CASE REPORT

Reversal of Quadriplegia with Ultrasound-guided Dry Needling of Muscles Affected by Critical Illness Polyneuromyopathy/Neuropathy

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Abstract

Case report: A 70-year-old developed critical illness polyneuropathy/critical illness polyneuromyopathy (CIP/CIPNM) quadriparesis with bilateral foot drop after a prolonged intensive care unit (ICU) stay. Systematic ultrasound-guided dry needling (USGDN) of axial and limb muscles reversed the low-grade co-contraction of agonist/antagonists of extremities surmised to cause CIP/CIPNM paresis to restore reciprocal inhibition essential for normal movements.

Disability of Arm, Shoulder, and Hand (DASH) score improved from 96 to 15, allowing daily activity resumption within 2 weeks and independent walking at 8 weeks with a walker for right foot drop. Sonography documented the reversal of structural disruption of muscle with USGDN.

Conclusion: CIP/CIPNM reversal with USGDN suggests low-grade co-contraction as a probable cause of paresis in this patient.

Keywords: Critical illness myopathy, Critical illness neuropathy, Critical illness polyneuromyopathy, Quadriparesis, Quadriplegia, Ultrasound-guided dry needling.

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Case Description

A 70-year-old man with a history of asthma and chronic obstructive pulmonary disease (12 years), ischemic heart disease (9 years) with a medicated stent in the circumflex branch, and a calcified plaque in the left coronary artery managed with oral nitroglycerin, diabetes managed on gliclazide, metformin, and sitagliptin (glycosylated hemoglobin 7 gm/dL) was admitted to the ICU with left lower lobe pneumonia. Reverse transcription polymerase chain reaction and coronavirus disease antibody tests were negative. He was intubated with fentanyl and midazolam sedation and intravenous vecuronium and ventilated through tracheostomy later. He had a turbulent ICU course with septicemia with various organisms (*Klebsiella pneumoniae, Acinetobacter baumannii*, *Pseudomonas aeruginosa*, and finally *Enterococcus faecium* treated with meropenem, colistin, and mycamine). The film array respiratory panel and *Mycobacterium tuberculosis* complex were negative. β-D-glucan test for *Candida* showed 133 pg/mL, but the galactomannan test for aspergillosis was negative (0.36).

He had oliguria from acute kidney injury, which resolved with furosemide infusion. He required ionotropic support with noradrenaline, dopamine, and adrenaline. Insulin infusion ensured tight glycemic control for 2-hourly nasogastric feed with a high-protein formula. But his worst problem was copious bronchial aspirate and continuous severe bronchospasm unresponsive to nebulized budesonide, salbutamol, and ipratropium bromide (Duolin®), a trial of vecuronium and different ventilation modes like volume control, pressure support, and synchronized intermittent mandatory ventilation. It was minimally responsive to nebulized adrenaline and ketamine infusion and remained thus for 2 days. AnaConDa™ with sevoflurane was planned. Fortunately, acupuncture (lung points 1–7) done at the family's behest reduced the bronchospasm. The patient perceptibly improved, and the ventilation improved steadily over 48 hours.

His C-reactive protein (CRP) at ICU admission was 36, and his white blood cell (WBC) count was 20,000. Later CRP varied between 399 and 145 and 120 and finally 7.5 and WBC count from 31,000 to 12,000. A transfusion of packed cells was administered to treat hemoglobin of 7 gm/dL.

The patient was uneventfully weaned off the ventilator at 21 days, the tracheostomy closed, and oral feeds resumed. His glycosylated hemoglobin was 7.6 gm/dL; total protein was 5.10 gm/dL, and albumin was 2.20 gm/dL with albumin:globulin ratio of 0.76. Creatine kinase was 40 U/L, procalcitonin was 1.5, and vitamin B12 levels were normal. Then he was found to have profound quadriplegia and extensive anasarca over the extremities. The neurologist diagnosed critical illness neuromyopathy based on electromyography findings (Table 1) and started coenzyme Q. He was discharged after 26 days in the hospital and 23 days in ICU.

