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Thoracic Outlet Syndrome: Evaluation and Management

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Abstract

Thoracic outlet syndrome is an umbrella term that describes the potential compression of the brachial plexus, subclavian vein or subclavian artery by different clinical disorders. This review covers the classification, clinical findings, physical examination findings and management of this challenging syndrome under the light of recent scientific research. Various medical specialties can encounter TOS within their field of expertise. TOS should not be viewed as a single clinical entity that simply manifests variations from one patient to another. TOS is composed of three very discrete subgroups, and treatment should be individualized after diagnosis is definite.

Keywords: Thoracic outlet syndrome; Cervical rib; Neurogenic; Brachial plexus

Introduction

Thoracic outlet syndrome (TOS) is a challenging term that describes the potential compression of the brachial plexus, subclavian vein or subclavian artery as it exits the thoracic outlet. The history of TOS extends back as far as to late 1700's when the cervical rib was first described. In 1956, the term 'thoracic outlet syndrome' was suggested to include several syndrome having similar symptoms under an umbrella term [1,2]. The cervical rib syndrome, scalenus anticus syndrome, scalenus medius syndrome, hyperabduction syndrome, costoclavicular syndrome, pectoralis minor syndrome, first thoracic rib syndrome, droopy shoulder syndrome may cause upper extremity symptoms due to compression of the neurovascular bundle in the area of the neck just above the first thoracic rib [3].

TOS can be classified based on the etiology (traumatic, repetitive stress, postural abnormality) or clinical presentation and anatomy (neurogenic, arterial, venous) [3]. Anatomical classification is simple and practical because the three subtypes can be differentiated easily with the help of history and physical examination. Another preferred classification suggested by a neurologist defines TOS subtypes as neurogenic, disputed neurogenic, venous, arterial and traumatic [4].

Neurogenic TOS	Venous TOS	Arterial TOS
Repetitive injury	Activity related effort thrombosis	Hypertrophy of Scalene muscles
Cervical spine extension injury	Droopy shoulder	Presence of anomalous first rib
Cervical whiplash injury	Congenital narrow subclavian vein	Presence of cervical rib
Hypertrophy of scalene muscles	Congenital variation in the course of subclavian veins	Congenital variation in the course of subclavian artery
Presence of anomalous first rib	Indwelling venous catheter	Secondary to hypercoagulability Malignancy
Presence of cervical rib	Secondary to hypercoagulability Malignancy	-
Congenital fibromuscular bands	-	-
*TOS: Thoracic Outlet Syndrome		

Table 1: Etiologic factors for development of TOS*.

The thoracic outlet is anatomically defined as the space between the first thoracic vertebra, first rib and the manubrium of the sternum. The thoracic outlet is a small space already occupied by subclavius, anterior scalene and cervical prevertebral muscles. The volume of thoracic outlet also changes dynamically with respiration and movement of neck and arm [5]. These dynamic changes may ease impingement of neurovascular bundle. Moreover, fibrosis or inflammation of the involved muscles or variations of bony structures may lead to impingement. Table 1 shows the etiological factors for the development of TOS.

Clinical Presentation

For simplification, we used the anatomical classification of TOS: neurogenic TOS, arterial TOS, and venous TOS.

Neurogenic TOS accounts for over 90% of all TOS cases [6]. It is seen predominantly in women aged 18-50 years. The most common etiological factor is a hyper-extension neck injury mostly whiplash during motor vehicle accidents [3]. The second most common cause is overuse injury such as incorrect keyboard us for a long time or hyperabduction of arms during work [7]. Anatomical etiological factors include anomalous first rib, cervical rib, congenital fibromuscular bands, scalene muscle hypertrophy, congenital narrow interscalene interval [3].

The symptoms of neurological TOS are a result of compression of the brachial plexus at the thoracic outlet. Involvement of lower brachial plexus fibers (C8, T1) is more prevalent and results in pain at supraclavicular and infraclavicular fossa, neck, scapula, and chest wall. Patients experience radiation of shoulder and neck pain to medial arm typically. An occipital headache and transient facial paresthesias may also be present [8].

The involvement of upper brachial plexus fibers (C5, C6, C7) results in pain at anterior neck, upper jaw, ear, and chest wall. Neck pain may radiate laterally on arm [9]. Subjective motor symptoms like fatigue and weakness may accompany pain and paresthesias. Patients mainly suffer from weakness and incompetence in fine motor activities of the hand. However, in patients with prolonged symptom duration weakness and atrophy primarily involving fourth and fifth flexor digitorum profundus, hypothenar muscles innervated by the ulnar nerve is detected. Weakness and atrophy involving flexor and extensor carpi ulnaris muscles may also be seen [2,3].

