CHAPTER 31

# TRACHEO-ESOPHAGEAL FISTULAS

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## INTRO

The trachea and esophagus, which are the two major tracts of the respiratory and digestive tract respectively, are in close proximity to each other in the cervical region and mediastinum. Defective embryological developments affecting these two organs, tissue or tissue damage due to trauma can create pathological pathways connecting the two organs. These pathologies, called tracheoesophageal fistulas, are life-threatening pathologies. It is important to determine the etiology and pathophysiology in order to carry out the diagnosis and treatment successfully. The esophagus and trachea develop from the embryonic foregut [1]. In the third week of gestation, tracheobronchial diverticulum buds form from the esophagus. The esophagus and trachea which were continuous with each other until the fourth week starts to diverge from each other during the 4-5th week starting from the level of the carina. A failure in this embryological process will result in congenital TEF [2]. In the case of acquired TEFs discussed in this section, the mechanism is different. Formation of acquired fistulas varies according to etiology.

## ETIOPATHOGENESIS

Acquired TEFs can be grouped into two main groups: malignant and benign [3]. Esophageal cancer is the leading cause of malignant TEF (mTEF). Esophageal cancer accounts for more than 50% of malignant TEFs [4]. The rate of TEF in esophageal cancers is around 5-10%. In a study conducted by Martini et al. which is one of the mTEF studies with a large case series to date, TEF developed in 4.94% of patients with esophageal cancer (n=1943) [5]. In another study by Balazs et al. mTEF developed in 12.5% (264/2113) patients with primary or secondary esophageal malignancy [6,7]. Both the esophagus and the trachea are ducts with a thin wall structure. Wall thickness is approximately 4 mm. Esophageal cancer penetrates directly into the trachea through the membranous wall because the cartilage wall of the trachea is more resistant to tumor infiltration. Local tumor invasion is followed by tumor necrosis, thus leading to the formation of mTEF [1]. Palliative therapies are preferred for advanced esophageal cancer. Treatment options include radiotherapy or esophageal stent placement. Unfortunately, both treatment modalities induce tumor necrosis and pave the way for TEF development. The risk of developing TEF is higher in tumors with high radiosensitivity. In addition to esophageal malignancy, TEF may develop in lung, trachea, larynx, thyroid and lymph node malignancies. It is also known that malignant-metastatic lymph nodes- especially in the subcarinal area- due to their proximity to the trachea and esophagus, can

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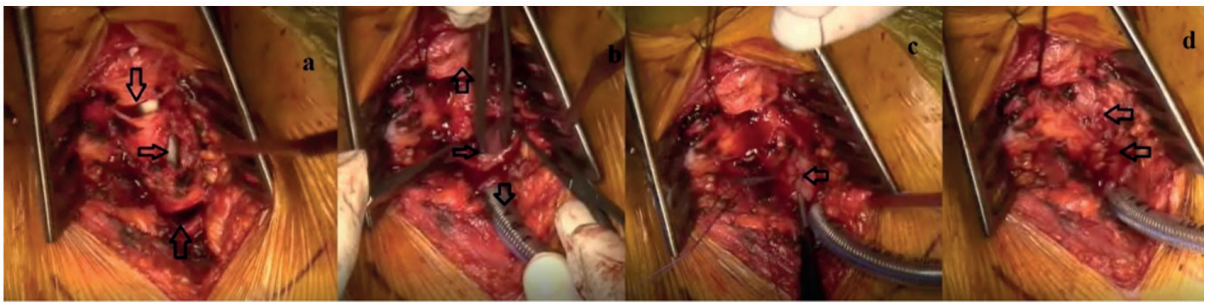
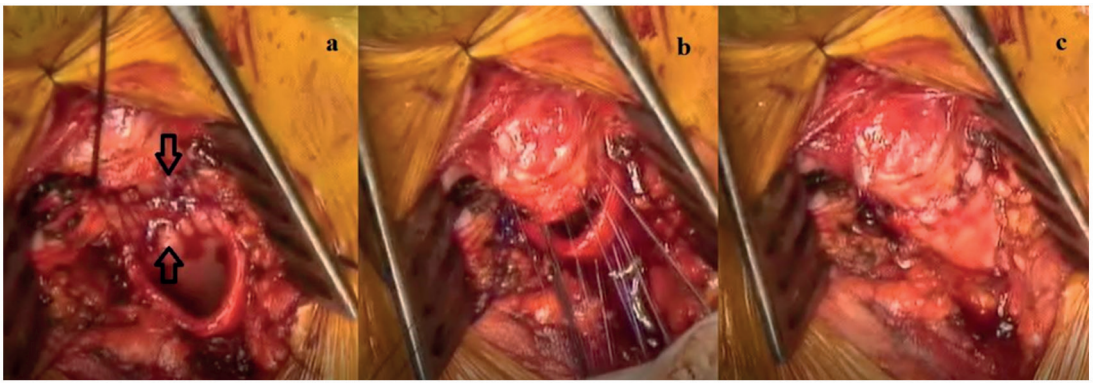



**Picture 4:** A) Upper border of trachea, tracheotomy and entubation tube, lower border of trachea. B) Excised tracheal segment, esophageal fistula. C) Repair of the posterior wall of the esophagus, endotracheal tube. D) Nasogastric tube placed distally after posterior wall repair, before closing of the excised fistula. Double layer closure of the esophagus




**Picture 5:** A) Posterior anastomosis of the trachea with continuous suture, closing of the posterior sutures, B) Anterior wall being repaired with interrupted sutures

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