



Neurogenic Thoracic Outlet Syndrome with Concomitant Musculoskeletal Injury

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Abstract

Background: Neurogenic thoracic outlet syndrome (nTOS) results from compression or irritation of the brachial plexus within the thoracic outlet, often associated with anatomical abnormalities, repetitive activity, or trauma. Symptoms include upper-extremity pain, paresthesia, weakness, and fatigue. Because symptoms are nonspecific, diagnosis relies on physical examination and selective diagnostic testing. This case describes the diagnostic and management challenges posed by symptoms of nTOS in the presence of concomitant shoulder pathology.

Case Report: A 24-year-old recreational marathon runner developed anterior left shoulder pain after abruptly extending both arms during a workplace incident. Initial examination and imaging supported a shoulder strain and acromioclavicular joint degeneration. Despite medication, injections, and physical therapy, symptoms persisted for more than four years and progressed to include periscapular pain, paresthesia, numbness, involuntary hand movements, upper-extremity tremors, fatigue, sleep disturbance, and functional limitations. Electromyography and nerve conduction studies demonstrated C6-C7 radiculopathy. Subsequent imaging identified rotator cuff and labral pathology and possible brachial plexus involvement. Arthroscopy revealed a Buford complex, unstable biceps anchor, multidirectional glenohumeral instability, and a superior labral tear. Biceps tenodesis, superior labral repair, anterior labral capsulorrhaphy, and capsular plication were performed, followed by structured rehabilitation.

Discussion: Coexisting orthopedic and neurologic abnormalities can obscure recognition of symptoms suggestive of nTOS, prolong symptoms, reduce quality of life, and delay appropriate intervention. Normal or nonspecific findings should not prevent reconsideration of the differential diagnosis when symptoms progress or respond poorly to treatment. This is especially important when structural abnormalities offer plausible but incomplete explanations for the evolving clinical presentation.

Conclusion: Clinicians should maintain a broad differential diagnosis, reassess persistent upper-extremity symptoms, and pursue timely multidisciplinary referral when neurologic features emerge. Improved continuity of care and standardized nTOS diagnostic criteria may facilitate earlier recognition and appropriate management.

Keywords: Neurogenic Thoracic Outlet Syndrome; Shoulder Instability; Cervical Radiculopathy; Brachial Plexus; Case Report

Background

The thoracic outlet is the anatomical space bordered by the clavicle and first rib through which the subclavian artery, subclavian vein, and brachial plexus traverse [1]. Thoracic outlet syndrome (TOS) encompasses a group of disorders resulting from compression of one or more of these neurovascular structures, often secondary to congenital anatomical variations, repetitive upper-extremity activity, or traumatic injury [2]. Diagnosis remains challenging because no universally accepted diagnostic criteria exist, contributing to variability in both clinical recognition and reported incidence [2]. Consequently, estimates of TOS prevalence vary widely in the literature, ranging from 3 to 80 cases per 1,000 individuals [2].

TOS most commonly affects adolescents and middle-aged adults and is reported more frequently in females between 20 and 50 years of age [2]. Neurogenic thoracic outlet syndrome (nTOS), the most common subtype, demonstrates a female predominance in the general population [3]. However, this epidemiologic pattern differs among overhead athletes, in whom younger males appear to have a higher incidence than females [4]. In younger individuals, TOS is frequently associated with congenital anatomical abnormalities or repetitive upper-extremity activities that place stress on the thoracic outlet [2].

This case report describes a 24-year-old patient whose initial symptoms and examination findings were consistent with a musculoskeletal shoulder injury. As symptoms persisted despite

conservative management, and progressive neurologic features emerged, making the diagnostic process increasingly complex. It remains unclear whether nTOS was present at the time of the original injury or developed later in association with prolonged musculoskeletal dysfunction and compensatory movement patterns. The case highlights the diagnostic complexity of TOS and emphasizes the importance of reassessing the differential diagnosis when a patient's symptoms evolve or fail to improve as expected.

