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Essentials of thoracic outlet syndrome: A narrative review

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Abstract

Thoracic outlet syndrome (TOS) is a group of diverse disorders involving compression of the nerves and/or blood vessels in the thoracic outlet region. TOS results in pain, numbness, paresthesia, and motor weakness in the affected upper limb. We reviewed the pathophysiology, clinical evaluation, differential diagnoses, and treatment of TOS. TOS is usually classified into three types, neurogenic, venous, and arterial, according to the primarily affected structure. Both true neurogenic and disputed TOS are considered neurogenic TOS. Since identifying the causative lesions is complex, detailed history taking and thorough clinical investigation are needed. Electrodiagnostic and imaging studies are helpful for excluding other possible disorders and confirming the diagnosis of true neurogenic TOS. The existence of a disputed TOS remains controversial. Neuromuscular physicians tend to be skeptical about the existence of disputed TOS, but thoracic surgeons argue that disputed TOS is under-diagnosed. Clinicians who encounter patients with TOS need to understand its key features to avoid misdiagnosis and provide appropriate treatment.

Key Words: Brachial plexus; Diagnosis; Review; Thoracic outlet syndrome; Treatment; Neuromuscular lesions

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Core Tip: Thoracic outlet syndrome (TOS) is a group of disorders involving compression of the nerves and/or blood vessels in the thoracic outlet region. TOS is classified into three types, neurogenic, venous, and arterial, according to the primarily affected structure. In true neurogenic TOS, the T1 nerve root is more susceptible to damage than the C8 nerve root. The disputed TOS is categorized as a type of

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neurogenic TOS, but the diagnostic criteria for disputed TOS are lacking and controversial. Before confirming a diagnosis of disputed TOS, clinicians should exclude all other disorders that share common symptoms and features with disputed TOS.

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INTRODUCTION

Thoracic outlet syndrome (TOS) is a group of disorders involving compression of the nerves and/or blood vessels in the thoracic outlet region (*i.e.*, the lower neck and upper chest areas)[1,2]. The structures involved in TOS are the first rib, scalene muscles, and clavicle. Local compression by these structures affects the brachial plexus and subclavian vessels[1,2]. This disorder was first reported by Rogers in 1949, although it was more precisely characterized and first termed “TOS” by Rob and Standeven in 1958[3]. TOS can be classified into three types, neurogenic, venous, and arterial, according to the primarily affected structure[4]. Neurogenic TOS accounts for over 90% of cases, followed by venous (approximately 3%-5%) and arterial (1%) TOS [5]. Recently, TOS has been classified as arterial vascular, venous vascular, true neurologic, traumatic neurovascular, and disputed types[6].

TOS is not a rare disorder. Several studies reported its incidence as 3-80 per 1000[7]. However, since its diagnostic criteria have not been elucidated, it is unclear whether the current epidemiologic data underestimate or overestimate the condition. TOS usually develops in patients in their third to fifth decades of life and more commonly in women[8]. The degree of TOS symptoms can be severe. In clinical practice, it is frequently misdiagnosed as radicular pain, other entrapment syndromes, or muscle- or tendon-origin pain[9]. Treatment for TOS varies depending on the different causes. Diagnostic accuracy is important because appropriate treatment for TOS shows good outcomes in many cases[5].

Here, we described the essentials of TOS by focusing on its pathophysiology, clinical evaluation, differential diagnoses, and treatment.

PATHOPHYSIOLOGY

TOS develops because of anatomical variations that compress the brachial plexus and the subclavian-axillary vessels in the area of the thoracic outlet. It most commonly occurs in the intercostal-scalene triangle, costoclavicular space, and retro-coracopectoral space[10]. As these areas are small, any deformities or variants of the muscle or bone can compress the nerves or vessels that pass through them, resulting in neurological and vascular symptoms[10]. Congenital muscle deformities or variants, such as hypertrophy of the scalene muscles, decreased tone, or shortening, contribute to the development of TOS in approximately 70% of cases. While those of the bone, such as ectopic cervical ribs and prominent C7 transverse processes, are known to cause the other 30% of cases[5,11,12].

Trauma can also induce TOS development[13,14]. Deformation of the bone or cervical plexus after trauma can trigger compression of the neurovascular structure. Fibrosis around the scalene muscles after trauma is known to cause mechanical compression[13,14]. Minor trauma from repetitive exercises or poor body posture can also induce TOS[15]. Muscle imbalances and postural abnormalities can cause TOS, which in turn may induce continuous mechanical irritations[15].

