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International Journal of Physical Medicine & Rehabilitation

Research Article

Comparison of Ultrasonic Therapy and Ischaemic Compression Therapy in Pain and Tolerance Threshold in Latent Myofascial Trigger Points 0f the Trapezius Muscle in the Age Group of 20-30 Years

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ABSTRACT

Purpose: Myofascial pain syndrome (MPS) is one of the most common musculoskeletal pain diseases and is characterized by myofascial trigger points, taut bands, and local twitch responses. The myofascial trigger points arise from overuse, overload, emotional stress or severe traumas. Although patho-physiology of MPS has not been completely understood, recent studies suggest that injured muscle fibers caused by overuse provide less oxygen and nutrition, and these deficiencies cause involuntary contractions. Myofascial pain symptoms usually involve muscle pain with specific "trigger" or "tender" points. The pain is aggravated with activity or stress. In addition to the local or regional pain, untreated and chronic cases might lead to symptoms like depression, fatigue and behavioral disturbances. There are various treatments for myofascial trigger points such as dry needling, local injection, ischemic compression, stretching, massage, and others. Of these methods, dry needling or local injection which physically stimulates trigger points is efficient for MPS by reducing muscles shortening and increasing blood flows. Ischemic compression helps tissue recovery by reperfusion after transient blood flow occlusion. The purpose of this study is to compare the effectiveness of ischemic compression therapy and ultrasound therapy on perception, pain and tolerance threshold of latent myofascial trigger point in upper trapezius.

Materials and method: 30 subjects with complaints of pain of the neck/ trapezius for a duration of up to 3 months were recruited on a conveniently random sampling based on the inclusion criteria and diagnostic criteria listed by travel and Simons and were randomly divided into two groups, Group A (N=15, 13 females and 2 males) received conventional ultrasound therapy and Group B(N=15, 13 females and 2 males) received ischaemic compression therapy for a period of seven days and threshold of feel, pain and tolerance (TF,TP and TT) were recorded on a daily basis pre and two minutes post treatment using Phyaction-787 stimulator- Galvanic mode and the readings were recorded in milliampere (mA) unit and the data is analysed statistically to compare the effects between Group A and group B.

Results: There is an immediate effect on TF, TP and TT on each day following treatment with ultrasound therapy and ischaemic compression therapy. Using paired t-test, pre and post treatment for Group A and Group B the results are statistically significant at $P<0.001$. However when compared for the difference between ultrasonic therapy and

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Received Date: June 06, 2021**; Accepted Date:** October 08, 2021; **Published Date:** October 18, 2021

Citation: Suresh AMR, Kashyap D, Behera TP, Tarsolia AK (2021) Comparison of Ultrasonic Therapy and Ischaemic Compression Therapy in Pain and Tolerance Threshold in Latent Myofascial Trigger Points 0f the Trapezius Muscle in the Age Group of 20-30 Years. Int J Phys Med Rehabil 9:p354.

ischaemic compression therapy using paired t-test on the data, the results are not significant at P<0.001 indicating that there is no statistical significance in TF,TP and TT between ultrasound and ischaemic compression therapy.

Conclusion: Both ultrasound and ischaemic compression are equally effective in treating trigger point. Both groups showed increase in TF, TP and TT thus subsequently there is reduction in pain sensitivity in trigger point. However ischaemic compression may be a preferred therapy for myofascial trigger point in a physical therapy setup as it is easily available, accessible, cost effective and non-dependent on any modality.

INTRODUCTION

Myofascial pain syndrome (MPS) is one of the most common musculoskeletal pain diseases and is characterized by myofascial trigger points, taut bands, and local twitch responses [1]. The myofascial trigger points arise from overuse, overload, emotional stress or severe traumas [2, 3]. Although patho-physiology of MPS has not been completely understood, recent studies suggest that injured muscle fibers caused by overuse provide less oxygen and nutrition, and these deficiencies cause involuntary contractions [4].

The causes for myofascial pain are structural inadequacies, tight constrictive clothing, systemic, alcohol toxicity, inflammatory diseases and relative growth hormone deficiency. There are various treatment modalities used for treating myofascial pain which includes individual treatment techniques under manual therapy, acupuncture, stress reduction, electrotherapy, body mechanics & ergonomic training, nutritional counselling and a wide range of pharmacological management [5].