Case Report

The patient's injury occurred in March 2021, and the diagnostic evaluation and management extended over approximately four years before definitive surgical intervention. The following chronology summarizes the patient's clinical course from the initial injury through diagnosis and treatment.

The patient was a 24-year-old recreational marathon runner who had been training regularly since 2020. Their medical history was significant for functional scoliosis and degenerative changes of the L4-L5 facet joints; however, these conditions were not believed by the treating physician to have contributed to the presenting complaint. The injury occurred while the patient was working and pushing carts outdoors during rainy conditions. After being startled by a vehicle horn, the patient abruptly jerked both arms backward and immediately experienced sharp pain localized to the anterior aspect of the left shoulder. Despite completing the remainder of the work shift and returning to work the following day, symptoms persisted.

Following the injury, the patient initially contacted a telemedicine nurse and was advised to manage symptoms with nonsteroidal anti-inflammatory drugs (NSAIDs) and cryotherapy. After reporting the injury to their employer, they were evaluated by a workers' compensation physician. Physical examination demonstrated tenderness over the left acromioclavicular (AC) joint, painful shoulder range of motion, and mildly positive shoulder impingement testing. Conservative management included naproxen (500 mg twice daily), cryotherapy, a corticosteroid injection, and referral to physical therapy three times per week. Despite treatment, the patient's pain progressively worsened and extended from the anterior shoulder into the trapezius, supraspinatus, and rhomboid regions. They also developed difficulty sleeping due to persistent pain.

Following several weeks of physical therapy without clinical improvement, magnetic resonance imaging (MRI) of the left shoulder without contrast demonstrated findings consistent with a left shoulder strain and degenerative changes of the AC joint. Radiographs demonstrated no acute osseous abnormalities.

Over the following year, symptoms continued despite conservative management. The patient discontinued formal physical therapy and instead performed self-directed cupping therapy at home. During this period, they reported the onset of intermittent involuntary twitching of the left hand and fingers with upper-extremity activity. Although the workers' compensation case was settled in July 2022, the patient continued to seek care through their primary care physician because of ongoing pain and functional limitations. By September 2022, they reported extreme difficulty tolerating work-related activities and decided to discontinue work. Although the patient also reported pain with cervical rotation at this time, no formal cervical-spine examination was documented and imaging was not ordered.

Between October 2022 and April 2023, the patient was awaiting establishment of care with a new primary care physician. During this interval, symptom management consisted of massage therapy and self-administered cupping without participation in a structured rehabilitation program.

In May 2023, worsening left shoulder pain accompanied by numbness and tingling extending into the left upper extremity prompted the patient to seek additional diagnostic evaluation. Repeat

radiographs of the left shoulder remained unremarkable, however, radiographs of the cervical spine and electromyography (EMG) with nerve conduction studies (NCS) were ordered. Scheduling delays postponed testing until August and September 2023. Nerve conduction studies were within normal limits; however, EMG findings were consistent with C6-C7 radiculopathy. Radiographs of the cervical spine showed decreased space at the C2-C3 level and overall reversal of the cervical lordosis.

The patient's symptoms continued to progress despite conservative management. At a routine follow-up in January 2024, she reported worsening shoulder pain with persistent numbness and paresthesia involving the left upper extremity. MRI of the left shoulder performed in March 2024 demonstrated subacromial impingement involving the supraspinatus, mild AC osteoarthropathy, mild supraspinatus tendinosis, and mild subdeltoid bursitis. MRI of the cervical spine, also performed in March 2024, showed decreased space at the C2-C3 level and disc bulges at the C5-C6 and C6-C7 level. No spinal stenosis and no compression of the traversing nerve roots was identified. One month later, the patient reported the development of severe episodic tremors involving the left hand, which occasionally progressed to involve the entire upper extremity and resulted in significant fatigue following activity.

Between April and August 2024, symptom management consisted of topical diclofenac gel, lidocaine patches, over-the-counter capsaicin cream, Tiger Balm, and heat therapy for muscle spasms. No formal rehabilitation exercises were performed during this period. Corticosteroid injections administered in September and December 2024 provided only transient symptom relief. Repeat EMG and NCS performed in November 2024 again demonstrated C6-C7 radiculopathy.

