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Invited Review

# Pectoralis minor syndrome

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***ABSTRACT***

Pectoralis minor syndrome (PMS) is defined as compression of the brachial plexus (BP) nerves, axillary artery, and axillary vein under the pectoralis minor muscle. The symptoms of PMS resemble supraclavicular compression of the neurovascular bundle, with shoulder, neck, chest, and arm pain, and paresthesia and weakness in the arm and hand. The diagnosis of PMS can be confused with other upper extremity pain syndromes. A detailed history, including occupation, daily activities, sports, and trauma, is critical in the diagnosis, together with physical examination findings. Radiological examinations, including direct radiography, computed tomography, magnetic resonance imaging, and electrophysiological tests, are also helpful for the differential diagnosis. Arterial and venous Doppler ultrasound, including dynamic investigation, can display arterial and venous compression. Injection tests are used to confirm the definitive diagnosis. Conservative treatment is successful in most patients, and surgical treatment is considered in unresponsive cases.

***Keywords:*** Axillary artery, botulinum toxin, brachial plexus, pectoral muscles, thoracic outlet syndrome.

Thoracic outlet syndrome (TOS) is the entrapment of the upper extremity neurovascular structures from the cervical region to the axilla, causing pain, paresthesia, weakness, numbness, swelling, coldness, tingling, and discoloration.[1] It is classified in several ways. According to one, neurovascular compression can occur at three anatomic levels: the interscalene triangle, the costoclavicular space, or the pectoralis minor (PM) space. The brachial plexus (BP) and subclavian artery can be trapped in the interscalene triangle. The subclavian vein is not located between scalene muscles; it runs anterior to the triangle. The costoclavicular space is the other entrapment area where the subclavian artery, vein, and BP pass through. Most subclavian vein compression is seen in this region. The PM space is just behind the PM muscle and where it attaches the coracoid process. Pectoralis minor syndrome (PMS) develops when the BP nerves, axillary artery (AA), and axillary vein (AV) compression occurs in the PM space.[1,2] According to another classification of TOS, compression occurs above or below the clavicle. Compression above the clavicle, occurs in the interscalene triangle and costoclavicular area, producing TOS; below the clavicle, compression occurs in the PM space, producing PMS.[2,3]

In general, neural structures are more affected than vascular structures. Sanders and Annest[1] reported more than 90% of cases involving BP, 5% involving venous obstruction, and 1% associated with arterial obstruction in TOS. Echoing this study, the literature has reported rare cases of vascular PMS.[3-6] The frequency of TOS in the general population is not known precisely. There are also quite contradictory results in the literature regarding the frequency of neurogenic and vascular TOS and PMS, since the diagnostic and classification criteria are not fully established.[1,2,7,8] The etiology is similar for TOS and

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PMS, except for abnormal cervical ribs. The causes of TOS and PMS include acute traumas, repetitive daily activities, exercises, sports, and occupation.[8,9] Commonly, PMS accompanies TOS. Recent studies have highlighted the potential importance of neurovascular compression under the PM muscle tendon as part of the spectrum of neurogenic TOS. Sanders and Rao[2] reported that TOS and PMS could be seen together in most cases, and pure PMS was present in 30% of the cases showing TOS symptoms. In another study emphasizing the importance of PMS in the presence of TOS symptoms, the researchers aimed to evaluate the effectiveness of isolated PM muscle tenotomy compared to supraclavicular decompression (scalenectomy, neurolysis, and first rib resection) combined with PM muscle tenotomy in patients diagnosed with neurogenic TOS.[9] The authors found similar outcomes between the two groups and emphasized the importance of recognizing PMS to treat neurogenic TOS successfully. These results are significant and emphasize the importance and frequency of PMS in patients presenting with symptoms of TOS; however, conservative treatment is usually sufficient in PMS.[4,10,11] It is essential to distinguish the entrapment site for appropriate treatment; therefore, diagnostic injection tests are used to confirm a diagnosis. Differential diagnosis is also crucial, which can be challenging in patients with other disorders causing neck, back, shoulder, and arm pain. In this review, we discuss the etiology, clinical presentation, diagnostic methods, differential diagnosis, and treatment of PMS in the light of the current literature.

