Thoracic Outlet Syndrome: Current Concepts, Imaging Features, and Therapeutic Strategies

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Abstract

Thoracic outlet syndrome describes a set of disorders resulting from compression of the brachial plexus and/or subclavian vessels. Early recognition is essential, as diagnostic or treatment delays are associated with significant morbidity.

In this article, we describe the imaging features of thoracic outlet syndrome with an emphasis on use of computed tomography and magnetic resonance imaging angiography and venography in association with postural maneuvers to demonstrate dynamic compression of the neurovascular structures. The pathophysiology, clinical features, and management options, which depend on the affected structures and degree of severity, are also presented in this article.

Thoracic outlet syndrome (TOS) was first described by Coot in 1861,[1,2] and the term was coined by Peet and colleagues[3] in 1956 to cover a spectrum of conditions caused by dynamic compression of the brachial plexus (neurogenic), subclavian artery (arterial), or subclavian vein (venous). The estimated incidence of TOS is 10 in 100,000.[4] However, cadaveric studies have suggested that up to 90% of the population may have what is considered abnormal anatomy of the thoracic outlet,[5] which in turn suggests a multifactorial etiology for symptomatic disease. TOS is most commonly diagnosed in patients 20 to 40 years of age, with females affected in a 4:1 ratio.[6] Although historically TOS is a clinical diagnosis, advanced imaging is often helpful in determining the nature and location of the structure undergoing compression and the structure producing compression, which help guide management. Computed tomography angiography (CTA) and magnetic resonance imaging (MRI) performed in association with postural maneuvers aid in the diagnosis in patients with dynamically acquired compression.[7]

Pathophysiology

The pathophysiology of TOS is attributable to the unique anatomy of the thoracic outlet. Compromise of the neurovascular structures can occur through congenital or acquired narrowing in 3 distinct compartments: the interscalene triangle, the costoclavicular space, and the retropectoralis minor space. The interscalene triangle is the most medial of the compartments. Containing the subclavian artery and the 3 trunks of the brachial plexus, it is bordered anteriorly by the anterior scalene muscle, posteriorly by the middle and posterior scalene mus-

cles, and inferiorly by the first rib. The interscalene triangle is the most frequent site of neurologic compression.[8] The middle compartment is the costoclavicular space, which is bordered superiorly by the clavicle, anteriorly by the subclavius muscle, and posteriorly by the first rib and the middle scalene muscle. The costoclavicular space is the most frequent site of arterial compression,[8] where the artery lies directly anterior to the subclavian vein and is surrounded by the 3 cords of the brachial plexus. The most lateral compartment is the retropectoralis minor space, which is bordered anteriorly by the pectoralis minor muscle, superiorly by the subscapularis muscle, and inferiorly by the anterior chest wall. Sources of neurovascular compression within any of the spaces include cervical ribs[9]; elongated C7 transverse processes; hypertrophy of the anterior or middle scalene, subclavius, or pectoralis minor muscles[10]; anomalous scalenus minimus muscle; repetitive overhead arm movements (pitching, swimming)[11]; anomalous fascial bands; degenerative spine disease; bone destruction from primary or secondary neoplasms (Pancoast tumor); hyperextension/flexion injury of the neck[12]; and malunion of clavicle fractures, among others.[13]

Classification

Three distinct TOSs have been described, individually or combined, depending on the injured component: neurogenic from brachial plexus compression, arterial from subclavian artery compression, and venous from subclavian or axillary vein compression.[14,15]

Neurogenic TOS has 2 reported types: *true (classic)* and *disputed*. True neurogenic TOS is rare, with an estimated incidence of 1 in

1 million.[16] First described in 1970 as a lower trunk plexopathy involving slowly progressive unilateral weakness of the intrinsic hand muscles and sensory abnormalities in the ulnar and medial antebrachial cutaneous nerve distributions, true neurogenic TOS was originally called *Gilliatt-Sumner hand syndrome*.[17] A congenital band extending between the first rib and an elongated C7 transverse process was thought to be the location of brachial plexus injury in true neurogenic TOS. Conversely, disputed neurogenic TOS is the most common form of TOS, occurring in 3 to 80 per 1000[18] and accounting for 90% to 95% of all TOS cases.[13,19] In contrast to true neurogenic TOS, in which anatomical and electrodiagnostic evidence supports the diagnosis, objective clinical findings are often lacking in the disputed form.[18] Patients with disputed neurogenic TOS present with a diverse array of symptoms, including pain, numbness, and weakness affecting the neck, shoulder, and arm, exacerbated by activities requiring elevation or sustained use of the extremity.[20]