Venous TOS accounts for 5% of all TOS cases. Venous TOS refers to compression and thrombosis of the subclavian vein and is frequently referred to as effort thrombosis or Paget-Schroetter Syndrome. Venous TOS presents with a sudden onset painful, blue and swollen upper extremity. History of prolonged upper extremity use is often present [10]. The thrombosis of the subclavian vein will end up with dilated superficial veins that represent a collateral flow to bypass obstructed segment. Dilated veins may also be detected at the shoulder and superior chest wall [11]. Patients may experience paresthesia secondary to compression of sensory nerves. In patients with intermittent obstruction, symptoms may be transient in nature.

Arterial TOS is the rarest form of TOS comprising 1% of all cases [10]. Arterial TOS refers to compression of a subclavian artery usually precipitated by the presence of anomalous first rib, cervical rib, hypertrophic scalenus muscles and congenital fibromuscular band [12]. Compression of the subclavian artery may lead to post-stenotic arterial dilatation, aneurysm formation, arterial thrombosis and eventually distal embolization. Patients may stay asymptomatic until distal embolization develops. Pain is less common in arterial TOS than venous and neurogenic forms. When present, pain is in the form of a deep, severe, ischemic pain. Patients report coldness and pallor after upper extremity use. Digital gangrene may be the presenting symptom in chronic cases.

Physical Examination Findings

The physical examination, as well as history, is an important clue for diagnosis and classification of TOS. Despite presenting with similar symptoms, physical examination findings of three anatomical subtypes differ from each other.

A thorough upper extremity neurological examination is necessary when any form of TOS is suspected in a patient. The neurologic examination may reveal global hand weakness especially in hypothenar and lateral thenar muscles. A patchy sensory loss mostly involving lower brachial plexus fibers is common. Palpation may reveal tenderness at scalene muscles, trapezius and anterior chest wall in neurogenic TOS [3]. Several provocative tests are conducted to reproduce symptoms.

Brachial Tinel's sign

Tinel's sign is a way to detect irritated nerves. Brachial Tinel's sign is performed by light percussion over the brachial plexus at supraspinous fossa. The test is positive if a tingling in the distribution of affected nerves is elicited. It is one of the most reliable provocative tests in the diagnosis of TOS [8].

Adson's test

The test was defined in 1927 by Adson and Coffey to assess the evidence of circulatory symptoms caused by the presence of a cervical rib [13]. Several modifications were suggested after its description. Adson's test is currently described in the following manner. The radial pulse is palpated by the examiner on the wrist. The affected arm is laterally rotated and extended by the examiner while the patients take a deep breath, holds it and rotates the face to tested shoulder. The disappearance of the radial pulse with final active neck extension is indicative of a positive test [14]. The test lacks sensitivity and specificity. Moreover, there are no inter-examiner reliability and validity reports of Adson's test in the literature [15].

Roos test

The test was first described in 1976 for the diagnosis of TOS. It is also named as abduction, external rotation test or stress abduction test. The patient is seated both arms in the 90° abduction, external rotation position and elbows in 90° flexion. The head is laterally rotated to the opposite side. This positions narrows the inter scalene and costoclavicular area and reproduces symptoms. If no symptoms occur, patients are instructed to open and close fingers making a fist slowly for 120-180 seconds. A gradual increase in pain at neck and shoulder, paraesthesia in forearm and fingers, inability to complete the test, dropping arms in lap in marked distress are indicative of positive test results. Mild fatigue during the test should not be interpreted as positive. Çalıs et al. investigated the diagnostic value of provocative maneuvers in TOS and reported that Roos test was the most sensitive test [16].

Costoclavicular compression test

The test is also named as Eden test or exaggerated soldier posture test. The test is applied in the sitting position. The radial pulse is palpated. The patient is asked to position his shoulders backward and downwards while protruding the chest, similar to soldier posture. The disappearance of radial pulse or presence of paresthesia, pain, and weakness radiating to arms are indicative of a positive test [17].

Halstead's test

The test can be applied in the sitting or standing position. The radial pulse is palpated. The patient is instructed to turn the head to the opposite side and extend the neck. The examiner pulls on the arm applying traction. The disappearance of radial pulse or presence of paresthesia, pain, and weakness radiating to the arm are indicative of a positive test [17].