## Etiology and pathophysiology

The PM muscle arises from the anterior surface of the third, fourth, and fifth ribs and attaches to the coracoid process of the scapula. The PM muscle forms a "bridge" over the neurovascular structures passing from the thorax into the upper extremity. In addition to protecting the neurovascular structures during their course, the PM muscle is a scapula protractor and stabilizes the scapula by drawing it downward.[1,2] Occupational and daily living activities, sports such as rowing, swimming, baseball, volleyball, and weightlifting with repetitive movements of the upper extremities can result in stretching of the PM muscles. Repetitive trauma leads to the formation of strain and myofascial trigger points in the PM muscle, further shortening the muscle. These pathologies are the potential mechanisms of underlying pain in PM muscle and neurovascular compression.[4,10-12] Repetitive sports or daily activity can also cause vascular PMS.[3-5] The PM muscle, itself, can compress some of the vessels, or the head of the humerus exert pressure on the vascular structures during throwing and forceful overhead movements of the upper extremity. Repetitive compression also leads to intimal injury and subsequent thrombosis.[13] It is usually associated with neurogenic symptoms of PMS due to the proximity of nerves to vascular structures, which causes pressure on the nerves.[4-6] Acute traumas, traffic accidents, hyperextension injuries to the neck, such as whiplash, can also have a role in the etiology of PMS. The pathogenesis of whiplash injury is complex, and reflex muscle spasms may occur in the surrounding tissues after injury. Chronically tight scalene or PM muscles can cause compression of the neurovascular bundle, producing symptoms of TOS.[1,2,10] In a study of 37 patients to investigate the role of myofascial trigger points in PM muscle following a whiplash injury, 27 with myofascial trigger points in the PM muscle had TOS symptoms, and all the complaints were relieved after conservative treatment.[10]

Postural disorders are also significant in the etiology of PMS. The upper crossed syndrome produces elevation and protraction of the shoulders, shortening of the pectoral muscles, and protraction of the head. Recently, this syndrome has been encountered more frequently with the increase in desk-bound occupations and the use of mobile phones. One of the crucial issues to remember is that all these problems do not only affect pectoral muscles. Shoulder joint stabilizers, paraspinal muscles, and surrounding muscles may be affected. Myofascial trigger point formation is encountered in all cervical and periscapular muscles; therefore, all these must be evaluated to tailor the most effective treatment.[14]

Spasticity is another cause of PMS. Spasticity in PM muscles can be observed in upper motor neuron lesions affecting the upper extremity and cervicothoracic region. A case of PMS has been reported in a hemiplegic patient with upper extremity flexor spasticity resulting from a stroke. The patient recovered with ultrasound (US)-guided botulinum toxin (BTX) injections in the PM muscle. The researchers suspected PMS due to numbness, tingling, and weakness in the fourth and fifth fingers of the affected upper extremity. The symptoms were aggravated by pressure applied to the PM muscle, and they confirmed the PMS diagnosis by US-guided PM muscle block.[15]

## Clinical presentation

The duration of the symptoms is reported to be protracted due to slow diagnosis.[1,2,16,17] To avoid overlooking PMS, it is necessary to recognize the symptoms and signs of this syndrome well. There is a broad spectrum of clinical presentation with pain in the arm, shoulder, neck, chest, and scapular region, and weakness, paresthesia, swelling and coldness in the upper extremity.[1,2,10,12] Another important issue is the age range of patients. Pectoralis minor syndrome can be seen in all ages and sexes.[18] When PMS symptoms are observed in children, carrying backpacks and sports activities should be questioned.

Pain is usually the leading symptom encountered in all upper parts of the body, including the chest, neck, and upper extremities. Patients often complain of pain in the supraclavicular area, anterior chest wall, scapular region, and shoulder toward the arm and hand. Chest pain can be present in most patients and, occasionally, mimics the symptoms of angina. Cases of PMS initially treated for ischemic heart disease have been described in the literature.[12,19,20]

A decrease in cervical lordosis is observed in most cases. Neck and chest pain may be accompanied by occipital pain in PMS patients.[1,2] Clinically, when BP branches are trapped above the clavicle, neck pain and headache are more prominent.