Myofascial Trigger Point (MTrP) has been described as an area of hyperirritability located in a taut band of skeletal muscle that is painful on compression and gives characteristic referred pain, tenderness, and an autonomic (functionally independent) response to a remote area [6] . Myofascial pain symptoms usually involve muscle pain with specific “trigger” or “tender” points. The pain is aggravated with activity or stress. In addition to the local or regional pain, untreated and chronic cases might lead to symptoms like depression, fatigue and behavioral disturbances.

Myofascial TrPs are extremely common and has become a painful part of nearly everyone's life at one time or another, yet poorly recognized, misdiagnosed and inadequately managed cause of musculoskeletal pain seen in medical practice with a point of prevalence from 10% to 18% and lifetime prevalence from 30% to 50% [12]. It is thought by some authors to be the main cause of neck and shoulder pain [7]. In todays urbanized, fast paced & competitive life the prime cause of neck pain is due number of lifestyle factors, psychological factor like anxiety, stress.

There are various treatments for myofascial trigger points such as dry needling, local injection, ischemic compression, stretching, massage, and others [8, 9]. Of these methods, dry needling or local injection which physically stimulates trigger points is efficient for MPS by reducing muscles shortening and increasing blood flows [10, 11]. Hong compared the efficacy of dry needling and lidocaine injection for myofascial trigger points and reported that those treatments are efficient only when induced with local twitch, regardless of drugs injected [11]. Ischemic compression helps tissue recovery by reperfusion after transient blood flow occlusion [12].

Latent Trigger points (TrPs), which often cause motor dysfunction, stiffness, restricted cervical range of motion without pain, are far more common than active TrPs that cause pain. Trigger points are activated directly by acute overload, overwork, fatigue, direct impact trauma, and radiculopathy and indirectly by other existing trigger point, visceral disease, arthritic joints, joint dysfunctions, and emotional distress anxiety [13]. Latent TrP can be recruited into causing pain, therefore it becomes very important to identify trigger points and treat it with appropriate effective treatment.There are various physical therapy treatment for treating latent & active TrP which includes electrotherapy modalities like TENS, Ultrasound, Laser, etc and manual therapy like myofascial release, ischaemic compression, muscle energy technique and other treatment include acupuncture, spray and stretch have been mentioned to be effective [7, 14].

The purpose of this study is to compare the effectiveness of ischemic compression therapy and ultrasound therapy on perception, pain and tolerance threshold of latent myofascial trigger point in upper trapezius.

REVIEW OF LITERATURE

Pain is defined as unpleasant sensory emotional experience associated with actual or potential tissue damage or described in terms of such damage. Pain involves both the sensation and emotions and it is a subjective experience unique to the individual [15].

Descriptions of myofascial pain date back to the mid-1800s when Froriep described muskelschwiele or muscle calluses.He described these calluses as tender areas in muscle that felt like a cord or band associated with rheumatic complaints.In the early 1900s,Gowers first used the term fibrositis to describe muscular rheumatism associated with local tenderness and regions of palpable a hardness. In 1938, Kellgren described areas of referred pain associated with tender points in muscle. In the 1940s, Janet Travell, began writing about myofascial trigger points. Her text, written in conjunction with David Simons, continues to be viewed as the foundational literature on the subject of myofascial pain [13].

Physiology of pain [16, 15, 13, 17, 18, 19, 20]

The sensory receptor that detects the painful stimulus is termed as Nociceptors, these are specialized free nerve ending that are found nearly in every tissue of the body. They are mostly

polymodal receptors i.e. they respond to various stimulus like mechanical (Aδ), thermal (Aδ, C fibre), Chemical (C fibre) activation of nociceptor also occur through release of certain chemical substance like Bradykinin (simon) Histamine, Prostaglandins, 5HT (Hydroxy tryptamine) from the damaged tissue. Afferent nerve fibers which carry pain signals to the CNS are of two types: A delta (Group III) and C (Group IV). Deltas fibres are large diameter (2-5 microns), myelinated, fast conducting (velocity 6-30 m/sec).These transmit acute sharp pain signals and are associated with initial response to trauma i.e. First pain. C fibres are small diameter (0.5-2 microns), unmyelinated, slow conducting (velocity 0.5-2 m/sec).These transmit slow chronic pain signals i.e. Second pain which enforces inactivity of damaged tissue in order to allow healing .

