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 CBP through peripheral vessels can be lifesaving in this situation.

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FEATURE ARTICLES

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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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

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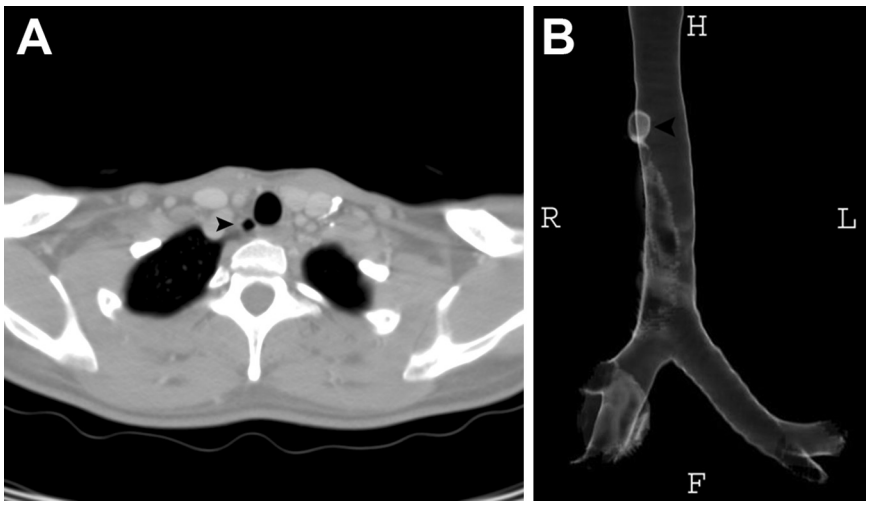

Fig 1. Computed tomography scan of the neck and thorax. (A) Axial and (B) three-dimensional reformatted images show a relatively small (2 cm) air-filled tubular structure (arrowhead in A and B), located posterolateral to the right side of the trachea. (H = head; F = feet; R = right; L = left.)

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

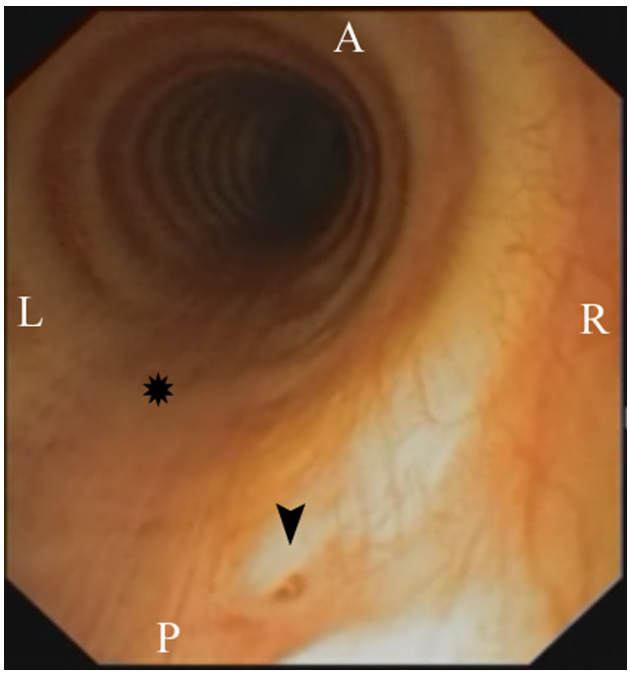

Fig 2. Bronchoscopic view. The narrow-mouthed opening of the tracheal diverticulum (black arrow) is located on the right posterolateral side of the trachea, adjacent to the pars membranacea (asterisk). (A = anterior; P = posterior; R = right; L = left.)