Shoulder pain is also very frequent in PMS. Pain all over the arm including muscles of shoulder girdle, trapezius and rhomboid muscles may be observed.[16] Numbness, tingling, and paresthesia are also widespread symptoms. Although paresthesia is common in the fourth and fifth fingers, complaints can be observed in the whole hand. Muscle wasting together with weakness is usually seen in prolonged disease. Weakness of the hand, dropping things, and poor grip strength can be seen.[1,2]

Although neurological symptoms are frequent in vascular entrapment due to the proximity of BP branches to the AA and AV, it shows some differences from pure neurogenic entrapment. Venous PMS is characterized by discoloration, swelling, pain, and heaviness of the affected arm. Venous collaterals of the upper arm, or chest wall can be seen.[6,8] As aforementioned, the subclavian vein can be compressed at the costoclavicular space, and the AV can be compressed under the PM muscle.[3,5,8] Therefore, when there is venous obstruction, these two regions should be considered for entrapment. Axillary artery stenosis due to PMS results in decreased blood flow to the extremity. Loss of endurance during exercise is an early sign of AA stenosis. Arm claudication and, eventually, pain, numbness, and coldness evolve, and digital ulcerations are the evidence of progression.[1,2,4] Intermittent symptoms are seen in non-thrombotic obstructions, which are prominent during the elevation of the extremity and disappear, when the extremity is at rest. The symptoms are more intense and persistent lasting for days or weeks, when thrombosis occurs.[1,4] Pulse examination is of utmost importance and, ideally, objective hemodynamic data with arms positioned to reproduce potentially ischemic symptoms are warranted.

## Physical examination

Pain, paresthesia, and weakness are common in pathologies causing neurogenic compression in several areas of the upper extremity.[21] Compression of the median nerve at the wrist and in the pronator muscle, ulnar nerve entrapment at the elbow, and entrapment of the upper extremity neurovascular structures at the interscalene triangle, costoclavicular, and the retropectoral space can be seen.[1]

Symptoms of nerve entrapments usually mimic each other. Therefore, physical examination should include palpation and a Tinel test at the aforementioned possible entrapment sites to detect tender spots and positive Tinel signs. If the neurovascular structures are trapped under the PM muscle, the symptoms increase by palpation and applying pressure on the pectoral muscles. In addition, pain induced by pressing on the coracoid process is a finding in favor of PMS.[1-3] It should always be kept in mind that double crush syndrome may also occur. In cases accompanied by supraclavicular entrapment, tenderness can be detected by pressure applied to the scalene muscles.[1,2]

Pain usually occurs with shoulder abduction and retraction, when PMS is present. Subacromial impingement maneuvers, such as Neer and Hawkins tests, can yield positive results.[16] Together with these physical examination findings and pain in the shoulder region, the patient may be misdiagnosed with subacromial impingement syndrome (SIS). Although shoulder impingement tests are insufficient for a definitive diagnosis of SIS, they are important in clinical evaluation.[22] A subacromial injection test can be performed for the definitive diagnosis of SIS.[23] Furthermore, SIS may accompany PMS. A study in patients with PMS indicated that subacromial impingement tests were positive in 66.6% of the patients, and 33.3% responded positively to a subacromial injection test.[16]

Subacromial impingement syndrome may be seen in relation to the damage to shoulder muscles and excessive overhead activity that leads to PMS and shoulder impingement.[24] Another reason is the deterioration of shoulder biomechanics due to the shortened or contracted PM muscle. In such painful pathologies, a subacromial injection test can be performed for a the differential diagnosis of SIS.[16,22,23]

