Literature Review

Optimizing Pain Relief in Refractory Thoracic Outlet Syndrome: The Role of Ultrasound-Guided Injections

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Background: Thoracic outlet syndrome (TOS) encompasses a range of symptoms originating from the compression of neurovascular structures, often leading to significant morbidity. Neurogenic thoracic outlet syndrome (N-TOS) frequently manifests as brachial plexus neuropathy, with a subset of patients experiencing refractory pain that does not respond to conservative treatments.

Objectives: This review aims to consolidate current evidence to evaluate the efficacy of available ultrasound (US)-guided injection techniques, including muscle injections, hydrodissection, regenerative therapies, and nerve blocks, in managing refractory pain associated with N-TOS. Additionally, this study aims to provide clinical guidance for pain management in refractory TOS through current treatment strategies, offering structured guides that physicians can use as practical tools.

Methods: A literature search was conducted across various academic databases to identify studies addressing US-guided interventions for refractory N-TOS. Relevant data regarding treatment efficacy, patient outcomes, and procedural details were extracted and synthesized narratively, as well as using structured tables and frameworks to aid in clinical decision-making.

Results: US-guided injection techniques have demonstrated effectiveness in managing refractory pain that occurs after TOS surgery. Muscle injections, particularly botulinum toxin and local anesthetics, target muscle spasms, while hydrodissection alleviates nerve entrapment. Additionally, nerve blocks, such as epidurals and stellate ganglion blocks, provide targeted pain relief by addressing specific nerve pathways. However, although regenerative therapies, including dextrose prolotherapy and platelet-rich plasma (PRP), show great potential for tissue healing, they remain under research and available data on them are limited.

Limitations: The effectiveness of these interventions may vary based on individual patient factors, practitioner experience, and the complexity of TOS presentations. Furthermore, while US-guided injections are well-established, the role of regenerative therapies requires further investigation due to a lack of standardized protocols and robust clinical trials, calling for future research.

Conclusion: US-guided injection techniques represent a promising approach for managing refractory pain in N-TOS, offering tailored pain relief strategies. However, ongoing research is essential to clarify the efficacy of regenerative therapies and to optimize treatment protocols, ultimately enhancing patient outcomes and quality of life.

Key words: Refractory thoracic outlet syndrome, pain relief, ultrasound-guided injections, nerve entrapment, muscle spasms, scar tissue, interventional pain management, regenerative therapies, brachial plexus, targeted interventions

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Thoracic outlet syndrome (TOS) is a complex collection of symptoms caused by compression of neurovascular structures as they pass through the thoracic inlet, shoulder girdle, and axilla and into the arm. First described in 1956 and with complex anatomy and pathophysiology behind it, TOS consists of 3 primary

compartments: the interscalene triangle (formed by the anterior and medial scalene muscles and the first rib), the costoclavicular space (between the clavicle and first rib), and the retropectoralis minor space (near the coracoid process). Traditionally, TOS are subdivided into neurogenic, venous, and arterial categories. In contrast, neurogenic thoracic outlet syndrome (N-TOS) manifests as brachial plexus neuropathy, which may be associated with the wasting of muscles and is confirmed by nerve conduction studies. N-TOS is commonly associated with traumatic injury, prolonged periods of repetitive activities, including athletics or working at computers, anatomical variations, or malignancy. Although most TOS patients respond to conservative treatment, a subset of such patients will ultimately require surgical intervention for persistent symptoms (1,2). Given the multifactorial nature of N-TOS, an effective method for the diagnosis and treatment thereof is needed.

TOS is uncommon, and the lack of defined diagnostic criteria limits its usefulness in population studies. Therefore, little is known about the prevalence of refractory TOS specifically. Bearing that limitation in mind, epidemiological estimates of TOS prevalence range from 3 to 80 cases per 1,000 individuals, depending on the population and diagnostic criteria used (3). Notably, among patients diagnosed with TOS, a subset of around 10-20% may experience refractory symptoms that do not respond to conservative treatments. These cases, in which symptoms persist despite standard management, are often considered candidates for surgery or US-guided injections (4).

TOS is still a very controversial entity in terms of its diagnosis and the best treatment modalities for the condition (5,6). Even after surgical decompression of a nerve or vascular structures, pain associated with TOS can persist, complicating the postoperative course and affecting the patient's quality of life. Importantly, surgical interventions can lead to long-term complications such as brachial plexus and phrenic nerve dysfunction, apical lung herniation, and graft occlusion after artery bypass (7). While US-guided injections show promise in managing TOS, current literature highlights the need for standardized protocols and further investigation into the clinical efficacy of TOS treatments. Positive outcomes have been reported from US-guided injection therapies, including botulinum toxin injections, but gaps remain that limit consistent and reliable relief for a wider general population (8). Recent evidence has also identified specific anatomical challenges in targeting the scalene muscles with precision, suggesting that better-defined injection points may improve results but require further study to validate these recommendations for diverse patient populations (9). The need for this review is underscored by recent research advocating the role of imaging guidance but calling for more focused studies to establish best practices by comparing and summarizing the evidence to enhance outcomes in refractory TOS cases (10). Therefore, this literature review aims to provide an overview of the role of US in managing refractory pain associated with N-TOS and to consolidate current evidence on US-guided injections. This review will evaluate the variability in interventions such as muscle injections, hydrodissection, regenerative therapies, and epidural and ganglion blocks when such techniques are used to target structures involved in TOS-associated refractory pain.

The main purpose of this review is to provide a comprehensive overview of the role of US-guided injections, covering all available techniques while highlighting limitations such as precision, safety, and patient outcomes. This review also emphasizes areas requiring further research. Additionally, it aims to provide clinical guidance for pain management in refractory TOS through current treatment strategies, offering structured guides that physicians can use as practical tools.

Methods

The literature search for this review was conducted across academic databases, including PubMed, Scopus, the Cochrane Library, Google Scholar, and EMBASE, to identify relevant studies on refractory N-TOS and the role of US -guided injections in its management. The search terms included but were not limited to: "thoracic outlet syndrome" or "brachial plexus neuropathy" or "thoracic outlet nerve compression" or "thoracic aperture syndrome" or "scalenus anticus syndrome" or "costoclavicular syndrome" and "refractory" or "chronic" or "persistent" or "treatment-resistant" and "ultrasound" or "imaging-guided" or "sonography" or "ultrasonography" and "injection(s)" or "nerve block" or "botulinum toxin" or "pain management" or "minimally invasive" or "pain relief" or "analgesia" or "anesthesia" or "muscle block" or "procedural" or "non-surgical." The search focused on any studies published until November 2024 that addressed the role of any sort of US-guided injections or other pain management interventions for N-TOS. Studies focused solely on surgical interventions. Papers that had methodological limitations, constituted animal studies, or did not address refractory or chronic N-TOS specifically were

excluded. The authors of the present review screened titles and abstracts individually, and full-text reviews were conducted to extract relevant data, including study design, sample size, interventions, outcomes, and major findings. The data were synthesized and provided narratively, summarizing the effectiveness of US-guided injections, identifying procedural gaps, and highlighting anatomical challenges. Limitations of this review include language restrictions to English and potential publication bias.

Pathophysiology of Refractory Thoracic Outlet Syndrome

Persistent TOS occurs when symptoms continue or worsen after surgery, while recurrent TOS refers to the return of symptoms after an initial period of improvement, often called the "honeymoon" phase. The usual endpoints of surgical success are for patients to be able to return to their baseline level of functioning and their willingness to undergo similar procedures again with similar results. Although uncommonly based on symptoms, the decision for surgical approach is more often influenced by surgeon preference. However, this variation in style does not make a significant difference to success rates based on the compression site. Importantly, early surgery is recommended for true N-TOS, since delaying treatment with conservative management may postpone effective management (11-13). A major factor in refractory TOS, particularly in women, is trauma, which often triggers neurological symptoms. Trauma-related TOS generally shows better surgical outcomes than work-related cases, making trauma history an important predictor of success. However, factors such as repetitive post-surgery arm movements, workers' compensation claims, and pre-existing joint or neurological issues in the upper extremity can affect outcomes negatively. These elements highlight the need for careful patient selection and accurate diagnosis to minimize the risk of recurrence (13-15).

