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Trigger Point Injections for Symptomatic Treatment of Median and Ulnar Neuropathy: A Case Report

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Background: Myofascial pain syndrome may coexist with other musculoskeletal conditions, including entrapment neuropathies. The relationship between myofascial dysfunction and entrapment neuropathy remains poorly understood, and the evaluation and management of patients presenting with overlapping symptomatology can be challenging.

Case Report: We report on the use of trigger point injections (TPIs) in a 49-year-old woman with median and ulnar entrapment neuropathies and concurrent myofascial pain. The patient underwent a series of 8 TPI sessions targeting muscles of the shoulder, arm, and hand, resulting in sustained improvement of both myofascial and neuropathic symptoms for up to 2 years. No complications were observed during the treatment course.

Conclusions: This case highlights the potential role of TPIs as a therapeutic option for patients with entrapment neuropathies and concomitant myofascial pain. Further investigation is warranted to better understand the relationship between the 2 conditions and to evaluate the efficacy and durability of this treatment approach.

Key words: Trigger point injections; myofascial pain; trigger points; neuropathy; carpal tunnel syndrome; cubital tunnel syndrome; case report

BACKGROUND

Mononeuropathies typically present as numbness, tingling, and neuropathic pain in the distribution of the affected nerve. They may occur from various etiologies, including chronic compression, which is a common cause in the absence of acute trauma or injury. Median neuropathy at the wrist and ulnar neuropathy at the elbow are the 2 most common entrapment neuropathies, with prevalence estimates ranging from 1%-8% (1,2) and 0.6%-5.9% (3,4), respectively. Their clinical presentations may have overlapping features with radiculopathy or plexopathy; thus, diagnosis requires a detailed history and physical examination that is often supplemented by electrodiagnostic testing and appropriate imaging studies (5).

Conservative management of mild entrapment neuropathies includes physical therapy and orthoses to reduce external compression on the nerve (6,7). Individuals with refractory pain and progression of disease can experience chronic paresthesia and atrophy of the affected muscles, resulting in functional impairment of the affected extremity (8). Various injection therapies consisting of corticosteroid, platelet-rich plasma, hyaluronic acid, and dextrose solution have been described in the literature, with mixed results (9-11). Surgical intervention is an option for persistent or severe cases; however, some patients may prefer to avoid surgery, while select individuals may experience symptoms despite undergoing surgery.

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Myofascial pain syndrome is a common condition that can exist independently or concurrently with various musculoskeletal and neuropathic pain disorders. Myofascial trigger points are hyperirritable spots within skeletal muscle or fascia that are painful to palpation and produce referred pain in a consistent pattern when compressed (12). Trigger point injections (TPIs) are a minimally invasive treatment option for myofascial pain and trigger points that are not responsive to conservative treatment (13). There is a paucity of literature exploring the use of TPIs for entrapment neuropathies that coexist with, or contribute to, myofascial pain. We describe the novel use of TPIs to treat symptoms of median and ulnar mononeuropathies.

CASE PRESENTATION

A 49-year-old woman presented with a 6-month history of right-sided neck and upper extremity pain. She reported a sedentary occupation and worked from home in a workspace with poor ergonomic design but otherwise denied any precipitating event. Her symptoms were described as neck pain with radiation into the right upper arm, elbow, forearm, and hand. Her pain was worse at night, and she experienced associated numbness in the elbow, forearm, and all digits of the hand. Pain was rated as 8/10 on the Numeric Rating Scale. Physical examination revealed multiple myofascial trigger points in the right levator scapulae, splenius capitis, upper trapezius, rhomboids, teres minor, infraspinatus, triceps, flexor carpi radialis, and pronator teres. Tinel's sign was positive at the right wrist and elbow. Cervical spine range of motion was preserved, and Spurling's test was negative. No focal weakness or sensory deficits were noted.