The cervical region should be also evaluated carefully. Supraclavicular entrapments and cervical radiculopathy should be excluded.[1,2] Cervical radiculopathy and PMS symptoms are similar, thus, PMS cases are often misdiagnosed as cervical radiculopathy. A case with neck pain and numbness in the upper extremity and positive magnetic resonance imaging (MRI) findings of cervical disc herniation was referred for an electrodiagnostic test. The electrodiagnostic examination was unremarkable in terms of cervical radiculopathy. Re-evaluation with provocation maneuvers and the reproduction of symptoms with pressure on the PM muscle led to an initial diagnosis of TOS. The final diagnosis was changed to PMS, confirmed with a diagnostic injection test.[4]

Swelling, discoloration, cyanosis, and coldness in the hand and entire arm are the main findings of vascular pathologies. Radial and ulnar pulses should be checked.[1,2] Provocation tests can be helpful for the diagnosis of TOS and PMS and should be a part of the physical examination. However, these tests alone should not be taken as definitive evidence of PMS or TOS. The provocation tests are based on the compression of neurovascular structures at the entrapment area. The Adson's test, costoclavicular maneuver, neck rotation test, and head tilt test all help diagnose supraclavicular entrapment of the neurovascular bundle. The physical examination in patients with pure PMS usually lacks vigorous responses and often shows minimal or no response to these maneuvers.[1,2,21] The Wright's test can be used as a provocation test for diagnosing PMS. The idea is to stretch and pull the muscle taut, causing it to further compress the BP and the AA and AV that run under the PM muscle. The Wright's test involves bringing the patient's upper extremity backward into abduction and extension, while the examiner palpates the strength of the radial pulse. An alternative position for the Wright's test is with the arm abducted and the forearm flexed. The test is considered positive, when there is a decrease in radial pulse and an increase in symptoms.[25]

The upper extremity tension test is another valuable test for PMS. This test is performed in three steps: First, the elbows are extended, and the arms are elevated to 90°, parallel to the floor (Figure 1a). The second position is the dorsiflexion of the wrists (Figure 1b). The third position is tilting the head to shoulder to the both side (Figure 1c). Pain and paresthesia start within a few seconds with this maneuver, if BP compression is present.[21,25,26] A 90° abduction or elevated arm stress test, also known as the Roos test, is another provocation test for PMS (Figure 2). The patient has both arms in the 90° abduction external rotation, elbow position to 90° flexion, and waits for 3 min. Flexion and extension of hand fingers are requested. A positive response is the onset of pain or paresthesia within 60 sec. Symptoms often become apparent within 5 to 30 sec, when there is moderate to severe compression.[1,2,4,21,25]

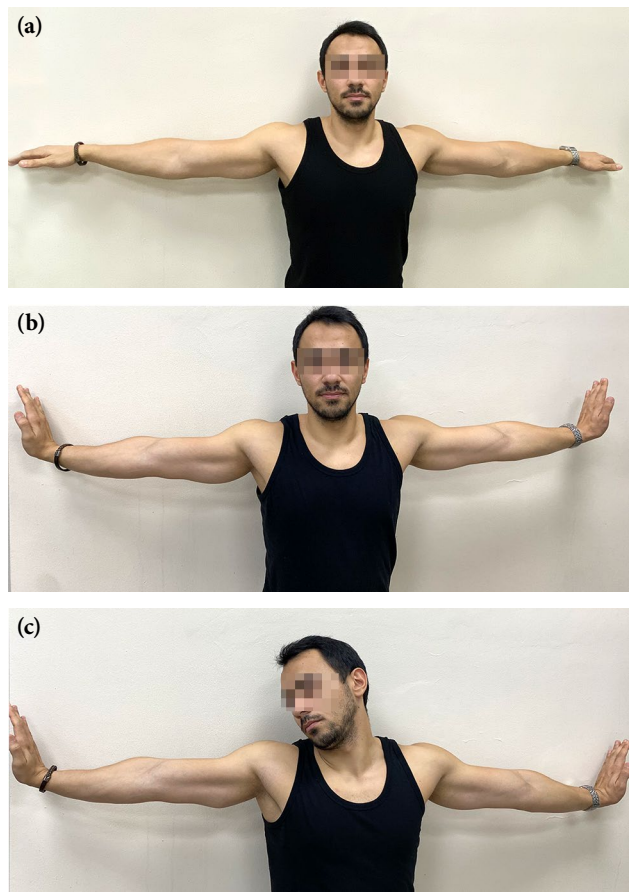


**Figure 1.** Upper extremity tension test. **(a)** Elbows are extended and the arms are parallel to the floor. **(b)** Wrists are dorsiflexed. **(c)** Head is tilted to both sides

