LETTER TO THE EDITOR

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OXFORD

Improvement in CRPS After Deep Dry Needling Suggests a Role in Myofascial Pain

Dear Editor,

Pain is a prominent feature of the clinical diagnostic criteria (CDC) of complex regional pain syndrome type 1 (CRPS-1) [1]. We have proposed a new myofascial etiology for explaining the reversal of CRPS-1 pain and CDC by ultrasound-guided dry needling (USGDN) [2–5]. Muscle pain might contribute to the overall pain syndrome, or there may be an interaction between muscle pains and the pain described in CRPS. Here we present data that suggest that cutaneous allodynia and hyperalgesia (generally considered sensory symptoms) might have some relation to the muscle hyperalgesia and allodynia described as a part of CRPS.

A 14-year-old weighing 35 kg presented with positive Budapest criteria (Table 1 and Figure 1) [1] and musculoskeletal ultrasonography (MSKUSG) conformed with CRPS. In view of his severe hyperalgesia, stellate ganglion block (SGB) with 5 mL of 2% lignocaine + 30 mg triamcinoclone was carried out under total intravenous anesthesia (TIVA). SGB reduced swelling and increased hand temperature but left pain and sensorimotor symptoms unaffected.

One week after SGB, the first USGDN performed under TIVA (2 mg midazolam + 50 mg ketamine + 40 mg propofol) allowed uneventful skin puncture, but the needle passage through the muscle elicited withdrawal movements/exaggerated dystonic movements. Incremental ketamine (20 mg) and propofol (100 mg) controlled these movements, but the movements recurred after needle removal, with additional violent opisthotonic movements of the trunk. Three attendants had to restrain him to prevent injuries. Intravenous midazolam (1 mg) and propofol infusion (100 mg over one hour) had no effect, but two incremental doses of ketamine (20 mg) abolished the opisthotonos, and extremity movements reduced to pre-TIVA levels.

Two days later, he returned with significant improvement. A second USGDN under TIVA again produced severe dystonia and opisthotonos, which were again controlled by ketamine. When he returned two days later, he had remarkable improvement of sensorimotor symptoms. A third USGDN session with slow incremental needle introduction could be performed uneventfully without sedation. After three more USGDN sessions every other day (Table 1), he recovered completely (Figure 2). He was advised to continue physiotherapy and was normal at follow-ups for next 18 months.

Discussion

CRPS in this adolescent was different from that seen in older patients. He had fewer MSKUSG findings. Shoulder, elbow, and arm were maximally affected but recovered very rapidly after USGDN. Older patients (>30 years) show severe loss of myo-architecture with a similar duration of CRPS [2], distal parts of extremities are more commonly involved, and these patients require 20 to 45 days for complete motor recovery [5,6]. The rapid reversal of CRPS and associated disability with USGDN in this teenager led to three surmises:

1. Younger patients might respond better to USGDN.
2. Patients with fewer USG changes show quicker CRPS recovery. He became normal within 12 days (just six USGDN sessions) in spite of initial severe motor impairment and disability.
3. Could hyperalgesia and allodynia (considered sensory features) actually be because of muscular hyperalgesia and allodynia? This is a common question we have asked ourselves because we routinely observe a similar resolution of sensory symptoms with USGDN in all our CRPS patients. We documented 40% to 50% improvement of his severe hyperalgesia-allodynia with concomitant motor improvement with just one session of USGDN.

Skin penetration and passage through subcutaneous fat is smooth and painless during USGDN (even in CRPS), but patients with hyperalgesia report severe burning. The skin feels tougher/thicker to needle passage, but after waiting for one to two minutes, pain/hyperalgesia/allodynia reduces, along with reduced resistance to needle passage. This resistance to needle passage is in the skin before the needle even touches the fascia over the muscle, as seen repeatedly on USG. We surmise that allodynia and hyperalgesia could well be manifestations of painful spasm of erector pili (dermal muscle elements that cause piloerection) [7]. The presence of the needle probably causes a reflexive relaxation of the painful erector pili spasm within the first one to two minutes to reverse hyperalgesia and allodynia. USGDN elicits an identical relaxation of painful spasms in deeper muscles [4,5].