Hyperabduction test

This test was defined in 1945 for diagnosis of TOS by Wright. The test is applied in sitting position. The radial pulse is palpated, and the patient is instructed to abduct arms in external rotation position. This position causes traction of neurovascular bundle around the coracoid

process. The disappearance of radial pulse or presence of paresthesia, pain, and weakness radiating to the arm are indicative of a positive test [14].

Calıs et al. investigated the diagnostic values of tests for TOS. They demonstrated that Roos test, Halstead's test, Brachial Tinel's sign, and costoclavicular test were most sensitive tests [16]. Nord et al. conducted another study to investigate the false-positive rate and specificity in normal subjects and carpal tunnel syndrome patients of provocative maneuvers used to diagnose the thoracic outlet syndrome. They concluded that current provocative maneuvers used to diagnose TOS resulted in a high false-positive rate in normal subjects and carpal tunnel syndrome patients [18]. Therefore, the results of the provocative tests should be interpreted with caution and should be combined with diagnostic clues in the patient history.

Diagnosis of venous TOS is fairly certain in most patients after an initial history and careful physical examination. A sudden swollen, cyanotic, painful upper extremity in an active young male is the hallmark of the presentation. In older patients, alternative causes for venous thrombus should be considered. Physical examination findings suggestive of Pancoast tumor or malignancy should be investigated in elderly patients [10].

Physical examination findings in arterial TOS are those of arterial occlusion. Absent or diminished distal upper extremity pulses, delayed capillary filling, pallor, sensory deficit secondary to ischemia, ischemia or gangrene at finger tips may be present. The clinician should palpate the supraclavicular area when arterial TOS is suspected. Palpation may reveal congenital palpable fibromuscular bands, bony prominence or pulsation of the supraclavicular artery [2,17].

Diagnosis

The diagnosis of neurogenic TOS is based on the clinical presentation in conjunction with objective physical examination findings, electroneuromyogram (ENMG) findings and radiological documentation of anatomical abnormalities and variations. The term 'Disputed neurogenic TOS' has become popular in late 1960's. The diagnosis of disputed neurogenic TOS is often challenging given the absence of objective physical examination or diagnostic test findings and presence of clinical presentation suggestive of neurogenic TOS. Most clinicians base their diagnosis on typical clinical history and positive provocative tests [11]. Diagnostic imaging serves to narrow rather than confirm the diagnosis in disputed neurogenic TOS cases. Plain radiograms of the cervical spine are the beginning imaging modality for neurogenic TOS.

The presence of cervical ribs or anomalous first rib can be visualized on the anteroposterior cervical radiogram. Whereas lateral cervical roentgenogram may show an unusual number of cervical vertebra defined as 'droopy shoulder syndrome'. Clein described the droopy shoulder syndrome in 1976. In normal lateral cervical roentgenogram, it is not expected to see seventh cervical vertebra. Only 6 and 1/2 cervical vertebra can be visualized. However, in patients with droopy shoulder syndrome, one sees not only the seventh cervical vertebra clearly but also the first and a part of the second thoracic vertebra [19]. The presence of obvious spondylosis excludes the diagnosis of droopy shoulder syndrome.

Computerized tomography (CT) may provide anatomic further details and assist in the preoperative planning of the rib resection. Scalene muscle hypertrophy may be seen on CT [20]. Magnetic resonance imaging (MRI) of the cervical spine is useful for the differential diagnosis of cervical disc herniation, cervical nerve root impingement, compression of the brachial plexus and scalenus muscle variations and abnormalities, and fibromuscular bands. MRI of the brachial plexus may show inflammation of the brachial plexus if impingement is prolonged [21,22].

The sensitivity and specificity of EMG are low in neurogenic TOS. Abnormalities in nerve conduction velocity, ulnar nerve action potentials, compound motor action potential and F-wave latency may be demonstrated in some patients. EMG similar to cervical spine MRI is helpful to rule out other neurological conditions such as motor neuron disease, polyneuropathy, upper extremity entrapment syndromes and cervical radiculopathy [6]. Medial antebrachial cutaneous nerve conduction (MAC) was first described in the 1990s and was reported to be capable of detecting milder forms of neurogenic TOS patients in whom EMG studies were normal.