On entering spinal cord via dorsal horn, the fast type Ad pain fiber terminates in laminae 1 and 5 of dorsal horn. These pain fibres excite 2nd order neuron that send long fiber immediately to the opposite side & then ascend to the brain via spinothalamic & spinorecticular tract (anterolateral pathway). The type C fiber transmitting slow pain terminates in laminae 2 & 3 of dorsal horns, called substantia gelatinosa. The last neuron in the series gives long axon which joins fibers from fast pathway n cross to opposite side & ascends in anterolateral sensory pathway.

Fibers of fast pain terminate mainly in ventrobasal complex of thalamus (3rd order neuron) from where signal are transmitted to other area of thalamus & somatosensory cortex. Slow pain fiber terminate mainly in reticular formation of brainstem, from here signal to interlamine nuclei of thalamus. The spinoreticular tracts also relay information to periaqueductal grey matter in midbrain and pons.

Pain modulation [16, 15, 17, 19, 21]

The sensation of pain can be interrupted at following four levels of ascending nociceptive pathway:

- Peripheral level
- Spinal segmental level
- Supraspinal level
- Cortical level

Peripheral level: interruption of pain by

- Removal of chemical irritants released in response to tissue damage which are responsible for nociceptive activation by application of heat which causes local vasodilatation and increased blood flow over the affected area
- Alteration of cell permeability by ultrasound, cryotherapy.

Spinal segmental level: segmental inhibition and physiological blocking are two mechanisms of neuromodulation of pain which occurs at this level. Intervention at this stage involves inhibition of activity of small diameter group III & IV fibres before the incoming information ascends further up.

Supraspinal level: ascending pathway makes important synaptic connection with brainstem structure involved with descending pain modulation system.

Cortical level: Intervention involved is individual perception modification.

Pain Gate control theory [15, 17, 16, 22, 19, 20]

It was 1st proposed by Melzack and Wall in 1965.Afferent input enters spinal cord via the posterior root and afferent information is passed through synapses in the substansia gelatinosa and nucleus proprious. For pain to pass through this gate there must be unopposed passage for nociceptive information arriving at the synapses in the substansia gelatinosa. However, if the gate is also concurrently receiving impulses produced by stimulation of thermo receptors or mechanoreceptors (transmitted via large diameter myelinated fibres), then this traffic predominates with resultant pre synaptic inhibition of the small diameter nociceptive information. Consequently for the 'gate' to be open to nociceptive traffic, the input has to be of a predominantly small diameter nociceptive nature; if the large diameter afferent information is superimposed then the 'gate' is closed to nociceptive traffic.

Descending pain suppression

If nociceptive information is allowed through the gate then this traffic will continue up the lateral spino-thalamic tract of the spinal cord to the thalamus, and from here to the cerebral cortex. As this stimulus passes through the brainstem it may cause an interaction between the periaqueductal area of grey matter (PAG) and the raphe nucleus in the mid-brain. These nuclei form part of the descending pain suppression system and their descending neurons can release an endogenous opiate substance into the substansia gelatinosa at a spinal cord level. The chemical nature of this endogenous opiate, which may be beta endorphin or enkephalin, is such as to cause inhibition of transmission in the nociceptive circuit synapses. This is achieved by blocking the release of the chemical transmitter (substance P) in the pain circuit.

Pathophysiology of MTrP [7, 13]

3 hypothesis are led, those are:

Energy Crisis Theory

This theory postulates an initial release of calcium [either from the sarcoplasmic reticulum or from the extra cellular fluid through injured sarcolemma. The ionic ca+ causes sustained sarcomere shortening and increased metabolism. The sustained shortening could compromise local circulatin, loss of oxygen and nutrient supply in the presence of an increased metabolic demand, thus the energy crisis. The lack of energy could compromise recovery of the calcium by the sarcoplasmic reticulum which would, at least temporarily, perpetuate the cycle. The more severe symptoms of chronic refractory TrPs and the onset of pathological changes may be caused by the development of such an energy crisis.