Arterial TOS accounts for 1% to 5% of all TOS cases.[21] Arterial TOS typically affects patients who perform repetitive movements of the upper extremities with their arms above their shoulders, resulting in compression of the subclavian artery. Symptoms of arterial TOS include pain, weakness, coolness, pallor, and paresthesia.[18,22] In severe cases of compression, subclavian artery damage can result in thrombosis with distal embolization, poststenotic aneurysm, or even retrograde extension causing stroke.[22,23]

Last, representing 2% to 3% of all TOS cases, venous TOS results from compression of the subclavian or axillary vein.[18,24] Two mechanisms for vascular compromise have been described. The first involves compression of the vein between the clavicle and the first rib with overhead activities.[18] Patients often experience intermittent "heaviness" of the extremity with repeated overhead use. The second mechanism involves repeated stress between the clavicle and vein, causing an intravascular thrombosis.[18] Patients may experience pain, edema, cyanosis, venous distention, and even spontaneous venous thrombosis, referred to as *Paget-Schroetter syndrome*, which can lead to pulmonary embolism.[6,25,26]

Clinical Features

In cases of suspected TOS, clinicians should take a thorough history and perform a thorough physical examination. The differential diagnosis for unilateral, upper limb pain, numbness, tingling, and/or weakness exacerbated by movement includes shoulder and rotator cuff pathology, cervical spine injury, cervical radiculitis, distal compressive neuropathies (carpal or cubital tunnel syndrome), and neuralgic amyotrophy (Parsonage-Turner syndrome/acute brachial radiculitis).[27,28] The clinician should pursue a history of trauma to the shoulder or neck as well as any occupational or recreational activities involving elevation of the upper extremity for extended periods.[29] Physical examination must include an evaluation of the contralateral side and may begin with visual inspection to assess for muscle asymmetry, atrophy, color changes, edema, or deformities.[18] Next, palpation should be used to assess for any

tenderness, texture changes, masses, or vascular pulsations. Attention should be directed at examination of the cervical spine as well as neurologic and vascular assessments of the bilateral upper extremities, including range of motion and strength testing,[18] to rule out alternative etiologies.

Four basic maneuvers—the Roos test,[30] Adson test,[31] Wright test,[32] and costoclavicular test—traditionally have been used to diagnose TOS. A positive Roos test involves symptom reproduction with the patient slowly opening and closing the hand for 3 minutes with the arm externally rotated and abducted to 90°.[33] However, the false-positive rate of the Roos test is as high as 77% in patients with carpal tunnel syndrome and up to 47% in normal subjects.[34] The Adson test is performed by having the patient inhale deeply while the arm is kept in the anatomical position with the head extended and turned toward the involved extremity. The examiner monitors the radial pulse; an absent or diminished radial pulse suggests compression of the subclavian artery. The Adson test is not very reliable, however, because the pulse diminishes even in normal subjects,[6,26] with a reported false-positive rate of 13.5%.[35] A positive costoclavicular compression test occurs when depressing a patient's shoulder reproduces symptoms. In one study, the false-positive rate of the costoclavicular compression test was 48% in patients with carpal tunnel syndrome and 16% in normal subjects.[34] Last, the Wright test is performed by hyperabducting and externally rotating the affected shoulder. It is positive with a diminished pulse or reproduction of symptoms. One study found that the Wright test had 70% to 90% sensitivity and 29% to 53% specificity.[36]

Clinically distinguishing between the various forms of TOS may be difficult, and occasionally multiple types exist in a single patient, exacerbating one another and adding to the diagnostic difficulty. For example, arterial insufficiency may lead to disruption of the neural microcirculation, leading to concurrent arterial and neurogenic TOS. Because most cases present with nonspecific symptoms, advanced imaging modalities are often required to establish a definitive diagnosis and to target therapy to the appropriate site of compression.

Imaging Features
Plain Radiography
First, cervical spine and chest radiographs should be obtained to assess for bone abnormalities, including cervical ribs, long transverse processes, rib/clavicle fracture callus, rib anomalies, degenerative spine disease, and neoplasm (Pancoast/apical tumor) (**Figure 1**).[18,25]

Ultrasonography
Ultrasonography is useful in evaluating arterial or venous TOS because of its low cost, noninvasive nature, and high specificity for vessel occlusion.[37,38] In arterial TOS, ultrasound may demonstrate increased flow velocity through a stenosis or an aneurysmal degeneration distal to the stenosis.[7] In venous TOS, duplex ultrasound can identify stasis and thrombus.[7] Obtaining duplex ultrasound with the upper extremity in multiple positions allows clinicians to correlate dynamically induced symp-

toms with ultrasonographic findings of altered blood flow.[39-41] Despite the purported benefits of ultrasound, its drawback is that it is operator-dependent,[42] with some studies reporting a high false-positive rate[24] for diagnosis of venous TOS.