Dystonia exaggeration by USGDN, and its elimination after ketamine analgesia, suggested a hyperalgesic response of the painful muscle to USGDN rather than a myoclonus induced by ketamine/propofol, especially since it was unresponsive to midazolam. We use slow incremental needle advancement (2–5 mm at a time as allowed by the patient) to minimize pain in CRPS patients, but

Table 1 The pre- and post- treatment details of the patient

Clinical symptoms	The symptoms had started after a fall on outstretched hand with impact on wrist and right elbow while playing 2 months prior	2 days after 1st USGDN	4 days after 2nd USGDN	After 3 more USGDN sessions
Pain	2-month history of continuous pain (8–9 on NRS) with spontaneous, intermittent shocks. He kept the hand in his pocket to reduce spontaneous dystonic movements, which exacerbated the pain to 10 on NRS.	Pain was 2–3/10 on NRS.	1/10NRS	No pain
Sensory	Severe cutaneous allodynia provoked a violent withdrawal of arm and trunk in response to light touch with a cotton ball. Hyperalgesia to pinprick was also severe. Both spontaneous and evoked sensations to touch and cold and hot sensations were severe in right shoulder, hemithorax down to iliac crest, arm, and forearm.	Hyper-algesia was less by 40%.	No sensory symptoms	No sensory symptoms
Sudo motor	He had presented to Physiotherapy Department of a teaching hospital with one month history of pain and swelling in the whole of right upper extremity with reluctance to use the hand.	No swelling	No swelling	No sudomotor symptoms
Vaso motor	They had documented temperature asymmetry of 1 °C.	No asymmetry documented		
Motor	Severely reduced ROM at shoulder, elbow, and wrist. DASH score was 75, indicating severe disability. Active and passive physiotherapy, electrical modalities or myofascial release had been impossible because of the extremely severe hyperesthesia and allodynia. Mirror box therapy initially appeared to improve ROM at wrist and fingers but later, the allodynia worsened and he developed a sudden displacement of right scapula on waking up one morning, and they referred him to us for further management.	Patient reported 40–50% reduction of symptoms.	USGDN could be performed uneventfully with no sedation.	Complete motor recovery, normalization of dynamometer reading and MSKUSG. DASH improved from 75 to 15, and pain DETECT became 5 from 23 in 12 days of USGDN. Schooling and full activities resumed. At 18 months, he is active in sports like any normal teenager.
pain DETECT	Score was 23, indicating a definite neuropathy.			
PHQ-9	Patient Health Questionnaire-9 score was 5, indicating absence of psychological issues.			
MSKUSG	Showed muscle hyperechogenecity and muscle size reduction in digital flexors/extensors and supinator conforming with CRPS. His supraspinatus was significantly smaller with hyperechogenecity.			
6 USGDN sessions performed once in 2 days over 12 days. USG ensured accurate targeting of the agonist and antagonist muscles performing the restricted/painful movements and in avoiding vital structures. USGDN protocol was one needle at 5 cm intervals both length- and breadthwise between the 2 ends of the muscle.				
Neck and shoulder	Trapezius, scalenes, levator scapulae, splenius and semispinalis, the cervicis and capitis parts of longissimus, iliocostlais cervicis, supra/infraspinatus, latissimus dorsi, teres major/minor, subscapularis, pectoralis major/minor, deltoid, coracobrachialis			
Arm and forearm	Biceps, brachialis and brachioradialis-anteriorly; triceps-posteriorly; flexor carpi radialis and ulnaris, flexor digitorum superficialis and profundus, pronator teres and quadratus-anteriorly; brachioradialis, extensors of the wrist and fingers, supinator and anconeus-posteriorly.			

CRPS = complex regional pain syndrome; DASH = disability of arm, shoulder, and hand score; MSKUSG = musculoskeletal ultrasonography; NRS = numeric rating scale; ROM = range of motion; USGDN = ultrasound-guided dry needling.