Muscle Spindle Concept

Active loci of TrPs to be located in the endplate zone and not in the taut band outside of the endplate zone. The potentials that we designate as spikes correspond to the spikes described in an

authoritative EMG text as arising in extrafusal muscle fibers at endplates. Brown and Varkey also attributed the SEA to potentials of the endplate zone and they attributed the positive-negative discharges [spikes] to postsynaptic muscle-fiber action potentials that were presynaptically activated by mechanical irritation. One other study, in addition to that of Hubbard and Berkoff, suggested that spikes arise from intrafusal muscle fibers. Those authors discussed why spikes are not ectopic discharges of motor axons but did not consider the possibility that spikes are the result of mechanically induced release of abnormal amounts of acetylcholine at the neuromuscular junction of an extrafusal fiber. Our experimental evidence also supports the origin of spikes in extrafusal muscle fibers. Muscle spindles may, at times, contribute to TrP phenomena, but it seems unlikely that muscle spindles are the primary site of the mechanism.

Motor Endplate Hypothesis

The motor endplate hypothesis identifies dysfunction in the region of extrafusal motor endplates as a major cause of myofascial TrPs. Hubbard and Berkoff first reported in 1993 that myofascial TrPs contain very minute loci that produce characteristic electrical activity..The loci are found among normal endplates. The localization of active loci in the endplate zone predominantly at the TrP has been confirmed experimentally.

Sympathetic changes associated with MTrP [13, 23]

The neurovascular bundle is oriented across the direction of the muscle fibers and contains nociceptor sensory nerve and autonomic sympathetic nerve that are closely associated with these blood vessel, this proximity of the structure to motor endplate at MTrP causes sympathetically mediated changes to take place.

Sympathetic efferent activity

The sensory afferent barrage produced by activated and sensitized MTrP nociceptor, on reaching spinal cord, activates sympathetic preganglionic neurons in the intermediolateral column present in the tissue separating the dorsal and ventral horns. This turn would cause noradrenergic postganglionic neuron in the sympathetic chain to become activated with the development of activity in sympathetic efferent & as a consequence,the release of noradrenaline (maintain the MTrP in an activated & sensitized state)

Pain Threshold [15]

Least stimulus intensity at which a subject perceive pain.

Pain tolerance threshold [15]

Greatest stimulus intensity causing pain that a subject is prepared to tolerate.

Ischaemic Compression Technique [7]

TrPs can be 'deactivated' by temporarily occluding their blood supply and causing a reactive hyperaemia (increase in blood supply), effectively flushing out the muscle of inflammatory exudate and pain metabolites, breaking down scar tissue, and reducing muscle tone. The muscle is nourished by the extra flow-through of blood, nerve endings are desensitised, and scar tissue is broken down so that the muscle fibres can move better. Ischaemic compression can be viewed as a sustained stretch to a specific point in the muscle, or as the most effective way possible to stretch because it gets right to the tight or restricted area in a muscle. It involves applying sustained pressure to the trigger point with sufficient force and for long enough to slow down the blood supply and force the tension out of the muscle. The muscle should be placed in a lengthened position so as to be more effective. The patient must be comfortable and relaxed & the compression gradually applied with the finger, thumb, and elbow. Such pressure should be relative to how much pain is being experienced & how much the patient can tolerate (ie, it will be painful but in most instances too much pain will cause tension in the muscle and negate the treatment).

The pressure is gradually applied, maintained, till discomfort ease by 50% perceived by patient and then gradually released. It can be held for as long as 90 seconds. Initiating pressure on to a trigger point must be done gradually in an effort to minimise increases in tone, so you can get closer to the core of the trigger point. It is important to reproduce the local thermal response for a better treatment result. Ischaemic compression can be used as a prophylactic or preventative measure in athletes with Latent trigger points. As the trigger point settles, there will be an accompanying decrease in referred pain and an improvement in other related issues such as weakness, muscle spasm, joint the trigger point itself should become less sensitive, and it will become harder to find a painful spot.

Ultrasound Therapy (UST) [17, 21, 24, 23]

It involves use of high frequency acoustic energy that is generated using the piezoelectric effect to produce thermal & non thermal effect in tissue.

