Vascular Thoracic Outlet Syndrome

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Two distinct terms are used to describe vascular thoracic outlet syndrome (TOS) depending on which structure is predominantly affected: venous TOS (due to subclavian vein compression) and arterial TOS (due to subclavian artery compression). Although the venous and arterial subtypes of TOS affect only 3% and <1% of all TOS patients respectively, the diagnostic and management approaches to venous and arterial TOS have undergone considerable evolution due to the recent emergence of minimally invasive endovascular techniques such as catheter-directed arterial and venous thrombolysis, and balloon angioplasty. In this review, we discuss the anatomical factors, etiology, pathogenesis and clinical presentation of vascular TOS patients. In addition, we use the most up to date observational evidence available to provide a contemporary approach to the diagnosis and management of venous TOS and arterial TOS patients.

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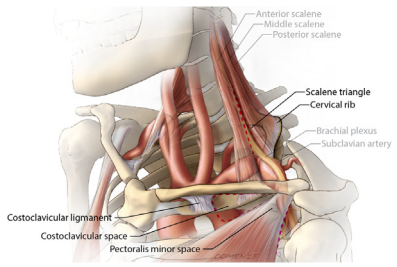

Arterial TOS: subclavian artery compression in the scalene triangle by a cervical rib.

Central Message

The approach to vascular TOS has undergone significant evolution given the emergence of endovascular therapies over the last decade.

Perspective

Vascular thoracic outlet syndrome (TOS) can be divided into 2 forms (arterial and venous) depending on which structure is compressed in the thoracic outlet. Clinical evidence for vascular TOS is limited to case series, with a paucity of randomized controlled trials. Our review focuses on the contemporary diagnostic and management approach to vascular TOS given the increased popularity of endovascular techniques.

See Editorial Commentary page 158–159.

OVERVIEW

TOS refers to a constellation of signs and symptoms attributable to compression of the neurovascular bundle in the thoracic outlet region of the upper extremity. A total of 3 distinct terms are used to describe TOS depending on which structure is predominantly affected: neurogenic TOS (nTOS) from brachial plexus compression, venous TOS (vTOS) from subclavian vein compression, and arterial TOS (aTOS) from subclavian artery compression. The most common form by far is nTOS, which accounts for more than 90% of all TOS cases. This review focuses on the venous and arterial subtypes of TOS, which are seen in 3% and <1% of TOS patients, respectively. During the past decade, the approach to vTOS and aTOS has undergone considerable evolution due to the emergence of minimally invasive endovascular therapies.

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ANATOMICAL FACTORS

The thoracic outlet area comprises 3 anatomic spaces: scalene triangle, costoclavicular space, and pectoralis minor (PM) space (Fig. 1). The borders of the anterior scalene muscle, middle scalene muscle, and first rib define the scalene triangle; trunks of the brachial plexus and the subclavian artery pass through this space (Fig. 1A). Cervical ribs and anomalous first ribs may compress the scalene triangle, resulting in symptoms of nTOS or aTOS. The costoclavicular space, which is the area between the first rib and the clavicle housing all 3 major structures (subclavian artery, vein, and brachial plexus), represents the most common site of subclavian vein compression (Fig. 1B). The PM space, defined by the PM muscle anteriorly and the chest wall posteriorly, is an extension of the thoracic outlet and a common site of neurovascular compression.

ETIOLOGY

Most patients with TOS are adults between the age of 20 and 50 years, although adolescents aged 18 years or younger present with vascular TOS more frequently than adults.[1]