Despite continued conservative management, including an additional corticosteroid injection in March 2025, the patient was diagnosed with nTOS due to persistent neurological symptoms and EMG and NCS results from November 2024. MRI of the left shoulder and arm performed in August 2025 demonstrated rotator cuff tendinopathy with a partial-thickness supraspinatus tear, a focal posterior labral tear with an associated paralabral cyst, mild subacromial-subdeltoid bursitis, findings suggestive of brachial plexus involvement, and lateral epicondylitis. Given the persistence of symptoms despite prolonged nonoperative treatment and the cumulative clinical and imaging findings, the patient was referred for surgical evaluation.

Treatment

The patient underwent arthroscopic left shoulder surgery in September 2025. Intraoperative findings included a Buford complex, an unstable biceps anchor, multidirectional glenohumeral instability, and a superior labral tear. The supraspinatus tendon was inspected and found to be intact without evidence of tearing or significant degenerative changes. Based on these findings, arthroscopic biceps tenodesis with debridement of the long head of the biceps tendon was performed, followed by superior labral repair. To address the multidirectional instability, an anterior labral capsulorrhaphy with capsular plication was also completed. Although subacromial debridement had been planned preoperatively, it was not performed because intraoperative assessment demonstrated that the completed procedures were sufficient to address the patient's pathology.

Following surgery, the patient reported resolution of the perceived "compression" in her shoulder, along with all neurologic symptoms. Her remaining pain was limited to discomfort associated with postoperative recovery. The patient was referred to physical therapy for a structured postoperative rehabilitation program focused on protecting the surgical repair while progressively restoring shoulder range of motion, strength, and functional capacity. The patient reported exercise-related soreness without exacerbation of pain or recurrence of neurological symptoms.

By May 2026, the patient was able to resume full functional daily activities. Although not yet working, she is hopeful to be able to start work again.

Discussion

This case highlights the diagnostic complexity of nTOS in the presence of concomitant shoulder pathology and prolonged conservative management. The patient's clinical course spanned more than four years and was characterized by persistent pain, progressive neurologic symptoms, multiple imaging studies, and repeated conservative interventions. The evolving presentation raises the possibility that nTOS developed as a sequela of chronic shoulder dysfunction, potentially related to altered movement patterns, postural adaptations, or ongoing neural irritation. However, the precise relationship between the shoulder pathology and subsequent neurologic symptoms cannot be definitively established. This prolonged timeline illustrates the challenges clinicians may encounter when evaluating patients with persistent upper-extremity symptoms that overlap with more common musculoskeletal conditions.

Neurogenic thoracic outlet syndrome (nTOS) is the most common subtype of thoracic outlet syndrome, accounting for approximately 90–95% of all cases [5]. It results from compression or irritation of the brachial plexus within the thoracic outlet, most commonly at the scalene triangle or beneath the pectoralis minor [6]. Clinical presentation is often variable and includes pain, paresthesia, weakness, fatigue, and impaired upper-extremity function [6, 7]. Because these symptoms frequently overlap with cervical radiculopathy, rotator cuff pathology, labral injuries, and other shoulder disorders, diagnosis remains challenging. The absence of universally accepted diagnostic criteria and the limited sensitivity and specificity of available diagnostic tests further contribute to delayed recognition [6].

The patient's initial presentation of shoulder pain, painful range of motion, and acromioclavicular joint tenderness was consistent with common shoulder pathology, supporting the initial diagnosis of shoulder strain. As the clinical course progressed, however, the patient developed paresthesia, numbness, tremors, and progressive functional loss involving the left upper extremity. Although subsequent electromyography demonstrated C6-C7 radiculopathy, these findings did not fully explain the patient's evolving clinical presentation. This case demonstrates the importance of reassessing the differential diagnosis when patients fail to respond to appropriate conservative management or develop progressive neurologic symptoms inconsistent with the original diagnosis.

