Neurogenic Thoracic Outlet Syndrome in Athletes — Nonsurgical Treatment Options

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Abstract

Neurogenic thoracic outlet syndrome (NTOS) is an etiologically and clinically diverse disorder caused by compression of the brachial plexus traversing the thoracic outlet. Athletes who perform repetitive overhead activities are at risk of developing NTOS with sport-specific symptoms. This article reviews the controversial NTOS nomenclature, common sites of anatomic compression, and red flag symptoms that require immediate intervention. It also reviews the congenital, traumatic, and functional etiologies of NTOS, with a discussion of the differential diagnosis, diagnostic criteria, and workup for NTOS. Nonsurgical treatment is highlighted with an emphasis on thoracic outlet syndrome-specific physical therapy and updates on injection options and ultrasound guided hydrodissection. This article compares nonsurgical versus surgical functional outcome data with an emphasis on athletes with NTOS. Functional assessment tools and performance metrics for athletes are reviewed, as well as return to sport considerations.

Introduction

Thoracic outlet syndrome (TOS) is a group of etiologically and clinically diverse disorders that share one key feature: compression of one or more of the neurovascular elements traversing the thoracic outlet (1–4). The thoracic outlet is an anatomical area through which the neurovascular bundle traverses in the interval between the neck and the axilla (2). The neurovascular bundle consists of the brachial plexus, subclavian artery, and subclavian vein (5,6). TOS has three subtypes based on the location of compression. Neurogenic TOS (NTOS) is defined by compression of the brachial plexus; venous TOS (VTOS), by compression of the subclavian vein; and finally arterial TOS (ATOS), by compression of the subclavian artery (3,6).

While NTOS is by far the most common type of TOS, compression of the brachial plexus varies in etiology, pathophysiology, clinical presentation, and subsequently requires further subtype classification, diagnostic workup, and surgical or nonsurgical treatment. As our knowledge of NTOS continues to evolve with increasing awareness, diagnosis, and treatment options, our retrospective review of NTOS aims to review the key points of NTOS evaluation and management, as well as update readers on management of athletes with NTOS.

NTOS Terminology

TOS nomenclature has sparked controversy to create a uniform vocabulary and diagnosis among a heterogeneous and multifaceted syndrome. Historically NTOS has been divided into "true" NTOS and "disputed" NTOS. True NTOS is estimated at one case per million population (7) as C8, T1 nerve roots or lower trunk of the brachial plexus fibers are stretched over congenital bands with an elongated C7 transverse process or rudimentary cervical ribs (8,9) that may include unilateral hand muscle wasting, especially the lateral thenar muscles and hand intrinsic muscles, sensory symptoms along mainly the medial forearm, and electrodiagnostic evidence of chronic, severe, axonal loss at the lower trunk of the brachial plexus (7,8).

In 1970 a case series of nine patients was described by Gilliatt and Sumner with unilateral hand muscle wasting associated with an elongated C7 transverse process or rudimentary cervical ribs, all of which had a sharp fibrous band during operation, causing compression of the C8 and T1 nerve roots or lower trunk of the brachial plexus (9). Dividing the fibrous band relieved pain and paresthesias in eight of nine patients and stopped muscle wasting in all patients with only a slight recovery of power in the muscles years later (9). This clinical presentation, including hand muscle and thenar atrophy, is referred to as the Gilliatt-Sumner hand and is associated with congenital anatomic entrapment of the lower trunk of the brachial plexus, requiring surgical decompression (10).

Disputed NTOS (also referred to as nonspecific, postural, and symptomatic NTOS) does not have these characteristics, is not well defined, and is, therefore, more challenging to treat as it is more nebulous (1,11). Disputed NTOS is thought by

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1537-890X/2006/319–326
Current Sports Medicine Reports

some clinicians and surgeons to be more of a musculoskeletal pain syndrome with primarily subjective symptoms, whereas others think that it is an underdiagnosed subdivision of NTOS (11).

