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NARRATIVE REVIEW

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Neurogenic thoracic outlet syndrome in the overhead and throwing athlete: A narrative review

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Abstract

Thoracic outlet syndrome is an important cause of shoulder pain and dysfunction due to compression of neurovascular structures as they traverse the thoracic outlet. Symptoms are most commonly due to compression of the brachial plexus called neurogenic thoracic outlet syndrome (nTOS). Throwing athletes are at increased risk of nTOS because of a variety of biomechanical factors. However, because nTOS symptoms are often nonspecific, delayed diagnosis is common. Neurogenic thoracic outlet largely remains a diagnosis of exclusion with advanced imaging ruling out vascular involvement and diagnostic injections gaining favor in helping localize sites of compression. Although rehabilitation alone may improve symptoms in some athletes, many require surgical treatment for long-term relief. This generally entails decompression of the thoracic outlet by some combination of muscle release, brachial plexus neurolysis, and first rib resection. Outcomes tend to be successful in athletes with most achieving resolution of symptoms and return to athletic activity. NTOS is an important cause of shoulder pain and dysfunction in throwing athletes. The history and physical examination should focus on activities that exacerbate symptoms. Treatment of nTOS generally requires surgical intervention and allows throwing athletes to return to sport.

INTRODUCTION

Thoracic outlet syndrome (TOS) is a relatively uncommon cause of shoulder pain characterized by pain, numbness, and paresthesias in the shoulder and arm, especially with activities such as raising the arms above the head. These symptoms manifest as a result of the compression of neurovascular structures as they pass through the thoracic outlet, the space between the clavicle and first rib.[1] There are three types of TOS, which are classified by the structures affected. Arterial TOS (aTOS) and venous TOS (vTOS) are caused by compression of the subclavian artery or vein, respectively. Venous TOS, also referred to as effort thrombosis or Paget-Schroetter Syndrome, results from venous compression during activity of the arm and shoulder resulting in swelling of the arm veins and increased risk for thrombosis, which can be life threatening if blood clots travel to the heart or lungs. Arterial TOS similarly results from overactivity of the arm and shoulder, which can result in transient painful ischemia of the upper extremity or an arterial clot that can break apart causing distal ischemia in the arm or hand.[1] The most common cause of TOS is neurogenic thoracic outlet syndrome (nTOS), which is due to compression or irritation of the brachial plexus as it traverses the scalene triangle at the base of the neck or in the pectoralis minor space below the clavicle.[2] When the neurovascular structures are compressed under the subcoracoid space by the pectoralis minor, this was initially termed "hyperabduction syndrome" by Wright in 1945 or more recently called "pectoralis minor syndrome" by Sanders.[3,4] Despite nTOS being described in the literature for decades, it remains a challenging diagnosis, given that upper extremity paresthesia, weakness, or pain are relatively nonspecific symptoms. Additionally, variability in brachial plexus anatomy can result in variable symptom patterns among patients with nTOS. Indeed, a lack of familiarity with nTOS among many clinicians is an additional barrier to timely workup and treatment for even those with classic symptomatology.[5] The presence of nTOS is important to recognize in athletes, including those who perform activities requiring upper extremity movements such as throwing. The biomechanics involved in throwing, with a high degree of mobility of the glenohumeral joint and delicate balance of stability versus mobility, all put the shoulder at increased risk of injury.[6] Although nTOS is a relatively uncommon cause of shoulder pain and injury, it should always be considered in throwing and overhead athletes presenting with pain, numbness, and paresthesias of the upper extremity, especially with repetitive activity. The purpose of the narrative review is to review and summarize the literature on the clinical presentation, exam signs, diagnostic tools, and available treatments for nTOS with particular emphasis on overhead and throwing athletes.

LITERATURE SEARCH

A nonsystematic search was conducted in the database Medline (PubMed). Search term combinations included: "neurogenic thoracic outlet syndrome," AND "overhead athlete" OR "throwing athlete" OR "diagnosis" OR "imaging" OR "treatment" OR "surgery" OR "outcomes." Types of literature included in this review were expert opinions, case reports and cohort studies, narrative reviews, retrospective cohort studies, and prospective comparative studies. No prospective multisite double-blinded randomized controlled trials were found. Each of these was then graded using the most recent classification scheme from the Oxford Center for Evidence-Based Medicine.[7] A search for the general diagnosis "thoracic outlet syndrome" was also completed. Such articles were reviewed by the authors only for information or further literature pertinent to nTOS and were otherwise excluded. The results and discussion of this review are presented in a format familiar to clinicians, starting with symptoms and signs followed by diagnostic testing and treatment outcomes, to help readers synthesize current evidence on evaluation and management of nTOS in a clinical context.

