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Dorsal scapular nerve entrapment: a systematic review

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ABSTRACT

Background: Periscapular and interscapular pain are common musculoskeletal complaints; however, dorsal scapular nerve (DSN) entrapment neuropathy remains an under-recognized and frequently overlooked etiology. DSN pathology may mimic multiple cervical, shoulder, and upper-extremity disorders, resulting in delayed diagnosis and treatment. To systematically review the current literature regarding the anatomy, etiology, clinical manifestations, diagnostic evaluation, and treatment of DSN entrapment neuropathy.

Methods: A systematic review was performed using PubMed/MEDLINE, Scopus, and Google Scholar databases through January 1, 2026. Search terms included combinations of “dorsal scapular nerve,” “dorsal scapular neuropathy,” “dorsal scapular nerve entrapment,” “scapular winging,” “scapulothoracic pain,” and “nerve decompression.” English-language clinical studies, case reports, case series, anatomical investigations, and diagnostic studies relevant to DSN pathology were included. Non-English publications, duplicate reports, unrelated shoulder conditions, and studies lacking clinical relevance were excluded.

Results: The literature primarily consists of case reports, small case series, anatomical studies, and technical reports. The DSN most commonly originates from the C5 nerve root and frequently traverses the middle scalene muscle, which represents the most common site of entrapment. Patients typically present with medial scapular border pain, scapular dyskinesis, rhomboid weakness, and subtle scapular winging. DSN neuropathy may clinically mimic cervical radiculopathy, thoracic outlet syndrome, myofascial pain syndrome, and rotator cuff pathology. Electromyography and nerve conduction studies may support diagnosis, although findings are operator dependent and technically challenging. High-resolution ultrasound and magnetic resonance imaging may demonstrate nerve edema, muscle denervation, or scapular asymmetry. Conservative management including activity modification, rehabilitation, physical therapy, and image-guided injections remains first-line treatment. Surgical decompression has been described in refractory cases, although evidence remains limited and heterogeneous.

Conclusion: DSN entrapment neuropathy is an uncommon but clinically significant cause of periscapular pain. Recognition of characteristic clinical features and understanding of the anatomical course of the nerve are essential for accurate diagnosis. Current evidence regarding management remains limited to low-level studies, highlighting the need for prospective investigations with standardized diagnostic criteria and outcome measures.

Level of evidence: Level IV; Systematic Review

The study was approved by the medical research and ethics committee of the Urmia University of Medical Sciences with registered Numbers: IR.UMSU.REC.1404.113. Available at: <https://ethics.research.ac.ir/form/ityu5dsz4jvpx857.pdf>.

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Keywords: Dorsal scapular nerve; Dorsal scapular nerve neuropathy; Dorsal scapular nerve entrapment; Levator scapula syndrome; Rhomboid pain syndrome; Periscapular pain; Midscapular pain; Interscapular pain

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Periscapular, interscapular, and midscapular pain are common patient complaints; however, dorsal scapular nerve (DSN) neuropathy remains an underdiagnosed and underestimated condition.^{2,5,8,9,16,17,23,26} In a study of 55 consecutive patients with interscapular pain, Sultan et al²⁶ reported neurophysiological abnormalities consistent with DSN lesion in 29 patients, with rhomboid muscles weakness and scapular winging in 9 patients.

Compared with other peripheral neuropathies, DSN neuropathy is infrequently reported.²¹ The purpose of this systematic review was to increase awareness of DSN neuropathy as a potential contributor to periscapular pain syndromes.

Materials and methods

The study was approved by the medical research and ethics committee of the University.

Study design

A systematic review of the literature was performed in accordance with Preferred Reporting Items for Systematic Reviews and Meta-Analyses principles.

Literature search strategy

A comprehensive search of PubMed/MEDLINE, Scopus, and Google Scholar databases was conducted through January 1, 2026. Search terms included combinations of the following keywords: “dorsal scapular nerve,” “dorsal scapular neuropathy,” “dorsal scapular nerve entrapment,” “scapular winging,” “periscapular pain,” “interscapular pain,” “scapulothoracic pain,” “nerve decompression,” and “rhomboid paralysis.” Reference lists of included studies were also manually screened to identify additional relevant articles.

Eligibility criteria

Inclusion criteria were as follows: English-language publications, clinical studies evaluating DSN neuropathy or entrapment, case reports and case series, anatomical investigations relevant to DSN entrapment, diagnostic imaging or neurophysiological studies involving the DSN, and surgical or nonsurgical treatment reports.

Exclusion criteria were as follows: non-English articles, duplicate publications, studies unrelated to DSN pathology, reports lacking clinical relevance, and studies focused exclusively on unrelated shoulder disorders.