In more advanced cases in which patients present with neurological deficits like weakness and atrophy, electromyography (EMG) studies are frequently abnormal. These cases are particularly challenging, since the surgical recovery might be incomplete, and as such, the time to intervention is internal before irreversible effects take place (12,13). If there are residual muscle spasms or hyperactivity, the occurrences thereof often happen in the scalenes or alongside other musculoskeletal issues like pectoralis minor tightness and trapezius overactivation or tension after TOS surgery, which will also be a contributor to ongoing pain and discomfort. These spasms are typically associated with surgical trauma, altered biomechanics, or neuromuscular compensations and result in poor recovery and referred pain to the arm and/or neck. The control of these spasms is essential in ensuring successful postoperative results (16).

Furthermore, fascial involvement plays a central role in TOS, particularly within the framework of the biotensegrity model, in which muscles do not function in isolation. Fascia, the connective tissue that surrounds muscles, viscera, and joints and coordinates neighboring muscle activity, plays a crucial role in musculoskeletal function and sensation of pain (17). In TOS, the scalene muscles, often implicated in both myofascial trigger points and fascial anomalies, can contribute to localized pain in the cervical region and refer symptoms to distant areas (e.g., migraines). Fascial alterations, such as densification or fibrosis, whether from poor posture or trauma, disrupt the mechanical properties of the deep fascia, leading to muscle dysfunction and chronic pain (15,18,19).

Surgical failure in TOS often results from incomplete or improper techniques, such as scalenectomy and neurolysis without removing the first rib, or incomplete resection of the first rib. Removing a cervical rib while leaving an aberrant first rib, or the inability to treat anatomical defects such as ectopic ribs or residual scalene muscles, might result in recurrent pain. The transaxillary approach has been associated with more frequent reports of persistent pain, while the supraclavicular approach offers advantages in reducing intraoperative damage. An often-overlooked factor in failed surgeries is the presence of congenital fibrous bands, which can cause neurovascular compression that remains undetected by standard preoperative imaging (15,20-22).

Spontaneous recurrence of TOS symptoms is frequently caused by scar tissue formation in the surgical area and exacerbated by the chosen surgical approach. Scar tissue can anchor nerves and vessels to the bone, worsening compression and leading to recurring symptoms. This issue reinforces the need for complete rib removal during the initial surgery. However, redo surgeries are more complex than initial surgeries due to distorted anatomy and extensive scar tissue (21,23-25). Scar tissue can develop adhesions that entrap nerves like the brachial plexus, supraclavicular nerve, or intercostobrachial nerve, resulting in persistent or worsening discomfort, sensory abnormalities, or mo-

tor impairments even after decompressive surgery. The fibrosis may compress or tie nerves, resulting in additional areas of compression. Inflammation at the surgery site can also result in swelling, which can compress surrounding nerves and cause neuropathic pain. Other causes of persistent pain include radial, median, or ulnar neuropathy, intercostal nerve entrapment (slipping rib syndrome), and suprascapular or cervical nerve entrapment (16,26). Understanding specific symptoms based on nerve entrapment in TOS helps clinical diagnosis and management (Table 1).

Joint and ligament instability surrounding the thoracic outlet, particularly the acromioclavicular, sternoclavicular, glenohumeral, and scapulothoracic joints, is a major cause of refractory TOS. Ligament laxity can cause subluxations in the thoracic spine or ribs, which worsens neurovascular compression. Furthermore, enthesopathies in muscles such as the levator scapulae, subscapularis, serratus anterior, and rotator cuff might contribute to chronic discomfort. The involvement of anterior chest joints and ligaments, particularly around the costochondral and costosternal areas, is another frequently neglected factor in refractory TOS (19,27).

Clinical Presentation of Refractory Thoracic Outlet Syndrome

TOS can often be misdiagnosed as other conditions that share similar symptoms, such as cervical disc herniation, shoulder joint problems, fibromyalgia, or Raynaud's disease. This diagnostic confusion can lead to unnecessary or ineffective surgical interventions. Although many patients experience gradual improve-

Table 1. *Symptoms of nerve entrapment in TOS to guide diagnosis and management.*

Entrapped Nerve	Symptom Guide for Diagnosis
Supraclavicular nerve	Pain: dull aching in the supraclavicular region, extending to the chest and shoulder. Paresthesia: tingling above the clavicle and upper chest. Hypersensitivity: increased sensitivity to touch, worsened by arm or shoulder movements. Numbness: around the clavicle, possibly extending to the shoulder. Secondary Effects: chronic irritation may cause shoulder and upper back muscle fatigue, limiting motion.
Suprascapular nerve	Pain: deep, dull ache in the posterior or lateral shoulder, worsened by movement. Weakness: difficulty with shoulder abduction and external rotation, affecting overhead or outward movements. Atrophy: chronic entrapment may lead to muscle wasting in the supraspinatus and infraspinatus, causing shoulder asymmetry. Sensory Symptoms: occasional tingling or numbness, though less common due to the motor function of the nerve
Dorsal scapular nerve	Pain: dull aching along the medial border of the scapula, often described as "burning" or "nagging." Radiation: pain may spread to the shoulder or neck, especially with overhead movements. Weakness: difficulty retracting or elevating the scapula, leading to poor posture or scapular winging. Tension: discomfort in the upper back or between the shoulder blades due to muscle fatigue. Sensory Symptoms: rare numbness or tingling, since the nerve primarily has motor function.
Spinal accessory nerve	Weakness: shoulder drooping and difficulty with shoulder elevation and scapular stability due to trapezius dysfunction. Pain: dull ache in the shoulder and neck, worsened by lifting or carrying weight. Impaired Movement: difficulty with shoulder shrugging and arm elevation above shoulder level. Atrophy: muscle wasting in the trapezius, leading to shoulder asymmetry and scapular winging. Secondary Discomfort: compensatory pain in the neck and shoulder, though sensory symptoms are rare.
Thoracodorsal nerve	Weakness: difficulty with shoulder adduction and internal rotation, affecting activities like rowing or swimming. Pain: dull, aching pain in the lower scapular or upper back during shoulder or arm movements involving the latissimus dorsi. Atrophy: chronic entrapment may lead to muscle wasting in the latissimus dorsi, altering the shoulder and upper back contour. Sensory Symptoms: rare, since the thoracodorsal nerve primarily serves motor function.
Long thoracic nerve	Winging: scapular winging, noticeable when pushing against a wall or lifting the arm. Weakness: difficulty stabilizing the scapula due to weakened serratus anterior, affecting overhead movements. Pain: dull ache or discomfort along the shoulder and upper back, especially after physical activity. Sensory Symptoms: rare, as the long thoracic nerve primarily has motor function.
Intercostobrachial nerve	Pain: numbness, tingling, or burning along the inner upper arm, from the armpit to the elbow. Hypersensitivity: discomfort or increased sensitivity in the upper arm, aggravated by arm movements or pressure in the axillary region. Radiating Pain: sharp or shooting pain may radiate from the chest wall to the upper arm. Sensory Function: the intercostobrachial nerve is sensory, causing discomfort along the upper arm.
Axillary nerve	Weakness: difficulty with shoulder abduction and external rotation due to deltoid and teres minor dysfunction. Atrophy: progressive muscle wasting in the deltoid, leading to visible shoulder asymmetry. Sensory Loss: numbness or tingling over the lateral shoulder, where the axillary nerve provides sensation. Exacerbation: repetitive overhead movements or poor posture may worsen pressure on the axillary nerve, causing impingement.

Table 1 cont. *Symptoms of nerve entrapment in TOS to guide diagnosis and management.*