TRUE NEUROGENIC AND DISPUTED THORACIC OUTLET SYNDROME

The most frequent cause of TOS is brachial plexus compression. If there are objective findings of nerve compression, it is called a true neurogenic TOS[6]. However, when

there is no specific pathological evidence of TOS, it is classified as disputed TOS. True neurogenic TOS is a very rare disorder with a prevalence of one in a million[16]. One out of 20000-80000 individuals with cervical ribs has a true neurogenic TOS. Thus, most people with cervical ribs are asymptomatic[1]. However, when clinical symptoms and electrodiagnostic tests suggest lower brachial plexopathy, the possibility of true neurogenic TOS due to cervical ribs increases. Nerve compression by the fibrotic band is also a common cause of true neurogenic TOS. The location where the fibrotic band and brachial plexus meet are more common in the distal part of the anterior primary rami of C8 and T1 rather than the proximal part of the lower trunk[17]. Since the fibrotic band is usually located below the T1 anterior primary rami, the T1 nerve root is more susceptible to damage than the C8 nerve root[17]. The atrophy in muscles innervated by the T1 nerve root is more pronounced than that innervated by the C8 nerve root. Therefore, thenar atrophy is more common than claw hand deformity. Likewise, the sensory symptoms tend to be more frequent in the T1 segment (medial side of the forearm) than in the C8 segment (the fourth and fifth fingers and medial border of the hand).

Disputed TOS is synonymous with various terms, such as nonspecific, aspecific, assumed, pejorative, symptomatic, and subjective TOS. The use of these terms needs to be unified in the future[18]. Disputed TOS refers to symptoms caused by dynamic compression of the brachial plexus. Most neurogenic TOS cases are known to be disputed TOS[2]. The pathophysiology of disputed TOS cannot be explained by a single theory, and various theories have been proposed[2,19]. First, it is explained that clinical symptoms can occur due to anatomical variations, such as true neurogenic TOS. Second, fibrosis of the scalene muscles after trauma can cause compression of the brachial plexus. Third, intermittent nerve compression due to muscle imbalance and postural abnormalities can cause a disputed TOS. However, it is difficult to determine the exact cause of the disputed TOS. In addition, since diagnostic criteria have not been established, all other disorders that share similar symptoms and features to disputed TOS should be excluded before the diagnosis is confirmed. Due to the difficulty of diagnosing disputed TOS, there are many cases where other disorders are misdiagnosed as disputed TOS in clinical practice.

CLINICAL EVALUATION

The clinical symptoms of patients with TOS vary according to their pathology. The symptoms of neurogenic TOS include neuropathic pain, numbness, and paresthesia in the fingers, and upper-extremity weakness[20]. On physical examination, pain is induced on palpation above the entrapped site, such as the brachial plexus, scalene muscles, and anterior chest wall. Symptoms in neurologic TOS are usually worse with exertion, such as upper-limb overloading or excessive extension[21]. In venous TOS, upper-limb edema is the most typical symptom, while severe non-radicular pain can precede a few days earlier[21]. Physical examination may reveal cyanotic coloration of the upper limb and dilated superficial veins in the upper limb and neck. In contrast, patients with arterial TOS can experience non-radicular upper-limb pain, numbness, and discomfort that increase with physical exercise and are relieved with rest[22]. Physical examination frequently revealed coldness and paleness of the involved upper limb. However, there are no pathognomonic signs or symptoms for any type of TOS.

On the basis that TOS symptoms are aggravated by a specific posture, a variety of triggering examinations, including the Adson, Wright, and Halsted maneuvers, the elevated arm stress test (EAST), and the upper limb tension test (ULTT), can be used to diagnose TOS (Table 1)[23,24]. The Adson, Wright, and Halsted maneuvers are tests in which the clinician palpates the radial pulse on the affected upper limb and evaluates for a decreased pulse in specific postures. In contrast, the EAST and ULTT are used to evaluate the presence of symptom provocation in specific postures. Of these tests, the EAST is the most commonly used. The EAST is performed with the bilateral shoulders in 90° abduction, external rotation, and 90° elbow flexion[25]. The patient then slowly opens and closes their hands at 2 s intervals for 3 min. The EAST posture constricts the costoclavicular space. If the test provoked a patient's usual symptoms, it was considered positive. However, there is still no consensus on the use and diagnostic accuracy of the above triggering maneuvers as the gold standard in the diagnosis of TOS[26]. In addition, the above maneuvers have high false-positive and false-negative rates[25]. Diagnosing TOS using only the findings of triggering maneuvers can lead to an inaccurate diagnosis. Therefore, a comprehensive approach combining clinical presentations and careful assessment of the triggering maneuvers is necessary. Electro-