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He was presented 2 days later at our daycare center for burning neuropathic pain in all four extremities distally. On examination, he had extensive distal extremity anasarca and had only sluggish and disjointed flickers in his shoulder in upper extremities (UE) but none in his lower extremities (LE) despite intensive physiotherapy twice daily for 6 days in the hospital.

In UE, touch sensation was intact, with reduced pain perception. The DASH score was 96 (Table 1). LE had no sensation of touch, pressure, pain, or vibration. Pregabalin 75 mg at night, daily USGDN, and physiotherapy were started.

The neck, back, limb-girdle muscles, UE extensors, and lateral and hamstring muscles of LE were systematically addressed with anatomical precision in two hemibody sessions of USGDN (Fig. 1). The third session addressed UE flexors, abdominal muscles, and the anterior LE muscles. All the muscles of the body were therefore addressed in 1 week. In one session, 150–200 needles of varying lengths depending on muscle thickness were used. For example, 13-mm needles were used for the thin tendons around the ankle and wrist, and 120-mm needles were used for the inner and outer thigh muscles. Such extensive needling was possible only because the

Table 1: Electromyography findings and sequential response to USGDN

	Sensory responses	*Motor responses*				
Upper extremity: left and right ulnar and median nerves	Low amplitude responses	Normal amplitude motor response in left ulnar nerve, borderline slow motor conduction, delayed motor responses, and borderline low amplitude motor responses in right ulnar nerve Slow motor, delayed F responses, low amplitude motor responses in left and right median nerves. Profuse fibrillations and positive sharp waves in left abductor pollicis brevis, flexor digitorum with the fallout of motor units				
Lower extremity: left and right sural and superficial peroneal nerves	Absent sensory potential	Normal amplitude motor responses, slow motor conduction, and delayed F responses in left tibial nerve Absent motor responses, slow motor conduction, absent F responses in right tibial nerve. Absent motor responses left and right peroneal nerves to extensor digitorum brevis and tibialis anterior. Profuse fibrillations and positive sharp waves in left tibialis anterior with no motor units, and in left quadriceps and in adductor hallucis with the fallout of motor units				
Time	Upper extremity			Lower extremity		
	Touch and pain sensation	Power	Grip strength/DASH	Touch and pain sensation	Power	Foot drop
D1 at Ashirvad	Reduced in both. R < L	Only flickers	Cannot make fists/96	Absent from groin	1–3 at hip 0 below the knee	Positive, both ankles and toes
1 week	Reduced in both. R < L	3 in left 3–4 right	Makes fists 0.5 PSI	Absent below mid-thigh	3–4 at the hip, 1–2 at the knee, and 0 at the ankle	Positive, both ankles and toes
2 weeks	Reduced in both below elbow. R < L	4–5 in both	1 PSI grip strength	Absent below knee in both	4–5 at the hip, 3–4 at the knee, 0–1 at the left, and 0 at the right ankle	Positive, both ankles and flickers at left toes
3 weeks	Reduced in both below wrist. R < L	5 in both limbs	3 PSI grip strength/15	Absent below mid-shin in both	5 at hip and knee, 1–2 left ankle, and stands 1 min with support	Positive, both ankles and I–II grade power at left toes
4 weeks	Normal sensation of touch and pain both upper limbs. USGDN reduced to weekly once	Lifts ½ kg for exercise	4 PSI grip strength/10	Present at intermalleolar area in both	Able to stand 10 min and walk 20–30 steps with two people supporting, walker, and a splint for foot drop	Positive, both ankles and I–II grade power at left toes
5 weeks	Burning over flexor/extensor tendons above wrist	Lifts ½ kg for exercise	All daily activities of life possible	Present at left hind foot but not right	Walks 100 steps with foot drop splint, walker, and minimal support	Positive, right but 2–3 left ankle and toes
6 weeks	Burning responds to USGDN but recurs by the 6th day	Lifts 1 kg weight as exercise	Totally self-sufficient in all activities	Present at left foot but at midleg on right	Walks 50–70 steps with splint and uses a walker but no added support	Positive, right. 40° left ankle and toes extension seen