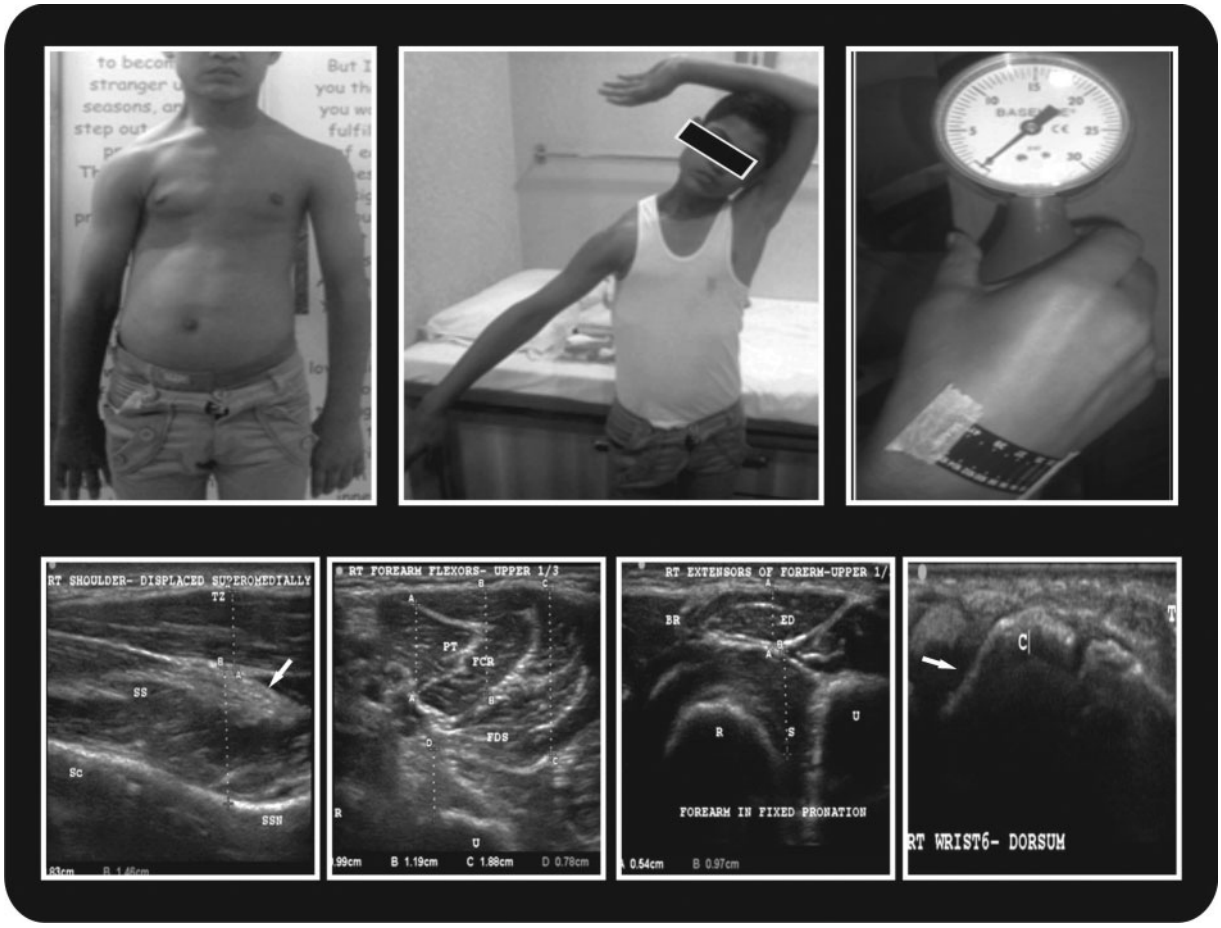

Figure 1 Row 1: Pre-treatment clinical images. Note the right shoulder at a lower level than the left, with the body tilting to the left to support the weight of the right limb. The right arm is adducted in fixed pronation, and the winging of the right scapula with the upper medial end is prominent. He had about 15° abduction at the shoulder, but no other movements were possible (no wrist flexion/extension, no elbow supination nor any other movements at the shoulder). The dynamometer reading was 0 psi in the right hand compared with 10 psi in the left hand. Row 2: Image 1: The supraspinatus muscle is 76 mm thinner on the right side and shows a definite area of hyperechogenecity (arrow). The trapezius, however, appears thicker; whether by being bunched by the deformity is uncertain. Image 2: The flexors of right forearm show mild hyperechogenecity compared with the left. This is unlike the very obvious hyperechogenecity seen in complex regional pain syndrome (CRPS) patients. The muscle bulk is reduced only in pronator teres (supination impossible), and his flexor digitorum profundus was 1 cm in the normal left hand compared with 0.79 cm in the CRPS hand, while the flexor digitorum superficialis appears to be similar in size in both hands. His dynamometer reading of 0 psi indicated a definite involvement of digital flexors/extensors. Image 3: In comparison with the normal forearm, the CRPS-affected extensor digitorum reading was 27 mm lower. Supinator was 12 mm lower. He was unable to extend his wrist, which had to be held up. Image 4: The digital tendons of right hand show effusion (arrow) as well as in-between the carpal bones.

those with hyperalgesia/allodynia might require topical prilocaine or analgesia with fentanyl lollipop or ketamine [4].