Study selection

Titles and abstracts were screened for relevance. Full-text articles meeting inclusion criteria were reviewed in detail.

Data extraction

Data extracted included the following: study design, patient demographics, etiology and mechanisms of entrapment, clinical presentation, imaging and electrophysiological findings, treatment modalities, and clinical outcomes.

Quality assessment

Given the predominance of case reports, technical notes, and small observational studies, formal meta-analysis was not feasible. The overall level of evidence was low and heterogeneous.

Results

Study characteristics

The available literature consisted primarily of case reports, retrospective case series, anatomical cadaveric studies, technical notes, and narrative reviews. No randomized controlled trials or high-level comparative studies were identified.

Anatomy of dorsal scapular nerve

The DSN typically originates from the ventral ramus of the C5 root within the posterior cervical triangle, deep to the pre-vertebral fascia. Contributions from the C4 and/or C6 roots may occur and the DSN may share a common trunk with the long thoracic nerve. The DSN usually penetrates the middle scalene muscle but may pass anterior or posterior to the muscle. The DSN then courses posteriorly between the posterior scalene muscle and the serratus posterior superior and levator scapulae muscles, traveling near the medial scapula border. The DSN reliably innervates the rhomboid minor and major muscles and may variably supply the levator scapulae. These muscles elevate and retract the scapula and contribute to scapular stability.^{4,5,16,19,22,27} Although traditionally considered a purely motor nerve, the DSN contains nervi nervorum capable of generating nociceptive pain.^{17,20,26}

The most common site of DSN entrapment is within the middle scalene muscle.^{5,7,27} Dynamic compression may also occur at the proximal medial border of the scapula, during overhead elevation above 90°.¹⁶

Etiology

The DSN may be stretched or compressed along its anatomical course. Dynamic compression along the medial proximal border of the scapula can occur in individuals performing repetitive or strenuous overhead activities including painters,



Figure 1 – This body builder athlete had bilateral medial scapular pain because of DSN entrapment. He had cupping on the superior medial angle and medial border of the left scapula to reduce pain. DSN, dorsal scapular nerve.

electricians,^{1,16,23} and overhead athletes such as volleyball or basketball players by the proximal medial border of the scapula.^{3,9} Traumatic stretching has been reported, including after shoulder dislocation.^{3,10}

Hypertrophy of the middle scalene muscle, particularly in bodybuilders, may lead to DSN compression. DSN compression related to an elongated C7 transverse process has also been described.^{5,26,28}

Iatrogenic DSN injury has been reported following interscalene brachial plexus block,^{11,24} painful trigger point injections into the rhomboid major muscle,³ and surgery or radiotherapy for neck malignancies.¹⁸ Debeer et al⁶ reported DSN neuropathy due to prolonged use of an improperly fitted thoracolumbar orthosis in a patient with idiopathic scoliosis.

Clinical manifestations

The primary complaint is medial border periscapular pain (Fig. 1). Although DSN is predominantly a motor nerve, injury or compression may still generate neuropathic pain.^{17,20,26} The following 3 mechanisms have been proposed:

1. Neuropathic trunk pain pattern, occurring because of stimulation of DSN *nervi nervorum*, that provide the intrinsic innervation of the nerve sheath. The *nervi nervorum* are sensitive to stretch, and nociceptive signals may initiate inflammatory reactions that can spread to the adjacent nerves via the common trunk of the C5 root. Since DSN primarily originates from the C5 root, pain may



Figure 2 – Oblique view better demonstrates a subtle asymmetry in scapular position and a subtle right scapular winging.

radiate to the lower neck, shoulder region, and upper arm through other nerves arising from C5.

2. Myofascial pain secondary to nerve entrapment by multiple taut bands that may develop within the rhomboids.
3. Scapular winging may stretch the cutaneous medial branches of the thoracic dorsal rami of the spinal nerves. Some patients may report dysesthesia, pruritus, and loss of pinprick sensation in the midscapular region, a condition known as *notalgia paresthetica*.^{17,26}

Patients may experience limited active shoulder range of motion, particularly during forward flexion and abduction. The scapular assistance test, in which the examiner stabilizes the scapula against the chest wall, may improve motion and decrease symptoms.⁷

Rhomboid weakness can produce medial scapular winging; however, it is more subtle than the winging caused by serratus anterior or trapezius weakness (Fig. 2). Rhomboid weakness produces winging of the medial border of the scapula, particularly its distal portion, accompanied by lateral rotation of the inferior angle. Unlike trapezius palsy, the scapula is not depressed but may assume a higher position due to preservation of the levator scapulae and compensation from the proximal fibers of the trapezius. Scapular winging due to DSN palsy may become more apparent when the patient's arm is placed in full forward

elevation and then progressively lowered.^{7,16} Scapular malpositioning may contribute to SICK scapula syndrome (Scapular malposition, Inferior medial border prominence, Coracoid pain, and Kinesis (dyskinesis) of scapular movement).^{8,17}