The presence of multiple concomitant shoulder pathologies further complicated the diagnostic process. Intraoperative findings included a Buford complex, multidirectional glenohumeral instability, an unstable biceps anchor, and a superior labral tear, while imaging also demonstrated rotator cuff tendinopathy and a partial-thickness supraspinatus tear. Although many of these findings are capable of producing pain and functional limitations independently, they may also obscure the recognition of nTOS by providing alternative explanations for persistent shoulder symptoms. The treating surgeon was unable to determine whether these structural abnormalities contributed to the development of nTOS or represented coexisting pathology.

The patient's shoulder abnormalities may provide a biomechanical context for the progressive neurologic presentation. A Buford complex is characterized by a cord-like middle glenohumeral ligament and absence of the anterosuperior labrum [8]. Although historically considered a benign anatomical variant, it may be associated with an increased risk of superior labrum anterior-to-posterior (SLAP) tears; however, it remains unclear whether it is congenital or may develop in association with early shoulder instability [8]. In this patient, the Buford complex occurred alongside multidirectional glenohumeral instability, an unstable biceps anchor, and a superior labral tear.

Together, these structural findings may explain the patient's persistent symptoms, including pain and instability, altering her upper-extremity biomechanics and functional movements. These changes may require compensatory muscle activation or altered movement patterns to maintain shoulder stability [9]. Over time, these adaptations could plausibly have contributed to irritation or compression of the brachial plexus. However, neither these studies nor the findings from this case establish a causal relationship between the shoulder pathology and nTOS. Therefore, it remains unclear whether nTOS developed as a sequela of chronic shoulder dysfunction or represented a coexisting condition that became clinically apparent as the presentation evolved.

Current recommendations emphasize that nTOS is primarily a clinical diagnosis requiring a comprehensive history and physical examination, consideration of competing diagnoses, and selective use of diagnostic testing [6]. When neurologic symptoms accompany persistent upper-extremity pain, examination should include cervical range of motion, strength, sensation, reflexes, upper-extremity neural tension, and assessment for symptom reproduction over the scalene triangle and brachial plexus. Diagnostic testing procedures may include the elevated arm stress test, upper-limb tension test, and Tinel sign over the supraclavicular brachial plexus [6, 10]. A recent multidisciplinary expert consensus identified five clinically important features for distinguishing nTOS, including 1) improvement following targeted pectoralis minor or scalene injections, 2) radiating symptoms reproduced by supraclavicular provocative maneuvers, 3) symptoms reproduced by infraclavicular provocative maneuvers, 4) pain reproduced with 90° of shoulder abduction and external rotation or upper-limb tension testing, and 5) participation in work or exercise involving repetitive upper-extremity stress [11]. Electrodiagnostic testing and cervical imaging may assist in identifying cervical radiculopathy, peripheral nerve entrapment, or brachial plexopathy, but findings should be interpreted within the complete clinical presentation.

In this case, the patient reported pain with cervical rotation in September 2022, but no formal cervical-spine examination was documented at that time. Subsequent radiographs demonstrated decreased disc space at C2-C3 and reversal of cervical lordosis, while MRI identified disc bulges at C5-C6 and C6-C7 without spinal stenosis or nerve-root compression. These findings did not provide a clear structural explanation for the C6-C7 radiculopathy identified through electrodiagnostic testing, further complicating differentiation among cervical radiculopathy, nTOS, and potentially coexisting sites of neural compromise. Additionally, no nTOS-specific examination or targeted diagnostic testing was documented, limiting the ability to determine whether the patient met proposed criteria to be diagnosed with nTOS. Earlier comprehensive examination of the cervical spine and thoracic outlet may have provided a clearer diagnostic pathway as the neurologic symptoms emerged.

The patient's prolonged and evolving clinical course also demonstrates the cumulative effect of persistent symptoms on functional outcomes. Progressive pain, weakness, paresthesia, and upper-extremity fatigue ultimately resulted in an inability to continue working, reduced participation in recreational activities, sleep disturbances, and diminished quality of life. These findings are consistent with previous reports describing chronic pain, functional disability, and work absenteeism among individuals with nTOS [3].