Entrapped Nerve	Symptom Guide for Diagnosis
Medial pectoral nerve	Weakness: difficulty with shoulder adduction, internal rotation, and movements involving the pectoralis muscles, affecting pushing or lifting. Pain: dull, aching pain or discomfort in the chest or upper shoulder, worsened by arm movements or postural changes. Atrophy: muscle wasting in the pectoralis major or minor, reducing pushing strength in chronic cases. Sensory Symptoms: rare, but mild tingling or numbness may occur in severe cases, since the nerve primarily provides motor innervation.
lateral pectoral nerve	Weakness: difficulty with shoulder adduction, flexion, and internal rotation, affecting pushing and cross-body movements. Pain: dull, aching pain in the chest or anterior shoulder, worsened by overhead movements, pushing, or poor posture. Atrophy: muscle wasting in the pectoralis major, leading to visible muscle loss and reduced shoulder function. Sensory Symptoms: rare tingling or numbness in the chest or shoulder, as the lateral pectoral nerve mainly provides motor function.
Sympathetic plexus	Vascular Symptoms: coldness and pallor in the affected hand or arm due to vasoconstriction and reduced blood flow. Sweating Abnormalities: excessive sweating (hyperhidrosis) or lack of sweating (anhidrosis) in the affected area (hand or forearm). Pain and Discomfort: burning pain or aching in the arm/hand, and tingling or numbness, potentially due to sympathetic plexus involvement. Temperature Regulation Issues: heightened sensitivity to temperature changes (cold or heat) in the affected limb. Superficial vs. Deep Plexus: Superficial Plexus: linked to sweating issues and temperature regulation. Deep Plexus: involvement can cause vascular changes, chronic pain, or muscle weakness.
Medial Cutaneous Nerve	Numbness/Tingling: felt along the medial arm and forearm, especially with certain arm positions or thoracic outlet compression (e.g., overhead movements). Pain: dull, aching or burning pain in the inner arm/forearm, worsened by lifting, reaching, or sustained arm postures. Hypersensitivity: excessive sensitivity to touch or temperature on the medial arm/forearm, causing discomfort even with light pressure or clothing. Skin Discoloration: rare vascular changes, such as paleness or flushing, if other brachial plexus or sympathetic fibers are involved. Muscle Weakness: not typical for the medial cutaneous nerve, but weakness may occur if other brachial plexus branches are affected.
Phrenic nerve	Breathing Difficulties: shortness of breath, especially with deep breaths or physical exertion. Paradoxical Breathing: abnormal abdominal rise during inhalation, indicating diaphragmatic weakness or paralysis. Decreased Diaphragmatic Movement: reduced chest expansion due to limited diaphragm movement, causing breathing difficulty. Cervical Pain: neck, shoulder, or upper chest pain due to phrenic nerve involvement (C3-C5 roots). Neurological Symptoms: tingling, numbness, or pain in the neck or shoulder region from nerve proximity. Autonomic Symptoms: in rare cases, symptoms like increased sweating or temperature regulation issues due to sympathetic dysfunction.

ment in numbness within the first 6 weeks after surgery, these symptoms can persist for months or even years, reflecting the prolonged recovery process associated with N-TOS surgery. This issue highlights the importance of patient education and setting realistic expectations about postoperative recovery and symptom resolution. Persistent pain can be caused by any of several factors, including lingering nerve compression (often affecting the brachial plexus or nearby nerves), myofascial tension or spasms (especially in the scalene and pectoralis minor muscles), scar tissue formation, or inflammation in the surrounding areas. Proper patient education reduces frustration and provides a better understanding of potential recurring symptoms (28) (Tables 2 and 3).

Secondary surgeries, particularly those involving first rib resection, carry a higher risk of complications, including nerve injuries. For instance, palsies of the long thoracic nerve (LTN) have been reported in a small number of patients undergoing secondary rib resection procedures. This increased risk emphasizes that the need for careful planning and execution in secondary interventions is even greater than in primary surgeries (23,29).

TOS manifests in 2 primary forms, organic compression of nerves or blood vessels, and functional compression related to muscle tightness and poor rib mechanics, particularly in the scalene and pectoralis minor muscles. Post-surgical pain often arises from undiagnosed or inadequately treated myofascial pain syndrome (MPS), with scalene muscle dysfunction playing a major role. Myofascial trigger points and fascial anomalies in these muscles can cause localized pain in the cervical region and even radiate to distant areas, like the head, resulting in migraines. Fascial alterations, such as densification or fibrosis from surgery or poor posture, disrupt muscle function and lead to chronic pain (30-33).

Nerve entrapment is a significant concern both as a primary source of TOS pain and as a potential post-surgical complication. During the first stages of recovery, patients typically experience mild to moder-

Table 2. *The factors contributing to refractory pain after TOS surgery and the complexity and interplay of various causes and steps in the treatment process.*

Cause of Refractory Pain Following TOS Surgery	Step	Details
First Step	Improper patient selection	Selection of patients unsuitable for surgery may lead to inadequate outcomes.
	Inconclusive diagnosis	Diagnosis may be unclear or mistaken for other conditions.
		Confused with other conditions that mimic its symptoms.
Second Step	Surgical failure	Issues during surgery may arise due to inadequate technique.
		Incomplete or improper surgical technique leading to unresolved symptoms.
		Failure to identify and address congenital bands during surgery
	Intraoperative nerve injury	Nerve injury occurring during the surgical procedure.
	After surgery	Complications after surgery may include scar tissue formation.
		Scar tissue can cause additional pain and complications.
	Redo surgery	Necessity for additional surgical intervention due to unresolved issues.

Table 3. *The various factors that can lead to pain after surgery and the potential sources of discomfort that patients may experience.*

Post-Surgical Pain Generators	Description
Residual nerve compression	Nerve compression that may occur after surgery, leading to pain and discomfort.
Myofascial tension	Tension in the fascia and muscles that can result from surgical trauma, causing pain.
Persistent muscle spasms	Ongoing muscle spasms that may arise as a response to surgery or nerve irritation.
Scar tissue development	Formation of scar tissue can create adhesions and restrict movement, leading to pain.
Inflammatory responses in surrounding tissues	Inflammation in tissues surrounding the surgical site can contribute to ongoing pain.

ate pain, frequently described as spasms in the neck, back, and shoulder, very close to the site of their N-TOS surgery. In addition, numbness may also occur notably at the incision site but can extend to the neck, chest, arm, or hand. Other postoperative conditions, such as hyperhidrosis, causalgia, and reflex sympathetic dystrophy, may lead to sympathetically maintained pain syndromes (21,34,35).

Scapular winging is a well-described complication that ensues after nerve injuries incurred during TOS surgery. This complication can occur in specific patterns, depending on the nerves that are injured. The most common cause of medial scapular winging is LTN palsy of the serratus anterior muscle, an effect that manifests as the protrusion of the scapula. On the contrary, lateral winging is often due to trapezius dysfunction (most commonly following spinal accessory nerve [SSN] palsy) or rhomboid paralysis associated with dorsal scapular nerve injury. These conditions not only cause pain and functional limitations but also contribute to cosmetic deformities, which can be distressing for most patients. As such, refractory symptoms after TOS surgery, such as scapular winging, often necessitate further therapeutic consideration, emphasizing the need for careful intraoperative nerve preservation and postoperative management (26,36). Conditions like costochondritis or ligament strain in the surrounding areas can mimic or worsen TOS, resulting in persistent chest wall pain that radiates to the shoulder or neck. Inflammation or degeneration at soft-tissue attachment sites can add to localized pain and increase neurovascular compression within the thoracic outlet. Additionally, ligament instability in the joints that connect the ribs to the sternum can cause abnormal movement and biomechanical stress, further intensifying TOS symptoms (33).

Diagnostic US in Refractory Thoracic Outlet Syndrome

Diagnosing N-TOS, particularly in refractory instances, is difficult due to a lack of defined diagnostic criteria, the low sensitivity of imaging modalities, and a broad differential diagnosis. High-frequency ultrasound (HFUS) emerges as a valuable tool in N-TOS diagnosis, allowing for the visualization and location of brachial plexus lesions, as well as the identification of muscle or bony abnormalities that cause nerve compression. HFUS provides precise insights into the perineural environment, nerve fascicular structure, and diameter, assisting with diagnosis and treatment (5).

HFUS is particularly beneficial for making preoper-

ative dynamic assessments, especially in cases of lower trunk compression of the brachial plexus. The tool allows for accurate evaluation of the required surgical approach. The dynamic nature of TOS symptoms can complicate diagnosis; however, when clinical suspicion of N-TOS is high, a dynamic US can capture symptoms that may not be present at rest. For instance, HFUS can reveal focal thinning at the point of lower trunk compression, along with significant thickening of the nerve distal to the compression site. This symptom can be quantified, showing a greater increase in the cross-sectional area of nerve cords on the affected side than on the healthy side, indicative of nerve compression. The US can also visualize hyperechoic fibromuscular structures, known as the "wedge-sickle sign," which may compress the inferior trunk of the brachial plexus. If abnormal bony structures are present along the deep surface of the nerve root, these should also be documented (13,37,38).

