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Twitch-Obtaining Intramuscular Stimulation in Reflex Sympathetic Dystrophy

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Abstract

Background: *Reflex Sympathetic Dystrophy (RSD) remains a painful disease entity of undetermined etiology and variable response to therapy.*

Findings: *Presented is a patient with left leg early RSD and chronic musculoskeletal back pain who received automated and electrical twitch-obtaining intramuscular stimulation (ATOIMS & ETOIMS) treatments.*

Diagnosis combined clinical symptoms of pain and allodynia, signs of motor/trophic changes and electromyographic evidence of especially of left L5 root level irritation, with chronic bilateral, multiple level partial lumbosacral nerve root involvement. Signs and symptoms of early RSD resolved with therapy.

Conclusions: *ATOIMS-ETOIMS have a promising role in the treatment of early RSD associated with neuropathic pain resulting from spondylotic radiculopathy.*

Keywords: RSD – Twitch – Radiculopathy – Chronic pain – ATOIMS-ETOIMS.

Introduction

Reflex sympathetic dystrophy, recently termed complex regional pain syndrome (CRPS) type I (27) is a painful disabling syndrome probably produced by sympathetic efferent innervation, pathologic changes in central spinal cord activation or processing, altered peripheral catecholamines or altered neural sensitivities (26). There are variable responses to RSD treatments. These treatments include early range of motion exercises, edema control, contrast baths, desensitization, fluidotherapy, transcutaneous electrical nerve stimulation, static/dynamic splinting, stress loading techniques used in occupational therapy, oral medications, stellate ganglion blocks, implantable electrical stimulation systems *and* amputation for non-functional limbs with uncontrolled infection (1, 9, 29). Presented is a case report of an effective new treatment for early RSD associated with neuropathic pain of radicular origin. This treatment's proposed mechanism of action may enhance understanding of RSD etiology.

Case report

History

A 46 year old, white female, referred to our clinic for evaluation and treatment of clinical suspicion of RSD had a 3-month history of increasing left leg pain that had presented suddenly, preceded by a 2-week history of heavy travel which included long hours of cramped sitting, lifting luggage, and excessive ambulation. The pain started acutely and was characterized by throbbing and aching over the left side of the lower back radiating diffusely down the back of the left leg with cramping, burning, pins and

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needles, and stabbing sensations localized to the medial aspect of the left calf and foot. Pain was aggravated even by light touch that she was unable to tolerate wearing trousers. Resting seemed to help the pain. Functionally, the patient was wheelchair-bound, unable to bear weight on the affected extremity. Initially prescribed Neurontin, up to doses of 2400mg daily, combined with Percocet, 1 tablet 4 times daily, and nonsteroidal anti-inflammatory medications did not provide relief. She tried but discontinued physical therapy due to the severity of the pain. Oswestry disability score was 100%, consistent with her physical examination.

Examination

The affected left calf, ankle and foot showed mild diffuse swelling, mottled skin appearance, disuse atrophy, and patchy hair loss at L4, L5 and S1 dermatomes. The left limb was paler and cooler to palpation than the right. There was profound weakness secondary to pain and guarding on muscle testing with diminished strength of the left extensor hallucis longus compatible with a left-sided L5 radiculopathy. Straight leg raise testing was limited to 25 degrees on the left and normal on the right. Sitting root sign was positive with severe tenderness diffusely over the low back and especially the left hamstring and tensor fascia latae muscles. Also, all muscles supplied by the left L2-S2 myotomes were diffusely tender even to light palpation. There were no recognizable pain referral characteristics to suggest presence of myofascial trigger points (MTrPs).

