Duplex Ultrasound in the Evaluation of Venous and Arterial Thoracic Outlet Syndrome

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Abstract

Thoracic outlet syndrome (TOS) is caused by neurovascular bundle compression in the thoracic outlet space. Three pertinent structures may be compressed: brachial plexus, subclavian artery, and subclavian vein. Clinical presentation may be variable depending on the anatomical structure involved. We report a 36-year-old man with chronic recurrent pressure-quality left chest discomfort. Comprehensive cardiac investigations were nonrevealing. A venous and arterial duplex ultimately revealed dynamic venous and arterial compression. Our paper is to illustrate the sonographic approach to evaluate vascular compression in the thoracic outlet. We review the mechanisms of compression, and examination maneuvers to facilitate a Doppler-based diagnosis.

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Duplex in Thoracic Outlet Syndrome

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Abstract

Thoracic outlet syndrome (TOS) is caused by neurovascular bundle compression in the thoracic outlet space. Three pertinent structures may be compressed: brachial plexus, subclavian artery, and subclavian vein. Clinical presentation may be variable depending on the anatomical structure involved. We report a 36-year-old man with chronic recurrent pressure-quality left chest discomfort. Comprehensive cardiac investigations were nonrevealing. A venous and arterial duplex ultimately revealed dynamic venous and arterial compression. Our paper is to illustrate the sonographic approach to evaluate vascular compression in the thoracic outlet. We review the mechanisms of compression, and examination maneuvers to facilitate a Doppler-based diagnosis.

Key word : thoracic outlet syndrome; duplex ultrasound; vascular imaging

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Introduction:

Thoracic outlet syndrome (TOS) is a constellation of symptoms in the shoulder and upper extremity that may present as pain, dysesthesia, weakness, edema, and pallor resulting from total or partial compression of the neurovascular bundle exiting the space between the first rib and the clavicle, known as the thoracic outlet. TOS is classified into three subgroups according to its pathophysiology: neurogenic TOS (nTOS), arterial TOS (aTOS), and venous TOS (vTOS). Among them, nTOS accounts for more than 90% of the cases, followed by vTOS accounting for 3-5%, and aTOS accounting for 1-2% [1]. Venous TOS, an uncommon subtype, occurs as a result of extrinsic compression and subsequent stenosis of the subclavian vein. It is a predominantly unilateral and is often associated with repetitive upper extremity motion. According to previous reports, anomalous anatomy, repetitive movement-induced swelling with microhemorrhage and subsequent fibrosis, compressive soft tissue masses, deep-venous thrombosis can be initiators of vTOS [2-5]. If the vTOS is caused by a spontaneous deep vein thrombosis (DVT), it is referred to as Paget-von Schröetter Syndrome.

Case Illustration:

A 37-year-old man with a past medical history of pulmonary sarcoidosis, melanoma in remission, and former tobacco smoking presented with recurrent pressure-quality left chest discomfort with radiation to neck and shoulder, accompanied by left arm numbness and tingling, intermittently aggravated by shoulder movement. Physical examination revealed tenderness along the medial scapula, reduced abduction of the left shoulder, minimal pain with impingement testing including Neer and Hawkins exam, negative cross-body and O'Brien's exams (Table 1). Complete blood count, comprehensive metabolic profile, and cardiac biomarkers were normal. Cardiac work up was negative, including electrocardiogram and stress echocardiogram. Computed topography of chest and abdomen showed noncompressive lung and liver lesions, in keeping with known sarcoidosis. Magnetic resonance imaging (MRI) of cervical spine, shoulder, and brachial plexus were negative. His symptoms failed to improve with physical therapy.

A vascular duplex of left upper extremity was performed showing compression of both the subclavian vein and artery. The subclavian vein was assessed with color and spectral Doppler in the neutral position, at 90° abduction, and at 180° abduction. At both 90° and 180° abduction, there was loss of cardiac pulsatility and respiratory phasicity, but continuous flow remained, indicating compression without obstruction (Figure

1). Halstead maneuver was also performed to illicit obstruction (Table 1). This position is performed by adopting a military posture with the arms pulled back and chest protruded forward. There is complete flow obstruction provoked by this maneuver (Figure 2). Interrogation of the subclavian artery was performed with shoulder abduction and Halstead maneuver. Diminished peak systolic velocities and pulse volume recordings with provocation indicate arterial compression (Figures 3 and 4).

The patient failed conservative therapy, including non-steroids anti-inflammatory drugs (NSAIDS), Lidocaine patch, and physical therapy. He underwent left thoracic outlet decompression with trans-axillary resection of the first rib and division of the anterior scalene muscle (Figure 5). Three months after surgery, he had drastic improvement in symptoms with near complete resolution.