DSN entrapment may lead to muscle spasm and the development of painful trigger points in the rhomboid and levator scapulae muscles. A trigger point is an area of metabolic and anatomic dysfunction that serves as a continuous source of neurologic irritability.^{3,8}

Given its varied presentations, DSN neuropathy may be clinically mistaken for various disorders involving the neck, shoulder, and upper arm. The pain may mimic cervical radiculopathy,⁸ fibromyalgia, myofascial pain syndrome, levator scapulae syndrome, rhomboid pain syndrome, thoracic outlet syndrome (TOS)⁵ and other neuropathies around the shoulder,²¹ complex regional pain syndrome,²³ neuralgic amyotrophy (Parsonage–Turner syndrome),³ impingement syndrome, rotator cuff injury,¹⁴ scapulothoracic bursitis or snapping scapula syndrome,¹⁵ and muscle detachment from the scapula.⁷

Diagnostic evaluation

The rhomboid muscles are located deep to the trapezius, making their evaluation challenging. Rhomboid strength can be assessed by asking patients to push their elbows backward while keeping their hands on their hips, or by instructing them to retract the scapulae medially. Weakness in these maneuvers suggests rhomboid dysfunction.^{7,16}

Chest radiographs may reveal scapular asymmetry, accentuated during forward flexion and abduction of the shoulder. Axial magnetic resonance imaging (MRI) may demonstrate abnormal scapular positioning, rhomboid muscles atrophy, or pathologic signal changes. Akgun et al¹ have indicated that MRI may detect rhomboid muscles denervation with reported sensitivity of 84% and specificity of 100%. However, fatty atrophy represents a relatively late manifestation of chronic neuropathy and that signal abnormalities may be subtle on standard shoulder MRI protocols. MRI findings of rhomboid denervation are nonspecific and the frequently cited sensitivity and specificity values derive from a single case report should not be interpreted as broadly validated diagnostic performance characteristics.

Nerve conduction studies (NCSs) and electromyography (EMG), are essential for confirming neuropathy, localizing lesion, assessing the degree of muscle denervation, and excluding alternative diagnoses. NCS and EMG have been used to identify DSN injury.^{1-3,7,9,16,26}

Side-to-side comparison is crucial. Even when NCS latency or amplitude falls within the normal limits, asymmetry may indicate pathology. Ultrasound-guided techniques enhance accuracy.

Lee and Chang¹³ reported ultrasound-guided NCS and EMG findings demonstrating amplitude reduction (5.2 vs. 1.6 mV), delayed latency (4.9 vs. 6.8 ms), and positive sharp waves (1+) with mildly reduced recruitment in the rhomboid major muscle on EMG.

Normal NCS with abnormal EMG changes in the rhomboid muscles suggest selective DSN involvement. The rhomboid major is the most important muscle for EMG evaluation.^{1,3,6,9}

However, electrophysiology studies have limitations. NCS is highly operator dependent. EMG of the rhomboid muscles is

technically demanding, and normative reference values vary between institutions. We therefore emphasize that DSN neuropathy remains primarily a clinical diagnosis supported, rather than definitively confirmed, by imaging and electrophysiological investigations.

High-resolution ultrasound may reveal fibrotic infiltration or an ill-defined, edematous DSN. Side-to-side comparison can help to confirm diagnosis.^{18,23,25}

Treatment

Conservative measures include pain control with anti-inflammatory medications, avoidance of aggravating activities, and trigger point injections. Injection risks include direct nerve injury¹⁷ and pneumothorax.⁸ Rehabilitation focuses on compensating for rhomboid weakness, whereas physical therapy emphasizes on trapezius strengthening.^{1,16}

Ultrasound-guided nerve block^{20,23} and hydrodissection (injecting local anesthetic, saline, or 5% dextrose in water) with or without steroids to separate DSN from the adjacent structures may provide symptom relief.^{12,18,25}

Surgery may be considered in carefully selected refractory cases. Published evidence regarding DSN surgical decompression is limited and consists mainly of small case series, interventional reports, and technical notes (Table I).