Ferrante and Ferrante (12) classify TOS as ATOS, VTOS, true NTOS, traumatic NTOS, and disputed TOS, noting different etiologies, clinical features, evaluation, and treatment for all except disputed TOS. Ferrante and Ferrante (12) recommend that Disputed TOS instead be considered a cervicoscapular pain syndrome managed with physical therapy and postural exercises (12). Illig et al. (13) recommends no longer defining NTOS as "disputed" for another reason, citing that it discredits the diagnosis for the patient with substantial symptomatology.

Anatomy

Sites of neurovascular compression within the thoracic outlet include the interscalene triangle, costoclavicular space, and subcoracoid space (2,5). The interscalene triangle is bordered anteriorly by the anterior scalene muscle, posteriorly by the middle scalene muscle, and inferiorly by the first rib. Contents include the brachial plexus and subclavian artery and exclude the subclavian vein (2,5,11). The subclavian vein is anterior to the anterior scalene muscle (5). The costoclavicular space is bordered anteriorly by the subclavius muscle, superiorly by the clavicle, and inferoposteriorly by the first rib and anterior scalene muscle (2). Contents include the brachial plexus, subclavian artery, and subclavian vein (2,11). The subclavian vein is most likely to be compressed at the costoclavicular space (13). The subcoracoid space (also known as the retropectoralis minor space) (2,5,11) is bordered anteriorly by the pectoralis minor muscle, posteriorly by ribs two to four, and superiorly by the coracoid process. Contents include the brachial plexus, and the subclavian artery and vein, now defined as the axillary artery and axillary vein (2,11). Trunks are included within the interscalene space, divisions in the costoclavicular space, and cords with some branches at the retropectoralis minor space (14) (Fig. 1).

TOS has been defined anatomically by the site of compression, and a variety of syndromes have been recognized. True NTOS includes a fibromuscular band extending from a cervical rib or elongated C7 transverse process to the first thoracic rib causing stretching and compression of the distal C8,T1 nerve roots or proximal lower trunk of the brachial plexus (8,9). There are seven subtypes that vary in origin and insertion around cervical ribs, 1st thoracic ribs, and scalene muscles (15). True NTOS not only includes sensory symptoms, but also hand muscle weakness and atrophy. True NTOS requires immediate surgical intervention to stop further damage to the brachial plexus and halt progressive hand muscle weakness and atrophy (10).

Neurogenic pectoralis minor syndrome (NPMS) is a subset of NTOS that is defined by compression of the axillary neurovascular bundle with narrowing in the retropectoralis minor space (1,16,17). The pectoralis minor muscle originates from ribs three through five and inserts on the coracoid process (14). Sanders and Rao (16) report that greater than 75% of patients diagnosed with NTOS were also diagnosed with NPMS once they integrated NPMS evaluation in their NTOS evaluation, and only 30% were diagnosed with NPMS without NTOS. NPMS is applicable to athletes with protracted shoulder posture and tightened pectoralis muscles. Factors

