate 110BPM; temperature -39°C; thorax - symmetric, with lagging of the left chest half; breathing on the left - absent below the level of the third intercostal space; no subcutaneous emphysema or pathological sounds are established on auscultation, abdomen - soft, not painful, with weak intestinal peristalsis. From the laboratory tests: HGB-118 g/l, RBC-3.97, HCT-0.35, WBC-27.3/10^9, PLT-383/10^9, CRP-269; AKP: pCO₂ - 43.3 mmHg, pO₂-49.9 mmHg, sO₂-87.7%. The rest of the laboratory indicators were within an acceptable range. On the computed tomography image, (Figures 1,2) - presence of multiple hydraemic levels and free air collections in the left chest half, with atelectasis of the lower lobe and lingual of the left lung. Mediastinum - not widened mediastinum with minimal mediastinal emphysema in the lower third in the para-aortic region. Bronchial system on the left - the inferior bronchus is not visualized. Right pleural cavity - small pleural effusion, without other pathology.

Additionally, due to the severe condition of the patient, an emergent FBS (fibre bronchoscopy) was performed to reject the assumption for oesophagobronchial fistula and aspiration of food. The FBS rejected the assumption, as endoscopic deconstruction was performed on the left with aspiration of a large amount of mucous-purulent matters. Immediately after that, an intercostal catheter No. 16 was placed in the left pleural cavity, and around 2500 ml of purulent secretion and pressurized air were evacuated. The control radiography on the third post-operative day (After thoracentesis and pleural catheter with vacuum aspiration) showed good radiographic control - bilaterally expanded parenchyma, a small capsulated pleural effusion in the left basal region.

In the following hours and days, the overall condition of the patient was improved along with conservative-medication therapy. Biochemistry of the pleural secretion -Specific gravity 1010, Rialto test +, Protein- 39, Glucose -0.03, pH - 7.0, Ly - 3%, Sg - 97%. Microbiology of the pleural secretion, Alpha-haemolytic Streptococcus, Enterococcus fascism, Candida tropicalis. In the days after the overall condition was controlled, a left posterolateral thoracotomy was performed for revision of the empyema’s cavity and the posterior mediastinum. A perforation aperture was found in the lowest part of the oesophagus (30 mm away from the diaphragmatic edge) with a diameter of 10 mm and round shape, near the anterior edge of the terminal part of the descending aorta. Due to severe fibrotic changes in the tissues around the perforation with developed picture of pleural empyema and impossibility for primary restitution, excision of the edges of the ulcer was performed, and then, using guiding fiberoesophagoscop, a T-tube Foley catheter No. 20 CH was inserted and fixed in the lumen of the oesophagus. The outer end of the catheter was taken out and fixed to the skin. Additionally, a pleural tent from parietal pleura was fixed around the perforation aperture. Due to the presence of pleural and visceral commissure and severe purulent-decaying inflammatory process, a thoracoplasty was performed with the following scope - 4intercostal spaces (with subtotal resection of rib VI, VII, VIII, IX). In the end of the surgery, a nasogastric probe No, 18 was fixed, and its tip was placed in the duodenal bulb. The pleural cavity was drained with tube catheters No. 18.

Figure 1: CT - Mediastinal window. Figure 2: CT- Pulmonary window.
Figure 4: Image of the T-tube catheter after a two-month period of time.

The histopathological result of the edges of the ulcer is acute perforated oesophageal ulcer with fibroid necrosis and chronic productive inflammatory process and it is demonstrated on (Figure 5) and (Figure 6).

Figure 5: Smear from the edges of Inflammatory process.

Figure 6: Fibroid necrosis and chronic perforated oesophageal ulcer.

The patient was discharged on the 30th post-operative day, in satisfactory overall condition, with removed pleural drainages, afebrile with recovered laboratory and clinical indicators. Nutrition was recovered through the nasogastric probe and the T-tube oesophageal intraluminal catheter is kept until a period of two months has passed. In our opinion, the two-month period for the T-tube catheter to be kept is desirable in order to give sufficient time for the formation of a fibrotic fistula with thick adhesion of the tissues around it. Computed-tomography image of the controlled oesophagocutaneous fistula after a two-month period is demonstrated on (Figure 7) and (Figure 8)

Figure 7 and Figure 8: Computed-tomography image of the controlled oesophagocutaneous fistula after a two-month period.

After the completion of the period, the T-tube catheter was removed gradually, under fiberoesophagoscopic control, and the manipulation is demonstrated in (Figures 9, 10, 11) respectively.

Figure 9: In the lumen of the oesophagus the T-tube catheter is visualized before the beginning of the manipulation.

Figure 10: The inner opening of the fistula in the oesophagus is marked after the removal of the drainage.
oesophageal mucosa [7].

Component of the duodenal juice, are capable of damaging the oesophageal mucosa. The majority of the duodenal reflux occurs at pH from 4 to 7, and then the bile acids, the main component of the duodenal juice, are capable of damaging the oesophageal mucosa [7].