Ketamine infusion has been a CRPS treatment because of its effects on the NMDA receptors blocking central sensitization. In general, the higher the ketamine dose, the greater the reduction of CRPS pain and duration of relief [8]. Low-dose infusions were found to be effective in patients with less severe distal disease and are ineffective in improving function [9]. Outpatient protocols utilizing low–subanesthetic dose (40 mg to maximum 80 mg) ketamine infusions administered over several days have reported pain relief but showed loss of effectiveness after six to 12 weeks [10]. Only the coma-inducing doses claim functional improvement. Our patient received 80 mg of ketamine over 90 minutes on just two occasions (instead of five- to 10-day infusion regimes). He also had complete functional improvement (Figure 2) sustained at 18 months. Moreover, he had significant disability even after the second TIVA, but improved

Figure 2 Row 1: Pre-treatment clinical images. After two sessions of ultrasound-guided dry needling under total intravenous anesthesia, pain was 1/10 on the numeric rating scale and there was no hyperesthesia or allodynia. The winging of right scapula seen in row 1 has disappeared. He has regained the full range of motion at the shoulder and wrist. Note that the dynamometer reading was also just less than 1 psi as compared with the 10 psi of the normal left hand. AA, BB, CC, DD = calipers used to measure the thickness of muscle; BR = brachioradialis; C = carpals; ED = extensor digitorum; FCR = flexor carpi radialis; FDP = flexor digitorum profundus; FDS = flexor digitorum superficialis; PT = pronator teres; R = radius; S = supinator; Sc = scapula; SS = supraspinatus; SSN = suprascapular nerve; T = tendon; TZ = trapezius; U = ulna.

dramatically over the next four USGDN sessions. So we believe that USGDN was instrumental in reversing his CRPS reversal rather than ketamine. However, the use of ketamine forms a confounding limitation for our surmise that our patient improved solely because of USGDN.

The efficacy of USGDN in reversing CRPS is based on a different understanding of CRPS pathophysiology: that in CRPS, abnormal co-contraction of digital flexors/extensors replaces normal reciprocal inhibition, causing the movement difficulties and disability of CRPS. Attempted movements with muscles tethered by constant co-contraction leads to friction and inflammation at the digital tenosynovial sheaths manifesting as pain/vasomotor/sudomotor abnormalities that form the CDC of CRPS. Sequel-like central sensitization and recruitment of the sympathetic system follow. In our patient, the more affected shoulder arm and elbow also responded to USGDN with CRPS reversal.

USGDN addresses this co-contraction to relieve not only pain and CDC but also the disability, which has been impossible with any other treatments. As dry needling is a specific treatment for myofascial trigger points (MTrPs), it is logical to assume that MTrPs (with compromised local blood supply, hypoxia, and acidosis) in both agonists and antagonists might cause co-contraction, and later ischemic fibrosis in CRPS. We have shown USG evidence of muscle edema, tenosynovial inflammation, and later fibrosis in various stages of CRPS [4,5,11].

USGDN produced a dramatic, lasting reversal of not just sensory but all aspects of CRPS in this patient. This is also our routine finding in all CRPS patients [2–5]. This report demonstrated the additional possibility of muscle pain referral to skin as hyperalgesia and allodynia, hitherto considered sensory symptoms.

Supplementary Data

Supplementary Data may be found online at http://painmedicine.oxfordjournals.org.

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LETTER TO THE EDITOR

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Spinal Cord Compression Related to Spinal Cord Stimulator

Dear Editor,

There is very little published data on the rare but serious complication of spinal cord compression associated with spinal cord stimulator (SCS) placement. We recently encountered a compelling case in which a patient developed subtle signs of spinal cord compression after SCS placement. This patient is a very pleasant 82-year-old lady who was referred by her neurosurgeon to consider an SCS trial. She has a history of lumbar radiculopathy and spinal stenosis. She unfortunately suffers severe persistent pain in the lower extremities despite extensive conservative treatments. The patient elected to have an SCS trial before considering surgical decompression. Her PE prior to SCS trial revealed an antalgic gait and ambulation with a single point cane. Motor strength testing was 5/5 in the lower extremities throughout. Proprioception, pin-prick, and light touch sensation were intact in the lower extremities. The SCS trial was uneventful with two percutaneous Medtronic Octrode spinal cord stimulators positioned at the T8 level. The insertion level for the percutaneous leads was at the L1-L2 interlaminar space. She reported greater than 50% relief during the five-day trial period. The permanent SCS system was implanted three weeks later. Her pain pattern and PE were unchanged before the permanent stimulator implantation. The permanent SCS procedure was performed in the same fashion while the patient was in a prone position with epidural needle entry sites at the L1-L2 interlaminar space. A magnetic resonance imaging (MRI)–compatible pulse generator and two percutaneous spinal cord stimulators were implanted with final lead placement at the T8 level. Adequate stimulation coverage in the lower back and both lower extremities was confirmed during intraoperative programing. Postoperatively, she denied any worsening pain, weakness in the legs, or pain in the thoracic back region. However, she was found unable to stand or ambulate without assistance. There was no swelling or hematoma at the surgical sites. Motor strength in sitting position revealed hip flexors 4/5, knee extensors 4/5,