Additionally, US is useful for identifying indirect signs of muscle spasms and hypertrophic changes. A US image can indicate muscle spasms by revealing increased stiffness or tension and may show chronic changes like thickened fascia or muscle swelling, with the color Doppler technique demonstrating altered blood flow caused by increased muscle tension. For hypertrophy, US can visualize increased muscle thickness and density, with changes in echotexture indicating chronic hypertrophy (39). Furthermore, muscle spasms are common in TOS, notably in the scalene and pectoralis minor muscles, which cause pain and worsen neurovascular compression. Hypertrophy may also result from persistent stress or compensatory mechanisms affecting these muscles. Hypervascularity is more associated with muscle spasms or inflammation than with hypertrophy itself; in hypertrophic muscles, there are typically no significant changes in blood flow detectable by Doppler US, particularly when the muscles are not inflamed. Whereas hypervascularity indicates inflammation or active processes, muscle hypertrophy alone generally does not lead to increased vascularity unless inflammation is present. Thus, both muscle spasms and hypertrophy can contribute to TOS symptoms, with hypervascularity linked primarily to spasms or inflammation. US is a noninvasive method for monitoring muscle thickness changes and detecting individual muscle patterns and superficial membranous layers during exercise. While muscles seem hypoechoic, the fascia connecting them appears hyperechoic, allowing for exact visualization (40).

Furthermore, US may detect the presence, topography, and thickness of superficial membrane layers, with the arrangement and thickness varied according to body area and individual characteristics. In advanced cases involving neurological impairments, an ultrasonography examination can detect muscular atrophy. However, a healing process for damaged muscle fibers may appear as hyperechoic alterations on ultrasonography, indicating muscle fiber remodeling. This dynamic evaluation helps to diagnose the level of damage and directs therapy interventions to maximize recovery. HFUS is a valuable tool for detecting muscle hypertrophy and fibrosis, particularly in the anterior and middle scalene muscles. In cases of TOS, HFUS can reveal specific manifestations of muscle hypertrophy that may contribute to nerve compression, even in the absence of other pathologies. For instance, brachial plexus echography might show isolated hypertrophy of the subclavius muscle, which can cause compression of the brachial plexus (37,38). This hypertrophy is often fusiform in nature, compressing the brachial plexus inside the interscalene triangle but not involving the pectoralis minor area. A typical subclavius muscle measures approximately 0.51 cm in thickness; however, in hypertrophic cases, the subclavius muscle can rise to 0.72 cm, underlining the usefulness of focused ultrasonography in identifying TOS-related muscular abnormalities (17).

In cases of refractory TOS, diagnostic US is essential for identifying entrapment neuropathy. The cross-sectional area of the brachial plexus is often larger on the affected side, indicating lower trunk compression. The distal part of the nerve may be thickened due to edema, with measurements of approximately 0.49 ± 0.12 cm on the affected side compared to 0.38 ± 0.06 cm on the healthy side. Abnormal US findings associated with pressure neuropathy include nerve enlargement at the site of compression, reduced echogenicity, and increased vascularity. Accurate quantification of nerve size is essential for diagnosing nerve entrapment and related conditions. US provides detailed measurements, such as cross-sectional area and swelling ratio in transverse views and nerve diameter in longitudinal images. Major pathological signs, such as alterations in nerve shape, disruption of the normal fascicular pattern, and increased blood flow, the last of which is identified by color Doppler imaging, indicate the severity and nature of nerve entrapment. Furthermore, post-surgical complications such as scar neuromas might develop at the site of nerve damage. US is useful in diagnosing these neuromas, which appear as discrete

hypoechoic fusiform masses with several longitudinal hypoechoic bands separated by hyperechoic tissue. Recognizing these different ultrasonography findings is crucial for addressing chronic symptoms after nerve injury (16,17,22,37,41).

Muscle wasting, atrophy, and degeneration often result from direct mechanical compression, anatomical variations, space-occupying lesions, or repetitive traction and irritation of nerves at focal entrapment sites. This phenomenon highlights the importance of early diagnosis and intervention to prevent permanent damage. For example, US can detect atrophy and weakness in the supraspinatus and infraspinatus muscles, which are innervated by the suprascapular nerve (SSN), further assisting in evaluating nerve compromise. US is particularly valuable in visualizing and diagnosing pathologies in the SSN, LTN, SAN, and phrenic nerve. In cases of LTN palsy, US imaging may reveal muscle edema, atrophy, or fatty degeneration, with the LTN having a mean diameter of 1.6 mm. Peripheral nerve lesions, such as neuromas, appear as discrete hypoechoic fusiform masses with multiple hypoechoic bands, distinguishing them from healthy nerve structures. Brachial plexus injuries are effectively assessed through sonography, which may reveal scarring, nerve thickening, or abnormal fascicular patterns, helping to identify the extent of nerve damage. High-resolution imaging, like MRI, further contributes to the radiologic diagnosis of peripheral nerve lesions, offering hope for advanced diagnosis and treatment planning in complex cases. Sonographic findings often correlate closely with operative findings, aiding surgeons in identifying nerve injuries or neuromas, such as spindle neuromas resulting from chronic irritation (36,42-45). These advanced imaging techniques are critical for guiding early and accurate intervention, preventing further nerve damage, and optimizing outcomes in refractory TOS and other nerve-related conditions (Fig. 1).

Considering the complexity of cervical anatomy and the role of fascial integrity in maintaining soft tissue balance, a comprehensive diagnostic approach is essential. Sonographic evaluations can assess both superficial and deep muscle layers accurately, facilitating the identification of myofascial trigger points (MTrPs) that may not be detectable through palpation. These MTrPs are characterized by fascial densification and impaired gliding, which illustrate the intricate support system of fascial structures that contribute to cervical myofascial pain and serve as a differential diagnosis. This advanced imaging methodology enhances diagnostic precision and informs targeted therapeutic interventions, ultimately improving patient outcomes and minimizing the risk of chronic post-surgical pain. By integrating US into clinical practice, health care providers gain a deeper understanding of the multifaceted nature of myofascial pain and its implications for surgical management (16,33).

Ultrasonography is instrumental in detecting fascial changes, particularly in the deep fascia of the neck, which are often implicated in TOS. The ability to visualize fascial planes allows for the detailed assessment of thickness, density, and structural integrity. Real-time US capabilities enable dynamic evaluations, providing insights into how fascial tissues respond to movement, tension, and mechanical stress. Clinicians can identify areas of fascial thickening, adhesions, or abnormalities that may contribute to musculoskeletal pain or dysfunction. In healthy individuals, the membranous layer of fascia appears as a sharply defined, continuous hyperechoic line. In contrast, unhealthy patients exhibit discernible adhesions among fascial layers, presenting a more heterogeneous and hypoechoic appearance, which may indicate increased susceptibility to compression and lateral translation (16,18,40).

Although the results of a systematic review highlight the lack of available evidence to support the assertion that US can be used as a diagnostic test for N-TOS, there do exist cues that may assist the physician in conducting a diagnostic workup at the patient's bedside to rule out the causes of refractory TOS (Table 4).

US-Guided Injections for Refractory Thoracic Outlet Syndrome Pain Management

US-guided therapies have proven critical in managing refractory pain after TOS surgery. The use of US guidance improves the precision and safety of treatments by assuring correct needle insertion and reducing the risk of complications. US-guided injection techniques enable precise administration of therapeutic medicines to areas of post-surgical pain, such as the scalene muscles, brachial plexus, or scar tissue regions. US guidance is simply an advanced, patient-centered method of managing refractory pain, reducing the risks and complications associated with blind injections while increasing efficacy (15). Different approaches are discussed in greater detail throughout in this section.

Muscle Injections

US-guided muscle injections involve delivering therapeutic agents directly into targeted muscle tis-