Diagnosis

Imaging studies obtained by the referring physician included a lower extremity venous ultrasound, MRI of the lumbar spine, and plain radiographs of the left lower leg that showed no abnormalities. MRI of the left leg showed non-specific findings for inflammation or infection over the mid to distal third of the left calf. A triple phase bone-scan showed increased uptake in the left medial calf soft tissues, interpreted as a non-specific finding. Complete blood count, protein electrophoresis, cytoplasmic and perinuclear staining for antineutrophil cytoplasmic antibodies, rheumatoid factor, extractable nuclear (Ro/SS-A) and cytoplasmic (La/SS-B) antibodies and Lyme titers were unremarkable.

Quantitative analytical electromyography (EMG), consisting of motor unit action potential (MUAP) analysis, was performed at our clinic over the bilateral sartorius (L2), adductor longus (L3), vastus medialis (L4), tensor fascia latae (L5), gluteus maximus (S1) and L4-S1 lumbosacral paraspinal muscles. Using a multi-MUAP program with a Keypoint electromyograph (Medtronic, formerly Dantec), twenty discrete MUAPs were collected from two insertion points of each muscle and classified according to duration, amplitude and number of turns and phases (6). Moderate ongoing partial axonal denervation/reinnervation changes in the left tensor fascia latae (L5), and L5 paraspinal muscles were found. This consisted of ≥25% occurrence of normal duration (5-15ms), normal amplitude (0.5-2mV), polyphasic (>4 turns/phases) MUAPs, in our laboratory (unpublished data, Chu 1996). Such polyphasic MUAPS are indicative of ongoing nerve root irritation with resultant low-grade denervation leading to muscle fiber atrophy and fiber size variation. These findings, in the absence of spontaneous activity, are suggestive of a stretch/distortion injury or transient compression of the left L5 nerve root against underlying bone possibly at varying levels which include disc, facet, foramina, or lateral recess, not visualized by this patient's imaging studies. Chronic stable reinnervation EMG changes characterized as ≥15% of MUAPs with <5phases/turns and size index (area/amplitude) >1.80 were noted over the left L4, L5 myotomes and lumbosacral paraspinal muscles. Borderline ongoing axonal involvement was also seen in the right tensor fascia latae (L5) and right L5 paraspinal muscles using our laboratory criteria.

Treatment

Our patient was begun on automated twitch obtaining intramuscular stimulation (ATOIMS) and electrical twitch obtaining intramuscular stimulation (ETOIMS) therapy once a week for the next thirty consecutive weeks (7, 8). Both therapy systems are approved for clinical use at the authors' institution. ATOIMS therapy was applied with an ATOIMS device that oscillated a monopolar electrode through

a distance of 20 mm forward and backward motions. The monopolar needle oscillated at 2Hz for two seconds to a depth of 37 or 50mm depending on the length of the needle chosen for treatment into appropriate muscles. Muscles treated included the bilateral rectus femoris (L3), adductor longus (L3), vastus lateralis (L4), vastus medialis (L4), gluteus medius (L5), tensor fascia latae (L5), gluteus maximus (S1), adductor magnus (S1) and bilateral C3-S1 paraspinal muscles. This was followed by ETOIMS therapy, applied with a TD20 electromyograph (Oxford Instruments, formerly Vickers, Medelec, Teca) using 5 volts of stimulus intensity, 0.5 ms pulse width, 2Hz frequency, through an MG37 monopolar needle (Oxford Instruments, formerly Viokers, Medelec, Teca) serving as the active electrode and a circular 250mm diameter metal plate as a reference electrode. Treatments were applied along the depressed edges of the intramuscular bands and along intermuscular or intramuscular grooves. Tender areas or swollen areas gave the best yield of large force twitches but the search for tender areas was not a requirement for treatment. The force of the twitches were graded and recorded for each muscle that received treatment. Twitches were graded according to the force generated, those twitches that moved the joint in the direction of action of the treated muscle were given the value 4; visible twitches without joint movements 3; multiple simultaneous, palpable twitches 2; and single palpable twitches 1. The most forceful twitch responses were recorded separately for ATOIMS and ETOIMS treatments. The twitch forcefulness had a negative correlation with the pain level using Spearman correlation analysis ($r=-0.61$, $p=0.005$) for ATOIMS but not with ETOIMS for the left tensor fascia latae muscle. Other significant correlation findings were that of a negative correlation between the number of treatment sessions of ATOIMS-ETOIMS and VAS pain levels ($r=-0.89$, $P<0.001$). There was a positive correlation ($r=0.41$, $P=0.01$) between the number of ATOIMS-ETOIMS treatment sessions, and the twitch force recorded during ATOIMS therapy of the left tensor fascia latae (L5). No significant correlations were noted between the twitch forces recorded during ATOIMS or ETOIMS therapy of the muscles innervated by adjacent nerve roots in the symptomatic side or asymptomatic side with the number of treatments and the pain levels. Significance was accepted when $p<0.01$.