CLINICAL PRESENTATION

The nonspecific nature of nTOS often leads to a delay in diagnosis and can be frustrating for athletes who have undergone extensive evaluations and previous surgeries without improvement in symptoms or overall function.[8] A combination of clinical suspicion, key historical features, and physical examination findings are all required in this diagnosis.

The typical history is an athlete presenting with pain at the base of the neck or anterior shoulder that radiates to the upper extremity. This is often accompanied by numbness and paresthesias that may reach the hand distally. In more advanced cases, weakness of the hand or loss of dexterity of the fingers may be present.[9] An athlete may report such symptoms as loss of ball feel, "dead arm" sensation, or loss of pitch control. Although Raynaud's phenomenon is more commonly associated with vTOS due to ischemia, characterized by symptoms of coldness and color changes in the affected extremity; it can also be seen in those with nTOS because of increased sympathetic activity. The sympathetic fibers run along the C8, T1 nerve roots and lower trunk of the brachial plexus and can become irritated with nerve compression.[10] The differential diagnosis for throwing athletes with neck and shoulder pain, sensory changes, and/or weakness in the upper extremity is broad. This includes cervical myelopathy or radiculopathy, Parsonage-Turner syndrome, cervical dystonia, rotator cuff disease, shoulder osteoarthritis, peripheral mononeuropathies specific to throwers, or complex regional pain syndrome (See Table 1).

OVERHEAD ATHLETE BIOMECHANICS

The anatomical structure of the brachial plexus and the surrounding anatomy play a central role in nTOS. As the brachial plexus traverses the thoracic outlet, limitations in this space can lead to compression either at rest or with arm movement. Given the anatomical basis for nTOS, there are several biomechanical factors that increase the risk of nTOS in throwing athletes. Previous studies in healthy adults have shown that maximal shoulder abduction and external rotation significantly reduce the space between both the clavicle and scalene and clavicle and first rib, key structures associated with brachial plexus compression and one mechanism thought to contribute to nTOS.[11] Immediately distal, the neurovascular bundle must traverse the posterior aspect of the pectoralis minor muscle, or retropectoralis minor space. During upper limb elevation the cords may be pressed tightly against the pectoralis minor musculature and predispose to compression.[12] In throwing mechanics, the late cocking and acceleration phases bring the shoulder into abduction and external rotation, which can be visualized in Figure 1 to further explain how these positions in the throwing motion can cause compression.[11] Biomechanical studies have shown that throwing athletes tend to have a throwing arc that is shifted further toward external rotation compared to their nonthrowing arms and the general population, increasing their predisposition for narrowing of the thoracic outlet and compression of the brachial plexus.[13] Given that adaptations in glenohumeral rotation are a significant contributor to power generated during a throwing motion, it is reasonable to conclude that throwing athletes, especially those who have undergone significant physiological adaptation throughout their time playing, are at increased risk for developing nTOS.[14,15] Some hypothesize that the high level of force produced by competitive overhead athletes during throwing may lead to hypertrophy of the scalene or

TABLE 1 Differential diagnosis for nTOS with brief history for each and comparison to nTOS