She had tried physical therapy, nonsteroidal anti-inflammatory drugs, acetaminophen, gabapentin, a course of prednisone, heat, and 2 rounds of cervical epidural steroid injections with only transient relief. Magnetic resonance imaging of the cervical spine obtained 3 months before presentation revealed multilevel mild-to-moderate foraminal stenosis, most notably at the C5-6 and C6-7 levels. Electromyography (EMG) and nerve conduction studies (NCS) performed shortly before presentation demonstrated mild bilateral median mononeuropathies at the wrist and mild right ulnar neuropathy at the elbow with no evidence of acute cervical radiculopathy. She was evaluated by a neurosurgeon, and nonoperative management was recommended.

The patient continued gabapentin 300 mg 3 times daily and initiated tizanidine 4 mg nightly and magnesium supplementation. Three weeks after the initial visit, trigger point injections (TPIs) were performed on the bilateral upper trapezius, right rhomboid, levator scapulae, infraspinatus, teres minor, flexor carpi radialis, and pronator teres. A 27-gauge, 1.5-inch needle was advanced into each muscle belly using a standard approach, and 0.5-1.0 mL of 1% lidocaine was injected after achieving appropriate local twitch responses. This resulted in complete resolution of the patient's neck, arm, and hand symptoms for 2 weeks. Although neck and periscapular pain eventually recurred, distal upper extremity symptoms remained minimal.

The patient ultimately underwent a total of 8 TPI sessions, as outlined in Fig. 1. The interval between sessions ranged from 6 weeks to 8 months. Target muscles were identified based on symptom localization and included cervical, periscapular, upper arm, forearm, and intrinsic hand musculature. During this period, the patient experienced progressively improving pain relief, ultimately achieving sustained pain improvement of 80% for 2 years after her final TPI session. During this period, the patient experienced progressively improving pain relief ultimately reporting sustained pain improvement of approximately 80%, which persisted for 2 years following her final TPI session. Although the patient reported more pronounced improvement in her neck and periscapular pain following the cervical epidural steroid injection, she had reported approximately 80% improvement in her distal upper extremity symptoms for several weeks following her fifth TPI session and prior to the epidural injection. The remainder of her symptoms following these injections were localized to the distal upper extremity. Before her final TPI session, repeat EMG/NCS demonstrated mild right median neuropathy at the wrist and mild right ulnar neuropathy at the elbow.

DISCUSSION

Neuropathic pain and paresthesia can be both a diagnostic and treatment challenge. It is not uncommon for individuals with symptoms of entrapment neuropathy to have concurrent musculoskeletal disorders, including myofascial pain, that contribute to the overall clinical presentation. In the case of our patient, she had electrodiagnostically confirmed entrapment neuropathies, along with elements of myofascial pain syndrome, cervical spondylosis, and shoulder pathology (i.e., osteoarthritis, bicipital tendinitis).

TPI Session	*Muscles*	*Response, Duration*
1	Upper Trapezius* (UT), Rhomboid (RB), Levator Scapulae (LS), Infraspinatus (IS), Teres Minor (TM), Flexor Carpi Radialis (FCR), and Pronator Teres (PT)	100%, 2 weeks
2 (6 weeks)	UT*, RB, LS, IS, TM, FCR*, PT, **Extensor Digitorum Communis (EDC)**	60%, 8 weeks
3 (10 weeks)	UT*, RB, LS, IS, FCR, EDC, **First Dorsal Interossei (FDI)**	70%, 6 weeks
4 (6 weeks)	UT*, RB, LS, IS, FCR, EDC, **Extensor Carpi Radialis (ECR), Splenius Cervicis (SC)**	70%, 6 months
5 (8 months)	UT, RB, LS, EDC, **Flexor Digitorum Profundus (FDP)**, **Flexor Digitorum Superficialis (FDS), Biceps Brachii (BB)**	80%, 6 weeks
6 (2.5 months)	UT, LS, EDC, FDS, PT, FCR, FDI, **Flexor Pollicis Longus (FPL), Abductor Pollicis Brevis (APB)**	80%, 6 weeks
7 (2 months)	UT, LS, EDC, FDS, PT, FCR, FDI, FPL, APB	80%, 12 months
8 (13.5 months)	BB, EDC, FDS, PT, FCR, **Triceps Brachii (TB)**, **Brachioradialis (BR)**	80%, 2 years

Fig. 1. Timeline of trigger point injection (TPI) sessions. Each session includes the interval from the preceding injection, muscles targeted (asterisk = bilateral; bold = new muscle targets), and percentage of pain relief and duration of improvement from that session.