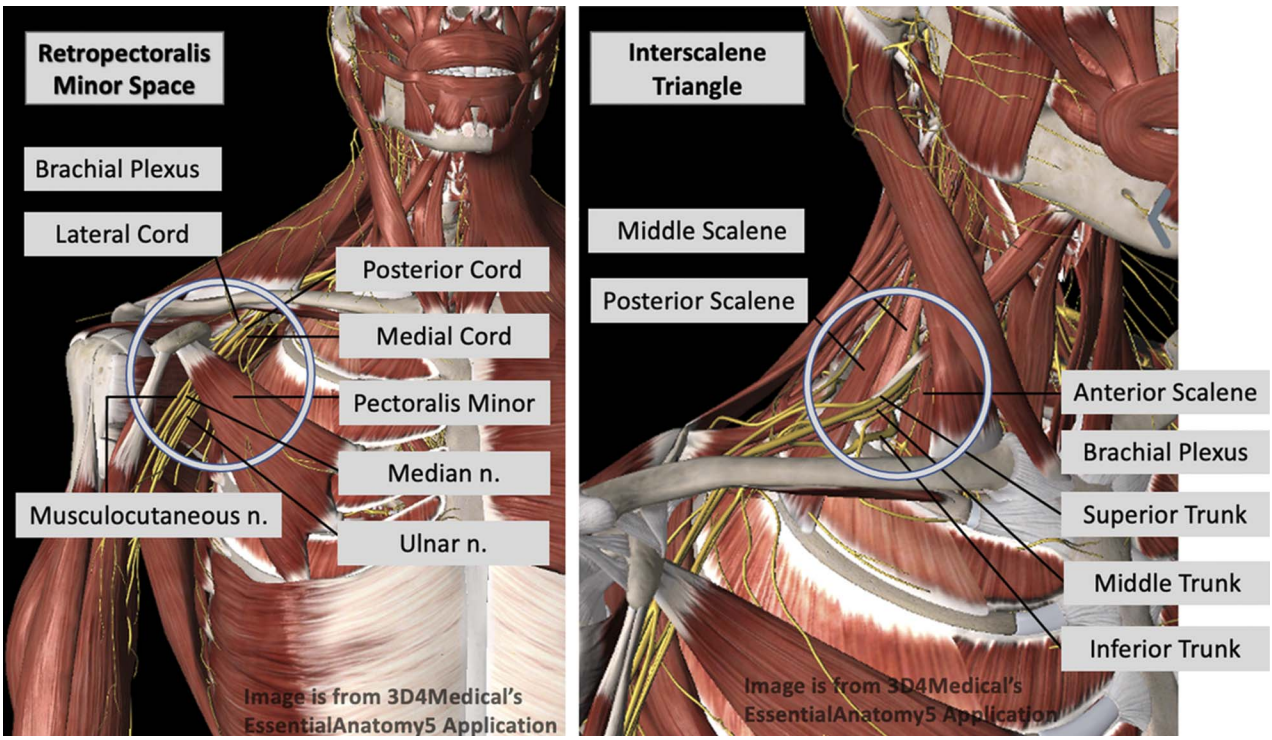

Figure 1: Retropectoralis minor space and interscalene triangle: sites of potential nerve compression.

predisposing or causing NTOS may further be defined as congenital, traumatic, and/or functional. The underlying the etiology of NTOS are reviewed.

Etiology and Pathophysiology of NTOS

Congenital, traumatic, and functional factors, or a combination of the above contribute to the potential development of NTOS (2,12). Congenital anomalies causing space occupying lesions at the interscalene space include fibromuscular bands (15,18), accessory scalenus minimus muscle (19,20), variations in scalene muscle position or insertions (scalenus anticus syndrome) (19), cervical ribs, elongated transverse process of the C7 vertebrae (19,20), and anomalous first thoracic ribs. Cervical ribs are estimated to be in approximately 1% of the general population based on radiographic images; however, ranges include 0.58% to 6.2% in Malawian and Turkish populations respectively and are more common in women than men (21).

Congenital anomalies do not necessarily correlate with a diagnosis of NTOS; most patients with cervical ribs are asymptomatic (22). A predisposing anatomical variation plus another inciting event such as neck trauma (such as from a motor vehicle accident) or repetitive stress injury may lead to scalene muscle scarring and hypertrophy, further impinging on the brachial plexus at the level of the interscalene triangle (23). Preceding neck trauma prior to the onset of symptoms occurs in 50% of people with cervical ribs and 75% of people with incomplete cervical ribs (22).

Traumatic injuries include unilateral midshaft fractures from a high speed motor vehicle accident or contact and speed sports (rugby, skiing) resulting in primary neural or vascular injury or secondary callus formation and a space occupying lesion in the costoclavicular space (11,12).

Athletes who perform high-velocity, repetitive overhead movements are at risk of developing NTOS due to functional causes (2,24). Sports include baseball, softball, volleyball, tennis, swimming, weightlifting, etc. (25). Repetitive stress injury can tear muscle fibers, cause hemorrhage and microscopic scar tissue within the scalene muscles leading to cumulative muscle fibrosis (22). Scar tissue incidence is three times greater in the scalene muscles of NTOS patients compared with controls (22). In addition, hypertrophied muscles lead to muscle imbalance, adaptive shortening of muscles, and alterations in joint biomechanics, which further contributes to the functional NTOS symptoms.