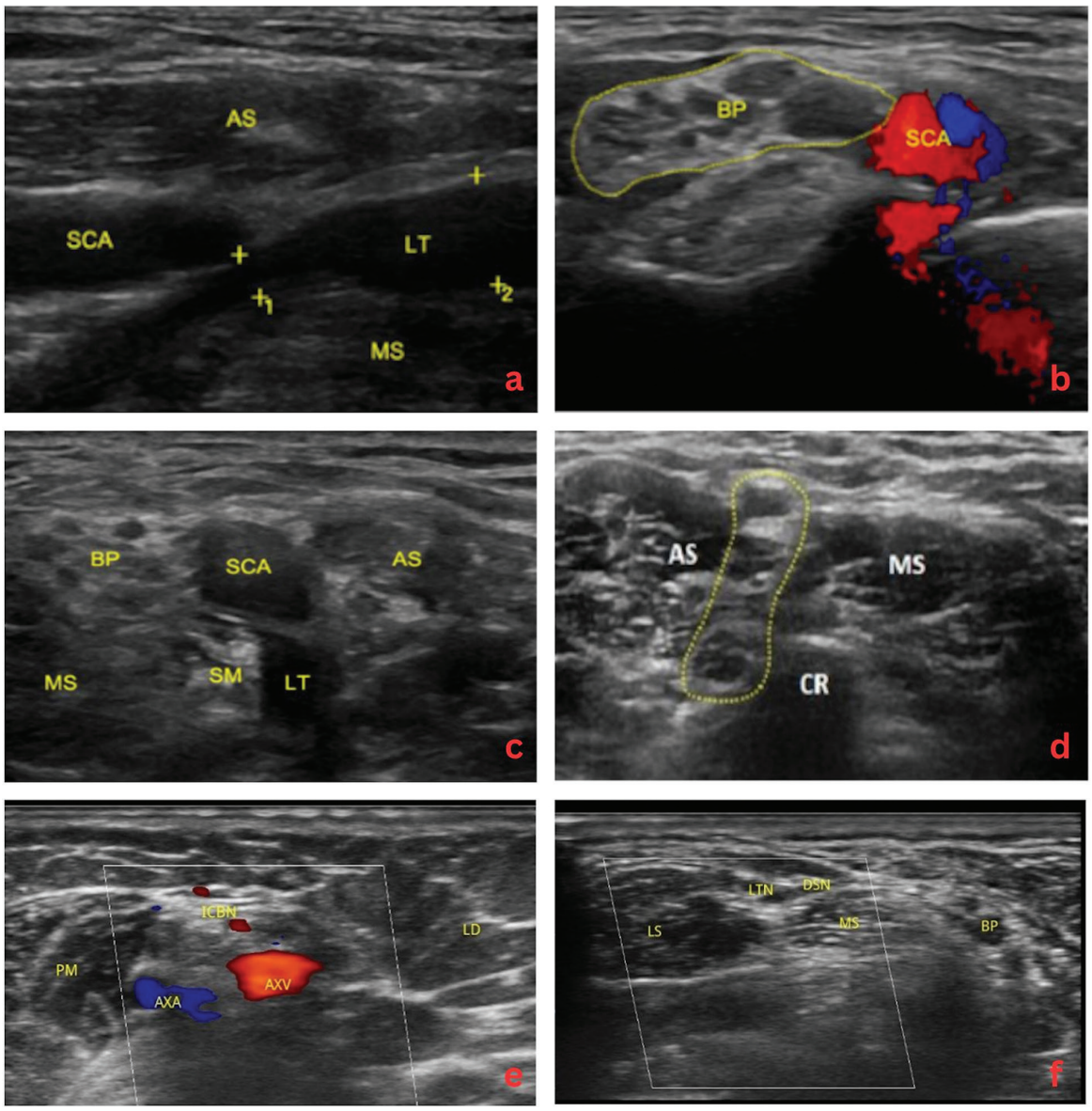

Fig. 1. *Diagnostic ultrasound in refractory TOS. a) Longitudinal view of the compressed lower trunk (LT) and the compression caused by the MS. Lower trunk compression. HFUS showed focal thinning at the level of lower trunk due to compression. BP, brachial plexus; LT, lower trunk, AS, anterior scalene muscle; SM, scalenus minimus muscle; MS, middle scalene muscle; SCA, subclavian artery (37). b) The cross-sectional area of the brachial plexus cords was markedly increased as an entrapment neuropathy (the distal nerve was thickened for edema). BP, brachial plexus; SCA, subclavian artery (37). c) Short-axis view showing LT compression caused by the SM. Hyperechoic fibromuscular bands (mostly in the scalenus minimus muscle) behind the compressed nerve. BP, brachial plexus; LT, lower trunk, AS, anterior scalene muscle; SM, scalenus minimus muscle; MS, middle scalene muscle; SCA, subclavian artery (37). d) Ultrasound images of the cervical rib. AS: anterior scalene muscle; CR: cervical rib; MS: middle scalene muscle. The yellow dashed area encircles the brachial plexus (62). e) The intercostobrachial nerve, often overlooked by surgeons during axillary procedures or during injections, can become a major source of distress for patients if damaged. ICBN, intercostobrachial nerve. AXA, AXV, axillary artery and vein. LD, latissimus dorsi muscle. PM, pectoralis minor muscle. f) The dorsal scapular nerve (DSN) emerges from the C5 anterior ramus, pierces the MS, and courses beneath the levator scapulae muscle (LS). The long thoracic nerve (LTN) also travels within the MS. Multiple tiny hypoechoic fascicles, belonging to the long thoracic and dorsal scapular nerves, pass through a cleavage plane within the MS, which may be injured during muscle injections or compromised during surgical procedures, leading to significant complications*

Table 4. *The ultrasound findings related to structural anomalies, muscle hypertrophy, fibrosis, and nerve compression in refractory neurogenic TOS as well as LTN, SAN, SSN, and DSN.*

Ultrasound Finding	Description	Possibility of Peripheral Nerve Entrapment
Cervical ribs/elongated C7 transverse processes and cervical rib protrusion	Presence of bilateral or unilateral cervical ribs or supernumerary ribs can contribute to compression and impingement of nerve roots. Protrusion of the distal tip of the cervical rib into the interscalene triangle narrows space, possibly impinging nerve roots.	Possible involvement of supraclavicular nerve, SSN, SAN, LTN, and phrenic nerve due to altered anatomy.
Brachial plexus trunk area	Cross-sectional view shows a larger area of the brachial plexus trunk on the injured side than on the normal side.	Inferior trunk (C8-T1), potentially involving supraclavicular nerve, LTN, SSN, and SAN.
Hypertrophy and spasm of anterior scalene muscle	Ultrasound can visualize muscle hypertrophy directly as an increase in muscle thickness. The muscle fibers may appear denser and bulkier, and changes in echotexture may indicate chronic hypertrophy.	Likely compression of supraclavicular nerve, SSN, phrenic nerve, and parts of the brachial plexus and cervical plexus Anterior scalene hypertrophy or spasm can indirectly compress or stretch DSN, SAN, and LTN.
Hypertrophy and spasm of middle scalene muscle	On ultrasound, a spasm of the muscles can sometimes reveal increased muscle stiffness or tension. In cases of chronic spasm, the clinician may see changes such as thickening of the fascia or muscle swelling. Color Doppler imaging may show hypervascularity due to the muscle's increased tension.	Phrenic nerve, SSN, **greater occipital nerve,** supraclavicular nerve, LTN, DSN, and SAN, and cervical plexus nerves.
Pectoralis minor muscle hypertrophy and spasm	Spasm/hypertrophy in the pectoralis minor muscle, contributing to brachial plexus compression.	Likely impact on the medial pectoral nerve, **lateral pectoral nerve,** axillary nerve subscapular nerve, thoracodorsal nerve, DSN, SSN, and LTN, possibly SAN.
Lower trunk compression (longitudinal view)	Compression of the lower trunk of the brachial plexus due to hypertrophy and/or fibrosis of the scalene muscle.	C8-T1 roots affecting the LTN, SSN, SAN, DSN, medial pectoral nerve, **lateral pectoral nerve,** axillary nerve, thoracodorsal nerve, and phrenic nerve.
Lower trunk compression (short-axis view)	Focal thinning and distal nerve thickening reflect compression at the lower trunk level, with nerve edema (affected side: 0.49 ± 0.12 cm vs. healthy side: 0.38 ± 0.06 cm).	
Wedge-sickle sign	Hyperechoic fibromuscular structure at the medial edge of the middle scalene muscle causing compression of the lower trunk, creating a swollen, sickle-shaped hypoechoic area.	Compression of lower plexus nerves, including supraclavicular nerve, DSN, LTN, SSN, medial pectoral nerve, **lateral pectoral nerve,** axillary nerve, thoracodorsal nerve, medial cutaneous nerve of arm and forearm, and possibly phrenic nerve.
Subclavius muscle hypertrophy	Ultrasound shows isolated fusiform hypertrophy of the subclavius muscle, causing lower brachial plexus compression. A normal subclavius muscle measures approximately 0.51 cm in thickness, but in hypertrophic cases, the subclavius muscle can increase to 0.72 cm.	DSN, SSN, SAN, LTN, and other nearby nerves may also be affected by compression.
Hypertrophy/fibrosis of scalenus minimus muscle	Scalenus minimus muscle hypertrophy and/or fibrosis contributing to brachial plexus compression.	Likely involves SSN, DSN, SAN, LTN, and sympathetic plexus.
Abnormal ligaments	Fibrous ligaments extending to the first rib or pleural cupula may displace and constrict the inferior brachial plexus (C8 and T1).	C8-T1 roots affecting LTN, SSN, SAN, and phrenic nerve.
Fibrous bands	Separate fibrous bands from C6 and/or C7 vertebrae to the first rib compress the brachial plexus.	SSN, SAN, LTN, DSN phrenic nerve, and sympathetic plexus potentially entrapped.
Myofascial trigger points (MTrPs)	Well-defined focal hypoechoic nodules appearing as myofascial trigger points in ultrasound imaging.	Possible involvement of SSN, SAN, and superficial sympathetic plexus due to fascial and muscle changes.
Increased fibrous component in neck muscle fascia	Ultrasonography reveals increased fibrous component and total thickness of the deep fascia of the neck muscles.	SSN, SAN, LTN, and superficial sympathetic plexus involvement.
Scar neuroma	Appears as a discrete hypoechoic fusiform mass with multiple longitudinal hypoechoic bands separated by hyperechoic tissue.	Likely affects SSN, SAN, LTN, medial pectoral nerve, **lateral pectoral nerve,** axillary nerve, thoracodorsal nerve, and sympathetic plexus, depending on scar location.