Improvement was significant by the 11th week and she regained the ability to bear full weight on the left leg. Pain level by visual analog scale (VAS), that initially was 10/10, was 3/10 by the 13th week and a pain-free state was achieved by the 19th week. As soon as improvement occurred, the patient participated in physical therapy for progressive ambulation and was able to resume work part-time. She continued with once a week ATOIMS-ETOIMS treatments for a total of 30 treatments to stabilize the pain free state and to prevent activity induced pain recurrences.

By the 30th week of treatment, a stable pain-free state was achieved without episodes of relapse, enabling her to work at full capacity. On her own volition, the patient started kick-boxing classes. Her treatments were carried through to the 34th treatment with 2, 3, 4 and 5-week intervals between treatments 30 and 31, 31 and 32, 32 and 33, and 33 and 34, respectively. Sporadic treatments were then given until the 37th treatment, at 13 months post initiation of therapy. All prior stigmata of RSD were reversed and clinical cure was achieved. On follow-up two years post-therapy, the patient remained RSD stigmata-free, pain-free without medication and had an Oswestry disability score of 0%.

Discussion

RSD has been reported to be associated with low lumbar radiculopathy (2). Analytical quantitative EMG of MUAPs confirmed the diagnosis of radiculopathy in our patient. Most of the critical changes on EMG were seen on the left L5 myotome and left L5 level paraspinal muscles, implicating the left L5 nerve root, consistent with the sensory and motor distribution of her signs and symptoms.

Recently proposed diagnostic criteria for CRPS I includes the following: 1) continuous pain disproportionate to any inciting event 2) reporting at least two signs and 3) reporting at least one symptom from each of the following four sub-categories: a) sensory, b) vasomotor, c) sudomotor/edema, d) motor/trophic changes (4, 23). Our patient was rated to have a sensitivity of 0.86 and specificity of 0.75 for the diagnosis of CRPS I using these criteria.

Automated and electrical twitch-obtaining intramuscular stimulation (ATOIMS and ETOIMS) developed for the treatment of nerve-related muscle

pain is an improvement of the dry needling procedure achieved with Gunn's intramuscular stimulation (IMS) method (7, 8). IMS provides pain relief by inducing focal muscle fiber contraction and relaxation during manual oscillation of an acupuncture needle inserted into a motor point (16). IMS found utility in the treatment of myofascial pain that was theorized to arise from a radiculopathic model. This treatment capitalizes on the phenomenon of denervation supersensitivity caused by spondylotic radiculopathy (2, 16). Denervation supersensitivity leads to a hyperreactivity of nerve and muscle tissue to chemical and physical stimuli (21, 24). This is secondary to expression of nicotinic acetylcholine receptors in noninnervated portions of the muscle fiber and to the infraterminal (subsynaptic) spreading of the nicotinic acetylcholine receptors into the depths of the junctional folds (10). Also, there is evidence that partial denervation induces a change in neuronal phenotype after lesions of mixed nerves or of the sympathetic post-ganglionic supply resulting in an increase in alpha-adrenergic receptors. This leads to hyperexcitability of the nociceptors and other primary afferent neurons by local or circulating epinephrine and nor-epinephrine. For central pathways, the adrenergic excitation would be equivalent to that produced by noxious events and would consequently evoke pain (24).