If treatment is started within 24 hours, the death rate is around 25%, which increases to 65% after the first 24 hours and to 75-89% after 48 hours [1,5]. The manifestations of Gastroesophageal Reflux Disease (GERD) are caused directly from the contact between the reflux gastric acid and the mucosa of the oesophagus. These manifestations include acids, peptic oesophageal erosion and ulceration, peptic oesophageal stricture and Barrett’s oesophagus. Peptic oesophageal erosions and ulcerations are excavation defect in the oesophageal mucosa, which occur when the epithelial cells are destroyed as a result of the caustic effects of the refluxed hydrochloric acid and pepsin. Most frequently, the oesophageal ulcers are complicated by haemorrhage, perforation and fistulisation in the respiratory tract. Oesophageal ulcers may stimulate the production of fibrous tissue and the deposition of collagen, which leads to formation of strictures [6]. The reflux of stomach contents due to GERD is not the only thing to participate in the genesis of oesophageal ulcers, but also the reflux of duodenal contents in the oesophagus. The reflux of duodenal juice at gastroesophageal reflux is more common than the pH examinations only suggest. The combined reflux of stomach and duodenal juices causes serious damage to the oesophageal mucosa. The majority of the duodenal reflux occurs at pH from 4 to 7, and then the bile acids, the main component of the duodenal juice, are capable of damaging the oesophageal mucosa [7].

The application of an effective treatment algorithm includes also the creation of a controlled oesophagocutaneous fistula. [2-5]. The method for drainage of the oesophagus with T-tube catheters, also known as the Abbott et al. Method, is technically very easy and safe, and avoids post-operative complications related to oesphagostomy with primary or secondary reconstruction. [4,7,8]. The use of T-tube catheters is effective, since it is a single stage surgery, which may be used with late diagnosed severe damage to the oesophagus with patients in severe overall condition [8]. The late control of the perforation of the oesophagus is a serious problem of the thoracic surgeon, who has to choose between the different treatment modalities. The factors, determining the choice of treatment are the clinical status of the patient, the vitality of the oesophageal wall and the extent of dissemination of the inflammatory process. As a modality of the surgical treatment, the following are offered – primary suture and external drainage, primary suture with reinforcement, drainage with a T-tube catheter through the perforation and oesphagostomy with primary or secondary reconstruction. [3,4,7,9]. The primary suture of the acute perforation and diversion of the salivary and oesophageal secretions, by placing T-tubes with late diagnosis, allows the successful healing of oesophageal perforations [10,11].

**Discussion**

Treatment of perforations of the oesophagus is mainly a surgical problem and despite the significant progress in surgery and intensive medicine, they have always been linked to high incidence rate and high death rate [1-3]. Late diagnosis leads to unfavourable result and death rate of 20-40% [4]. According to other authors, death rate varies between 5-89% depending on the delay time and the etiology of the perforation. Post-emetic perforation (rupture or Boerhaavesyndrome) has higher death rate of 25-89% in total. If treatment is started within 24 hours, the death rate is around 25%, which increases to 65% after the first 24 hours and to 75-89% after 48 hours [1,5]. The manifestations of Gastroesophageal Reflux Disease (GERD) are caused directly from the contact between the reflux gastric acid and the mucosa of the oesophagus. These manifestations include acids, peptic oesophageal erosion and ulceration, peptic oesophageal stricture and Barrett’s oesophagus. Peptic oesophageal erosions and ulcerations are excavation defect in the oesophageal mucosa, which occur when the epithelial cells are destroyed as a result of the caustic effects of the refluxed hydrochloric acid and pepsin. Most frequently, the oesophageal ulcers are complicated by haemorrhage, perforation and fistulisation in the respiratory tract. Oesophageal ulcers may stimulate the production of fibrous tissue and the deposition of collagen, which leads to formation of strictures [6]. The reflux of stomach contents due to GERD is not the only thing to participate in the genesis of oesophageal ulcers, but also the reflux of duodenal contents in the oesophagus. The reflux of duodenal juice at gastroesophageal reflux is more common than the pH examinations only suggest. The combined reflux of stomach and duodenal juices causes serious damage to the oesophageal mucosa. The majority of the duodenal reflux occurs at pH from 4 to 7, and then the bile acids, the main component of the duodenal juice, are capable of damaging the oesophageal mucosa [7].

**Conclusion**

The late diagnosed perforation of oesophageal ulcers can be successfully treated with the Abbott method with a T-tube catheter for the diversion of the oesophageal contents outside and the diversion of the secretions of the mediastinum and the pleural cavity through their adequate drainage. Waiting for two months before the extraction of the T-tube catheter is a guarantee for protection against complications, such as mediastinal paraesophageal abscess or empyema.

**References**


Figure 11: The inner opening and the catheter are marked after the extraction in the fistula course.

The controlled oesophagocutaneous fistula was closed by primary intention, without additional complications.

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