**Figure 2.** Roos test.

## Diagnosis

Pectoralis minor syndrome is often overlooked, as it is rarely based on a physician's differential diagnosis. Therefore, the actual frequency of PMS in patients with upper extremity pain and paresthesia symptoms is unknown. An accurate diagnosis is essential for treatment. A detailed history and physical examination are critical to determine the location of the compression of neurovascular structures and differentiating from the diseases that show similar symptoms.[1,2,21] To discover an underlying cause of repetitive stress and injury patients should be questioned about their occupation, sports activities, and exercise habits.

Direct radiography, computed tomography (CT), and MRI are useful for demonstrating structural lesions. The anterior-posterior and lateral X-ray of the cervicothoracic region is a simple and inexpensive way to identify cervical or anomalous first rib.[1,2,21] This finding is significant, when supraclavicular entrapment is suspected. Additionally, the lateral view can show the straightening of the normal lordotic neck curve. A decrease in cervical lordosis may be positive in cervical vertebra problems as well as in PMS and TOS. Although MRI of the BP can show findings of nerve compression, it reveals normal structure in most PMS cases. This imaging tool gives valuable information for cervical disc herniation and spinal or foraminal stenosis.[1,2,4]

Electrodiagnostic studies are diagnostic tools, including electromyography and nerve conduction. In TOS, the reduced amplitude of medial antebrachial cutaneous nerve and ulnar nerve sensory nerve action potentials correlate with the diagnosis.[27] A positive electrodiagnostic test indicative of BP involvement cannot differentiate the compression at the scalene triangle from the PM space. Besides, most electrodiagnostic study findings are within the normal range in subjects with neurogenic PMS and TOS, although the patient has neurological symptoms. Formerly, these patients were diagnosed with disputed neurogenic TOS. Disputed neurogenic TOS was a clinical diagnosis in patients with typical symptoms of TOS and provocation tests eliciting TOS symptoms, but lacking objective evidence of electrodiagnostic studies. Currently, clinical criteria and diagnostic injections have gained importance in diagnosing TOS and PMS. According to the latest criteria, neurogenic TOS/PMS is defined by the presence of three of the following four criteria:[28]

1. Symptoms consistent with irritation at the site of compression; scalene triangle in the case of TOS and pectoralis muscle site in the case of PMS and pain on palpation of the affected area
2. Brachial plexus compression findings such as numbness, pain, paresthesia, vasomotor changes, and weakness (with muscle wasting in extreme cases)
3. Absence of other possible diagnoses (cervical disc disease, shoulder disease, carpal tunnel syndrome, chronic regional pain syndrome, brachial neuritis)
4. Positive injection test.

Electrophysiological studies can be ordered to rule out neurological conditions such as cervical disc herniation, ulnar and medial nerve entrapments, brachial plexopathy, and polyneuropathies.[1,2,4]

Electrodiagnostic studies can detect mild median nerve entrapments at the wrist and ulnar nerve entrapment at the elbow using accurate, sensitive test techniques.[27] In addition, high-frequency US can be used to diagnose entrapment neuropathies correctly. The cross-sectional area of the nerve at the suspected localization, echostructure of the nerve, instability in a dynamic US examination, and pain reaction to nerve compression with an US probe provide valuable information for peripheral nerve entrapment diagnosis.[29]

Documentation of venous and arterial compression at the scalene triangle, the costoclavicular junction, and the retropectoral area by US is essential. Compression of neurovascular bundle by the PM muscle can be visualized

sonographically during shoulder hyperabduction and normalizing in neutral positioning.[30]