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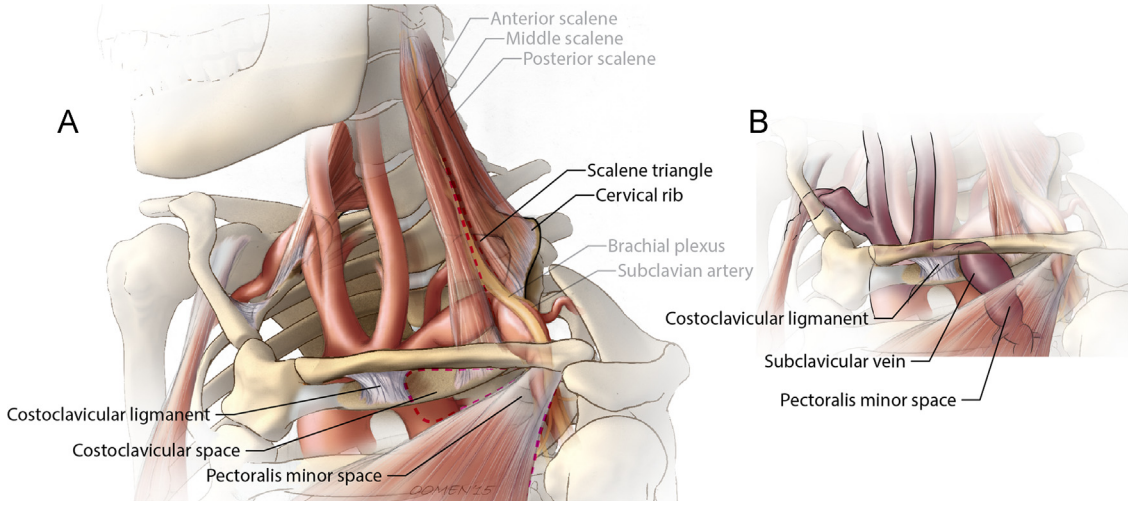

Figure 1. Illustration of the thoracic outlet showing the 3 common sites of vascular compression: scalene triangle, costoclavicular space, and pectoralis minor space. The subclavian artery is most commonly compressed in the scalene triangle with the presence of an accessory cervical rib, leading to arterial TOS (A). Venous TOS commonly occurs due to compression of the subclavian vein in the costoclavicular space between the costoclavicular ligament, first rib and subclavius tendon (B). (Adapted with permission from Oomen et al.)

Factors that increase the risk of neurovascular compression and development of symptomatic TOS include congenital anomalies in the thoracic outlet, repetitive motion or stress activities, and traumatic injuries. Arterial TOS is associated with bone abnormalities or trauma in nearly all cases, whereas vTOS is most often the result of repetitive overhead arm and shoulder activities, such as swimming, throwing, or weight-lifting in combination with anomalies of the costoclavicular space.

PATHOGENESIS

Arterial TOS is most commonly associated with bone abnormalities of the thoracic outlet, with cervical ribs being present in up to 85% of patients with aTOS in contemporary series.[2-5] Cervical ribs are present in $<1\%$ of the general population, and about 70% of individuals with cervical ribs are women. Cervical ribs that cause symptoms are often large, and can have bony fusion to the first rib that results in subclavian artery compression.[6] This causes injury to the third segment of the subclavian artery, leading to intimal damage, thrombosis, distal embolism or poststenotic dilation, and aneurysm formation. Rarely, complete occlusion of the subclavian artery may occur. Other less common anatomic abnormalities causing aTOS include anomalous first rib, prominent C7 transverse process, callus formation from an old clavicular or first rib fracture, and fibrocartilaginous band.

In vTOS, the subclavian vein is often compressed between the first rib, costoclavicular ligament and subclavius tendon within the costoclavicular space (Fig. 1B). Repetitive arm movements traumatize the vein, causing posttraumatic inflammation, focal intimal fibrosis, stenosis, blood flow stasis, and eventual thrombosis leading to acute symptoms of upper extremity deep venous thrombosis. This clinical condition is referred to as effort thrombosis or Paget-Schroetter syndrome, and is often observed in young individuals and competitive athletes who engage in physical activities requiring repetitive arm and shoulder movements.[7] Chronic compression and repetitive trauma also cause inflammation external to the subclavian vein. This point is underscored by the fibrotic, hypertrophied and relatively fixed surrounding anatomic structures often observed during vTOS surgery.

CLINICAL PRESENTATION

Early in the disease process, aTOS patients most commonly present with chronic arm, shoulder, or neck pain with increasing activity due to subclavian artery stenosis or thrombosis. Hand or arm ischemia due to arterial embolization is the most common acute presentation of aTOS. A subset of aTOS patients present with concomitant symptoms of nTOS, such as chronic arm or hand paresthesias, numbness, or weakness from coexisting brachial nerve compression.[8] Physical examination for aTOS focuses on measuring bilateral blood pressures of the upper extremities to asses for a marked discrepancy between the symptomatic and asymptomatic side, palpating for cervical ribs or a pulsatile mass in the