Contd…

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	Sensory responses	*Motor responses*				
7 weeks	Normal sensations No burning. USGDN once in 2 weeks for upper limbs	Lifts 1 kg weight for exercise	Applicable for week 7–10	Left foot hyperalgesic but normal touch. No sensations at midleg on right	Stands independently without support/ walker. No splint, walking with a walker for all daily activities	Positive, right. On the left, 90–100° extension and toe extension
8 weeks	Normal sensations. No burning. Once in 2 weeks for upper limbs. USGDN discontinued	Lifts 2 kg weight for exercise		Left foot hyperalgesic but normal to touch. No sensation in the right midleg	No splint is needed for all daily activities. Walks for about 15–20 min. Weight training all four limbs	Positive, right. On the left, 90–100° extension and toe extension
9 weeks	Normal sensations. No burning. Once in 2 weeks for upper limbs	Lifts 2 kg weight for exercise		Left foot hyperalgesic but normal to touch. No sensation in the right midleg	Stands for 10 min without support. Walk with a walker. Climbs 1 flight of stairs with handrails	Positive, right. On the left, 90–100° extension and toe extension
10 weeks	Normal sensations. No burning. Once in 2 weeks for upper limbs	Lifts 2 kg weight for exercise		Normal touch sensation on the left but no sensations on the right till midleg	Stands for 10 min without support. Walks for 20 min with a walker. Climbs one flight of stairs with handrails	Positive, right. On the left, 90–100° extension and toe extension

Strength, walking ability, and independence continued to improve over the next 6 months. He goes to work twice weekly. No foot drop on the left, but his right foot drop necessitates the continued use of a walker.

patient was extremely cooperative and highly motivated to become self-reliant. Of course, the lack of sensations in LE and reduced pain sensation in UE made the treatment less arduous.

Results

The UE showed rapid weekly progress with USGDN (Table 1). By 21 days, his grip strength increased to 3–4 PSI, which is sufficient for typical daily activities like bathing, dressing, and eating on his own (Fig. 1), allowing USGDN discontinuation at 8 weeks. LE functional recovery was slower, with grade III–IV strength in his hip and knee muscles by 4 weeks. By 14 days, he could stand for a minute with three people supporting him. But by 30 days, he could stand up from a sitting position by himself and walk about 100 steps with just a walker (Table 1) (Fig. 1). USGDN was discontinued after 12 weeks. The left foot drop improved by 8 weeks, but the right foot drop has persisted to date.

Initially, extremity sonoanatomy showed structural disruption and low muscle bulk. UE muscle bulk improved within 15 days of USGDN, but the LE lagged behind, particularly in the area supplied by the common peroneal nerve (CPN) on the right side (Fig. 2). The right sciatic, common peroneal, and tibial nerves (TNs) showed a vacuolated appearance compared to the normal appearance on left (Fig. 1).

Discussion

The incidence of CIP, critical illness myopathy (CIM), and CIPNM vary according to patient population, definition, and timing of evaluation. Sepsis, multiple organ failure, or prolonged ICU stay contribute to CIP/CIM/CIPNM. Individuals with sepsis or systemic inflammatory response syndrome may show a 70% incidence which increases to 100% if complicated by multiple organ failure. About 25% of ICU patients seem to develop weakness at 7 days, and 49–84% have electrophysiological abnormalities.[1]

The diagnosis relies on clinical, electrophysiological, and muscle biopsy investigations. Concurrent issues such as sedation, neuromuscular blockers, and neuromuscular weakness (after severe infections and antibiotics) obscure the onset time. Profound quadriparesis may present with bulbar, facial and pupillary musculature preservation, and autonomic stability, or there may be bulbar involvement as well with difficulties in ventilator weaning ophthalmoplegia, etc.[1]