Discussion:

TOS is usually caused by extrinsic compression and subsequent stenosis of the neurovascular structure. An anatomical abnormality is the typically fundamental prerequisite, including a cervical rib, anomalous first rib, or anomalous scalene muscle [6]. Additionally, injuries such as hyperextension/flexion injury of neck and bony fracture have been described [7, 8]. Thrombosis and tumor are also potential etiologies. The vTOS composed of 3-5% cases of TOS. It is more common in younger (aged 14-45), able-bodies individuals, and most often affects the dominant upper extremity [9]. The vTOS is also grouped into 3 categories: intermittent positional stenosis/obstruction, secondary subclavian vein thrombosis due to catheter placement, and effort thrombosis [3].

The symptoms of nTOS include pain, dysesthesia, numbness, and weakness involving the distribution area of C5 through T1 brachial plexus nerves. The aTOS can present with hand ischemic symptoms like pain, pallor, paresthesia, and coldness. The vTOS presents with upper extremity edema, accompanied by pain and cyanosis. Symptoms may be atypical and elusive, as illustrated in our patient with recurrent chest pressure, masquerading as angina pectoris. A clue to diagnosis was the precipitant of symptoms triggered by arm movement. As such, he was referred for an upper extremity venous duplex with examination maneuvers to temporarily occlude the neurovascular structure. Electrodiagnostic testing via nerve conduction and electromyography (EMG) are indicated for suspected nTOS. For vTOS and aTOS, Doppler ultrasound is the primary test of choice due to real-time assessment of dynamic Doppler signals associated with compressive maneuvers, yielding a high sensitivity and specificity[10]. Secondary tests include CT or MRI to define anatomical defects, such as prominent cervical rib or compressive tumors and their anatomical relationship to the thoracic outlet[10].

Treatment strategy of TOS depends on the underlying etiology, and can involve conservative or surgical measures. Cervical rib or anomalous rib without symptoms only need observation. Therapeutic strategies for nTOS include physical therapy with option for interscalene injection, corticosteroids, and botulinum toxin type A. Anticoagulation can be given for thrombosis complicating VTOS or aTOS. Surgical intervention such as thoracic outlet decompression remains the final method conservative management fails. Surgical intervention is often the initial approach for vTOS and aTOS in current practice, with the first rib being resected to decompress the brachial plexus. The procedure is usually performed by transaxillary, supraclavicular, or infraclavicular techniques. Blocking the scalene muscle or pectoralis minor may be done to predict the effect of surgery.

Conclusion:

Buried among a long list of different diagnoses for atypical chest pain is TOS, and a heightened clinical suspicion is an absolute prerequisite. There are a number of physical examination maneuvers to illicit symptoms. If concern remains, a duplex ultrasound with provocative maneuvers can be an efficient and invaluable tool to solidify the diagnosis.

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Table 1: Diagnostic maneuvers:

Test	Maneuver	Interpretation
Special tests for TOS	Special tests for TOS	Special tests for TOS
Adson's test	The shoulders are adducted to 30° in fully extended position. The neck is extended and turned towards symptomatic side. With the patient taking and holding deep breath, the radial artery is palpated and compared to baseline.	Positive test: diminished or loss of radial artery pulsation.
Cyriax release test	With patient in sitting or standing position, the forearms is grasped from under, the elbows is held at 80° of flexion with the forearms and wrists in neutral. The trunk is leaned posteriorly and the shoulder girdle is passively elevated. This position is held for up to 3 minutes.	Positive test: paresthesia and/or numbness (release phenomenon) occur, including reproduction of symptoms.

Test	Maneuver	Interpretation
Halstead maneuver (costoclavicular maneuver or exaggerated military brace test)	With patient in standing position, both shoulders are drawn back, the humerus is extended back, and the chest is protruded forward. The radial artery is palpated and compared to baseline.	Positive test: diminished or loss of radial artery pulsation.
Roos test (Elevated arm stress test)	Both shoulders are adducted to 90°, then the elbows are flexed to 90°. Hands are pumped over 3 minutes.	Positive test: symptoms reproduced; inability to complete test due to symptom limitation; arm pallor in aTOS; cyanosis and swelling in vTOS.
Supraclavicular pressure	Examiner places fingers between the anterior scalene and upper trapezius muscle, and squeeze for 30 seconds.	Positive test: paresthesia indicates brachial plexus compression through the scalene triangle.
Wright test (hyperabduction test)	With patient in sitting position, the arm is passively brought into abduction and external rotation to 90° without tilting the head, the elbow is flexed no more than 45°, then the arm is held for 1 minute.	Positive test: decrease in the radial pulse and/or reproduction of the patient's symptoms.
Other tests in the article	Other tests in the article	Other tests in the article
Cross-body test	The shoulder is forward flexed to 90°, then adducted horizontally across the chest.	Positive test: pain over the acromioclavicular joint, indicating acromioclavicular joint degeneration/arthritis.
Hawkins test	The patient's arm is placed in 90° of shoulder flexion with the elbow flexed to 90° and then the arm is internally rotated.	Positive test: pain with internal rotation, indicating subacromial impingement.
Neer test	With patient in standing position and arm at side of body with elbow fully extended, the arm is internally rotated and forcefully moved through the full range of forward flexion or until reports of pain.	Positive test: pain in the anterior – lateral aspect of the shoulder, indicating subacromial impingement.