The muscle fiber shortening/contracture associated with denervation causes a mechanical traction effect on nerves, blood vessels, tendons, bones and joints causing pain (16). A recently proposed mechanism for the etiology of myofascial pain also includes dysfunctional muscle endplates causing central sensitization in the spinal cord (17, 18, 25). There is supportive evidence of axonal degeneration and denervation in adjacent and contralateral large peripheral nerves of loosely ligated motor nerves in animal studies. This suggests that peripheral motor nerve degeneration has effects over to motor neurons at the level of the spinal cord (11).

Electron microscopic analysis of the soleus and gastrocnemius muscle in patients with chronic RSD showed decreased numbers of type I fibers, increase of lipofuscin, atrophic fibers, afferent C-fiber abnormalities, and significant capillary changes ranging from severely thickened basal membranes to necrosis (29). A recent animal study induced RSD from a chronic constriction-injury model, produced denervation supersensitivity in subcutaneous resistance arteries to catecholamines and decreased contractile response to electrical field stimulation (20). Decreased contractile response to electrical field stimulation is well known in late stages of denervation of skeletal muscle. The primary author also notes this phenomenon in conditions with increased muscle tightness, stiffness and tension from presence of taut myofascial bands.

Observations in animals and humans have documented that shortened muscle fibers reduce peak contractile tension (3, 6, 14). With radiculopathic involvement, the shortened muscle fibers due to denervation occur in the paraspinal muscles as well as in the limbs, necessitating treatments that can restore muscle fiber length to be directed towards the myotomes. The proposed mechanism of pain control of ATOIMS and ETOIMS therapy is by focal exercise and stretch-induced alteration of muscle shortening, stiffness, and probable induction of fatigue of susceptible abnormal or immature motor end-plate zones MEPZS due to denervation or early reinnervation respectively. We believe that the twitches that occur from different depths and areas of the muscle from contraction of many muscle fascicles or bundles produce exercise-induced stretch to the adjacent areas with shortened muscle fibers, especially those in the contraction knots (5, 13, 28). These stretch effects from the induced twitches are very effective especially for the shortened fibers deep within the muscle to restore the muscle fibers to optimal length. The effectiveness of such stretches is from ability to concentrate the stretch forces to occur close to the involved muscle fibers. The reduction in muscle shortening and stiffness leads to pain relief from reduced mechanical traction effect of muscles on nerves, blood vessels, tendons, bones and joints (16). The exercise effects and stretch effects from the twitches may also reduce the energy crisis component proposed in the integrated hypothesis of Travell and Simons (28). Twitches are related to reflexes at the spinal cord level (18). Therefore, the twitch-induced reduction in muscle shortening/stiffness may also effect to normalize supersensitive reflexic spinal pathways. Potentially, ATOIMS-ETOIMS counters the harmful effects of denervation supersensitivity. As there is evidence of the existence of this phenomenon in RSD, ATOIMS-ETOIMS has potential in RSD therapy.

The presence of more forceful twitches in affected muscles during ATOIMS and ETOIMS treatments may indicate areas that have less degree or less numbers of shortened muscle fibers. This type of twitches may also represent areas with reversible muscle shortening and hence indicate areas with new onset nerve irritation. If such twitches can be elicited, the forces can stretch the shortened muscle fibers towards the original length and also reduce or prevent further muscle fiber shortening by interrupting the vicious cycle of "energy crisis" occurring at these areas. The presence of forceful twitches can thus be duly used as signs for prediction of a good treatment outcome for pain relief.