Therapies using insulin, hormones, immunoglobulins, and antioxidants have shown inconclusive results.[1] A total of 88% of patients with CIM and 55% of patients with CIPNM/CIP may take 1 year to recover.[2]

Critical illness myopathy, CIP, and CIPNM affect respiratory and most skeletal muscles with gradual loss of reflexes as weakness progresses. CIM manifests with proximal > distal weakness, sensory preservation, and atrophy depending on illness duration. CIP/CIPNM manifests with distal > proximal weakness, sensory changes, and limited atrophy. CIPNM manifests proximal > distal weakness, distal sensory loss, and variable atrophy. Initial preservation of reflexes is common, but a gradual loss will occur as weakness progresses in all types.

Our patient had distal > proximal weakness, muscle atrophy confirmed by USG, and sensory deficits indicating CIP/CIPNM. His muscle recovery also was from proximal to distal muscles.

Based on our 18 years of experience of extensive and successful reversal of many neuropathic conditions at the Ashirvad Institute of Pain Management and Research with USGDN, we believe that motor nerves are as vulnerable to neuropathy as sensory nerves. Motor neuropathy appears to lead to the development

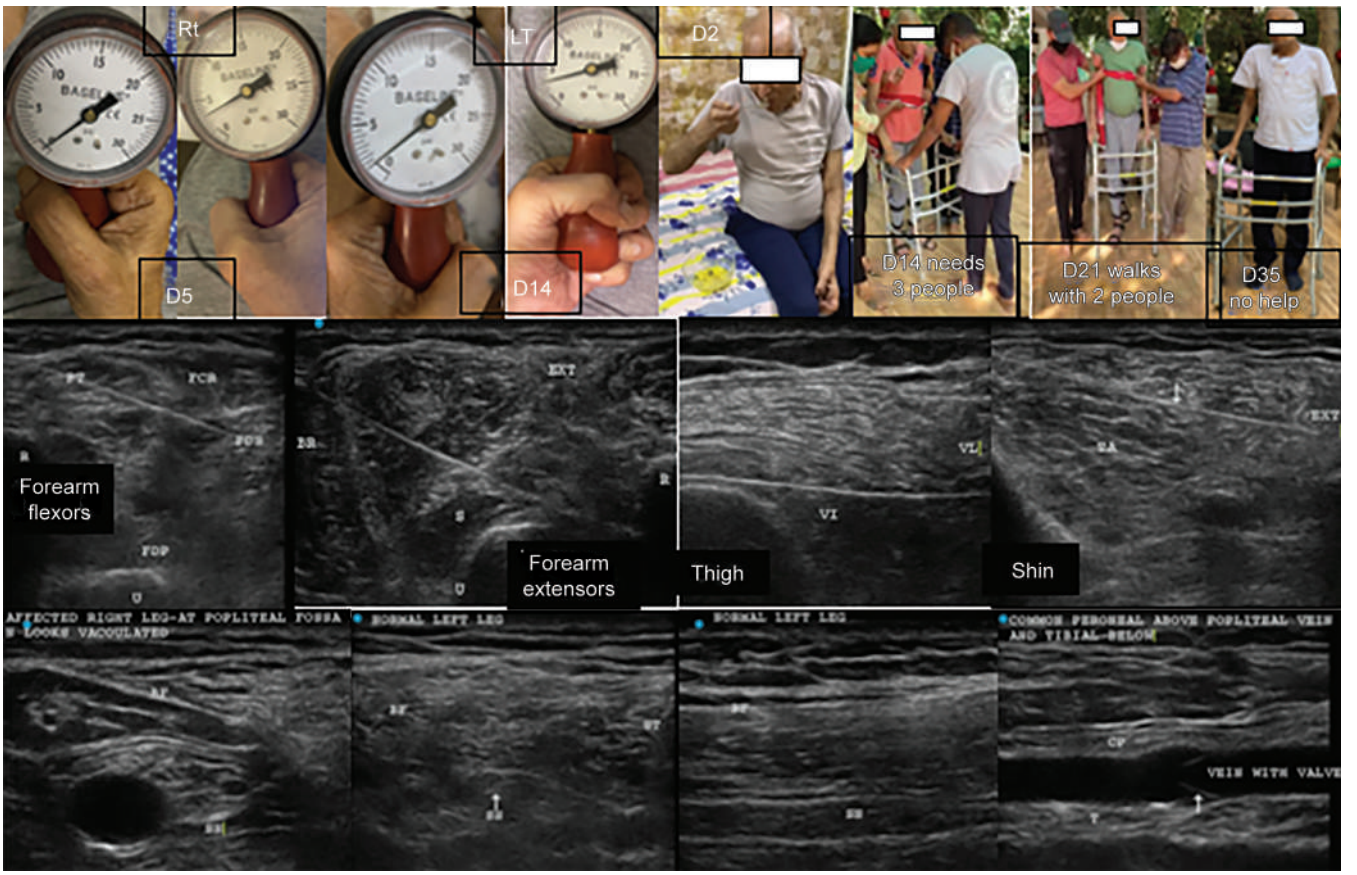

Fig. 1: Clinical improvement after USGDN. First row: after 14 days of USGDN. Grip strength increased to 3 PSI, wrote, dressed, and ate by himself at 21 days. Improvement in standing and walking from day 14 to 35 when he could take 100 steps with a walker. Second row: USGDN of upper and lower extremity muscles. Third row: sciatic nerve of the foot drop side looks vacuolated as compared with normal sciatic nerve in axial and longitudinal section. R, Radius; U, Ulna; PT, Pronator teres; FCR, Flexor carpi radialis; FDS, Flexor digitorum superficialis; FDP, Flexor digitorum profundus; BR, Brachioradialis; Ext, Extensor; S, Supinator; F, Femur; VI, Vastus intermedius; VL, Vastus lateralis; TA, Tibialis anterior; EXT, Extensor digitorum; SN, Sciatic nerve; BF, Biceps femoris; ST, Semitendinosus; CP, Common peroneal nerve; TN, Tibial nerve