Electrodiagnostic Testing

Ruling out etiologies such as cervical radiculitis (Parsonage-Turner syndrome), cervical radiculopathies, brachial plexus lesions, and other distal compressive neuropathies requires nerve conduction studies and electromyography.[18,43-46] In true neurogenic TOS, a combination of decreased sensory nerve action potentials in the ulnar and medial antebrachial cutaneous nerves and decreased compound motor action potentials in the median nerve is often found.[18] Specifically, an abnormal ulnar sensory nerve action potential suggests the lesion is situated away from the intraspinal canal, which argues against a diagnosis of radiculopathy or myelopathy.[43,44] In the disputed form of neurogenic TOS, the role of electrodiagnostic testing is less clear.[18]

Conventional Arteriography and Venography

Although CTA has superseded conventional arteriography and venography in most treatment centers, it may still be used in patients with acute symptoms requiring immediate thrombolytic therapy. Catheter angiography and venography with postural maneuvers are often the first invasive treatment modality in cases of thoracic outlet vascular compression.[22,24] Presence of intraluminal thrombus, vessel dilatation, and collateral vessels is readily demonstrated (**Figure 2A**). Recanalization of occluded vessels can be attempted using balloon angioplasty and venoplasty (**Figure 2B**), but it is usually only temporarily successful if the cause of extrinsic compression is not corrected (**Figures 2C, 2D**). CTA or conventional angiography, used if sophisticated CTA with 3-dimensional (3-D) reconstruction is unavailable, is the gold standard in diagnosis of TOS.

CTA and Venography

Computed tomography (CT) is a valuable modality because it can be performed rapidly and effectively to depict the relation-

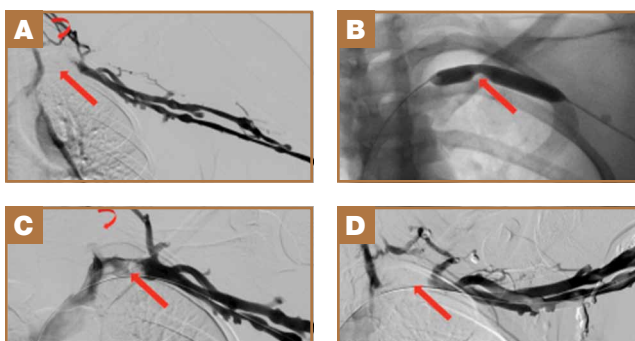**Figure 1.** Frontal radiograph shows cervical rib (arrow).

Figure 2. (A) Conventional venogram shows thrombosis of subclavian vein (arrow) at level of costoclavicular space with collateral circulation (curve arrow) reflecting vessel compression chronicity. (B) Balloon venoplasty shows decrease in focal area of subclavian vein stenosis. (C) After venoplasty, there is partial recanalization (arrow) of subclavian vein with persistent partial intraluminal thrombosis but decreased collateral flow (curved arrow). (D) Recurrent complete subclavian vessel occlusion with arm abduction reflects recurrent vessel compression at costoclavicular space.

Figure 3. (A) Computed tomography venography 3-dimensional reconstruction shows Paget-Schroetter syndrome (effort thrombosis). During arm elevation (abduction), costoclavicular space compression of neurovascular bundle is accentuated, causing dynamic narrowing of subclavian vein (arrow). On right, venous collateralization reflects chronic occlusion (curved arrows). (B) Magnetic resonance imaging venogram reconstruction on same patient has same findings.

ship of vascular structures to surrounding bone and muscle.[47] In addition, CTA and venography provide high-quality representations of the vasculature, and 3-D reconstruction reliably identifies areas of neurovascular compression in patients with TOS.[47,48] Furthermore, CT may be performed in a dynamic fashion, with the upper extremity in various positions to reproduce dynamic compression of the neurovascular structures (**Figure 3A**). Comparison of the images with the upper extremities in the anatomical position and elevated allows the physician to evaluate narrowing of the compartments and dynamic compression of neurovascular structures.[8] CT is particularly valuable in arterial and venous TOS. In arterial TOS, the cross-sectional area or diameter of the artery can be measured to calculate the degree of stenosis.[8,47] In venous TOS, dynamic narrowing of the vein can be visualized and may be associated with venous thrombosis or collateral circulation (**Figure 3B**). Although a variety of maneuvers is possible during CTA, the size of the CT tunnel as well as mandatory supine positioning of the patient may limit the series. Drawbacks of CT for diagnosing TOS include difficulties in analyzing the brachial plexus because of limited contrast resolution. In addition, the risks of CT (ionizing radiation, administration of iodinated contrast medium) must be considered before image acquisition.