sues. For TOS-related pain, this technique addresses tense or spastic muscles to alleviate associated nerve pressure and reduce pain. When persistent muscle tension or compensatory spasms develop in the scalene or pectoralis minor muscles, US-guided injections offer relief by targeting these areas. Therapeutic agents, including local anesthetics, botulinum toxin (Botox), or dextrose, are administered precisely to reduce muscle tension, alleviate pressure on adjacent nerves, and relieve pain. In cases wherein persistent pain involves the pectoralis minor muscle, targeted injections can also reduce muscle tension and modulate pain transmission. Functional issues related to muscle tightness and poor rib mechanics, particularly in the scalene and pectoralis minor muscles, contribute to pain and dysfunction. The myogenic plexopathy variant, characterized by muscle-related nerve compression, often responds well to US-guided myofascial release and trigger point therapy (33,41,46-48). Dramatic pain relief is often achieved through US-guided dextrose hydrodissection, which releases entrapped nerves, scar tissue, and fibrosis that develop in muscles or soft tissues and can cause mechanical irritation. Sites of muscle remodeling, indicated by hyperechoic changes on US, can be targeted by hydrodissection, proving effective in reducing pain and enhancing function (33,34,49).

Hydrodissection

In simple terms, hydrodissection involves using fluid to separate tissue layers, alleviating nerve entrapment and associated pain. When used for neuropathic pain, US-guided hydrodissection targets specific nerves or nerve groups, such as the brachial plexus, with solutions like dextrose or local anesthetics to reduce irritation and reduce inflammation (17). In cases of persistent neuropathic pain, such as supraclavicular nerve entrapment, supraclavicular nerve hydrodissection may provide relief. Targeting the brachial plexus with dextrose, a local anesthetic, or their combination with corticosteroids can reduce nerve irritation and modulate inflammation. If pain radiates toward the upper arm or chest, especially in patients with referred pain after TOS surgery, intercostobrachial nerve hydrodissection may also be beneficial. Additionally, US-guided hydrodissection of peripheral nerves, such as the brachial plexus and the radial, median, ulnar, intercostal, suprascapular, and superficial cervical plexus, offers another avenue for pain relief injections (50-52). Recent research also supports the use of US-guided perineural injection of botulinum toxin-A (BT-A), a promising and safe treatment for painful peripheral nerve injuries, providing pain relief for several months without causing sensory disturbances. Interestingly, this intervention appears to deliver significant analgesic benefits even for patients who do not exhibit signs of hyperalgesia. US-guided injections, particularly subepineural BT-A injections, represent a novel approach to managing unresponsive neuropathic pain. Although intraneural injections have traditionally been discouraged due to concerns about potential neurological complications, recent evidence suggests that BT-A injections into nerves are safe, with no adverse effects on nerve architecture or myelination. In particular, targeting the pectoralis minor and scalene muscles has demonstrated potential in alleviating the symptoms of chronic neuropathic pain, including entrapment of the supraclavicular nerve. These injections may also aid in predicting how patients might react to more invasive procedures, such as first rib resection, thus providing a novel, elective therapy option for those with chronic neuropathic pain and opens new therapeutic options in cases wherein conventional therapies have failed. This review underscores the importance of conducting further studies with larger sample sizes to strengthen the confidence in these findings (48,53,54).

Regenerative Therapies

Regenerative therapies, such as dextrose prolotherapy, platelet-rich plasma (PRP), and stem cell prolotherapy, stimulate tissue repair and promote long-term pain relief. These treatments target the fascia, muscle-fascia interfaces, and areas of degeneration, helping to strengthen weakened tissues and reduce chronic pain. Regenerative therapy for TOS can treat muscular atrophy and nerve entrapment by stimulating cellular repair. Dextrose injections, particularly, show potential for restoring muscle function and integrity. While intriguing, the use of PRP and stem cells in TOS treatment remains under-researched. Further studies are needed to standardize techniques and establish their efficacy (55,56). Moreover, US-guided interventions, such as perimysium dissection with dextrose, hydrodissection, and myofascial release, can target the muscle-fascia interface, which is particularly beneficial in cases of residual or secondary myofascial pain following surgery or injury. By addressing the fascial layer that surrounds muscles, these treatments promote healing, release tension, and reduce inflammation in the muscle tissues. This targeted approach can lead to pain relief and muscle function improvement (33,57-59). These regenerative treatments

promote the recovery of tissues compromised by chronic pain or nerve compression. They provide significant long-term comfort by strengthening the fascia and stimulating tissue regeneration. However, more research is needed to develop standardized protocols and perform randomised trials to support the wider use of these treatments in clinical practice.

Epidurals and Ganglion Blocks

Nerve and spinal injection techniques are employed to reduce pain and inflammation by targeting specific nerve pathways and spinal structures. Thoracic epidural and peripheral nerve blocks are used in cases wherein conservative treatments have failed or persistent pain remains after TOS surgery. US-guided thoracic epidural steroid injections can be used to address inflammation at the spinal level. However, safer, less invasive alternatives—such as erector spinae plane blocks, paravertebral blocks, and pectoralis plane blocks—are becoming preferred options. These US-guided blocks target the neural pathways and fascial layers involved in the thoracic outlet, providing long-lasting pain relief, reducing the risk of chronic pain, decreasing opioid dependence, and supporting better post-surgical recovery outcomes (17,34,60).

The US-guided stellate ganglion block (SGB) is another effective intervention for cases of recurrent or sympathetic-maintained pain syndromes. By targeting the stellate ganglion, this block reduces sympathetic nervous system outflow, providing pain relief, improved blood flow, and the alleviation of other associated symptoms. Beyond its pain-relieving effects, the SGB also serves as a diagnostic tool to assess the role of the sympathetic nervous system in chronic pain, guiding further treatment strategies (61) (Table 5).

In summary, US-guided injection techniques provide focused, low-risk therapies for managing refractory pain after TOS surgery. These techniques include muscle injections—such as botulinum toxin, local anesthetics, and dextrose—that target muscle spasms and tightness in areas like the scalene and pectoralis minor. Hydrodissection, which uses fluid injection to alleviate nerve entrapment, can relieve pressure on the brachial plexus and supraclavicular nerve. Additionally, regenerative therapies like dextrose prolotherapy and PRP aim to stimulate tissue healing and address nerve or muscle degeneration, while nerve blocks, including epidurals and stellate ganglion blocks, provide targeted pain relief by addressing specific nerve pathways.

Current literature supports the effectiveness of US-guided injection techniques in managing refractory pain associated with N-TOS. Botulinum toxin injections and hydrodissection have shown significant promise in alleviating symptoms related to muscle spasms and nerve entrapments. US-guided epidurals and ganglion blocks further enhance pain management by addressing inflammation and nerve pathways. However, regenerative therapies, while potentially beneficial in aiding long-term relief, remain under-researched, with insufficient literature to fully establish their efficacy. The lack of standardized protocols and robust clinical trials complicates the assessment of these therapies, indicating a need for further investigation into their role in patient management.

Despite their advantages, US-guided injections have limitations. The effectiveness of these interventions can vary based on the complexity of the TOS presentation, individual patient factors, practitioner experience, and patient response. Moreover, US-guided injections may not resolve all underlying structural issues, such as severe anatomical compression, and may need to be integrated with other treatment modalities for comprehensive management.