A more objective diagnostic test is scalene muscle block with lidocaine. The test was described in 1939 as injection of 1% lidocaine into the belly of the anterior scalene muscle. The test can be performed under imaging or electromyography guidance [23-25]. Patients with neurogenic TOS would have decrease or relief of symptoms for four hours. Lum et al. conducted research to examine the effects of the selective use of lidocaine and botulinum toxin for anterior scalene blocks on the outcomes of the patients undergoing trans-axillary decompression with first rib resection and scalenotomy for neurogenic TOS. They concluded that selective use of lidocaine blocks was more beneficial in predicting surgical success in patients ≥ 40 years old [26].

The combination of clinical presentation and imaging is adequate for the diagnosis of venous and arterial TOS [10]. In the diagnosis of venous TOS, color Doppler sonography is a rapid and noninvasive technique. Color Doppler sonography has high specificity (82-100%) and sensitivity ratios (78-100%) for the diagnosis of venous thrombus [27].

Significantly increased flow velocities would suggest a narrowing at the thoracic outlet where the absence of flow suggests total occlusion. Both the subclavian and axillary veins should be completely evaluated using both gray-scale imaging and Doppler spectral waveform analysis. Evidence of thrombus or complete occlusion of the subclavian vein with a loss of flow are findings that are suggestive of venous TOS. The entire portion of the vein must be examined to avoid missing a potential thrombus and to assess extensiveness of the thrombus if present. If initial imaging is in the normal range, the flow in subclavian and axillary veins should be evaluated during arm abduction maneuvers [10].

Contrast venography is indicated in patients with symptoms and normal Color Doppler sonography findings. Contrast venography is also indicated in patients whom the initial endovascular intervention is planned. The venous system is accessed through the brachial or basilic veins with ultrasound guidance during contrast venography [28]. A positive contrast venography for venous TOS will demonstrate occlusion of the subclavian vein at the costoclavicular junction. Alternative imaging techniques such as CT venography and magnetic resonance venography can offer higher anatomic details of the adjacent structures. However due to the ease and economic burden of duplex ultrasound they are not recommended for the initial diagnosis of venous TOS [10].

Using ultrasound for diagnosis of arterial TOS is similar. Arterial pulse waveforms are reduced and flow; velocities will be increased with

stenosis or absent in complete occlusion. CT angiography or conventional angiography can be utilized to identify more clearly the occlusion, aneurysm, or distal embolization [5].

TOS is often a diagnosis of exclusion. Many other clinical situations may mimic symptoms of TOS and must be considered in differential diagnosis (Table 2).

Diseases of nervous system	**Traumatic**	**Musculoskeletal diseases**
Cervical radiculopathy	Cervical Vertebra fractures	Shoulder bursitis/ tendinitis
Ulnar nerve entrapment neuropathy	Clavicular Fractures	Inflammatory joint disease
Carpal tunnel syndrome	Brachial plexus traction injury	Myofascial pain syndrome
Brachial neuritis	-	Complex regional pain syndrome
Peripheral nerve injuries	-	-
Systemic Diseases	Venous vascular disorders	Arterial vascular disorders
Supraclavicular fossa tumors	Thrombophlebitis	Arteriosclerosis
Pancoast Tumor	Mediastinal serous obstructions	Arterial aneurysm
Cervical spinal cord tumors	Venous aneurysm	Raynaud's disease
Gastroesophageal reflux	-	Thromboangitis obliterans
Coronary arterial disease	-	Arterial Embolism
Panniculitis	-	-
Vasculitides	-	-
Mixed connective tissue disease	-	-
*TOS: Thoracic Outlet Syndrome		

Table 2: Differential diagnosis of TOS.

Treatment

TOS is one of the most controvertable diagnoses in clinical practice. Despite many reports of conservative and surgical interventions, rigorous scientific investigation of this syndrome leading to evidence-based management is lacking. A Cochrane database review was conducted to evaluate the beneficial and adverse effects of the available operative and non-operative interventions for the treatment of TOS [29]. The report was updated in 2014. However both of the reviews were complicated by a lack of generally accepted criteria for the diagnosis of TOS and had to rely exclusively on the diagnosis of TOS by the investigators in the reviewed studies. The first review included only one study and the second review included two studies [29,30].

In general, conservative treatment for at least three months is recommended in patients with neurologic TOS before considering surgery. Despite literature lacking the effects of the conservative non-invasive treatment, we apply combined conservative treatment for neurogenic TOS. Combined conservative treatment includes the modification of daily living activities, providing good posture, cryotherapy, nonsteroidal anti-inflammatory drugs, myorelaxants, and electrotherapy.