The relationship between the pathogenesis of myofascial pain and entrapment neuropathy remains unclear. Nerve irritation from entrapment syndromes may result in secondary hyperalgesia and myofascial trigger points in muscles supplied by the entrapped nerve (14). On the other hand, myofascial trigger points may themselves contribute to nerve entrapment by increasing muscle tension and altering biomechanics (15). Given this possible bidirectional relationship, it is not surprising that these conditions are challenging to diagnose and treat. Myofascial pain may not only coexist with neuropathies but may also perpetuate them.

The pathophysiology of entrapment neuropathies is complex and includes inflammatory changes in the nerve due to compression itself, noninflammatory fibrosis of surrounding connective tissues, and biomechanical variables such as increased tendon loading (16-18). TPIs are used to treat myofascial pain, in which hypersensitive trigger points contain increased inflammatory mediators, vasoactive substances, and pronociceptive neurotransmitters (13). These substances contribute to peripheral sensitization and abnormal spontaneous discharges at lower thresholds to both painful and nonpainful stimuli.

TPIs may alleviate perceived neuropathic symptoms in entrapment neuropathies through several mechanisms. Reduction in muscle and tendon tone may decrease mechanical compression on the adjacent entrapped nerve (16). Alleviating this imposed tension may improve local microcirculation and mitigate biochemical disturbances associated with chronic entrapment (19). Injection into active myofascial trigger points may also decrease spontaneous electrical activity and the release of local inflammatory mediators that contribute to peripheral sensitization (20,21). Finally, TPIs may reduce amplification of neuropathic pain perception by diminishing nociceptive input from myofascial tissue. Attenuation of peripheral nociceptive input may also modulate central sensitization processes that sustain neuropathic symptoms in patients with concurrent myofascial dysfunction and nerve entrapment (22).

The existing literature on addressing the myofascial component of entrapment neuropathies is sparse.

Dry needling of the thenar muscles for mild-to-moderate carpal tunnel syndrome has demonstrated promising short-term outcomes in function (23), as well as reductions in the likelihood of requiring surgery (24). To our knowledge, this is the first reported case highlighting the successful use of TPIs to treat the symptoms of concurrent electrodiagnostically confirmed median and ulnar entrapment neuropathies. TPIs are a low-risk, repeatable alternative to surgery, and their targets can be modified based on real-time patient feedback. In individuals with variable adherence to rehabilitation or home exercise programs, symptomatic relief from TPIs may facilitate improved compliance with prescribed therapies.

Our conclusions are based on observations from a single case report, and further investigation with randomized controlled trials is warranted to establish

efficacy and long-term outcomes. Future studies should determine which muscle targets provide the greatest benefit for specific entrapment neuropathies and compare the efficacy of different injectates. TPIs are a low-risk, repeatable treatment option that may have a role in conservative management of entrapment neuropathies, with targets that can be modified based on real-time patient feedback.

CONCLUSIONS

Our case highlights the potential role of TPIs as a minimally invasive therapeutic option for the symptomatic treatment of electrodiagnostically confirmed entrapment neuropathies. Our patient experienced a gradual reduction in both myofascial and neuropathic symptom severity with repeated TPI sessions, with sustained relief for up to 2 years. Clinicians should be aware of the potential bidirectional relationship between myofascial trigger points and entrapment neuropathies and consider myofascial dysfunction in the differential diagnosis of mononeuropathy-like symptoms. Although our findings are promising, further controlled trials with larger cohorts are warranted to validate the efficacy and durability of this approach.

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