The twitch force during ETOIMS treatment was uniformly graded as visible throughout the treatment course. The large visible twitch forces with ETOIMS may be due to volume conduction from the applied electrical current from the tip of the monopolar needle that also excites adjacent nerve and muscle fibers within the electrical field, consistently recruiting a larger number of muscle bundles than with ATOIMS. Therefore, even if there was a significant change in the degree of muscle shortening, there may not be a significant change in the externally visualized twitch force with ETOIMS, unless the joint moves. In contrast, the twitches induced by ATOIMS is presumably generated from stimulation of selective afferent fibers involving reflex arcs of a defined population of motor neurons that innervate a specific population of muscle fibers at each treatment point. Since it acts over a discrete area of muscle with a limited capacity to affect neighboring areas in the muscle, observing twitch force during ATOIMS may give a more adequate reflection of presence of shortened muscle fibers. *Measurement of twitch forces in ATOIMS and ETOIMS therefore need different grading systems since there seems to be differences in the mechanism of twitch elicitation with these two techniques.* After this study, we have indeed changed the description of grade two forces in ETOIMS which must not only be visible but must have enough force to shake the joint although the forces are not strong enough to move the joint in the direction of contraction of the treated muscle.

In both treatments, the points to stimulate are found along depressed edges of the tight intramuscular bands or intermuscular or intramuscular grooves in the paraspinal and lower limb muscles. Our assumption is that these intramuscular bands may indicate the areas with the greatest concentration of MEPZs. Therefore, when the muscle fibers in these zones shorten, stiffen or become fibrotic, they become palpable as myofascial bands. The depressed edges of the palpable intramuscular bands/nodes or intermuscular and intramuscular grooves may be the trajectory along which the axons supplying the MEPZs travel.

There is more pain during twitch elicitation with ATOIMS than with ETOIMS due to oscillating movements of the needle that occur with ATOIMS. There is more tendency for forceful twitches to deflect a moving needle from its original trajectory than that of a stationary needle. Therefore ATOIMS in its present mode has more potential for tissue trauma. In ETOIMS the needle remains stationary after the initial muscle penetration and the twitches are electrically induced making this method less painful. The twitches induced with ATOIMS are etiologically similar to the twitches obtained at MTrPs (17, 18, 25). With ATOIMS, the muscle fibers supplied by the stimulated nerve "jumps". This jumping motion occurs from contraction of the muscle bundles due to stimulation very close to the nerve fibers supplying these muscle fibers. The mechanism may also involve that of a stretch reflex from stimulation of afferent fibers with motor neuron discharge (18, 28). The twitches associated with ETOIMS are derived from a combination of the stretch reflex and from direct stimulation of nerve terminals at or close to the MEPZs. Direct nerve stimulation of larger nerve bundles also close to the involved MEPZS is more probable especially on occurrence of very strong twitch forces that are strong enough to mobilize the joint over which the treated muscle acts. The direct electrical stimulation of the most peripheral and terminal axons will produce only a pumping action of the muscle.

The twitches elicited with ATOIMS and ETOIMS are used therepeutically to relieve myofascial pain rather than for diagnostic localization of the MTrP (17-19, 25). Although inactivation of MTrPs have been used to treat CRP, the hypodermic needle used for injecting trigger points has a beveled edge, which can be destructive to tissues. Injecting local anesthetics into a trigger point suppresses twitch elicitation and twitch associated pain relief. Therefore, the

tremendous therapeutic significance of a twitch remained unrecognized with trigger point injections. Generally, the local twitch response has been used mainly as a diagnostic criterion for critical localization of the MTrP or inactivation of some MTrP mechanisms with such injections (17). However, when the effects of local anesthetics dissipate, the muscle pain returns because the cause of the pain which is the muscle tightness and stiffness, has not been treated.