Diagnosis	History and etiology	Examination	Imaging/diagnostics
Cervical myeloradiculopathy	Both myelopathy and radiculopathy commonly present with neck and/or shoulder pain, paresthesias, and weakness of the upper extremity, including fourth and fifth digits as seen in nTOS.	Patients with spinal cord or nerve root pathology will generally exhibit limitations in cervical motion, nerve root irritation with axial pressure during cervical extension (Spurling's sign). Clumsy gait, positive Hoffman's sign, and other upper motor neuron signs would suggest a lesion of the central nervous system rather than nTOS.	Cervical MRI without contrast has high sensitivity and specificity for cervical myelopathy of compressive neuropathy of the roots. Electromyography can be helpful in determining radiculopathy affected motor function but may miss purely sensory radiculopathy.
Idiopathic brachial plexopathy (Parsonage-Turner syndrome)	Abrupt onset shoulder pain followed by weakness and paresthesias of usually the upper trunk of the brachial plexus. Sensorimotor changes can persist for months before reinnervation takes place. This may be preceded by a viral infection prior to pain symptoms. In contrast to nTOS, these symptoms are unrelated to activity and would not change with provocative maneuvers or exertion. Also, nTOS tends to affect the lower trunk instead of the upper trunk.	Weakness and paresthesias of muscles of the upper trunk of the brachial plexus with associated sensorimotor changes present.	This is a clinical diagnosis, with imaging and electrodiagnosis only providing supportive information to clinicians when needed that may show abnormal motor and sensory NCS and/or spontaneous activity.
Cervical dystonia	This is characterized by often painful spontaneous neck-turning or writhing of the cervical musculature. These movements may result in positional numbness or pain of the upper extremity which mimics nTOS. Those born with cervical dystonia would generally not be referred for evaluation of nTOS. However, some develop occupational cervical dystonia in adulthood with more subtle, painful posture.	Examination for asymmetry in cervical and shoulder girdle musculature, including tightness are generally indicative of dystonia and not nTOS.	This is a clinical diagnosis with diagnostic tests looking only for a cause of new onset dystonia, such as advanced imaging of the brain.
Rotator cuff tear	Either acute or degenerative tears are possible. The former generally entails a high force injury to the shoulder. Degenerative tears commonly occur in people over age 40 who repeatedly perform the same shoulder movements, leading to pain and weakness similar to that of nTOS. However, rotator cuff disease tends to be more painful with activity and less commonly with rest, particularly while lying on the affected side.	Examination of the shoulder for decreased range of motion, pain with muscle activation, positive signs of rotator cuff tears including special tests such as drop arm test, Jobe's test, weakness with rotator cuff maneuvers, and painful arc of motion worst from 60-120°.	Ultrasound or MRI evaluation of the shoulder will readily reveal a tear of the rotator cuff, if present.
Shoulder osteoarthritis	Degeneration of the glenohumeral or	Examination of the shoulder will reveal decreased range of	Radiographs of the shoulder will show joint space narrowing,

(Continues)

TABLE 1 (Continued)

Diagnosis	History and etiology	Examination	Imaging/diagnostics
	acromioclavicular joints can cause shoulder pain that may radiate down the arm similar to nTOS. However, osteoarthritis tends to be associated with stiffness and pain after inactivity that improves with some movement, which may be limited due to joint degeneration.	motion with pain notably increased at end range. Crepitus can be noted with arm motion.	osteophyte formation, subchondral sclerosis, and cyst formation.
Mononeuropathy of the upper extremity	Mononeuropathies of the upper extremity, especially compression neuropathy of the ulnar or median nerves, are a common cause of radiating pain in the arm and hand. Compression of the ulnar nerve at the cubital tunnel can cause numbness, tingling, and weakness in the ulnar forearm and the palmar fourth and fifth digits. Similarly, compression of the median nerve at the wrist as it traverses the carpal tunnel can cause paresthesias and weakness of the hand, especially the palmar surface of the first three digits. In contrast to peripheral mononeuropathy, patients with nTOS will generally have paresthesias of the whole hand and both thenar and hypothenar atrophy.	Examination of mononeuropathy would typically show decreased sensation and motor nerve function that correlates with the nerve being tested. Severe cases of these may show thenar atrophy (carpal tunnel syndrome) or intrinsic atrophy of the fourth and fifth digits (cubital tunnel syndrome). Additionally, reproduction of paresthesias with percussion, called Tinel's sign, will be positive at the wrist or elbow for peripheral mononeuropathy due to nerve entrapment, whereas in nTOS, Tinel's sign would be positive at the interscalene triangle over the brachial plexus.	Electrodiagnostic testing will identify a mononeuropathy of the upper extremity if present and solely affect a single nerve compared to multiple that may be present with nTOS.
CRPS	Characterized by regional pain with increased sensitivity to a variety of stimuli along with a combination of associated features, such as dystonia, sudomotor dysfunction, swelling, skin discoloration, nail dystrophy, or contracture. In nTOS, the sensory changes tend to be limited to paresthesias because of arm movement without these secondary features or abnormal sensitivity to stimuli.	Thorough physical inspection of the entire affected extremity, including sensory exam will help to differentiate. The modified Budapest criteria were developed to support physicians in diagnosing complex regional pain syndrome. Domains of interest include Pain, Sensory, Vasomotor, Sudomotor, and Motor/trophic.[64]	CRPS is clinical diagnosis with diagnostic testing performed solely to rule out similar conditions that may mimic those of CRPS.