Psychosocial factors are also relevant when evaluating and managing the effects of prolonged nTOS symptoms. Throughout the four-year course of care, the patient intermittently discontinued supervised physical therapy and instead relied on self-directed symptom management strategies, including massage, cupping therapy, topical analgesics, and heat. Previous literature has demonstrated that psychosocial factors, including depression, self-efficacy, pain catastrophizing, functional disability, and reduced quality of life are associated with poorer adherence to rehabilitation and less favorable

outcomes in individuals with chronic pain [12, 13]. Although the reasons for inconsistent adherence cannot be definitively established, prolonged pain and limited clinical improvement may have influenced the patient's willingness to continue rehabilitation. These factors were not formally assessed in this patient but may represent important considerations when managing individuals with persistent symptoms.

This case also illustrates the challenges that can occur during transitions between healthcare systems and providers. Multiple healthcare providers, delays in specialty referrals and diagnostic testing, and continued reliance on the initial diagnosis contributed to an extended diagnostic timeline. Therefore, the prolonged clinical course cannot be attributed solely to the workers' compensation system or the initial treating clinicians. Rather, it underscores the importance of continuity of care, communication among providers, and comprehensive reassessment whenever symptoms evolve or fail to respond as expected. These principles apply across workers' compensation, private medical, and rehabilitation settings. When combined with the absence of standardized diagnostic criteria for nTOS and the presence of concomitant shoulder pathology, these factors likely contributed to the prolonged time to diagnosis observed in this patient.

As a single case report, these findings are not intended to be generalized to all patients with nTOS. However, the extended longitudinal follow-up provides insight into the diagnostic and management challenges that may occur when nTOS presents alongside other musculoskeletal disorders. This case underscores the importance of maintaining a broad differential diagnosis in patients with persistent upper-extremity pain and neurologic symptoms that fail to improve with appropriate conservative treatment. Early recognition of nTOS, ongoing reassessment of the clinical presentation, and timely referral to clinicians experienced in the evaluation of thoracic outlet syndrome may reduce diagnostic delays and improve patient outcomes.

Conclusion

This case highlights the diagnostic complexity of nTOS, particularly when they present alongside coexisting musculoskeletal shoulder pathology and cervical radiculopathy. Overlapping clinical findings, the absence of standardized diagnostic criteria, and an initial focus on more common musculoskeletal conditions contributed to a prolonged diagnostic course. The resulting delay was associated with persistent pain, progressive functional limitations, and reduced quality of life. The patient reported resolution of the perceived shoulder compression and neurologic symptoms following surgical management of the identified shoulder pathology. However, the relationship between the shoulder abnormalities and the nTOS symptoms cannot be definitively established.

For athletic trainers and other rehabilitation professionals, this case underscores the importance of maintaining a broad differential diagnosis and recognizing when a patient's clinical presentation deviates from the expected course of recovery. Ongoing reassessment, comprehensive history taking, and timely referral for additional evaluation are essential when symptoms persist despite appropriate conservative management. Attention to both physical and psychosocial factors throughout rehabilitation may further optimize patient outcomes.

Although the findings from a single case cannot be generalized, this report provides clinically relevant insight into the challenges of evaluating and diagnosing possible nTOS in the presence of multiple coexisting conditions. Clinicians should recognize that TOS may coexist with other shoulder or cervical disorders and that reliance on a single diagnosis may delay identification of contributing pathology.

A multidisciplinary approach incorporating clinical examination, diagnostic imaging, electrodiagnostic testing, and specialist consultation may improve diagnostic accuracy and facilitate more timely intervention.

Future research should focus on developing more standardized diagnostic criteria for TOS and identifying clinical features that distinguish it from other causes of upper-extremity pain and neurologic dysfunction. Additional investigation into the interaction between TOS and concomitant musculoskeletal disorders, as well as the psychosocial consequences of prolonged diagnostic delay, may further inform patient management and rehabilitation strategies.

Conflict of Interest: The authors declare no conflict of interest.

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