of muscle spasms which facilitate the generation of myofascial trigger points (MTrPs).[3–6] MTrPs could be active, generating spontaneous and referred pain, or latent, where the pain is only produced on palpation,[5] but can disturb motor function by causing mechanical effects such as localized areas of stiffness and disordered recruitment of muscle fibers that results in weaker, painful, and incoordinate movements due in part to interference with reciprocal inhibition.[3] Myofascial pain syndromes (MPS) seem to have a ubiquitous presence in neuropathic pains.[4] Therefore, we treat all neuropathies as neuromyopathies, and it has been our routine observation that most neuropathies that manifest with MPS improve consistently with USGDN. The introduction of a needle causes immediate, complete analgesia by a physical "needle effect,"[3] probably due to reflex relaxation of muscles upon needle introduction. It also reverses the motor effects of MTrP mentioned above.

We treated him with USGDN based on our 18 years' success in treating neuropathy as neuromyopathy in poststroke pains, chronic postsurgical pains[7,8] complex regional pain syndrome (CRPS),[9,10] and writer's cramp[3] where an element of abnormal co-contraction replaces the reciprocal inhibition essential for smooth functioning of agonist and antagonist muscles of extremity movements. This co-contraction likely produces a relative impairment of movements that presents clinically as weakness/paresis. USGDN elicits a local twitch reflex on needle introduction with immediate, complete analgesia by a physical "needle effect"[3,6] presumably due to reflex muscle relaxation. This relaxes the abnormal co-contraction to reestablish the normal reciprocal inhibition, thereby enabling and streamlining normal movements.