Note: Some information adapted from book chapter by Jordan in Illig's *Textbook on Thoracic Outlet Syndrome*.[63]
Abbreviations: CRPS, Complex regional pain syndrome; MRI, magnetic resonance imaging; NCS, nerve conduction study; nTOS, neurogenic thoracic outlet syndrome.

pectoralis minor musculature, which could further decrease the size of the thoracic outlet. Indeed, baseball players tend to have significant asymmetry in pectoralis minor musculature between nonthrowing and throwing arms.[16] This has also been reported clinically in the case of a high-level collegiate baseball player with paresthesias and pain during throwing refractory to arthroscopic labral repair. He was found to have imaging evidence of chronic neurovascular impingement due to moderate hypertrophy of the anterior scalene and pectoralis minor musculature, which improved with structured rehabilitation focused on decreased weight

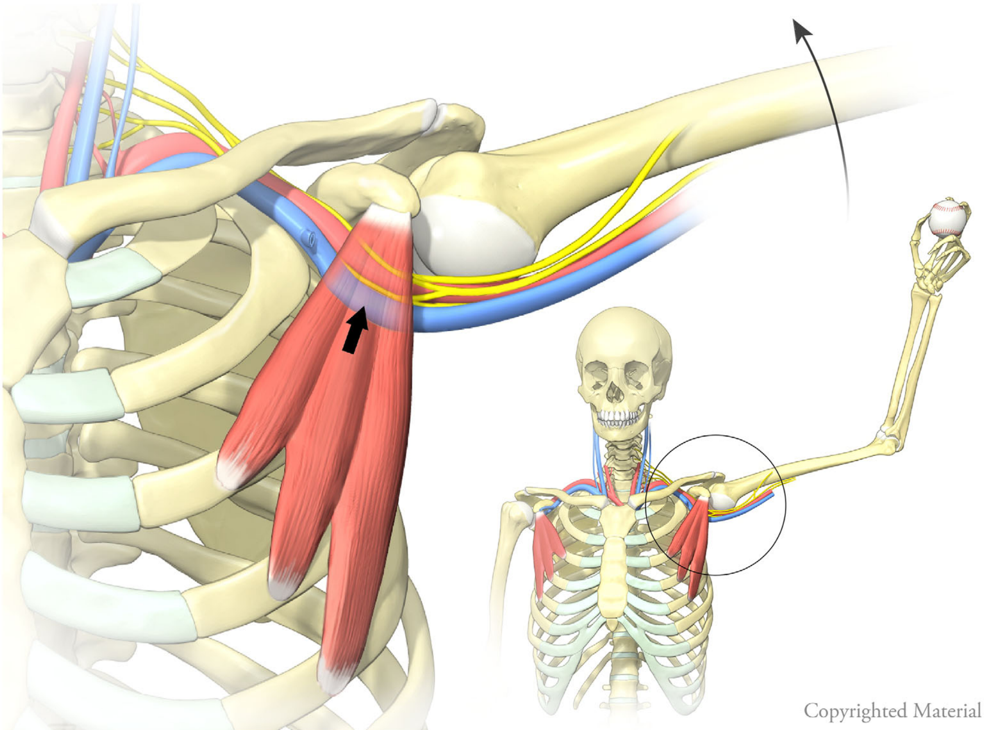

FIGURE 1 Anatomical rendering that depicts how the overhead throwing motion with abducted, externally rotated positioning places the neurovascular structures at risk for compression under the pectoralis minor musculature in the overhead athlete. Used with permission of Mayo Foundation for Medical Education and Research, all rights reserved

training, daily stretching, and gradual return to sport.[17] Additionally, the number of the overhead repetitions required for high-level play further increases the risk for symptomatic nTOS.[18,19]

Prevalence studies of nTOS in throwing athletes compared to the general population support the anatomical and biomechanical arguments suggesting that throwers are at increased risk for nTOS. Otoshi et al. showed an incidence of symptomatic TOS in 32.8% of Japanese high school baseball players, which they hypothesized was primarily nTOS.[20] This rate of TOS is three times higher than the incidence found in industrial workers by Sallstrom et al.[21] A study analyzing U.S. collegiate athlete injury data found that baseball and softball were most associated with nTOS, followed by swimming and water polo.[18]

PHYSICAL EXAMINATION

Physical examination of the patient with suspected nTOS should start with a combination of evaluation of the shoulder and cervical spine to rule out pathology of these regions that can have a similar presentation. The physical exam for identifying nTOS should begin with a thorough inspection of the shoulder girdle and upper extremities, looking particularly for muscle hypertrophy or atrophy and posture that can give a clinician insight into biomechanical factors that may be contributing. Active range of motion of the cervical spine and shoulder should be assessed to rule out more common pathology.