Peripheral Nerve Compression and Connective Tissue Changes

Repetitive, tensile stress on a nerve can alter the physiology and structure of the nerve. External compressive stress over a long time course can impair blood flow, lead to inflammation, endoneurial edema, and fibrotic changes (26). When nerves undergo chronic inflammation, a remodeling response seeks to add mechanical stability, and collagen is deposited in connective tissue, leading to decreased nerve compliance with nerve elongation (26). Vigorous, chronic, repetitive motion may form scar tissue or adhesions (4). Scar development may impinge upon the already narrow thoracic outlet, perpetuating the cycle that causes NTOS symptoms (4,25).

Neurogenic TOS

Incidence

Estimated incidence of NTOS varies widely, stemming from the challenges of diagnosing a complex spectrum disorder. Disputed NTOS is by far the most common type of TOS, accounting for more than 90% to 95% of all TOS cases; true NTOS is 1% of all neurogenic cases (25). In comparison, VTOS is 3% to 5%, whereas ATOS is less than 1% (2,19,27,28). Incidence is between 3 and 80 TOS cases per 1000 population (18,28). Illig et al. estimates a regional incidence of NTOS between two and three cases per 100,000 people per year (29).

Epidemiology

While athletes are an important subgroup that experiences NTOS, the patient population most recognized as presenting with NTOS symptoms are mostly women between 20 and 45 or 50 years of age (1,2,30). The female to male ratio presenting with NTOS is 3.5:1 (28).

Diagnosis of NTOS

Clinical Red Flags — TOS Subtypes

While the clinical presentation of NTOS will be discussed in reference to different subtypes, workup, and treatment plans, it is important to first convey the critical nature of excluding red flag symptoms that require urgent vascular surgical referral and additional imaging. True NTOS include hand muscle weakness and atrophy, particularly the lateral thenar muscles, indicating severe axonal loss (8,10). ATOS include arterial ischemia in the upper extremity with acute pain, digital ischemia, ulcerations, nonhealing wounds, claudication, pallor, and coldness due to arterial emboli (27,31). VTOS include arm swelling, dilation of superficial veins, cyanosis, and paresthesias in the hands due to subclavian vein obstruction (27,28,31). Paget-von Schroetter syndrome (effort thrombosis) is a rare subset of VTOS (incidence of 1 to 2 per 100,000 individuals per year) that includes physically active individuals between 15 and 45 years of age with repetitive overhead motion (with the subclavian vein compressing during the extremes of shoulder abduction and/or external rotation) (32).

Clinical Presentation

The clinical presentation of NTOS reflects the diverse nature of the syndrome. The lower brachial plexus is affected in approximately 80% of NTOS cases, and corresponds to compression of the C8 and T1 nerve roots (2). Subsequently, most patients with NTOS present with symptoms in the hand and forearm within a C8 and T1 nerve distribution. Symptoms include upper-extremity pain, paresthesias, and weakness in the hand, forearm, and shoulder (2,27). Symptoms may worsen with effort, especially with overhead movements that decrease the space within the interscalene space or activities that stretch the plexus with arms extended and abducted (12). Altered biomechanics may cause myofascial pain more proximally with head, neck, and shoulder pain (5,6,19). Sanders et al. (27) report that among 50 patients, symptom distribution in NTOS included upper-extremity paresthesia (98%), trapezius pain (92%), shoulder pain (88%), arm pain (88%), neck pain (88%), supraclavicular pain

(76%), occipital headache (76%), chest pain (72%), and paresthesias (98%) in all five fingers (58%), fourth and fifth fingers 26%, first through third fingers 14%.

High-performance athletes tend to have minimal rest symptoms and only be symptomatic during or after athletic activities; this is in comparison to NTOS patients in the general population that may have significant symptoms at rest (23). A baseball pitcher may present with a decrease in pitch velocity, grip strength, throwing stamina, and overall feel for the ball (33).