MRI

MRI is a noninvasive and nonionizing technique that offers good resolution of the anatomical components of the thoracic outlet[8] and that, because of its superior soft-tissue contrast, is the modality of choice for imaging brachial plexus nerve compression in TOS (**Figure 4**). Neurologic compression is identified with MRI when the fat surrounding the brachial plexus disappears.[8] MRI reliably identifies the source of compression, which may include bony structures, muscle hypertrophy (scalenus, scalenus minimus, subclavius, pectoralis minor), and fibrous bands.[49] Because of their craniocaudal direction, the sagittal plane is often most useful in demonstrating neurovascular compression.[42] Analyzing the caliber of the vessel along its course may evaluate vascular compression, and magnetic resonance (MR) angiography and venography (**Figures 5A, 5B**) can often complement the findings.[50] Specifically, in arterial TOS, poststenotic aneurysmal dilatation may be seen, whereas thrombosis and collateral circulation can be visualized in cases of venous TOS.[50] Limitations of MRI in the diagnosis of TOS historically were similar to those of CT, and included supine positioning as well as restricted upper extremity maneuvers because of the size of the tunnel and the presence of surface coils.[42] However, newer higher channel surface coils and wider bores allow for imaging in a wider range of motion, including arm hyperabduction (**Figures 5C, 5D**), which is often necessary to elicit pathology.

Management

Generally, therapeutic options for TOS are aimed at relieving the source of neurovascular compression. It is important that treatment be directed only toward symptomatic patients, as many patients have anatomy consistent with TOS and remain

Figure 4. Sagittal proton-density magnetic resonance imaging shows normal anatomy at level of costoclavicular space with preserved fat planes between neurovascular structures. Abbreviations: c, clavicle; a, subclavian artery; v, subclavian vein; s, subclavius muscle. Bracket indicates cords of the brachial plexus.
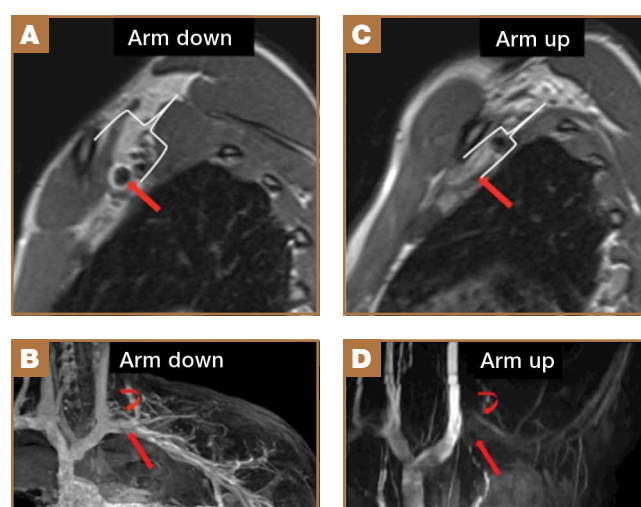

Figure 5. (A) Sagittal proton-density magnetic resonance imaging (MRI) and (B) MRI venogram show no evidence of subclavian vein (arrow) compression at level of costoclavicular space and normal costoclavicular distance (bracket) with arm down (adduction). Abundant collateral veins suggest chronic shunting of blood even though stenosis is not directly demonstrated in arm adduction (curved arrows). (C) Sagittal proton-density MRI and (D) MRI venogram with arm up (hyperabduction) show severe compression of subclavian vein (arrow) in costoclavicular space, narrowing of costoclavicular space (brackets), and significantly decreased collateral flow (curve arrows) in patient with thoracic outlet syndrome.

asymptomatic.[5] Treatment of TOS is predominately conservative and involves a combination of patient education, activity modification, medication, and rehabilitation to promote appropriate body mechanics and posture.[18]