Test	Maneuver	Interpretation
O'Brien's test	With the patient in sitting or standing, the arm is placed in 90° of shoulder flexion and 10-15° of horizontal adduction, then the shoulder is fully internally rotated and the elbow is pronated with a distal stabilizing force is applied by examiner as the patient is applying an upward force. The test is then repeated but with the arm in neutral rotation.	Positive test: pain and/or clicking when the arm is in full internal rotation but not when the arm is in neutral rotation, indicating labral (SLAP Lesion) or acromioclavicular lesions.

Figure 1. Left subclavian vein with adduction

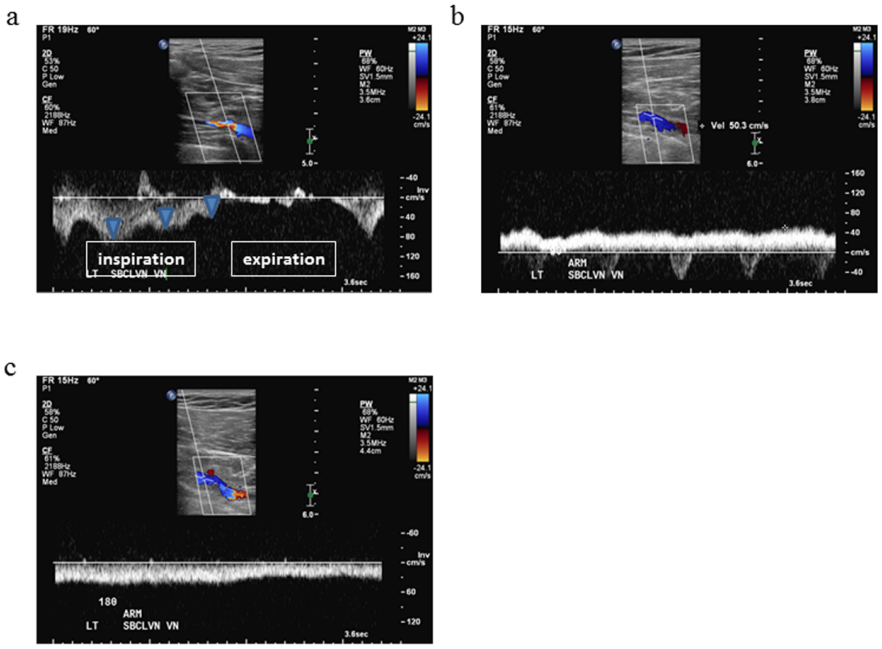

Figure 2. Left subclavian vein with military position

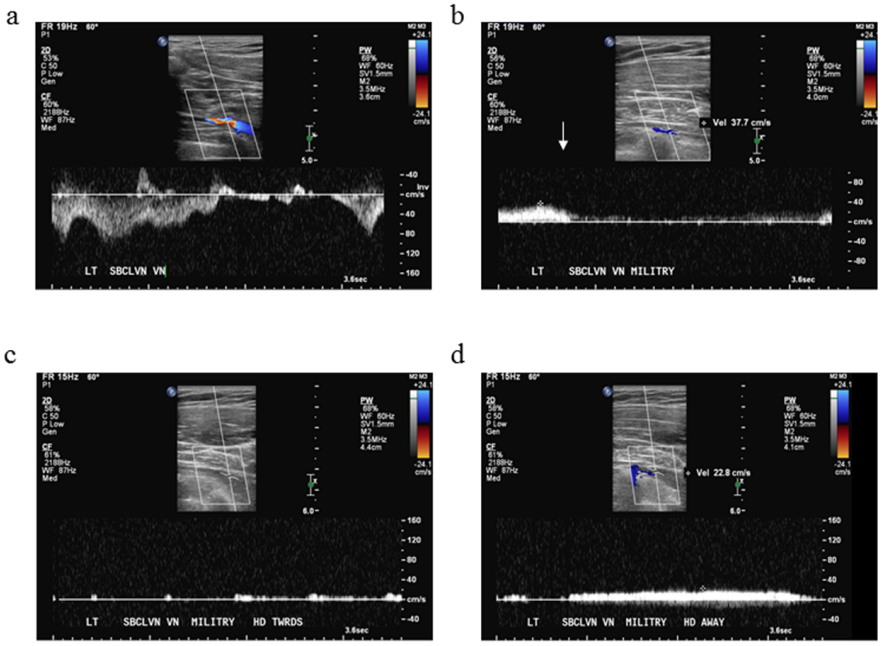

Figure 3. Left subclavian artery with Adson's maneuver

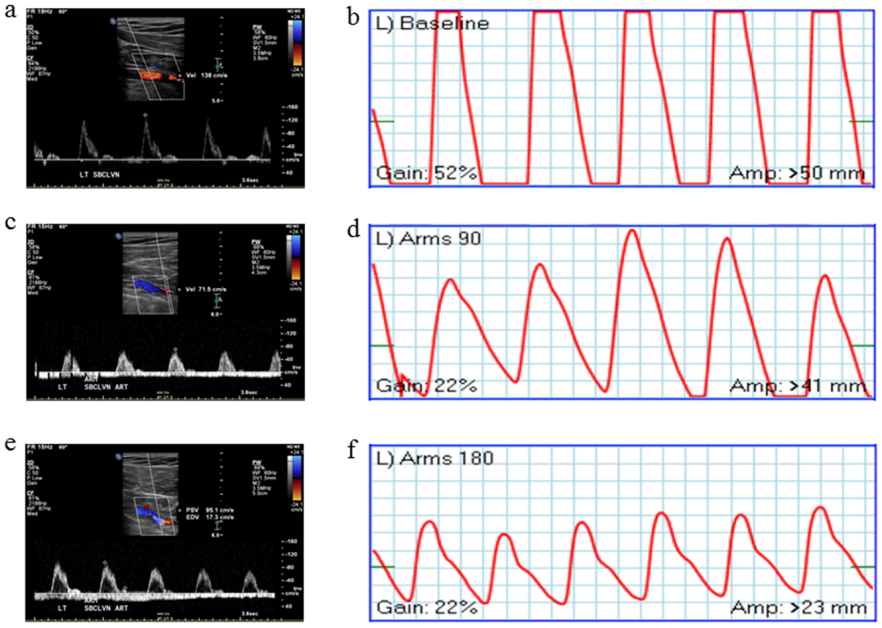

Figure 4. Left subclavian artery military position

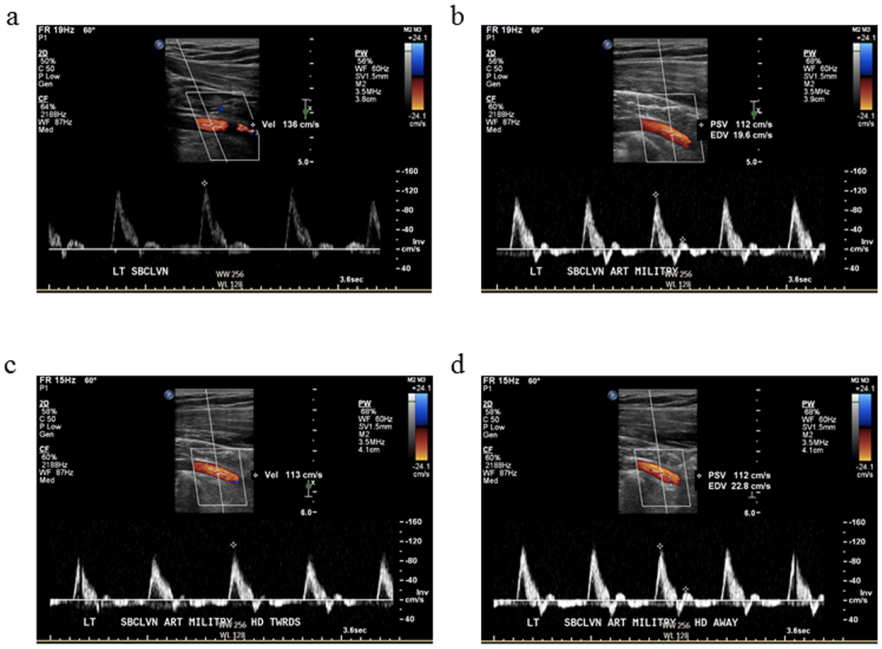

Figure 5. Preoperative computed tomography 3D reconstruction