Exercise recipe is planned after initial symptomatic control. An exercise program should include posture exercises, stretching exercises for neck and shoulders especially cervical flexors and pectoralis major. Isometric or isotonic strengthening exercises for neck, shoulders and back are also included gradually in exercise protocol. Strengthening of shoulder elevators is crucial in the rehabilitation of TOS. Hanif et al. performed a study to highlight the role of therapeutic exercises in patients with neurogenic TOS. They included fifty patients with TOS and recommended a therapeutic exercise program for six months. 17 (34%) of patients showed full recovery, 14 (28%) had marked improvement, 16 (32%) had partial improvement while 3 (6%) patients reported severe persistent symptoms [31]. The study did not include a control group. Despite methodological insufficiency, they reported that a trial of therapeutic exercises provided relief of symptoms in the majority of patients with neurogenic TOS [31].

Buonocore et al. conducted another study that included thirteen patients with neurogenic TOS. Kinesitherapy involving the cervical spine and shoulder girdle and massage were applied to the participants. All the patients were satisfied with the treatment outcome This study, despite the lack of a control group and a small number of participants also suggested that conservative treatment may be efficient in patients with neurogenic TOS [32].

There is a general opinion among clinicians that approximately 60-70% of patients with neurogenic TOS can be successfully treated with combined conservative treatment [5]. Patients with refractory symptoms should undergo a lidocaine scalene muscle injection. If they respond to this block, they may be evaluated for surgical treatment. Progressive neurological dysfunction and acute vascular insufficiency associated with venous or arterial TOS are also indications for surgical treatment.

The goal of surgery is to decompress the brachial plexus by relieving the tension of the scalene muscles. Scalene muscle excision or first rib excision are the most recommended surgical procedures. It is unlikely that the first rib itself is a causative factor in the majority of neurogenic TOS patients.

First rib resection and scalenectomy are applied through a transaxillary incision under general anesthesia. Positioning and retraction are the most important aspects of obtaining an adequate surgical field of view [33]. Sheth et al. involved 55 participants with the 'disputed type' of TOS in their research. They compared the efficacy of transaxillary first rib resection with supraclavicular neuroplasty of the brachial plexus. Transaxillary first rib resection was more effective in pain control than supraclavicular neuroplasty of the brachial plexus. There were no adverse effects in either group [34].

Anticoagulation and symptomatic treatment is the mainstays of venous TOS treatment. Symptomatic treatment includes compression and pain control. Complications related to anticoagulation are a significant rate of residual disability, recurrent thrombosis, and persistent symptoms. Prolonged anticoagulation is limiting the lifestyle of the most affected young males. Catheter–directed thrombolysis became available as a treatment option in the 1970s [35]. The vein is accessed percutaneously in an antegrade fashion into the clot with an attempt to cross the thrombus. A catheter is placed across the lesion.

Recombinant tissue plasminogen activator is infused from the catheter for clot lysis. A control venogram should be performed to demonstrate vein patency [10].

Pharmacomechanical thrombolysis is an alternative to catheter–directed thrombolysis that involves a catheter coupled to a mechanical device that assists clot fragmentation. Efficacy of thrombolysis is reported to be similar for both methods. However, pharmaco-mechanical thrombolysis is the method of choice as it requires less time for clot lysis [36]. The most important factor for the effectiveness of thrombolysis is the symptom duration. The rate of clot dissolution decreases to 29% in patients treated 2-12 weeks after symptom development [37]. Short term benefits of thrombolysis are apparent. However, there is a high recurrence rate. A recent research reported 23% recurrence within 13 months after thrombolysis [38]. First rib resection and scalenectomy are currently recommended for patients with residual venous stenosis or in the presence of demonstrable extrinsic venous compression.

The goal of arterial TOS treatment is to restore distal arterial blood flow. First rib resection and scalenectomy and sometimes reconstruction of the diseased segment of the subclavian artery are the surgical management options. Delays in treatment may lead to a sympathetically mediated chronic pain syndrome.

Conclusion

TOS is a group designation for several different disorders that affect axillary artery or vein or components of the brachial plexus. Therefore, TOS is almost unique in that it is used to describe disorders that affect both blood vessels and nerve fibers. Various medical specialties can encounter TOS within their field of expertise. TOS should not be viewed as a single clinical entity that simply manifests variations from one patient to another. TOS is composed of three very discrete subgroups, and treatment should be individualized after diagnosis is definite.

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