Strength and sensation of the affected and contralateral extremity should be examined to identify asymmetry with particular attention to musculature innervated by the upper versus lower brachial plexus. Upper plexus (C5-C7) involvement may present with weakness of the deltoid, biceps, and brachialis muscles whereas lower plexus (C8-T1) involvement should be suspected with weakness of wrist and finger flexors.[22] Advanced cases may even exhibit Gilliatt-Sumner hand, with signs of denervation and atrophy of the abductor pollicis brevis, hypothenar, and interossei muscles (Level V).[23] It is also important to perform neurological testing, such as upper extremity reflexes, assessing for Hoffman's sign and Spurling's test, which if positive, would suggest more proximal disease at the nerve roots or cervical spinal cord. Additionally, rotator cuff exam maneuvers or even point of care ultrasound can help to rule out rotator cuff disease mimicking symptoms of nTOS.

Next, palpation and provocative maneuvers should be performed over pertinent structures to measure reproducible pain and paresthesia. A key finding is the ability to provoke symptoms with direct pressure placed over the brachial plexus at the areas of nerve compression including the interscalene triangle or below the pectoralis minor or with overhead arm use (Level V).[10,24,25] Other commonly used provocative maneuvers are evaluating for presence of Tinel's sign at the coracoid process, the Elevated Arm Stress Test (EAST), Upper Limb Tension Test (ULTT), and Adson's Test.[1]

In the EAST, the arm is abducted to 90° with elbows flexed. The shoulder is externally rotated to bring the

TABLE 2 Description, reliability, sensitivity, specificity, and likelihood ratios of clinical tests for nTOS

Test	Description	Reliability	Sensitivity	Specificity	LR+	LR−	References
Elevated arm stress test	Patient seated with arms above 90° of abduction and in full external rotation with head in neutral position. Patient opens and closes fists while holding this position for 3 minutes. Positive test: pain and/or paresthesia that abates when arms are lowered.	1.0	52-84	30-100	1.2-5.2	0.4-0.53	27,61-65
Adson's Test	Patient seated with arms at sides. The radial pulse is palpated. Patient inhales deeply and holds breath, extends and rotates the neck toward the side being tested. Positive test: change in radial pulse and/or pain, paresthesia reproduction.	NT	79	74-100	3.29	0.28	27,62,63,65
Wright's test	Patient seated with arms at side. The radial pulse is palpated. Examiner places the patient's shoulder into abduction above the head. The position is held for 1–2 minutes. A positive test: Change in radial pulse and/or symptom reproduction.	NT	70–90	29-53	1.27-1.49	0.34-0.57	27
Upper limb tension test	Patient supine with examiner on the side to be tested. Examiner depresses the shoulder girdle and abducts the shoulder to 110° with slight extension and elbow flexion to 90°. The forearm is then maximally supinated and the wrist and fingers extended. Finally, elbow extension is applied. The neck is laterally flexed to the contralateral side. Positive test: reproduction of symptoms with distal arm movement or neck movement and/or restricted elbow extension range of motion.	0.03	90	38	1.5	0.3	65,66

Note: Adapted from similar table in Hooper et al.[47]
Abbreviations: LR, likelihood ratio; NT, not tested; nTOS, neurogenic thoracic outlet syndrome.

forearm past the head with the patient opening and closing the hand for several minutes. This position closes the scalene triangle and should reproduce symptoms.

In the ULTT, the patient abducts the shoulder to 90° with the elbow nearly fully extended. The patient then extends the wrist and tilts the neck away from the tested extremity. This should stretch the brachial plexus as it traverses the thoracic outlet and reproduces pain and paresthesias in the affected extremity.

Adson's test is meant to check for compromise of the brachial plexus as it runs through the scalene triangle by extending the neck past neutral and then turning the head toward the tested extremity while the patient holds a deep inspiration.