In CRPS and writer's cramps, an element of abnormal co-contraction of agonist and antagonist muscles of extremity movements replaces the normal reciprocal inhibition essential for smooth movements. Constant co-contraction leads to tenosynovitis manifesting as inflammation and hypoxic changes in muscle, presenting as a structural disruption on sonoanatomy of the muscles in CRPS, which gets reversed by USGDN.[3,7–10] We surmise that in our patient, a similar but intense co-contraction caused severe movement impedance that presented as weakness/paresis and muscle structure disruption. It also produces secondary tenosynovial inflammation, which causes pain, burning, warmth, and swelling of the feet and wrist.

All of these symptoms were alleviated by USGDN; the rapid and specific improvement with every session of USGDN was obvious to the patient, physiotherapist, and us. This post-USGDN improvement was in sharp contrast to the initial severe quadriparetic disability, where he could not even scratch his nose, could not shift in the bed and required manual shifting, and had to be fed despite 10 days of twice-daily intensive dedicated physiotherapy.

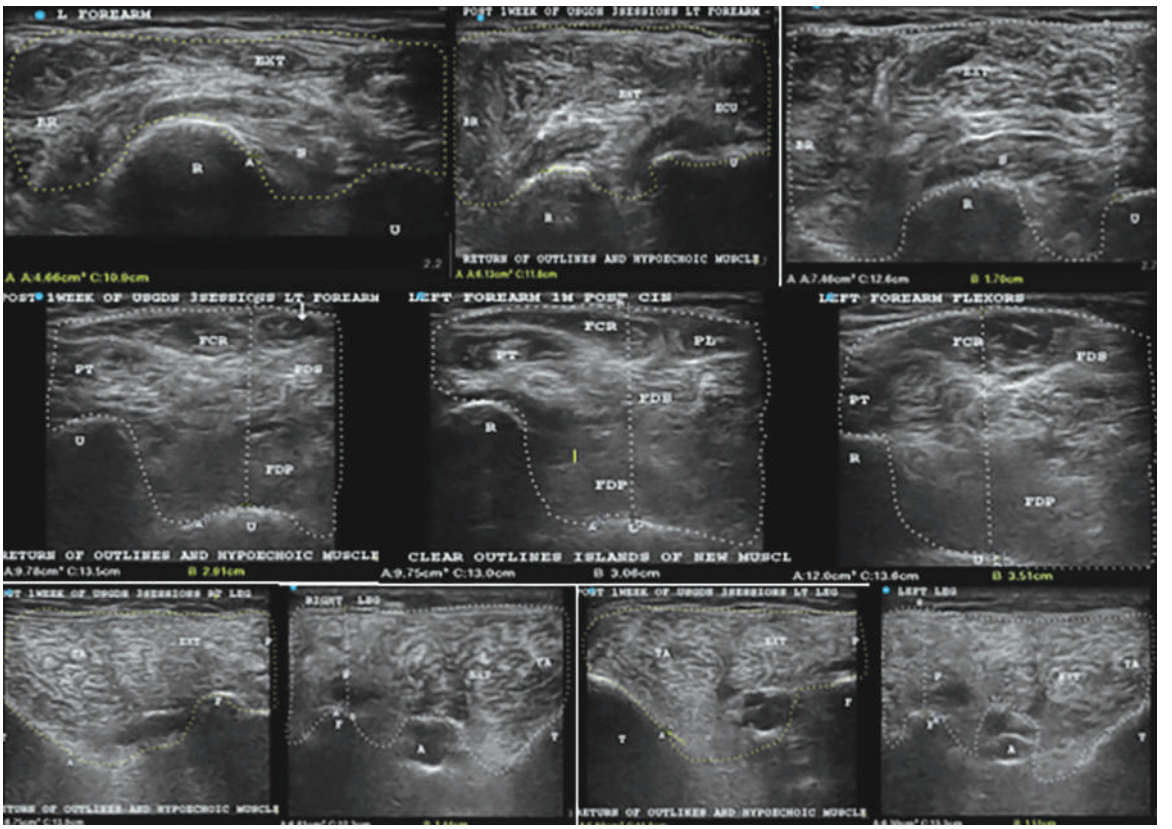

Fig. 2: Normalization of muscle bulk and echogenicity after USGDN rows one and two—show the flexor and extensor compartments of the forearm at 1 week, 1, and 2 months after USGDN. The muscle outlines have become clearer in the images on the middle and right. The muscle bulk, as measured by the caliper, has also increased significantly in the images in the middle and right, which are taken after 4 and 8 weeks of USGDN. There are islands of hypoechoic muscle in the images in the middle and on the right, particularly in pronator teres (PT), flexor digitorum superficialis (FDS), and flexor digitorum profundus (FDP). Similar changes were seen in the muscles of arm, thighs, and calf as well. Third row—the dorsiflexor muscles of the leg after 1 week of USGDN and after 2 months show a clear improvement of muscle outlines and bulk on the left, but in the right leg with continuing foot drop, there is some improvement in outlines but no improvement in muscle bulk

The results of USGDN were very specific, with an objective improvement of DASH score from 96 to 10 in 3–4 weeks, demonstrable muscle bulk increase, and reversal of structural disruption (Fig. 2).