Table 1 Diagnostic tests for thoracic outlet syndrome

Test	Maneuver	Positive result
Adson maneuver	The affected arm is abducted 30° at the shoulder and maximally extended. The patient extends the neck, turns the head toward the symptomatic shoulder, and inhales deeply	Decrease or absence of ipsilateral radial pulse
Wright maneuver	The shoulder on the symptomatic side is abducted above 90° and externally rotated	Decrease or absence of ipsilateral radial pulse
Halsted maneuver	The affected arm is abducted, extended to 45°, and externally rotated. The examiner applies downward traction to the arm and turns the patient's neck away from the affected side	Decrease or absence of ipsilateral radial pulse
EAST (Roos test)	The arms are placed in the surrender position with shoulders abducted to 90° and in external rotation and the elbows flexed to 90°. The patient slowly opens and closes the hands for 3 min	Provoking pain, paresthesia, heaviness, or weakness
ULTT	Position 1: Arms abducted to 90° with elbows flexed; Position 2: Active dorsiflexion of both wrists; Position 3: Head is tilted ear to shoulder in both directions	Positions 1 and 2 elicit symptoms on the ipsilateral side, while position 3 elicits symptoms on the contralateral side

EAST: Elevated arm stress test; ULTT: Upper limb tension test.

diagnostic and imaging may also help confirm the diagnosis of TOS.

In a nerve conduction study (NCV) in patients with TOS, slow conduction velocity can manifest in the medial cutaneous nerve of the forearm and the motor part of the median nerve. In electromyography (EMG), denervation potentials can be revealed in muscles innervated by the C8 and T1 roots (the latter more than the former)[27,28]. These abnormal findings in the electrodiagnostic test can be manifested in the true TOS. However, in patients with disputed TOS, no abnormal findings may be found on the NCV and EMG tests. Most cases of TOS are disputed. Therefore, electrodiagnostic tests are used to differentiate other disorders that share similar symptoms and features with TOS, such as cervical radiculopathy, cubital tunnel syndrome, or ulnar tunnel syndrome[27,28].

Ultrasonography (US), magnetic resonance imaging (MRI), and computed tomography (CT) can be helpful in the diagnosis of TOS. US is a relatively inexpensive and easily accessible diagnostic tool. Therefore, it is frequently used as an initial imaging examination[29]. With US, clinicians can identify causative lesions, such as tumors, fibrosis, and posttraumatic sequelae. Dynamic US examinations can also help diagnose TOS. Chang *et al*[30] found that the lateral cord was interposed between the subclavius muscle and subclavian artery in a patient with TOS. When the patient abducted their shoulder to more than 90°, the lateral cord flattened between the two structures. The injection of local anesthetics under US guidance into the supposed pathological lesion can also be used to successfully diagnose neurogenic TOS. In addition, Moore *et al*[31] reported that duplex scans have a high diagnostic accuracy for detecting venous stenosis or occlusion. Radiographs are taken to detect ectopic cervical ribs or prominent C7 transverse processes. MRI can identify a fibrotic band, edema, or high signal intensity of the brachial plexus due to compression by the former[32-34]. Moreover, CT angiography can be useful for localizing compression of the subclavian artery and inferring compression of the brachial plexus[35]. Furthermore, local stenosis or upward angulation of the subclavian artery are suggestive findings of TOS[35].

DIFFERENTIAL DIAGNOSES

TOS is frequently misdiagnosed because other disorders show similar symptoms. Therefore, to confirm TOS, other disorders such as cervical radiculopathy, inflammatory neuropathy, and other nerve entrapment syndromes should be excluded[9]. The disorders that should be ruled out before confirming the diagnosis of TOS (along with their distinguishing clinical features) are shown in Table 2.