The conical tip of the monopolar needle used to elicit twitches with ATOIMS and ETOIMS is ideal for eliciting twitches from multiple superficial and deep MEPZs located within the muscle in a three dimensional manner and inactivates supersensitive muscle and nerve without use of chemicals. In both ATOIMS and ETOIMS, the twitches are difficult to elicit when the muscle fibers are shortened and/or stiff. On the contrary, when the muscle is more normal, the twitches will be generally brisk but the force will not be strong enough to move the joint. In the presence of supersensitivity of nerve and muscle tissue, the force generated from contraction of the whole muscle will be strong enough to move the joint in the direction of direction of action of the treated muscle. However, when the muscle is very tight or tense, twitch elicitation is very difficult even with electrical stimulation.

The twitch points within the muscle that produces such large force twitches are diligently sought for during the treatment since they produce the most therapeutic pain relieving effects clinically. These points are usually very tender but this is not a requirement for treating deep MEPZs. Swollen areas in the muscle can also be objectively sought for. In both ATOIMS and ETOIMS, the pain relief that occurs especially with large force twitches is probably from producing the most effective stretching of the shortened and stiff muscle fibers (14). This leads to effective reduction of the mechanical traction effect on intramuscular nerves, blood vessels, tendons, bones and joints and also from relief of the energy crisis component proposed in the integrated hypothesis (28). These mechanisms can occur together with needle stimulation of the MTrP with inactivation of the sensitized nociceptors responsible for the twitch response reflex as proposed by Hong and Simons (17, 19, 28). ATOIMS and ETOIMS can produce immediate pain relief. Sustained pain relief occurs from cumulative effects of reduced muscle stiffness as treatments continue which allow progressive healing of nerve and muscle tissues.

Although surface electrical stimulation used by physical therapists can produce muscle twitches, this type of stimulation is superficial and non-specific and unable to excite deeply located nerves and MEPZs. Similarly, "stress loading" which is used in the treatment of reflex sympathetic dystrophy consists of active traction and compression exercises (30). These exercises provide stressful stimuli without joint motion and therefore act as non-specific and diffuse stimuli to the muscles involved. Active muscle contraction of focal areas in individual muscles due to stimulation at supersensitive MEPZs at varying depths and locations within the muscle are specific only to ATOIMS/ETOIMS.

Transcutaneous electrical stimulation (TENS) achieves pain relief by stimulating large nerve fibers in the skin that close a "pain gate" in the spinal cord (22). Closing this gate prevents painful impulses from reaching the brain. As muscle has larger nerve fibers than skin (12), stimulation of these nerves by ETOIMS-induced twitches may be more effective in closing the pain gate. The pain relieving effects of percutaneous electrical nerve stimulation [PENS] (15) or electrical acupuncture may be similar to ETOIMS. With PENS, electrical stimulation of only very superficial MEPZs occurs, due to the lack of strength of the acupuncture needle for deeper muscle penetration and stimulation. The acupuncture needles used with these methods are unable to treat deeper tissues since these needles do not have tensile strength. Also the stainless steel shaft of the acupuncture needle conducts electricity and stimulates tissues surrounding the needle shaft. The treatment time with these procedures is arbitrary and is usually in the range of 10-20 minutes. With ETOIMS, the stimulation is 2 seconds/point and the micro-stimulation is aimed to focus only on MEPZs close to the tip of the needle since the entire shaft is coated with Teflon. In theory, this would increase therapeutic effectiveness of ETOIMS while decreasing the potential for tissue damage, compared with electrical stimulation using non-coated stainless steel needles.

The key in the treatment of our patient is timely intervention, prior to the development of more chronic and irreversible denervation changes in the muscles of the spine and the lower limbs. The body

of evidence presented strengthens the proposed mechanism of action of ATOIMS and ETOIMS therapy. ATOIMS and ETOIMS have been found to exert a positive therapeutic effect in chronic pain patients, such as seen in this patient (7, 8). Spontaneous remission of RSD does occur but is usually accompanied by residual deficits (31). As our patient has no residual deficits, and has regained her prior quality of life after our timely diagnosis and therapeutic intervention, ATOIMS-ETOIMS treatment may have had a significant role in the positive outcome in this patient. These methods have a place in the consideration of treatment strategies for RSD.

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