Diagnostic Challenges and Diagnostic Criteria

Establishing a diagnosis of NTOS is challenging. Symptoms may be vague, physical examination maneuvers are nonspecific, and there is no uniform test to unequivocally confirm the diagnosis (34). NTOS is often a diagnosis of exclusion (4,6). In 2016, The Society for Vascular Surgery for TOS developed the Clinical Diagnostic Criteria for NTOS. With three of four criteria met, a diagnosis of TOS can be made. These criteria include, "signs and symptoms of pathology occurring at the thoracic outlet (pain and/or tenderness), signs and symptoms of nerve compression (distal neurologic changes, often worse with arms overhead or dangling), absence of other pathology potentially explaining the symptoms, and a positive response to a properly performed scalene muscle test injection" (35).

Differential Diagnosis

NTOS clinical symptoms overlap with symptoms from other clinical diagnoses. Nocturnal numbness, for example, is a common complaint in patients with NTOS that overlaps with nocturnal numbness in carpal tunnel syndrome and ulnar neuropathy. A "dead arm" sensation during vigorous exercise may also be associated with superior labral anterior and posterior lesions as well as rotator cuff weakness (23). Nerve symptoms may originate more proximally or distally than the lower brachial plexus including C8/T1 radiculopathies, medial cord injuries, and mononeuropathies involving the median or ulnar nerve, however clinical symptoms in NTOS are not localized to one cervical nerve root or peripheral nerve distribution (6).

Muscle, tendon, and ligamentous injuries also are common in athletes. The differential diagnosis for anterior shoulder pain includes Pectoralis minor syndrome, acromioclavicular joint pathology, biceps tendinopathy, and cervical radiculopathy (36). The differential diagnosis for posterior shoulder pain includes suprascapular nerve entrapment, quadrilateral space syndrome, scapular dyskinesia/winging, acromioclavicular pathology, as well as cervical radiculopathy (36). Additional diagnoses include medial collateral ligament strain at the elbow, epicondylitis, cubital tunnel syndrome, pronator syndrome, vasculitis, fibromyalgia, multiple sclerosis, pancoast tumor, compartment syndrome, and complex regional pain syndrome (18,33).

Physical Examination and Diagnostic Workup

A thorough cervical spine, shoulder, and upper-extremity examination is warranted, including inspection of head, neck, and thoracic posture (2). A workup for NTOS would not be complete without evaluation of the scapula, as scapular dyskinesia can be due to poor muscle recruitment, and a tight pectoralis minor tendon inserting on the coracoid process can contribute to mechanical dysfunction (22). A comparative evaluation between contralateral extremities, as well as hair distribution, temperature, and skin color changes, can reveal muscle wasting and raise concern for serious pathology (2,10). Palpate over the anterior and middle scalene muscles and perform a Tinel's at the interscalene triangle just inferomedial to the coracoid process to evaluate for NTOS symptoms (34,36). Palpate the pectoralis minor tendon just inferior to the coracoid process (6,36). Tenderness suggests potential NPMS. Perform a Spurling's test and deep tendon reflexes to evaluate for cervical radiculopathy (25).

False-positive results are common in provocative testing for TOS such as Adson's test (2%), Costoclavicular test (10%), and Wright Tests (16.5%) (37). The upper limb tension test is a nonspecific neural tension test that includes shoulder abduction to 90 degrees, with elbows in full extension, with forearms pronated and wrists in a neutral position. With bilateral wrist extension there is reproduction of NTOS symptoms (6,31). Additional modifications include tilting the head from ear to shoulder (31).

The elevated arm stress test (also known as the Roos test) is limited in its utility by positioning in a seated position with shoulder abduction at 90 degrees and elbow flexion to 90 degrees with opening and closing of the fist rapidly for 3 min, because this will exacerbate conditions other than NTOS. An inability to complete the test due to pain or reproduction of NTOS symptoms yields a positive result (6,31). This test also may create a steal phenomenon and lead to one limb demonstrating less vascular flow than the other.