Conclusion

The effective management of refractory pain following TOS surgery necessitates a comprehensive and multifaceted approach. Accurate diagnosis is essential and can be enhanced greatly through the detailed visualization of relevant anatomical structures, using US and other imaging modalities. Targeted US-guided interventions play an essential role in addressing trigger points, fascial anomalies, nerve entrapments, neuromas, sympathetic trunk involvement, fibrosis, scar tissue, joints, tendons, and muscle dysfunction, allowing for treatments that are precisely tailored to each patient's unique presentation. The incorporation of regenerative therapies may further enhance this approach by promoting healing and alleviating chronic pain; however, additional research is required to establish their long-term outcomes. By embracing these innovative pain management strategies, clinicians can significantly improve patient outcomes, offering relief from persistent post-surgical pain. This integrative approach not only addresses the intricacies of post-surgical pain but also fosters a thorough awareness of the interrelated anatomical components involved in TOS. Finally, a comprehensive and individualized therapy plan is critical for enhancing the quality of life for individuals with refractory pain following TOS surgery.

Table 5. *Overview of various US-guided injection techniques used to address refractory TOS, detailing their specific purposes and benefits in managing pain and facilitating recovery.*

Ultrasound-Guided Injections in Refractory TOS	Description	Indications
Muscle injection for persistent spasm/inflammation	Injection of corticosteroids or anesthetics into specific muscles (e.g., scalene or pectoralis) to alleviate ongoing spasms and inflammation	Persistent muscle spasms or inflammation affecting shoulder and arm function
Myofascial tension release	Techniques involve injecting local anesthetics or corticosteroids into trigger points within myofascial structures to relieve tension in the surrounding nerves.	Myofascial pain contributing to TOS symptoms.
Hydrodisection of entrapment nerves	Involves the injection of saline or local anesthetic around entrapped nerves (e.g., brachial plexus) to separate them from surrounding tissues.	Nerve entrapment symptoms due to compression or fibrosis.
Scar tissue adhesion release	Targeted injection techniques to break down scar tissue around nerves and muscles using saline or steroid injections to reduce adhesions and improve nerve mobility.	Pain and dysfunction associated with previous surgeries or trauma.
Fascia hydrodissection	Injection of fluid into the fascia to create a plane of dissection, which helps to decrease tension and restore movement in affected areas.	Restricted movement and pain due to fascial tension or adhesions.
Regenerative therapies	Includes injecting PRP or stem cells into affected areas to promote healing and tissue regeneration.	Chronic pain and tissue degeneration in joints and ligaments around the thoracic outlet.
Epidural injection	Injection of corticosteroids or anesthetics into the epidural space to provide relief for thoracic or arm pain.	Persistent pain radiating into the upper extremities.
Paravertebral block (PVB)	Injection targeting the paravertebral nerves to provide analgesia for thoracic pain by blocking pain transmission.	Chronic thoracic pain related to TOS.
Erector spinae plane block (ESPB)	A technique that targets the erector spinae muscles and associated nerves to provide analgesia for thoracic pain.	Thoracic pain and muscle spasms.
Pectoral nerve block (PCTB)	Injection that targets the pectoral nerves to alleviate pain in the anterior chest and shoulder region.	Pain localized to the pectoral region and anterior shoulder.
Stellate ganglion block (SGB)	Injection at the stellate ganglion to block sympathetic nerve activity, reducing pain and improving blood flow to the arm.	Complex regional pain syndrome or other sympathetic-mediated pain conditions.

References

1. Capodosal G, Holden D, Maloy W, Schroeder JD. Thoracic outlet syndrome. *Curr Sports Med Rep* 2024; 23:303-309.
2. Tafler L, Borkowski S, Javaid G, Gandolfo D, Kleyn D. Novel diagnosis and treatment for neurogenic thoracic outlet syndrome. *Cureus* 2024; 16:e71434.
3. Li N, Dierks G, Vervaeke HE, et al. Thoracic outlet syndrome: A narrative review. *J Clin Med* 2021; 10:962.
4. Chang MC, Kim DH. Essentials of thoracic outlet syndrome: A narrative review. *World J Clin Cases* 2021; 9:5804-5811.
5. Peek J, Vos CG, Ünlü Ç, Schreve MA, Mortel RHW van de, Vries JPPM de. Long-term functional outcome of surgical treatment for thoracic outlet syndrome. *Diagnostics (Basel)* 2018; 8:7.
6. Larsen K. How to truly identify and treat thoracic outlet syndrome (TOS). MSK Neurology. Published 02/18/2018. Accessed 11/14/2024. www.mskneurology.com/how-truly-treat-thoracic-outlet-syndrome/
7. D'Souza D. Thoracic outlet syndrome. Radiopaedia. Updated 05/09/2024. Accessed 11/06/2024. www.radiopaedia.org/articles/thoracic-outlet-syndrome
8. Farrell ME, Alter KE. Ultrasound-guided botulinum toxin injections for thoracic outlet syndrome. In: Zachary C, Truong D, Dressler D, Hallett M, Pathak M (eds). *Manual of Botulinum Toxin Therapy*. 3rd ed. Cambridge University Press, 2023, pp. 299-310. Accessed 11/06/2024. www.cambridge.org/core/books/manual-of-botulinum-toxin-therapy/ultrasoundguided-botulinum-toxin-injections-for-thoracic-outlet-syndrome/E73FD5B98FF7742EF3E951D50C409AA3
9. Lee KL, Lee JH, Huh HW, Kim HJ. Novel ultrasound-guided injection method for thoracic outlet syndrome based on anatomical features: A cadaveric study. *Pain Physician* 2023; 26:E163-E169.
10. Rizzo S, Talei Franzesi C, Cara A, et al. Diagnostic and therapeutic approach to thoracic outlet syndrome. *Tomography* 2024; 10:1365-1378.
11. Goeteyn J, Pesser N, Houterman S, van Sambeek MRHM, van Nuenen BFL, Teijink JAW. Surgery Versus Continued Conservative Treatment for Neurogenic Thoracic Outlet Syndrome: The first randomised clinical trial (STOPNTOS

Trial). *Eur J Vasc Endovasc Surg* 2022; 64:119-127.