Positive provocative maneuvers such as these are supportive of a diagnosis of nTOS, but their standalone utility is variable. High false-positivity rate is the principal concern that arises in the literature for the use of provocative maneuvers. In one study 58% of healthy volunteers had at least one positive provocative maneuver (Level II).[26] However, others have demonstrated that multiple positive provocative maneuvers improved the validity for the diagnosis of nTOS. In one study, having two positive tests improved the sensitivity to 90% and a combination of five positive tests reached a specificity of 84% (Level II).[27]

There is an excellent table summarizing the specificity and sensitivity of many provocative maneuvers in an article by Hooper et al., which is adapted in Table 2.[28]

DIAGNOSTIC TESTS

As there are currently no gold standard tests for the diagnosis of nTOS, it largely remains a clinical diagnosis of exclusion. In patients with concerning history or positive findings on physical examination, imaging studies can help confirm sites of suspected compression, abnormal anatomy, or potential causes for the patient's symptoms.[29] Imaging starts with ruling out other causes of nerve entrapment, such as cervical pathology that can present with similar symptoms. Anterior-posterior radiographs looking at the cervical spine, chest, and shoulder give insight into anomalous bony structures including the incomplete or complete cervical ribs, first rib aberrations, clavicular deformities, post-traumatic changes that may make a diagnosis of nTOS more likely. Weber et al. showed that the presence of bony anomalies in patients with TOS was 29%, with cervical ribs accounting for a high number of these cases and a much higher rate than exists in the general population.[30] Radiographs are also important in ruling out other pathology, such as shoulder osteoarthritis.

Angiography with either computed tomography (CT) or magnetic resonance imaging (MRI) remains the preferred imaging modalities to rule out vascular TOS. CT angiography (CTA) has the added benefit of contrast to both arterial and venous structures, along with repeat imaging in positions of provocation to give more insight into how these structures are affected with narrowing of the thoracic outlet. However, the lack of a gold standard for diagnostic imaging in vascular TOS or even a precise definition for what constitutes arterial stenosis makes calculating the sensitivity and specificity for CTA or magnetic resonance angiography (MRA) difficult to quantify.[31] One recent study of 221 thoracic outlets, for which >50% arterial stenosis was considered significant, found that 97% of those with arterial stenosis showed symptoms of aTOS. However, only 40% of the symptomatic group were found to have significant stenosis, which suggests that CTA is specific but not sensitive for the identification of aTOS.[32] Some investigators argue that MRA is the preferred method of imaging for vascular structures involved in TOS, as it has the inherent lack of ionizing radiation. Indeed, studies have shown that MRA provides quality images and is particularly useful for postsurgical follow-up.[29,33] Studies comparing MRA and CTA for the diagnosis of vascular TOS are scant but will be necessary in building a gold standard approach to diagnosis. Although CTA and MRA remain important in diagnosis of vascular TOS or combination of neurogenic/vascular TOS, MRI of the brachial plexus remains ideal in the visualization of structures that may contribute to nTOS symptoms.[34] MRI is also useful in ruling out other causes of neural compression in the cervical spine or musculoskeletal conditions that may mimic the symptoms of nTOS. In the case of nTOS, MRI provides superior localization of neural compression suspected on physical examination and provocative testing that cannot be seen on plain radiographs or CT imaging.

Given that nTOS is due to compression of neurovascular structures, it is reasonable to hypothesize that electrodiagnostics would assist with diagnosis. Although the evidence for use of electrodiagnostics for diagnosis of nTOS remains controversial, these studies can be used to rule out many conditions with a similar presentation, such as brachial plexopathy, cervical radiculopathies, peripheral mononeuropathies, and polyneuropathies. One study showed that a combination study of the medial antebrachial cutaneous sensory nerve (T1) and median motor (T1 > C8) was abnormal in 89% of those with nTOS, whereas response combinations that primarily assessed the C8 fibers were less frequently affected (Level III).[35] However, it should be noted that the most recent TOS guidelines from the Society for Vascular Surgery in 2018 do not recommend nerve conduction studies as they can often be normal in those without prolonged symptoms (Level IV).[25]

Diagnostic ultrasound may also aid in diagnosis of nTOS by visualizing key structures. Although not specific to throwing athletes, ultrasound has been shown to have utility in identifying anomalous structures that can be associated with nTOS symptoms, such as fibromuscular bands of the scalenes, variations of the scalene musculature, and brachial plexus branching variations.[36-38] One proposed paradigm suggests a dynamic ultrasound evaluation with the arm abducted to measure the "pectoral bowing ratio," measured as a percentage of the deformation of the vertical pectoralis minor muscle compared to its length (Level IV).[39] This may be of particular interest given concerns that pectoralis minor musculature has been implicated as an area of compression in the overhead athlete. The authors found that the amount of "bowing," measured as a percentage or ratio, was substantially greater in the nTOS participants (17.1%) than in the normal participants (1.8%). Although this is only a small study of 15 patients with nTOS, the authors do illustrate the utility of diagnostic ultrasound in identifying objective findings consistent with nTOS. An important limitation of this technique is that formal training is not readily available for this advanced ultrasound evaluation. Therefore, one would need initial basic instruction and subsequent experience to become comfortable when performing this type of neuromuscular ultrasound evaluation.