The UE improved faster than LE, mainly because of the foot drop and sensory loss from concomitant CPN damage during the ICU stay. Early postural correction of the externally rotated leg with a partially everted foot might have reduced/prevented CPN damage, which extended even to the TN on the right side. The vacuolation was seen in the right sciatic, common peroneal, and TNs from the midthigh level as compared to the left (Fig. 1).

This is the first reported study of the sonoanatomy of muscle disruption in CIP/CIPNM and its reversal by USGDN, a novel treatment modality. The suggestion that low-grade co-contraction contributed to paresis in CIP/CIPNM in this patient has to be reproduced in a large cohort of patients for scientific confirmation.

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References

1. Hermans G, De Jonghe B, Bruyninckx F, et al. Interventions for preventing critical illness polyneuropathy and critical illness myopathy. Cochrane Database Syst Rev 2014;2014(1):CD006832. DOI: 10.1002/14651858.CD006832.pub3
2. Koch S, Wollersheim T, Bierbrauer J, et al. Long-term recovery in critical illness myopathy is complete, contrary to polyneuropathy. Muscle Nerve 2014;50(3):431–436. DOI: 10.1002/mus.24175
3. Vas LC. Ultrasound guided dry needling: relevance in chronic pain. J Postgrad Med 2022;68(1):1–9. DOI: 10.4103/jpgm.jpgm_710_21
4. Vulfsons S, Minerbi A. The case for comorbid myofascial pain—a qualitative review. Int J Environ Res Public Health 2020;17(14):5188. DOI: 10.3390/ijerph17145188
5. Lucas KR, Polus BI, Rich PA. Latent myofascial trigger points: their effects on muscle activation and movement efficiency. J Bodyw Mov Ther 2004;8(3):160–166. DOI: 10.1016/j.jbmt.2003.12.002
6. Dommerholt J, Bron C, Franssen J. Myofascial trigger points: an evidence informed review. J Man Manip Ther 2006;14(4):203–221. DOI: 10.1179/106698106790819991
7. Vas L, Khandagale N, Pai R. Successful management of chronic postsurgical pain following total knee replacement. Pain Med 2014;15(10):1781–1785. DOI: 10.1111/pme.12508
8. Vas L, Pai R. Ultrasound-guided dry needling as a treatment for post mastectomy pain syndrome—a case series of twenty patients. Indian J Palliat Care 2019;25(1):93–102. DOI: 10.4103/IJPC.IJPC_24_18
9. Vas LC, Pai R, Radhakrishnan M. Ultrasound appearance of forearm muscles in 18 patients with complex regional pain syndrome 1 of the upper extremity. Pain Pract 2013;13(1):76–88. DOI: 10.1111/j.1533-2500.2012.00539.x
10. Vas LC, Pai R, Pattnaik M. Musculoskeletal ultrasonography in CRPS: assessment of muscles before and after motor function recovery with dry needling as the sole treatment. Pain Physician 2016;19(1):E163–E179.