Imaging

Imaging may include X-rays, magnetic resonance imaging (MRI), magnetic resonance (MR) neurography, computed tomography (CT) scans, ultrasound, electrodiagnostic studies, and vascular studies (6,19). NTOS workup starts with evaluating for a potential cervical rib, elongated C7 transverse process, and anomalous first rib with an anterior-posterior chest X-ray or cervical X-rays (31). If there is no osseous abnormality, this greatly decreases the likelihood of ATOS (27). True NTOS may include a cervical rib or fibromuscular bands; therefore, further evaluation includes an MRI or CT. As technology has evolved, the visualization of nerves has improved to even demonstrate nerve fascicles on MR neurography (31). Dynamic MRI is gaining attention to evaluate the thoracic outlet dynamically with arms adducted and at 90 degrees of abduction. Ultrasound has utility in cases of acute thrombosis in the upper extremity, as does MR Angiography and MR venography (31). Vascular studies need to be ordered for any patient with concerning symptoms for ATOS or VTOS, and provocative positioning is beneficial to dynamically evaluate the thoracic outlet.

Electrodiagnostic testing includes electromyography (EMG) and nerve conduction studies, which are usually normal in patients with NTOS, and have limited clinical utility outside of detecting more severe cases of demyelination, conduction block, or axonal disruption to sensory and/or motor fibers (1). Electrodiagnostic testing evaluates the worst, or symptomatic fascicles together with the best, or large and myelinated fibers, and therefore there may be no evidence of nerve slowing as the myelinated fibers continue to demonstrate normal

conduction velocities. Electrodiagnostic testing should focus primarily on C7 through T1 nerve fibers. Part of electrodiagnostic testing includes median and ulnar nerve conduction studies, both motor and sensory, as well as EMG testing of C7 through T1 innervated muscles, and F waves. In NTOS the sensory medial antebrachial cutaneous nerve will sometimes demonstrate conduction velocity slowing when all other electrodiagnostic testing is normal (27).

Ultrasound has limitations in field of view with shadowing artifact below the clavicle. It does not visualize the costoclavicular space, and deeper structures such as C8 and T1 nerve roots are incompletely visualized. Sucher (38) reports that a tight Pectoralis Minor Muscle visualized on ultrasound during abduction can be seen to deform around the brachial plexus. Because of the pectoralis minor muscle shortening there is no slack to account for the pressure from the neurovascular bundle pushing on the muscle and the Pectoralis Minor muscle is seen to bow around the neurovascular bundle. The degree of pectoralis minor deformation is described as bowing with a pectoral bowing ratio seen more commonly in cases of NPMS (38). The pectoral bowing ratio is expressed as a percentage with a normal pectoral bowing ratio of <10% (39). A case series of 15 patients included a pectoral bowing ratio of 17.1% as compared with the people without NPMS with a pectoral bowing ratio of 1.8% (39).

Overview of Nonsurgical Treatment

Nonsurgical management includes activity modification, relative rest of the affected upper extremity, postural mechanics, stretching, and specific muscle strengthening (2).

Physical Therapy

Physical therapy is a core component of conservative treatment for NTOS. Physical therapy focuses on improving posture, biomechanics, modifying activities, stretching tightened muscles, stabilizing the scapula, strengthening weakened muscles, educating athletes about the goals of care, and developing a home exercise program.

Activity modification is needed to limit repetitive, overhead movements. An athlete's posture and biomechanics have likely evolved for sport-specific and individual patterns of use with adaptive muscle use and tendon shortening occurring over time. Head forward posture and scapular protraction tightens the anterior and middle scalenes, sternocleidomastoid, trapezius, levator scapulae, pectoralis muscles, suboccipital muscles, and elongates the middle and lower trapezius muscles (40,41). Muscles in a shortened or lengthened position are at a mechanical disadvantage (41). Postural correction includes reducing forward head and protracted shoulder posture that narrows the thoraco-coraco-pectoral space (42). Scapular retraction opens up the thoraco-coraco-pectoral space (Fig. 2) (42). Postural restoration

Figure 2: Scalene and pectoralis muscle stretching and scapular retraction. Scalene stretch (A, B, C). (A) Sit upright, hold ipsilateral arm on seat of chair to depress ipsilateral shoulder. Place contralateral arm on head. (B) Lateral head tilt with contralateral arm. (C) Head rotates with chin toward ipsilateral shoulder. (D) Pectoralis muscle stretch in corner with arms below shoulders. (E) Pectoralis muscle stretch in corner with arms above shoulders. (F) Scapular retraction.

techniques include neuromuscular re-education to restore proper kinematics. Emphasis has been on stretching the muscles that close the thoracic outlet such as the scalene and pectoralis muscles and strengthening the muscles that open the thoracic outlet such as the scapular stabilizing muscles, the rhomboids and the middle/lower trapezius muscles (3).