Table 2 Differential diagnoses for thoracic outlet syndrome and their distinguishing clinical features

Disorder	Distinguishing features
Raynaud's syndrome	Cold fingers, color changes in the skin in response to cold or stress that are relieved by warmth
Vasculitis	Severe sudden-onset pain involving more than one limb, elevated C-reactive protein level, skin lesion (<i>e.g.</i> , purpura, petechiae, ulcer)
Rotator cuff tear	Pain during shoulder movement that is easily differentiated by ultrasound
Cervical radiculopathy	Acute pain (disc rupture), insidious onset (spinal stenosis), spurling sign (+), denervating potential of cervical paraspinalis on electromyography
Cubital tunnel syndrome	Tinel sign (+) over cubital tunnel; Differentiated by nerve conduction study
Guyon's canal syndrome	Tinel sign (+) over Guyon's canal; Differentiated by nerve conduction study
Neuralgic amyotrophy	Extreme sudden-onset pain followed by rapid motor weakness and atrophy
Pancoast tumor	Pain in the shoulder radiating to the inner part of the scapula, possible Horner syndrome, tumor on the apex of the lung
Complex regional pain syndrome	Diffuse pain, predominant vasomotor features, history of stroke, trauma, or peripheral nerve injury

TREATMENT

Many patients with TOS symptoms are relieved by conservative treatment. Therefore, they are initially treated conservatively. Therapeutic strategies depend on the type of TOS. A consensus on appropriate conservative therapeutic methods is lacking. However, rehabilitation therapy, including patient education (postural mechanics, relaxation techniques, and weight control), exercise (stretching and strengthening of targeted muscles), and activity modification, are usually applied as initial treatment. Oral pain medications can be used to relieve neuropathic pain[36]. Botulinum toxin injections into the scalene muscles and/or pectoralis muscle and corticosteroid injections into the pathologic areas have also been shown to be useful for managing neurogenic TOS[37]. If the patients do not respond to these conservative treatments, surgical treatment, such as first rib and/or cervical rib resection, may be considered.

Thrombolysis with continuous infusion of a plasminogen activator is most commonly used for the treatment of venous TOS[38,39]. However, anticoagulants have resulted in several complications, such as recurrent thrombosis events, persistent pain, and restricted movement of the upper limbs[40].

However, thrombolysis or embolectomy is the most effective treatment for arterial TOS[9]. For patients with mild acute arterial ischemia due to embolization, catheter-directed thrombolysis is appropriate prior to surgical treatment. However, the presence of more severe ischemia usually requires embolectomy with or without intraoperative thrombolysis, in conjunction with thoracic outlet decompression.

In the minority of patients who do not respond to the less invasive treatments above, surgical treatment such as vascular and/or nerve decompression should be considered.

ONGOING DEBATES ON DISPUTED TOS

The diagnosis of a disputed TOS is based on clinical features. Even in recently published papers, consistent diagnostic criteria for disputed TOS have not been suggested[41-43]. Most articles demonstrating the results of surgical treatment for disputed TOS have described that the diagnosis of disputed TOS should be made after identifying structural abnormalities and discriminating other diseases by performing cervical spine and chest radiographs, electrodiagnostic tests, CT angiography, and cervical MRI, before surgery. A few studies have reported that diagnostic nerve blocks using local anesthetics may be helpful for diagnosis[42]. Advocates of disputed TOS stress that provocative tests are important for diagnosis. However, the provocative tests used for each study are different. Among the provocative tests, the EAST test is the most commonly used but is known to have a very high false-positive rate. According to one report, when a provocation test was performed on 150 normal subjects, almost all patients developed symptoms within the 2 min[44]. In addition, it is questionable whether the induced posture performed in the EAST increases tension in the lower part of the brachial plexus. This test was designed by Roos[45] in 1966 to

check for the presence or absence of arterial obstruction according to posture and is currently used as an important provocative test for neurogenic TOS. Due to the ambiguity of the available diagnostic tests, there have been many controversies in the diagnosis of disputed TOS. To resolve this, consistent diagnostic criteria are warranted. Moreover, the treatment results according to these criteria should be presented.

Advocates argue that disputed TOS is justified for surgical treatment because the clinical manifestations may be worsened. However, there are few reports on the natural course of disputed TOS and the long-term prognosis of conservative treatment. Laulan described that in 30 years of clinical experience, only one patient progressed from disputed to true neurogenic TOS[19]. Considering that true neurogenic TOS is a very rare disease with a prevalence of approximately one per one million individuals [2,46], it can be assumed that the probability of clinical progression of disputed TOS is likely to be very low. To justify surgical treatment, a long-term follow-up study involving randomization of conservative treatment and surgical treatment is necessary.

CONCLUSION

In clinical practice, we may encounter many patients with upper limb pain with or without motor weakness. Of these, only a few are likely to have TOS. To ensure diagnostic accuracy and appropriate treatment, clinicians should have some knowledge of TOS. This review aimed to provide the essential knowledge for clinicians to diagnose and manage TOS in clinical practice.

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