Physical Therapy

Physical therapy should be aimed at decreasing pressure on the neurovascular structures of the thoracic outlet by relaxing the scalene muscles, strengthening the shoulder muscles, and working on postural exercises to help the patient sit and stand straighter.[51] The scalene muscles are the primary targets for TOS rehabilitation, but focus should also be given to the upper trapezius, levator scapulae, sternocleidomastoid, pectoral, and suboccipital muscles.[18] Physical therapy is often combined with hydrotherapy, massage, nonsteroidal anti-inflammatory drugs, and muscle relaxants for maximal symptomatic relief. Some patients have found relief with selective anesthetic or botulinum toxin A injections in the scalene muscles.[18] A minimum of 4 to 6 weeks (often 4-6 months) of physical therapy and conservative treatment should be attempted before consideration of any invasive intervention.[13,18]

Anticoagulation

In venous TOS with evidence of thrombus but no obstructive clot, conservative management is typically sufficient. In rare cases, however, intimal damage secondary to vascular compression in arterial and venous TOS leads to thrombus formation, impairing upper extremity perfusion and producing symptoms. Treatment guidelines for venous TOS involve catheter-directed thrombolysis within 2 weeks of symptom onset.[15] Thrombolysis replaced the prior recommendation of systemic anticoagulation combined with extremity rest and elevation because anticoagulation and rest alone result in up to 75% morbidity,[52,53] whereas thrombolysis reestablishes vessel patency in nearly all patients.[54] After thrombolysis, patients should receive intravenous heparin, and conversion to oral anticoagulation should occur as soon as manageable. In patients with arterial TOS, the goal of treatment is revascularization to prevent or decrease ischemia. In mild arterial ischemia, catheter-directed thrombolysis can be attempted. However, the threshold for surgical thromboembolectomy must remain low, as acute upper extremity ischemia may result in compartment syndrome and permanent loss of function.[13] Fixed arterial lesions, whether occlusive or aneurysmal, are an absolute indication for thromboembolectomy with possible thoracic outlet decompression.[13]

Thoracic Outlet Decompression

Indications for surgical decompression are controversial. They include symptomatic patients who have vascular (arterial or venous) TOS and are not at high risk for surgery, patients with true neurologic TOS and acute progressive neurologic weakness or disabling pain,[55] and patients who have disputed neurologic TOS and have failed conservative management—keeping in mind that high recurrence rates and iatrogenic brachial plexopathy have been reported in this population.[56] In

general, surgical procedures are aimed at reducing soft-tissue compression (scalene release or neurolysis) or bony compression (cervical or first thoracic rib excision). Three surgical approaches (transaxillary, supraclavicular, infraclavicular) are commonly used for decompression, and surgeons choose one over another depending on the anatomical abnormality causing the compression. The transaxillary approach requires limited dissection but still allows for adequate visualization of the rib during resection.[57] In this approach, a transverse incision along the inferior border of the axilla extends from the pectoralis major to the latissimus dorsi. After dissection of the axillary vessels and the first thoracic nerve root, the first rib is identified and can be removed, when indicated. In contrast, the supraclavicular approach provides a wide exposure, and the site of compression is directly visualized, allowing for arterial reconstruction.[58] Through this approach, the anterior and middle scalene muscles can be resected, and neurolysis of the brachial plexus can be performed. Last, the infraclavicular approach allows for exposure of the central veins through extension of the incision medially, which allows for venous reconstruction. Some patients with neurogenic or arterial TOS present with symptoms of sympathetic overactivity, in which case cervical sympathectomy can be used with decompression.

Outcomes of surgical decompression for TOS depend on the clinical type but are generally good. For instance, in cases of disputed neurogenic TOS, symptom resolution after decompression is reportedly between 80% and 90%.[59] However, major depression, work-related injuries,[60] and diffuse preoperative arm symptoms[61] all influence long-term results. In true neurogenic TOS, postoperative pain relief is often substantial, though recovery of strength can be slow because of the axonal injury.[55] In arterial TOS, outcomes are influenced by time to surgical intervention, with early surgery demonstrating better outcomes than later surgery.[62] In one study, Cormier and colleagues[14] evaluated 47 patients who underwent correction of subclavian-axillary artery compression; 91% were asymptomatic a mean of 5.7 months after decompression. Last, outcomes of successful thrombolysis and decompression for venous TOS demonstrated patency rates higher than 95% at 5-year follow-up.[54,63]

Conclusions

TOS is a spectrum of disorders caused by compression of the brachial plexus, subclavian artery, or subclavian vein. Early recognition of TOS is imperative, as diagnostic or treatment delays may be associated with significant morbidity. Clinical examination alone is often inadequate for determining the compression site and the structure causing compression. CTA and MRI performed in association with postural maneuvers may demonstrate dynamic compression of the neurovascular structures in the thoracic outlet. These imaging modalities reliably identify the structures causing compression and can be crucial for effective management.

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