Performing diagnostic injections at common sites of compression such as the anterior scalene or pectoralis minor is now common practice in the workup for nTOS. Alleviation of symptoms with these injections would suggest a diagnosis of nTOS.[40] Studies examining the utility of anterior scalene injection are the most abundant. One such study reports that, for those with anterior scalene musculature predominant nTOS for whom physical therapy was not successful, diagnostic lidocaine injections were positive in 89% of the <40-year-old group and 93% of the ≥40-year-old group. After multivariate analysis was performed to consider patient presenting factors, the impact of a successful lidocaine injection in patients ≥40 years old was greater than in patients <40 years old and was associated with improved surgical success in the older group (Level III). Botulinum toxin blocks were less successful in patients than lidocaine injections as a predictor of symptom relief after transaxillary decompression (Level IV) but can still be a valuable tool in diagnosis of treatment of nTOS.[41] More recent studies have shown botulinum toxin injections to have a positive predictive value when it comes to surgical success as well as providing short-term symptomatic relief, which was attributed to inclusion of more patients with pectoralis minor pathology.[42,43] Importantly, a report from Bottros and colleagues suggests that high-level overhead athletes be observed exercising both before and after diagnostic blocks, as their symptoms are often dynamic and subtle outside of high-level activity.[44] Their paper reports two cases using a method for simultaneous blocks to the anterior and middle scalene as well as the pectoralis minor for successful diagnostic evaluation of nTOS (Level IV). Although large studies measuring results of only the pectoralis minor muscle for nTOS are sparse, the method for such an injection is described in a 2017 report by Sanders et al.[45] They propose starting with a pectoralis minor muscle block followed by repeat physical exam for provocative testing. If symptoms persist then a block of the anterior scalene muscles is subsequently performed with repeat physical examination repeated to assess for residual pain symptoms. Of note, the current authors' diagnostic protocol involves a similar approach to diagnosis, beginning with the pectoralis minor muscle block followed by anterior scalene if there are residual symptoms (Level V).

CLINICAL MANAGEMENT

Though there is no consensus on conservative treatment for nTOS, a multimodal plan involving education, pharmacological interventions, and physical rehabilitation is the generally accepted initial treatment plan. Rehabilitation for nTOS should focus on scalene and pectoralis minor relaxation and stretching and also emphasize shoulder girdle, scapular, and glenohumeral mechanics (Level IV).[46] Because sensorimotor control of the periscapular muscles is often dysfunctional in nTOS, special attention should be given to scapular retraining.[47] Caution should be taken with strengthening exercises, especially of the pec minor or scalenes, as these can often exacerbate nTOS symptoms (Level V).[19] Although patients may improve with a comprehensive rehabilitation program alone, the return to sport rate after nTOS surgical treatment is also favorable. Data indicate that the effectiveness of physical therapy alone is mixed.[36-39] However, an emphasis should be placed on the careful selection of surgical patients as this has been shown to significantly increase the rate of success within high-performance athletes who undergo surgery from 50 to 90%[14] (Level III).[18,48-51] The rehabilitation of scapular kinematics should be emphasized as it appears to be beneficial both as a conservative measure prior to surgery and in the postsurgical rehabilitation population.