Scapular kinematics is key, and evaluation of joint mobility and parascapular control of the rhomboids, serratus anterior, and the middle/lower trapezius is necessary. There may be weakness in these lower scapular stabilizers and hypertrophy in scapular elevators that are overused with arm movements, especially the upper trapezius, levator scapulae, and upper rhomboids (41). Stretching tends to include the upper trapezius, levator scapulae, pectoralis minor, scalenes, sternocleidomastoid, suboccipitals, and pectoralis major muscles (41). The scalene muscles may be stretched using the contralateral arm while in the seated position. Examples are depicted in Figure 2.

Physical therapy aims to recruit the lower scapular stabilizers (42). Nonspecific strengthening does not focus on efficient muscle recruitment and can potentially worsen symptoms. Targeted, progressive strengthening aims to improve muscular endurance with low weight and a high number of repetitions (3,41).

Cognizant use of muscle relaxants, anti-inflammations, neuropathic pain medication, and possible trigger point injections can act as an adjunct to relaxing tight muscles (25,33). If modalities can temporarily improve discomfort and compliance with an exercise program, then they are recommended. Modalities may include transcutaneous electrical nerve stimulation unit, thermal agents, ultrasound, and iontophoresis (41). Neural tissue gliding aims to improve neural mobility relative to surrounding tissue and ameliorate adhesion formation (42).

Injections

One of the Clinical Diagnostic Criteria for NTOS includes "a positive response to a properly performed scalene muscle test injection," which is indicated by relief of NTOS symptoms by an anesthetic injection to the scalene muscles (35). While this test injection can diagnose scalene muscle etiology of NTOS, it would not alleviate a cervical rib or a tight pectoral muscle. Over time, the diagnostic test injections have expanded to include the diagnostic injections around the pectoralis minor tendon as part of NPMS evaluation. In athletes this test injection may be beneficial to evaluate if the source of symptoms is from the interscalene triangle, pectoralis minor space, or both. A test injection is not necessary for a diagnosis of NTOS, and there is the risk of iatrogenic injury.

Anesthetic Injections

Anesthetic injection into the anterior scalene, middle scalene, and/or pectoralis minor muscles can aid in diagnosis (34) and predict response to potential surgery (23,33). There are variations in the literature about which anesthetic to use (lidocaine, ropivacaine, bupivacaine, for example), the ideal amount of injectate (ranging between ~1 mL and 5 mL into each muscle), and if the injection should be done in one or multiple muscles simultaneously (43).

Botulinum Toxin Injections

Botulinum toxin injections have fallen out of favor since a randomized, placebo controlled, double-blinded trial of botulinum toxin to the anterior and middle scalene muscles did not demonstrate a clinically or statistically significant improvement compared with placebo for patients with TOS (44).

Ultrasound-Guided Hydrodissection

Ultrasound-guided hydrodissection of the brachial plexus is an emerging nonsurgical tool using fluid injected under pressure to separate nerves from fascial compression (45). Scant literature exists on ultrasound-guided hydrodissection as a treatment modality for NTOS, however there is substantial literature on ultrasound-guided deep regional nerve blocks around the neck and shoulder for analgesia (45). Releasing the fascial compression on nerves, may relate to the release of nervi nevorum or vasa nervorum compression and may ameliorate neuropathic pain (45). Lam et al. (45) describes four approaches for ultrasound-guided hydrodissection of the brachial plexus using 5% dextrose water +/– anesthetic.