Thermal effect

It is used to accelerate healing, extensibility of collagen is increased, and stretching of scar or adhesion is easier following ultrasound, for pain reduction. Mechanical or micro massage in which the longitudinal compression waves of the ultrasonic beams produces compression/ rarefaction of cells, and affect the movement of tissue fluid in interstitial spaces, it help reduce odema. Combined with thermal effect, it could help reduce pain.

Non thermal effect

Cavitation, Acoustic streaming, standing waves. Cavitation is oscillatory activity of highly compressible bodies within the tissue such as gas or vapour, which is potentially dangerous to the tissue as the collapse of bubble causes great local rise in temperature. Therefore it avoided by using low intensity (below 3W/cm2 and high frequency (1 or 3 MHz), and by moving the treatment head to prevent standing waves.

Treatment Intensity of 1-2 W/cm2 gives a desirable therapeutic result in reduction of pain [17]. Frequency depends on the amount of penetration required. Higher the frequency more superficial is the depth. Therefore for deeper structure 1MHz is commonly used. Continuous mode is selected when thermal effect is desired to treat general musculoskeletal disorder [21].

STUDY DESIGN AND METHODOLOGY

Myofascial trigger point: Diagnostic criteria for MTrP (simon 1999) [38]

- Presence of a palpable taut band in a skeletal muscle,
- Presence of a hypersensitive tender spot in the taut band.
- Local twitch response provoked by the snapping palpation of the taut band,
- Reproduction of the typical referred pain pattern of the MTrP due 2 compresion
- Spontaneous presence of the typical referred pain pattern and/or patient recognition of the referred pain as familiar.

If only the 1st four first criteria were satisfied, the MTrP was considered to be latent. If all of the aforementioned criteria were present, the MTrP was considered to be active [7, 14].

Types of trigger point [7, 13, 38]

Latent (passive) TPs are tender on palpation. They may be found in clinically normal patients and are associated with restricted movement (guarding) and weakness/fatigue of the affected muscles. Passive TPs can be activated easily by many factors, especially overstretching/overuse, and can then trigger clinical pain or dysfunction. The fitter the muscle, the more difficult it is to activate its passive TPs.

Active TPs are very tender on palpation and associated with existing pain or other dysfunction.

Satellite TPs are ones which develop activity when the muscle in which they are situated is in another muscle's zone.

Primary TPs are TPs in the muscle or group of muscle whose nociceptors activity is primarily responsible for the development of the MTrP pain.

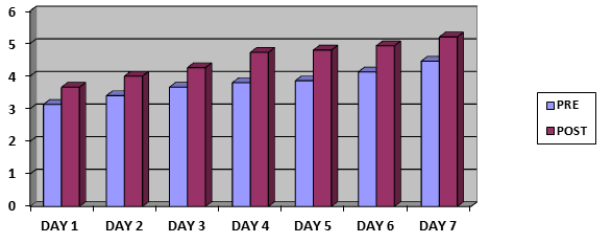
Secondary TPs seen in affected muscle's synergist or in their antagonist or both.

Symptoms [13]

- Pain, headache (upper trapezius TrP)
- Disturbance of motor function included muscle weakness, loss of coordination
- Disturbance of autonomic function included peripheral hypothermia, imbalance, dizziness, tinnitus
- Sleep disturbance

Physical signs

- Taut band
- Tender nodule: experimental data indicate many active loci in any one TrP
- Pain recognition
- Local twitch response
- Jump and shout reactions
- Peau d'orange cutaneous and subcutaneous thickening
- Restricted range of motion: increased muscle tension, muscle shortening, enthesopathic tenderness and TrP induced pain is liable to restrict ROM.



Location of TrP in upper trapezius: Mid way between C7spinous process & acromion [13].

Referral zone of Pain: Are up to the side of neck, base of skull, Side of the head to reach the temple and back of eye [13].

30 subjects were recruited on a conveniently random sampling basis who reported to Pandit Deendayal Upadhyay National institute for the persons with Physical Disabilities (Divyangjan), PDUNIPPD on OPD basis with the complaints of unilateral upper trapezius pain/ latent trigger point as per diagnostic criteria listed by TRAVELL AND SIMONS.