Subclavian and axillary arterial and vein Doppler US examination is a non-invasive technique showing real-time compression of vascular structures with dynamic maneuvers.[1,2,4,28] Dynamic venography and arteriography are diagnostic in uncertain cases.[1,2,5,6,8] Magnetic resonance neurography may be used to visualize BP, and MR/CT angiography is useful in diagnosing arterial or venous TOS and PMS.[1,2] Hemodynamic testing, including finger plethysmography and pulse oximetry, may be a helpful, non-invasive, rapid, and inexpensive clinical tool in diagnosing TOS. It shows hypoperfusion of the upper extremity during provocative activities or exercise.[28,31]

### Diagnostic injection tests

Diagnostic injection tests, such as scalene muscle and PM muscle blocks, are employed to confirm a diagnosis suspected on clinical findings. Identifying the location of the neurovascular compression is vital to tailor the most effective treatment. Physical examination and provocation tests are repeated after each muscle block, and the degree of reduction in symptoms including pain, tenderness, numbness and weakness is recorded. Ultrasound should be used for diagnostic muscle injections.[1,2,32]

### Pectoralis minor muscle block

For the diagnosis of PMS, a PM muscle block should be performed. A positive response is accepted as loss of tenderness over the PM muscle and significant relief of symptoms in 30 to 40 min after injection.[1,2,4] The US-guided block is reliable, practical, and inexpensive, allows visualization of neurovascular structures to avoid complications. During the procedure, the patient is placed in the supine position.[1,2,4,15] The US probe is placed on infraclavicular region. The pectoralis major muscle, PM muscle, AA artery, and AV are visualized on the pectoral region. The needle is visualized during injection with in-plane technique (Figure 3). Assessment provides recognition of anatomical variations, PM thickness, and relationship with the surrounding structures.[30,32]

A short-acting local anesthetic of lidocaine 1%, 4 mL is often used in diagnostic blocks. The injection is performed via the in-plane technique with a linear probe at an angle of 45° (Figure 3, Figure 4) to the point of maximum tenderness, and the needle size varies according to the depth of the muscle. For the diagnosis of TOS, we prefer blocking the PM muscle initially, if symptoms and signs subside after

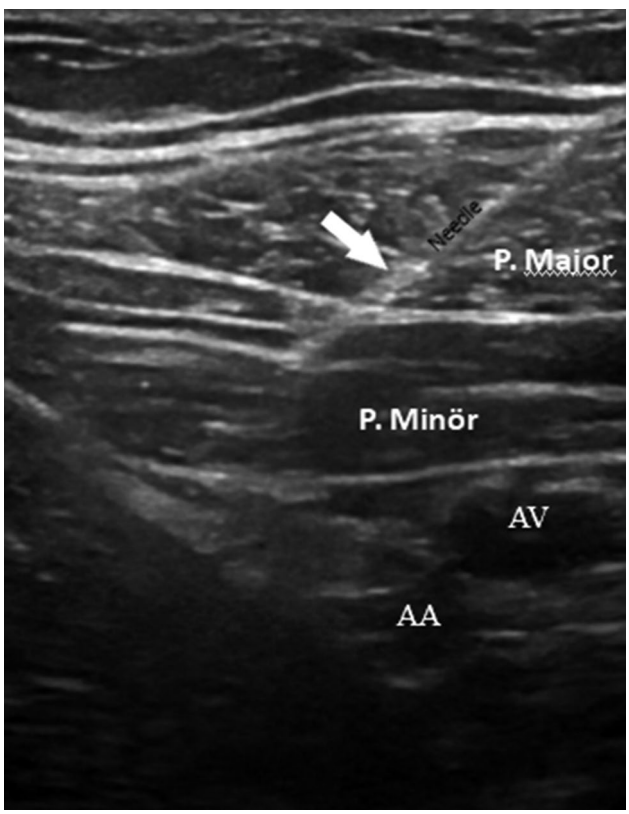


**Figure 3.** Pectoralis major (P. Major), pectoralis minor (P. Minor) muscles, axillary artery (AA), and axillary vein (AV) are visualized on the pectoral region. The needle (white arrow) is viewed via in-plane injection technique.

