postoperatively and was discharged 10 days after the operation without major complications. He was still doing well at the end of 8-month follow-up.

Comment

Next to the isthmus of the thoracic aorta, the innominate artery is the second most vulnerable for blunt injury. In civilian patients, mechanisms of injury are motor vehicle accidents in 90%, crush injuries in 8%, and falls in 2% [1]. It was proposed that 2 types of forces are responsible for the innominate artery injury. One is transverse compression between the sternum and the spine, and the other is longitudinal shear stretch when the neck is hyperextended and the heart is displaced downward. Both forces cause increased tension on the innominate artery, especially in the proximal segment, because it is relatively fixed on the arch. Bovine arch anatomy is a predisposing factor for innominate artery injury, which is seen in 11% of the general population but in 29% of patients with innominate artery injury [4]. The underlying cause for this association is that bovine arch anatomy results in fewer aortic arch fixation points and consequently more concentrated force on the origin of the innominate artery.

In the majority of patients, the diagnosis of innominate artery injury after blunt trauma can be established in the acute phase. However, delayed presentation—up to 34 years after the injury [2]—has been reported in 12.3% of patients. The most common types of innominate artery injury are intimal tears and pseudoaneurysm formation. Concomitant respiratory manifestations, mainly resulting from pneumothorax, can be seen in about 15% of patients [5]. Airway distress caused by direct compression from the pseudoaneurysm, as seen in this patient, is rare.

Several operative approaches have been reported for innominate artery injury, including primary repair, graft replacement, and bypass. Protective measures for cerebral perfusion, such as shunting and CPB, may or may not be used. Most patients with a normal circle of Willis can tolerate temporary clamping of the innominate artery. However, shunting must be used when backflow pressure is insufficient (<50 mm Hg). CPB is usually reserved for patients whose pseudoaneurysm cannot be safely exposed or those with concomitant arch injury, the pending or contained rupture of the pseudoaneurysm requiring anastomosis under circulatory arrest. Recently, endovascular repair has been applied for posttraumatic innominate pseudoaneurysm [6]. Endovascular repair is less invasive and more expeditious than open surgical repair, but its success is highly dependent on the site of rupture and the operator’s expertise. Endovascular repair was not suitable for our patient for 2 reasons: (1) life-threatening airway compression that mandated prompt use of CPB and (2) bovine arch anatomy that precluded an adequate landing zone for a stented graft.

In conclusion, pseudoaneurysm of the innominate artery is rare. CPB is useful for surgical repair if the pseudoaneurysm cannot be safely accessed. The unique feature of the present case is severe tracheal compression, which is potentially lethal and tricky to manage. Early detection and prompt institution of CPB through peripheral vessels can be lifesaving in this situation.

References


Tracheal diverticulum (TD) also referred to as tracheocele, right paratracheal cyst, or tracheogenic cyst, is a rare benign entity with only limited reports in the literature [1]. It is characterized by single or multiple peripheral vessels can be lifesaving in this situation.

Recurrent Laryngeal Nerve Paralysis by Compression From a Tracheal Diverticulum

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Tracheal diverticulum is a rarely encountered entity, located usually on the right side of the trachea as an outpouching of the wall. Based on mainly histologic features, it can be classified in a congenital or an acquired form. It is usually an incidental finding in an asymptomatic patient. When symptoms are present, they are mostly nonspecific such as chronic cough, dyspnea, or pulmonary infections. We describe a case of dysphonia due to right recurrent laryngeal nerve compression from a tracheal diverticulum. Computed tomography scanning and bronchoscopy revealed the lesion and surgical resection resolved the symptom.

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Invaginations of the tracheal wall and is typically an incidental finding as most of the cases remain asymptomatic. Symptoms that have been reported in association with TD are dysphagia, dyspnea, hemoptysis, or pulmonary infections [1, 2]. We report a case of dysphonia as a consequence of recurrent laryngeal nerve (RLN) paralysis due to compression from a TD.