Functional Assessment Tools and Performance Metrics

Several functional assessment tools can be used to evaluate function before embarking on physical therapy or an intervention, and later can then be used to monitor progress.

The disability arm shoulder hand (DASH) questionnaire is a 30-item self-reported questionnaire that assesses a person's ability to perform upper-extremity activities, with difficulty rated on a five-point Likert scale (13). The QuickDASH is a shorter, 11-item questionnaire (24). Both include a Sports/Performing Arts Module for high-performance athletes (24). Quality of life measures include the Cervical Brachial Symptom Questionnaire (14 questions plus pain diagram), the Short-Form 36 (36 questions), and the Short-Form 12 (12 questions) (4,13). Performance metrics in baseball pitchers include a strikeout/walk ratio, hits per inning pitched, and earned run average (46).

Surgical Treatment

Surgical decompression may include one or more of the following surgeries: first rib resection (transaxillary vs supraclavicular vs posterior approach), anterior and/or middle scalenectomy, cervical rib resection, release of fibromuscular bands (6,19), and pectoralis minor tenotomy (a minimal-risk surgery for NPMS) (17). A prospective, observational cohort of NTOS who received surgical decompression (supraclavicular decompression with or without pectoralis minor tenotomy) versus physical therapy demonstrated an improvement in QuickDASH scores. Surgical decompression demonstrated 47.9% improvement in QuickDASH score (median, 12 months of follow-up for surgery) compared with 29.5% improvement in physical therapy (median, 21.1 months of follow-up for physical therapy) (47). There were no factors that predicted which NTOS patients would benefit from physical therapy versus surgery (47).

Similarly, another study comparing surgical decompression (supraclavicular first rib resection and brachial plexus neurolysis) versus physical therapy in patients with NTOS demonstrated an improvement in Mean QuickDASH score of 87% versus 73.5% respectively (evaluated at a mean of 34.5 months) (24). Selection of surgical candidates was biased toward those who responded best to physical therapy, noting this as a predictor of surgical success (24). Eighty-two percent of all NTOS athletes (surgical and nonsurgical athletes) returned to full athletic competitive levels (24). Major League

Baseball pitchers who received surgical decompression for NTOS demonstrated similar performance metrics compared between the preoperative and postoperative career time periods. (46). Among competitive athletes post first rib resection and scalenectomy, 70% reported the same or improved athletic activity prior to surgery and 82% demonstrated symptom resolution (evaluated at a mean follow up of 3.9 years) (48).

Return To Sport Considerations

Considerations for returning to sport include evaluation of the athlete's range of motion, muscle strength, and functional athletic ability (49). Sport-specific testing is recommended to recreate similar athletic activities. Medical staff can evaluate the athlete's functional ability and monitor for any discomfort on the part of the athlete (49). Assessing mental readiness to return to play is included in the safety evaluation, especially for contact sports (49). Finally, communication with athletic trainers, coaches, team physicians, and other medical personnel can help gather information necessary for return to play decision making.

CONCLUSIONS

Athletes who perform repetitive overhead activities are a unique patient population at risk of developing NTOS. Nuanced, sport-specific symptoms make it challenging to diagnose NTOS in athletes. Requesting that the athlete perform sport-specific activities prior to evaluation can aid in diagnosis. Treatment varies depending upon the subtype of NTOS. Considering the diverse congenital, traumatic, and functional etiologies of NTOS and excluding red flag symptoms during clinical presentation is part of a comprehensive and prioritized workup. While most cases of NTOS include nonsurgical treatment and physical therapy, True NTOS requires surgery. Core components of physical therapy include targeting scapular stability, posture, altered biomechanics, and muscle imbalance with TOS-specific stretching and strengthening to open the thoracic outlet. Athletes seek to improve their functional impairment and return to their high level of athletic performance. Nonsurgical versus surgical treatment options have both demonstrated improvement in functional outcome data in patients with NTOS to varying degrees. Comprehensive treatment plans involve multidisciplinary care, physical therapy, activity modification, functional assessment tools, performance metrics, and return to sport considerations.

The authors declare no conflict of interest and do not have any financial disclosures.

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