

Tracheal compression caused by an innominate artery aneurysm after thoracic aortic aneurysm repair in a patient with Marfan disease

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Tracheal compression caused by innominate artery anomalies is rare, and because of congenital anomalies, it is more recognized in children¹⁻³ than in adults.^{4,5}

Innominate artery aneurysms are even more uncommon than anomalies and are mainly described in relation to atherosclerosis⁶ or *Treponema pallidum* infection.⁷ We report the case of a patient with Marfan disease and repeated sternotomy for aortic arch surgery in whom difficult weaning after repair of a thoracoabdominal aortic aneurysm led to the diagnosis of severe tracheal compression caused by an innominate artery aneurysm. Reoperation with exclusion of the aneurysm and transection of fibrotic tissue compromising the patency of the trachea improved tracheal stenosis and led to complete resolution of symptoms after successful extubation. To our knowledge, tracheal compression caused by an innominate artery aneurysm and fibrotic tissue after repeated sternotomy for aortic arch surgery in a patient with Marfan disease has not previously been reported.

Clinical Summary

A 43-year-old man was admitted to the intensive care unit because of weaning impairment. His medical history revealed hypertension, Marfan disease, Bentall procedure, partial aortic arch replacement, and replacement of the aortic arch with bypasses to the left common carotid, proximal innominate, and left subclavian arteries. Now a thoracoabdominal aortic aneurysm (Crawford extent II) with dissection necessitated aortic replacement from the previous graft to the iliac bifurcation. Extubation attempts early after surgical intervention failed. After transfer to the intensive care unit, weaning was impaired by desaturation caused by sputum retention. Resistance during advancement of a suction tube into the trachea was noted. Bronchoscopy showed 90% stenosis caused by external compression. A computed tomographic (CT) scan (Figure 1) visualized tracheal compression between the spinal column and the aneurysmatic innominate artery (maximal diameter of 3 cm). During reoperation, the innominate artery aneurysm was excluded, new bypasses to the right carotid and subclavian arteries were constructed, and fibrotic tissue compromising the patency of the



Figure 1. CT scan of the cervical region showing 90% tracheal stenosis caused by compression of the trachea between the spinal column and the aneurysmatic innominate artery (\varnothing 3 cm).

trachea was transected. During the operation (positive-pressure ventilation), bronchoscopy revealed spontaneous deployment and a fully patent lumen. Five days postoperatively, bronchoscopy during spontaneous breathing (zero positive pressure) revealed tracheomalacia and a remaining stenosis of 60%. Several hours after extubation, coughing and increased inspiratory effort resulted in desaturation and stridor. Reintubation was necessary for another week. After 14 days, the patient was discharged from the hospital without stridor or dyspnea. A CT scan performed after 5 months showed persistent tracheomalacia with a diameter reduction of approximately 60% (Figure 2). The patient was free of symptoms.

Discussion

Innominate artery aneurysms are rare and described in relation to atherosclerosis⁶ or syphilis⁷ and, to a lesser extent, to Takayasu disease, trauma, chronic dissection, bacterial infection, and collagen disorders.⁸ Three percent of the total number of cases of aneurysms correspond to aneurysms of the innominate artery. Tracheal compression caused by an innominate artery aneurysm has been reported before⁹⁻¹² but never in a patient with Marfan disease after repeated sternotomy for aortic arch surgery. The development of an innominate artery aneurysm after a Bentall procedure has been reported once.¹³ In addition to clinical manifestations caused by tracheal compression, as in our patient, manifestations caused by compression of other structures have been reported, such as dysphagia; dysphonia; brachial plexus dysfunction with pain or with weakness of the upper extremity, or both; and superior vena cava syndrome with upper extremity edema. Other manifestations are due to embolism or thrombosis, causing (transient) cerebral ischemia, ocular deficits, or vertebro-

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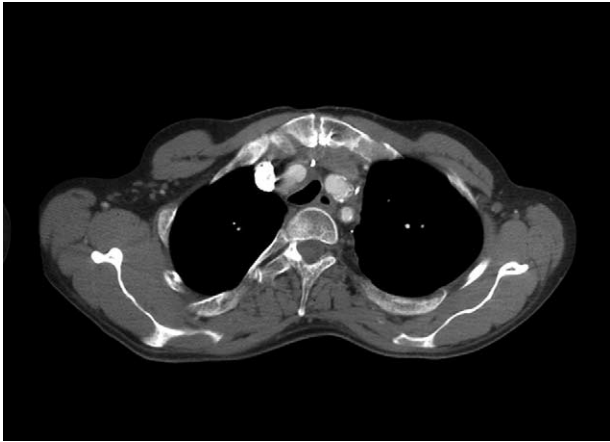


Figure 2. CT scan of the cervical region 5 months after exclusion of the innominate artery aneurysm and construction of new bypasses to the right carotid and subclavian artery. Persistent tracheomalacia with a diameter reduction of approximately 60% is visible.

basilar syndrome. A pulsatile mass might be present in the supraclavicular region, and the neck veins might be prominent. CT or angiography usually confirms the diagnosis. Surgical treatment is always necessary in symptomatic cases and must be seriously considered in asymptomatic and otherwise healthy patients.⁸ Surgical approach through a median sternotomy and bypass grafting from the ascending aorta to the subclavian and carotid arteries has good long-term patency.⁵ Surgical treatment can be especially difficult in patients with Marfan disease because of the usual habit of these patients, which is characterized by minimal dissection space in the neck and mediastinal region. In an elective setting, the mortality risk is 4% to 5% and increases up to 50% in case of emergency surgery for a ruptured aneurysm.⁸ Persistence of some degree of tracheomalacia is common but usually needs no further

treatment. In case of recurrent episodes of dyspnea, stenting of the trachea can be considered.

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