12. Bhattacharya V, Hansrani M, Wyatt MG, Lambert D, Jones NAG. Outcome following surgery for thoracic outlet syndrome. *Eur J Vasc Endovasc Surg* 2003; 26:170-175.
13. Jones MR, Prabhakar A, Viswanath O, et al. Thoracic outlet syndrome: A comprehensive review of pathophysiology, diagnosis, and treatment. *Pain Ther* 2019; 8:5-18.
14. Green RM, McNamara J, Ouriel K. Long-term follow-up after thoracic outlet decompression: An analysis of factors determining outcome. *J Vasc Surg*; 14:739-745.
15. Lim C, Kavousi Y, Lum YW, Christo PJ. Evaluation and management of neurogenic thoracic outlet syndrome with an overview of surgical approaches: A comprehensive review. *J Pain Res* 2021; 14:3085-3095.
16. Bordoni B, Varacallo M. Anatomy, head and neck, scalenus muscle. In: *StatPearls*. StatPearls Publishing, 2024. Accessed 11/06/2024. www.ncbi.nlm.nih.gov/books/NBK519058/
17. Gharaei H. *Clinical Pearls in Interventional Pain Management: Atlas of Ultrasound Guided Interfascial Plane Hydrodissection*. Vol 3. Aryateb Publishing; 2023.
18. Stecco A, Stern R, Fantoni I, De Caro R, Stecco C. Fascial disorders: Implications for treatment. *PMR* 2016; 8:161-168.
19. Levine NA, Rigby BR. Thoracic outlet syndrome: Biomechanical and exercise considerations. *Healthcare (Basel)* 2018; 6:68.
20. Dahlin LB, Backman C, Düppe H, et al. Compression of the lower trunk of the brachial plexus by a cervical rib in two adolescent girls: Case reports and surgical treatment. *J Brachial Plex Peripher Nerve Inj* 2009; 4:14.
21. Suzuki T, Kimura H, Matsumura N, Iwamoto T. Surgical approaches for thoracic outlet syndrome: A review of the literature. *J Hand Surg Glob Online* 2023; 5:577-584.
22. Vuoncino M, Humphries MD. How I do it. Thoracic outlet syndrome and the transaxillary approach. *J Vasc Surg Cases Innov Tech* 2023; 9:101128.
23. Teijink SBJ, Goeteyn J, Pesser N, van Nuenen BFL, Thompson RW, Teijink JAW. Surgical approaches for thoracic outlet decompression in the treatment of thoracic outlet syndrome. *J Thorac Dis* 2023; 15:7088-7099.
24. Likes K, Dapash T, Rochlin DH, Freischlag JA. Remaining or residual first ribs are the cause of recurrent thoracic outlet syndrome. *Ann Vasc Surg* 2014; 28:939-945.
25. Hosseinian MA, Loron AG, Soleimanifard Y. Evaluation of complications after surgical treatment of thoracic outlet syndrome. *Korean J Thorac Cardiovasc Surg* 2017; 50:36-40.
26. Rustagi SM, Sharma M, Singh N, Mehta V, Suri RK, Rath G. Peripheral communications of intercostobrachial nerve Peripheral communications of the intercostobrachial nerve in relation to the alar thoracic artery. *Adv Biomed Res* 2015; 4:51.
27. Streit RS. NTOS symptoms and mobility: A case study on neurogenic thoracic outlet syndrome involving massage therapy. *J Bodyw Mov Ther* 2014; 18:42-48.
28. Pfnür A, Antoniadis G, Wirtz CR, Pedro MT. Langzeitergebnisse nach Operation des neurogenen Thoracic-Outlet-Syndroms bei Jugendlichen. *Handchir Mikrochir Plast Chir* 2024; 56:32-39.
29. Phillips WW, Donahue DM. Reoperation for persistent or recurrent neurogenic thoracic outlet syndrome. *Thorac Surg Clin* 2021; 31:89-96.
30. Sucher BM. Thoracic outlet syndrome--a myofascial variant: Part 2. Treatment. *J Am Osteopath Assoc* 1990; 90:810-812, 817-23.
31. Laulan J. Thoracic outlet syndromes. The so-called "neurogenic types." *Hand Surg Rehabil* 2016; 35:155-164.
32. Bordoni B, Mahabadi N, Varacallo M. Anatomy, fascia. In: *StatPearls*, StatPearls Publishing, 2024. Updated 07/17/2023. Accessed 11/06/2024. www.ncbi.nlm.nih.gov/books/NBK493232
33. Hauser R. Neurogenic and Nonspecific-type thoracic outlet syndrome - Diagnosis and treatment. Caring Medical Florida/Hauser Neck Center. Accessed 11/06/2024. www.caringmedical.com/prolotherapy-news/really-thoracic-outlet-syndrome-treatment/
34. Urschel HC Jr. The transaxillary approach for treatment of thoracic outlet syndromes. *Semin Thorac Cardiovasc Surg* 1996; 8:214-220.
35. Leffert RD. Complications of surgery for thoracic outlet syndrome. *Hand Clin* 2004; 20:91-98.
36. Wu F, Ng CY. Long thoracic nerve palsy: When is decompression indicated. *J Hand Surg Glob Online* 2023; 5:519-524.
37. Chen D, Gong W, Wang J, Hao J, Zhao R, Zheng M. Diagnosis of thoracic outlet syndrome with the lower trunk compression of brachial plexus by high-frequency ultrasonography. *BMC Musculoskelet Disord* 2023; 24:690.
38. Pesser N, Teijink JAW, Vervaart K, et al. Value of ultrasound in the diagnosis of neurogenic thoracic outlet syndrome. *Eur J Vasc Endovasc Surg* 2020; 59:852-855.
39. Dollinger P, Böhm J, Arányi Z. Combined nerve and vascular ultrasound in thoracic outlet syndrome: A sensitive method in identifying the site of neurovascular compression. *PloS One* 2022; 17:e0268842.
40. Wilson MP, Low G, Katlariwala P, Jacques L, Jack AS. Ultrasound for neurogenic thoracic outlet obstruction remains theoretical. *Diagnostics (Basel)*; 10:205.
41. Cavallieri F, Galletti S, Fioravanti V, Menozzi E, Contardi S, Valzania F. Botulinum toxin treatment for thoracic outlet syndrome induced by subclavius muscle hypertrophy. *NeuroSci* 2021; 2:135-140.
42. Reece CL, Varacallo M, Dulebohn SC, Susmarski AJ. Suprascapular nerve injury. In: *StatPearls*, StatPearls Publishing, 2024. Updated 01/26/2024. Accessed 11/7/2024. www.ncbi.nlm.nih.gov/books/NBK559151
43. Patel Z, Franz CK, Bharat A, et al. Diaphragm and phrenic nerve ultrasound in COVID-19 patients and beyond: Imaging technique, findings, and clinical applications. *J Ultrasound Med* 2022; 41:285-299.
44. Casaletto E, Lin B, Wolfe SW, et al. Ultrasound imaging of nerves in the neck: Correlation with MRI, EMG, and clinical findings. *Neurol Clin Pract* 2020; 10:415-421.
45. Haber HP, Sinis N, Haerle M, Schaller HE. Sonography of brachial plexus traction injuries. *AJR Am J Roentgenol* 2006; 186:1787-1791.
46. Beason AM, Thayer JA, Arras N, Franke JD, Mailey BA. Anterior scalene muscle block response predicts outcomes following thoracic outlet decompression. *Hand (N Y)* 2024; 19:361-366.
47. Lum YW, Brooke BS, Likes K, et al. Impact of anterior scalene lidocaine blocks on predicting surgical success in older patients with neurogenic thoracic outlet syndrome. *J Vasc Surg* 2012; 55:1370-1375.
48. Woodworth TT, Le A, Miller C, Conger A,

Mahan MA, Cushman DM. Botulinum toxin injections for the treatment of neurogenic thoracic outlet syndrome: A systematic review. *Muscle Nerve* 2024; 70:28-35.

49. Lee JH, Lee HJ, Yi KH, Lee KW, Gil YC, Kim HJ. Ideal injection points for botulinum neurotoxin for pectoralis minor syndrome: A cadaveric study. *Toxins (Basel)* 2023; 15:603.
50. Kemper KM, Mailey BA. Thoracic outlet decompression unmasks symptoms of suprascapular nerve compression. *Plast Reconstr Surg Glob Open* 2022; 10:e4429.
51. Wu YT, Wu CH, Lin JA, Su DC, Hung CY, Lam SKH. Efficacy of 5% dextrose water injection for peripheral entrapment neuropathy: A narrative review. *Int J Mol Sci* 2021; 22:12358.
52. Siahaan YMT, Herlambang J, Putri C, Tiffani P. Ultrasound-guided perineural dextrose injection for treatment of superior cluneal nerve entrapment: Serial case report. *Open Pain J* 2021; 14:32-37.
53. Ascensio Mercado MP, Olea MS, Teles Rodrigues A, et al. Intraneural injection of botulinum toxin-A in palliative care and unresponsive neuropathic pain. *APJP* 2023; 33:25-30.
54. Meyer-Frießem CH, Eitner LB, Kaisler M, et al. Perineural injection of botulinum toxin-A in painful peripheral nerve injury - a case series: Pain relief, safety, sensory profile and sample size recommendation. *Curr Med Res Opin* 2019; 35:1793-1803.
55. Navani A, Li G, Chrystal J. Platelet rich plasma in musculoskeletal pathology: A necessary rescue or a lost cause? *Pain Physician* 2017; 20:E345-E356.
56. Alderman DD. Regenerative Injection therapies for pain: Traditional, platelet rich plasma and biocellular prolotherapy. In: Bonakdar RA, Sukiennik AW (eds). *Integrative Pain Management*, Oxford University Press, 2016, pp 345-360.
57. Lai YC, Tsai SH, Chiou HJ. Ultrasound-guided dextrose solution perimysium dissection for posterior shoulder myofascial pain. *J Chin Med Assoc* 2021; 84:650-654.
58. Everts P, Onishi K, Jayaram P, Lana JF, Mautner K. Platelet-rich plasma: New performance understandings and therapeutic considerations in 2020. *Int J Mol Sci* 2020; 21:7794.
59. Kuhns L. ASPN issues guidelines on use of regenerative medicine therapy for chronic pain. Clinical Pain Advisor. 2024. Published 10/22/2024. Accessed 11/08/2024. www.clinicalpainadvisor.com/features/aspn-issues-guidelines-on-use-of-regenerative-medicine-therapy-for-chronic-pain/
60. Motyl CM, Dohring C, Wang ML, et al. Opioid-sparing effects of the bupivacaine pleural catheter in surgical decompression of the thoracic outlet. *Ann Vasc Surg* 2023; 88:283-290.
61. Urschel HC, Kourlis H. Thoracic outlet syndrome: A 50-year experience at Baylor University Medical Center. *Proc (Bayl Univ Med Cent)* 2007; 20:125-135.
62. Hsu PC, Chang KV, Mezian K, et al. Sonographic pearls for imaging the brachial plexus and its pathologies. *Diagn Basel Switz* 2020; 10:324.