A 38-year-old Caucasian female was referred to the Department of Otorhinolaryngology with a sudden onset of dysphonia without obvious reason. Medical history revealed 10 years of moderate smoking (5-6 cigarettes/day) and recurrent episodes of seasonal allergic rhinitis and tracheobronchitis.

Clinical and pulmonary examination findings were within normal limits. A subsequent laryngoscopy revealed a paralysis of the right vocal cord. To exclude a malignant process compressing the RLN, a computed tomography (CT) scan with three-dimensional imaging reconstructions of the neck and thorax revealed an air-filled cyst of 2 cm in diameter, adjacent to the right side of the trachea (Figs 1A, 1B). Bronchoscopy confirmed the paralysis of the right vocal cord and showed a narrow-mouthed origin of the TD at the right side of the posterior wall of the trachea (Fig 2). Finally, the diagnosis of a partial axonotmesis of the right RLN was withheld by an electromyography of the vocal cords. In order to resolve this incapacitating paralysis, the patient was referred to the Department of Thoracic Surgery. A lateral cervical approach confirmed compression of the RLN from a TD (Fig 3). The latter was resected and the patient’s voice fully recovered.

Histologic examination demonstrated a fibrotic cystic wall with an epithelial lining of ciliated respiratory cells and absence of cartilage rings (Fig 4).

Comment
Since the first report of a TD in 1838 by Rokitansky, few cases have been described [1]. The largest series of 65 patients was reported by Goo and colleagues [3], based on the retrospective finding of a paratracheal air cyst on CT scan. The overall prevalence is estimated on 1%, according to a series of 867 consecutive autopsies [4].

Tracheal diverticulum can be classified in an acquired and congenital form, varying in anatomic position, characteristics, and histology. The acquired form usually presents in adulthood and is characterized by a wide opening in the trachea and a relatively large diverticulum. On histologic examination the TD is formed solely of respiratory epithelium, lacking smooth muscle or cartilage as in our case. The latter can be attributed to the...
mechanism by which the TD originates [1, 5]. It is thought that increased intraluminal pressure, by chronic coughing or recurrent tracheobronchitis, might result in a mucosal outpouching of the tracheal wall between the cartilaginous rings. Predominantly, it appears on the posterolateral right side of the trachea, as the left side is supported by the esophagus. Goo and colleagues [3] confirmed that 98% of the lesions were located in the right paratracheal region. The acquired form can present as multiple diverticula, which is the hallmark of tracheobronchomegaly or Mounier-Kuhn disease [1]. The congenital form, in contrast, is usually found single. It most often occurs on the right, posterolateral side of the trachea, 4 to 5 cm below the vocal cords. It is rather small and the communication with the trachea is narrow, resulting in poor drainage of the mucous secretions, predisposing to secondary infection. The congenital TD is thought to be a remnant from a vestigial supernumerary lung [1]. This is confirmed on histologic examination as the diverticulum possesses a complete tracheal anatomy (respiratory epithelium, smooth muscle, and cartilage).

Most TD cases evolve asymptptomatically and are found incidentally. Only less than 20 symptomatic cases have been reported in the literature in which the disorder was accompanied by symptoms like hemoptysis, dyspnea, dysphagia, tracheobronchitis, or stridor [1, 2]. Tracheal diverticulum has also been reported to cause difficulty with endotracheal tube positioning causing failed lung isolation or accidental pneumomediastinum [6]. To our knowledge, only 1 other clinical case of dysphonia due to RLN compression from a TD has been described in a 70-year-old female with a history of recurrent tracheobronchitis [5]. Laryngoscopy revealed right vocal cord paralysis and CT scan diagnosed a TD on the right side. Due to the patient’s age and comorbidities, medical treatment with antibiotics and physiotherapy was preferred.