Ottstad and Wilson described decompression of fibrous arches, scar tissue, and taut bands, with significant improvements in pain and disability. The authors described ultrasound-guided interventional management, which appeared useful diagnostically and therapeutically in the short term, but long-term efficacy remains uncertain.²⁰

Lohre et al detailed an arthroscopic decompression technique including releasing fascia overlying the rhomboid major, allowing direct visualization of the DSN and scapulothoracic space. The authors specifically emphasize that the publication is a “Technical Note” describing indications and operative technique rather than an outcomes study. It also acknowledges that arthroscopic DSN decompression is technically demanding and lacks evidence addressing proximal compression sites such as the scalenus medius. The paper lacks a clinical outcomes cohort that limits conclusions regarding effectiveness.¹⁵

Chen et al treated DSN compression in 36 patients via release of the middle scalene insertion and removal of tendinous structures. Improvement occurred in 19 patients (20 sides). Many cases were classified as forms of TOS, with some isolated DSN lesions termed atypical TOS. The authors reported symptomatic improvement following surgical decompression; however, the study was limited by small patient numbers, subjective outcomes, and lack of controls.⁵ Overall, the literature is heterogeneous, low-level in quality, and lacks standardized outcome measures and long-term follow-up. Further prospective studies with standardized patient-reported outcomes and longer-term follow-up are required.

Discussion

This systematic review demonstrates that DSN entrapment neuropathy remains an uncommon yet clinically important cause of periscapular pain. Despite increasing recognition,

Table I – Summary and comparison of published surgical and interventional series for dorsal scapular nerve compression: techniques, follow-up, and clinical outcomes.

Authors	Design/level of evidence	Patient numbers	Technique	Follow-up	Outcomes	Limitations/critical appraisal
Chen et al ⁵	Case series	Small series	Open decompression/ neurolysis of the dorsal scapular nerve	Short- to mid- term	Improvement in medial scapular border pain and scapular function reported	Limited sample size, no control group, heterogeneous diagnostic criteria, and outcomes largely subjective. Lack of standardized Patient-Reported Outcome Measures and objective postoperative neurophysiology limits interpretation.
Ottestad and Wilson ²⁰	Interventional case report/series	Very small series	Ultrasound-guided hydrodissection/perineural injection	Short-term	Temporary or partial symptom relief following image-guided intervention	Primarily diagnostic and therapeutic adjunct rather than definitive treatment. Follow-up limited, reproducibility uncertain, and durability of symptom relief unclear. Potential placebo and selection bias.
Lohre et al ¹⁵	Technical note/ surgical technique paper	No clinical outcome cohort presented	Arthroscopic dorsal scapular nerve decompression with scapulothoracic neurolysis ± superomedial scapular border resection	Not applicable for outcomes	Described minimally invasive arthroscopic decompression technique and indications	Primarily a technical description without prospective clinical outcome data. No validated patient-reported outcomes, comparative arm, or complication analysis. Generalizability may be limited by technical complexity and specialist expertise required.

diagnosis remains challenging because symptoms overlap substantially with more common cervical and shoulder disorders.

The current literature highlights several important themes. First, entrapment most commonly occurs at the middle scalene muscle, emphasizing the importance of cervical anatomy in symptom generation.⁵ Second, scapular winging associated with DSN pathology is often subtle and may therefore be overlooked during routine examination.^{7,16} Third, available diagnostic modalities including MRI, ultrasound, EMG, and NCS may support diagnosis but are limited by technical variability and lack of standardized criteria.^{1-3,7,9,16,25,26}

The evidence base remains low quality overall. Most available studies consist of isolated case reports and small retrospective series. Surgical literature is particularly limited, with most publications focusing on technical descriptions rather than validated clinical outcomes.

Current evidence supports conservative treatment as first-line management.^{1,16,17} Image-guided procedures may provide temporary symptom relief and diagnostic confirmation in selected cases.^{12,18,20,23,25} Surgical decompression may be considered for refractory symptoms, although high-quality comparative studies are lacking.^{5,15}

Future research should focus on the following: standardized diagnostic criteria, prospective cohort studies, validation of imaging and electrophysiological findings, comparative treatment studies, long-term patient-reported outcomes, and development of evidence-based treatment algorithms.

Limitations

This review has several limitations. The available literature consisted predominantly of low-level evidence, limiting the strength of conclusions. Significant heterogeneity existed regarding patient selection, diagnostic criteria, treatment methods, and outcome reporting. Additionally, publication bias likely favored positive outcomes.

Conclusion

DSN entrapment neuropathy is an under-recognized cause of periscapular pain that may mimic numerous cervical and shoulder disorders. Careful clinical evaluation, combined with selective use of imaging and electrophysiological studies, is essential for diagnosis. Conservative management remains the primary treatment strategy, while surgical decompression may be beneficial in carefully selected refractory cases. Current evidence remains limited and heterogeneous, underscoring the need for higher-quality prospective investigations.

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