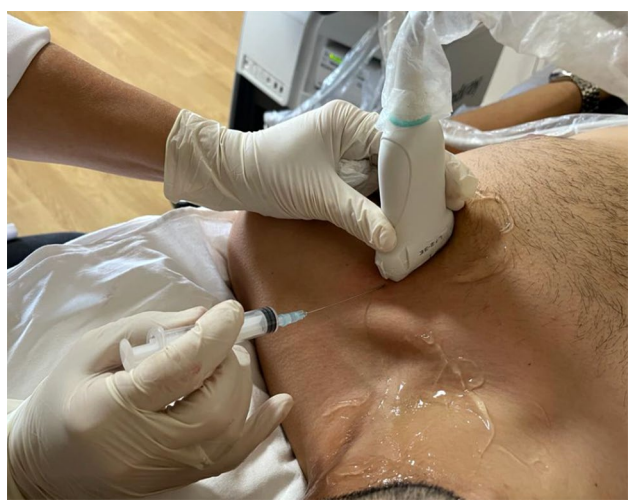

**Figure 4.** Ultrasound probe position for infraclavicular imaging of the pectoral muscles and neurovascular structure. Ultrasound-guided pectoralis minor injection with in-plane technique.

the injection, diagnosis of PMS is confirmed, and the scalenus anticus (SA) block is not performed. If there are persistent signs or symptoms, SA block is performed and the physical examination is repeated.[1-4,15,32]

### Scalenus anticus muscle block

The patient is placed in the supine position (Figure 5a). Initially, supraclavicular BP is visualized with a linear US probe. The probe is, then, directed cephalad to view the interscalene area. The BP at the level of C6 and the adjacent SA are identified, the jugular vein and carotid artery are visualized (Figure 5b). The needle is visualized with an out-of-plane or in-plane (medial to lateral or lateral to medial) approach, as it is placed into the body of the SA (Figure 5a, b). After negative aspiration, lidocaine 1% is injected into the SA under real-time imaging to ensure that the injection solution remains contained within the muscle.[32]

## Treatment

### Conservative Treatment

When there is no arterial and venous thrombosis, conservative treatment is preferred. Early diagnosis and treatment are important. The most crucial component of treatment is activity modification and the regulation of daily living activities. Most cases benefit from non-operative treatment. Non-operative management consists of physical therapy and rehabilitation combined with pharmacological agents including, analgesics, muscle relaxants, and anti-inflammatory drugs.[1-4,12] A rehabilitation program including PM muscle stretching, shoulder stabilization, and postural correction constitutes the core of conservative treatment. Each patient is instructed a home-based self-treatment program tailored to individual needs. Good posture and self-treatment PM muscle stretching exercises are essential to a successful treatment. It is pivotal for treating PMS and should be recommended for all patients diagnosed with PMS.[1,2,4]

A study evaluating the effects of a stretching protocol of PM muscle on function, muscle length, and scapular kinematics in subjects demonstrated that a daily home-based stretching protocol significantly decreased pain and improved function in patients with shoulder pain.[33] An effortless way of stretching PM muscles is standing in an open doorway, with the hands at shoulder level resting on the door jambs. Patients are instructed to stretch three times a day/three repeats at each session/seven days a week, hold each stretch for 15 to 20 sec, rest for the same length of time, and repeat. This should be performed for two to three months. Patients who are unable to do the exercises correctly or do not benefit enough are included in the physical therapy and rehabilitation program. Hot packs, conventional transcutaneous electrical nerve stimulation, and therapeutic US can be applied to the pectoral area and shoulder girdle in each session.[4,15] The therapist applies gentle stretching to