Diagnosis is made by CT scanning and bronchoscopic evaluation. The CT scan is characterized by an air-filled tubular structure, often located posterolateral to the right side of the trachea. Differential diagnosis includes laryngocele, pharyngocele, Zenker's diverticulum, apical lung hernia, or bullae [7]. A CT scan may help in differentiation between the acquired and congenital form, depending on the presence of cartilage and the size of the neck of the diverticulum [3]. Bronchoscopy may help in localizing the opening, although diverticula with a narrow opening can be missed.

Various methods of treatment have been reported in the literature; surgical resection via a lateral cervical approach, endoscopic electrocoagulation, and conservative medical management of the symptoms with antibiotics, mucolytic agents, or physiotherapy [1, 5]. The latter is generally recommended for the elderly.

In conclusion, a TD is a rare entity, mostly asymptomatic and commonly seen on the right side of the trachea. It can be classified in an acquired or congenital form. A CT scan or endoscopic evaluation will help in diagnosing a TD. Surgical therapy is preferred in the younger patients and has proven to be an effective solution in our case of RLN paralysis and dysphonia.

References


Successful Management of Anastomotic Leakage and Lung Fistula After Esophagectomy

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We report the successful management of a case of anastomotic leakage with a lung fistula as a complication of esophagectomy by use of a double elementary diet tube. (Ann Thorac Surg 2014;97:1071–3) © 2014 by The Society of Thoracic Surgeons

A nonneoplastic fistula between the gastric conduit and the lung as a complication of esophagectomy is a very rare but life-threatening condition. We report a patient in whom management with a double elementary diet tube (W-ED tube) for a lung fistula as a complication of esophagectomy was successful.

A 62-year-old man with epigastric discomfort was referred to our hospital. Upper gastrointestinal endoscopic examination showed an esophageal type 3 tumor in the middle portion of the thoracic esophagus, and biopsy specimens revealed squamous cell carcinoma. He received a diagnosis of esophageal cancer T3N1M0 (International Union Against Cancer stage III). He received neoadjuvant chemotherapy comprising 5-fluorouracil and cisplatin, and underwent video-assisted thoracoscopic esophagectomy, anastomosis of the cervical esophagus with a gastric conduit through the posterior mediastinal route, and three-field lymphadenectomy.

He suddenly experienced shock and was admitted to an intensive care unit on postoperative day 4. Chest radiography showed a left-sided, infiltrative shadow. Chest computed tomography (CT) demonstrated a 3.0 × 2.5 cm lung abscess of the left lung located beside the esophagogastric anastomosis site and infiltrative shadow around the lung abscess (Figs 1A, 1B). He was diagnosed with septic shock, lung abscess, and severe pneumonia, and received “early-goal” treatment according to the surviving sepsis campaign guidelines [1]. He survived the shock, which required de-escalation antibiotic therapy, mechanical ventilation, and endotoxin absorption treatment. After 6 days in the intensive care unit, a radiologic contrast examination demonstrated anastomotic leakage and a lung fistula (Fig 2). We considered that the anastomatic leakage induced mediastinal abscess, and contents of the abscess penetrated the pulmonary bulla wall and led to severe pneumonia. Pre-operative chest computed tomography demonstrated a pulmonary bulla of the left lung located beside the superior portion of the thoracic esophagus. For the drainage of digestive fluid, pressure reduction of the gastric conduit, and enteral nutrition, we used a W-ED tube (Nippon Sherwood, Tokyo, Japan). The W-ED tube was composed of a separated double lumen, which facilitated a reduction in the pressure of the digestive cavity 45 cm from the end of the tube and the delivery of enteral nutrition administered nasally (Fig 3A). He recovered rapidly, the lung fistula closed on postoperative day 21 (Fig 4), and oral intake was reinitiated on postoperative day 31. Computed tomography of the chest on postoperative day 24 showed improvement of the lung abscess and pneumonia (Figs 1C, 1D). He was discharged on postoperative day 42 in a favorable condition.

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