Therapeutic injections with anesthetic agents, botulinum toxin, or steroids into the anterior scalene or pectoralis minor have also been performed with varying success.[52] One limited cohort study by Torriani showed that 69% of botulinum toxin injections performed to the anterior scalene and pectoralis minor muscles had >50% pain relief with a mean of 31 days of relief (Level IV).[43] However, a randomized controlled trial investigating scalene injection of botulinum toxin A did not show benefit compared to placebo for the treatment of nTOS (Level I).[42,53]

Although conservative treatments may alleviate symptoms of nTOS in some athletes, surgery may be required for definitive treatment and more long-term relief. Surgical treatment of nTOS entails decompression of the thoracic outlet by some combination of scalenectomy and/or pec minor release, brachial plexus neurolysis, and first rib resection.[54] Literature has described different approaches, such as supraclavicular or transaxillary approach, with additional approaches available but seldom used unless for isolated nTOS.[55] Although surgery can be indicated in many cases where conservative management and physical therapy fail, these surgical techniques are not without their own complications ranging from more common complications, such as pneumothorax, recurrence of symptoms, and scar tissue formation to more serious complications seen when addressing arterial or venous structures involved within the thoracic outlet. Complication rates have been noted as high as greater than 20% in several studies included in a systematic review by Peek et al.[54] Although outcomes of surgical decompression for nTOS have not always been as successful given the lack of uniform indications for surgery, similarity to several other neurologic conditions of the upper extremity, and poor long-term functional results, appropriate patient selection criteria and highly selective treatment algorithms have helped improve the percentage of patients who benefit from decompression.[56,57] After undergoing first rib resection in a highly specialized surgical practice, positive outcomes were reported in 93% of patients in the first 5 years and 96% in the second 5-year period (Level II).[58] Importantly, diagnosis of nTOS in these patients was made based on a history of pain and paresthesia with arm activity and a positive EAST. Although the utility of diagnostic blocks of the anterior scalene are mentioned, they were not reported as a requirement for diagnosis in this study. Additionally, only those with refractory nTOS symptoms after 8 weeks of physical therapy were referred for first rib resection.

One retrospective study of high school, college, and professional athletes who presented to a tertiary referral center with symptomatic nTOS showed that 81% returned to sport within 4–5 months following surgical treatment (Level III).[18] The authors of this study used a previously published nTOS algorithm for selection of patients to undergo surgical decompression and emphasize the importance of thorough conservative treatment prior to discussing surgery and the use of therapy as a diagnostic tool to predict which athletes would benefit from decompression.[8] Chandra et al. used a combination of baseline QuickDASH (Disabilities of the Arm, Shoulder and Hand) scores followed by nTOS specific physical therapy based on the Edgelow protocol, a combination of breathing, relaxation, posture, and positioning that mimics decompression of the thoracic outlet.[8,18,59] Patients were noted to undergo physical therapy sessions three times a week for at least 2 months with diligence of home exercises in between sessions before they were reassessed clinically and with repeat QuickDASH scores. Those patients who showed subjective symptomatic improvement as well as improvements in QuickDASH scores and a commitment to physical therapy were offered surgery. Foundational to the surgical success in these studies were the rigorous and methodological patient selection. It is noteworthy that not all selection algorithms listed in the described studies are identical, but commonly a combination of diligence with nTOS specific physical therapy and diagnostic blocks was used to determine who would benefit from surgical decompression.

Another retrospective study of 67 competitive athletes at an average of 3.9 years after undergoing first rib resection and scalenectomy for nTOS found that 82% of survey respondents had durable resolution of nTOS symptoms following surgery with 94% stating they were able to perform all daily activities without symptoms. Additionally, 70% returned to a similar level of activity or better within 1 year of surgery (Level III).[60]

A recent outcome report for professional baseball players with nTOS followed key performance metrics in 10 Major League Baseball (MLB) pitchers and showed essentially no decrease in performance following decompression and rehabilitation for nTOS. Metrics measures included maximal and average pitch velocity, earned run average, and strikeouts to walks ratio (Level III).[61] A similar, but larger study of MLB pitchers over 2001-2017 showed that 20 pitchers with nTOS underwent surgical decompression. Of those, fastball velocity and strike percentage were equivalent postoperatively, but pitcher earned run average remained inferior to preoperative baseline (Level III).[62]

CONCLUSION

Overhead and throwing athletes are particularly susceptible to nTOS given the intense repetitive actions of the upper extremity required for high-level competition. Diagnosis of nTOS can be difficult and often delayed in throwing athletes. A thorough history and a physical exam focused on provocative maneuvers is essential to arriving at the correct diagnosis. Injections into the anterior scalene and pectoralis minor musculature have gained favor as an important diagnostic tool, but high-level studies evaluating their utility are needed. Multiple level II-III studies show that structured conservative treatments followed by surgical decompression and rehabilitation are generally successful in allowing throwing athletes to return to sport at all levels with little or no change in performance.

DISCLOSURE

None.

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