**Introduction**

Cervical ribs were first noted by Galen in the second century A.D., and by Vesalius in the 16th century [1]. The incidence of cervical ribs is estimated to be two percent of the general population [2]. Subclavian thrombosis as a result of cervical rib compression was described as early as 1927 by Symonds in the case of a patient who developed hemiplegia due to an embolism [3]. In 1956, Peet used the term thoracic outlet syndrome to describe upper limb symptoms arising from neurovascular compression in the interscalene triangle [4]. A recent retrospective analysis revealed that as many as two-thirds of patients with arterial compression at the thoracic outlet may have associated bone anomalies, cervical ribs being chief among these, followed by anomalies of the first rib [5]. Symptoms of upper extremity ischemia, paresthesias, and hand coldness are common [6,7].

There are still few detailed reports of cervical ribs as a cause of subclavian artery occlusion and thoracic outlet syndrome. This case report seeks to provide a clinical correlation with surgical findings and radiographic imaging.

**Case Report**

A 39-year-old female presented to the emergency room with a 2-day history of unilateral headache, facial tingling, and slurred speech, along with a 2-month history of left upper extremity weakness and coolness of the left hand. A stroke workup that included carotid duplex ultrasonography revealed findings suggestive of left subclavian occlusion, later confirmed on CT angiography. Neurologic workup suggested migraine, rather than ischemia, as the cause of headache; however, the subclavian occlusion prompted further investigation, including a chest radiograph that revealed bilateral cervical ribs. The patient was taken to surgery where her left cervical rib was excised and the chronically occluded left subclavian artery was found to have significant post-stenotic dilatation. Given that the chronic nature of her left upper extremity symptoms were not limiting, the decision was made not to undertake bypass. Recommendation was made to remove her right cervical rib prophylactically at a future date.

**Conclusions:** Cervical ribs are a rare cause of vascular thoracic outlet syndrome, with compression leading to post-stenotic dilatation and symptoms of ischemia in the involved extremity.

**Keywords:** Vascular thoracic; Ischemia; Subclavian thrombosis
Intraoperatively, a large subclavian artery aneurysm was found just anterior to the cervical rib, and the cervical rib was fused to the first rib just underneath the subclavian artery. On inspection, the subclavian artery aneurysm appeared to result from post-stenotic dilatation stemming from compression by the cervical rib. Immediately proximal to the dilatation, a water hammer pulse was dopplered, with loss of pulse in the artery distal to the rib extending into the axillary artery.

Given that the patient’s left extremity symptoms were chronic in nature and had not interfered with her ability to work, the decision was made to forgo thrombectomy or bypass.

Discussion

Patients with cervical ribs resulting in stenosis often remain asymptomatic until post-stenotic dilatation with thrombus formation and embolization occurs. The pathogenesis of post-stenotic dilatation is not fully understood, but likely results from stasis, increased lateral pressure, cavitation, abnormal shear stresses and turbulence [8,9].

Based on a recent review, the role of cervical ribs in causing thoracic outlet syndrome may be underappreciated [5]. Cervical ribs should be considered in any patient presenting with thoracic outlet syndrome, and their presence can be suggested by physical exam findings on palpation of the supraclavicular fossae with subsequent confirmation via cervical radiographs.

In patients with bilateral cervical ribs who have already had one of their cervical ribs removed due to symptomatic occlusion, consideration should be given for prophylactic removal of the contralateral cervical rib. Some practitioners argue for regular surveillance imaging of the asymptomatic side to monitor for the development of subclinical arterial occlusion in these patients [10].

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References