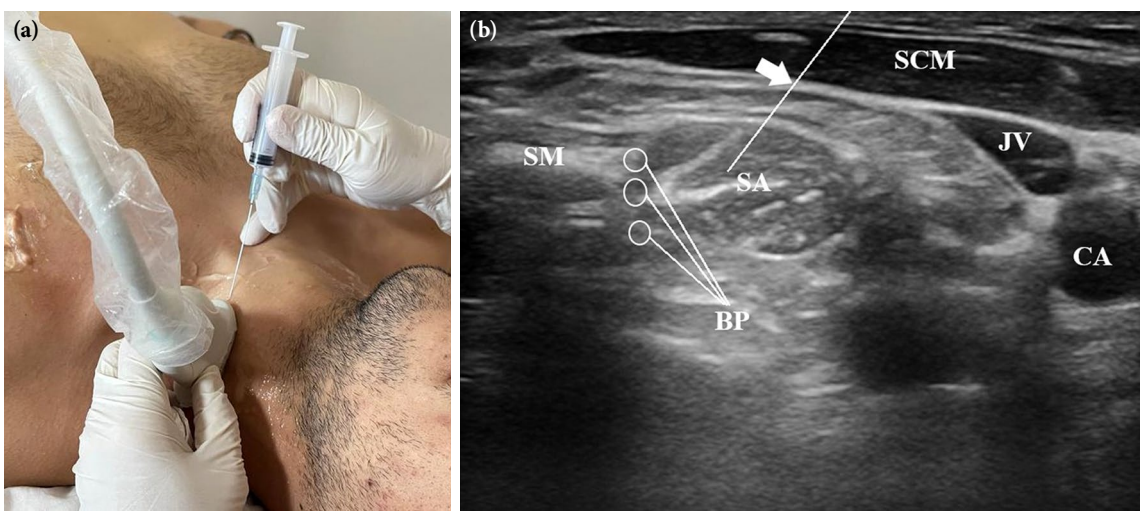


**Figure 5.** **(a)** Patient in the supine position during scalenus anticus muscle block with in-plane technique. **(b)** Ultrasound image showing the sternocleidomastoid muscle (SCM), scalenus anticus (SA) muscle, scalenus medius (SM) muscle, carotid artery (CA), jugular vein (JV) and branches brachial plexus (BP). The injection route (white arrow) is viewed with in-plane injection technique for left SA (medial to lateral approach).

the pectoral and scalene muscles, shortened shoulder girdle, and cervical muscles. Shoulder proprioceptive neuromuscular facilitation and post-isometric relaxation exercises are also helpful.[1,2,4]

Local anesthetic injections or dry needling of PM muscle can be performed if necessary, and corticosteroids injection is not recommended.[10] Intramuscular BTX injections, commonly used in treating local spasticity, reduce muscle spasms by inhibiting the release of acetylcholine at the neuromuscular junction. A case with hemiplegia with upper extremity spasticity resulting from a stroke was reported to have adductor spasticity and symptoms of PMS. Symptoms were revealed with BTX injections in the PM muscle and the pectoralis major muscle, as well as the subscapularis and latissimus dorsi muscles.[15] Botulinum toxin injection can be performed for spasticity and in resistant cases where muscle relaxation is targeted. An observational cohort study of patients with PMS who underwent US-guided BTX injection in the PM muscle revealed good results of symptom relief in patients without spasticity.[34]

In recent years, kinesio taping (KT) applications have been widely used to loosen fascial loading and relieve muscle tension. A study on KT of the PM muscle indicated that KT of the shoulder with stretching of the PM muscle significantly increased the PM muscle length and significantly decreased the supine measurement of rounded shoulder posture.[35]

### Surgical treatment

Surgical treatment should be considered in cases unresponsive to conservative methods. Pectoralis minor muscle tenotomy with partial myomectomy is performed via the transaxillary or transthoracic approach. The remaining body of the PM muscle adheres to the anterior chest wall after the operation. Postoperatively, up to two to three months, the patient should avoid elevating the arm above shoulder level to facilitate this adherence. However, once a day, shoulder elevation up to 180° is allowed to avoid frozen shoulder.[21] Anatomical variations in the PM muscle can be encountered. The clinical consequences of variants are usually minor; however, there is some evidence that PM muscle insertion has the potential to cause neurovascular compression.[36] Surgical treatment should be planned after adequate examination with appropriate imaging modalities in patients unresponsive to conservative treatment.

In conclusion, PMS is an overlooked syndrome which is easily overlooked and confused with other painful neurovascular compression syndromes of the upper extremity. A detailed history, physical examination, and diagnostic blocks are the cornerstones of the diagnosis. Conservative treatment is usually successful, and surgical treatment should be considered in unresponsive cases.

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