Inclusion criteria

- Age group: 20-30 yrs.
- Both males and females.
- FROM (Full range of motion of neck and shoulders).
- Pain which is <6 on VAS scoring.
- No history of high velocity neck or shoulder injury
- Mechanical neck pain.
- Subject with Latent trigger point for duration of up to 3 months.
- No treatment (medical or physical therapy) received in past 3 months.
- No history of chronic inflammatory, auto-immune and hereditary diseases.

Exclusion criteria

- Fibromyalgia.
- Whiplash injury.
- Cord compression.
- Instability of cervical spine.
- Vertebro-basiliar insufficiency.
- Permanent tattoo in the region of cervical and shoulder area.
- Fractures or surgeries of the neck and shoulder region.
- Known history of oral or injectable medications for more than 2 weeks.
- Duration of the present complaints for more than 3 months.
- Unwilling to participate in the study.
- Criteria other than the inclusion criteria.

MATERIAL USED

- Phyaction-787 stimulator- Mode choosen was galvanic current.

- Rubber electrode and a point/pen electrode.
- Velcro strap, Sponge, Plinth, Tap water, Electrode gel.
- Ultrasound machine (Combination therapy machine).

METHOD

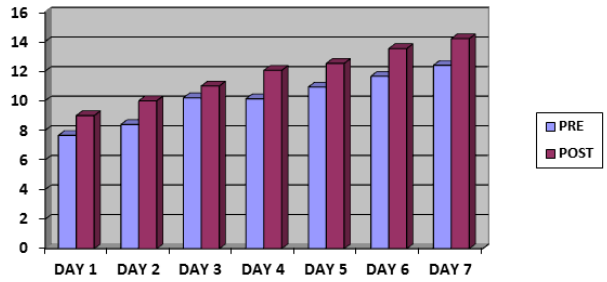
- 30 subjects, who had complaints of neck/trapezius pain for a duration of 3 months with demonstratable latent trigger points were randomly divided into 2 groups.
- Group A: 15 subjects (13 females and 2 males to be treated with ultrasound therapy).
- Group B: 15 subjects (13 females and 2 males to be treated with Ischaemic compression therapy).
- Each subject was assessed thoroughly by taking history, examining and performing special tests on cervical spine and shoulder to rule other condition. Trigger point on upper trapezius was identified as per diagnostic criteria by Travell and simons & marked by a marker.
- Subjects were explained in detail about the procedure of pain assessment and treatment plan. Subjects were instructed not to carry out any unaccustomed work like lifting heavy things, straining activities of upper limb etc for 7 days (duration of the study).
- In this study assessment of pain was done by use of direct (Galvanic) current considering the principle that the concentration of constant current over a small area of skin produces pain due to polar effect.It is the most suitable stimulus as it is simple, easily reproducible, accurate, shows consistent thresholds values, and is less subjective compared to VAS.
- Galvanic current from phyaction-787 stimulator was applied on the marked trigger point by a point electrode connected to the cathode and an indifferent pad connected to the anode was placed over lower lateral 1/3 of the arm. The resistance of the skin was reduced prior to application of current by wetting it (tap water) with wet gauze. The intensity of current was gradually increased. The subject was asked to report:When they start feeling the current sensation (TF) ie. Feel threshold. When the current sensation become painful (TP) ie. Pain threshold.When the painful stimulus becomes intolerable (TT)ie. Tolerance threshold.
- All the 3 readings were documented from the mill ampere reading. This was done before and after the treatment.
- : Subjects were treated with conventional ultrasound in relaxed sitting position, with intensity of 1.5 Watts/cm2 for 5 minutes on continuous mode with frequency of 1 MHz from combination therapy machine.
- : Subjects were asked to lie prone, with side to be treated stretched slightly by lateral flexion of cervical spine to the opposite side. The upper limb of the side to be treated was placed behind (on the back) and upper limb of the other side was placed under the forehead. Compression was given by therapist's thumb directly on the marked trigger point. Subjects were instructed to inform once the pain felt on the compression site reduces by 50% of the beginning of procedure and then the compression pressure was gradually increased .This procedure was carried over a period of 90 seconds[3].
- A gap of 2 minutes was given before assessing post treatment pain by galvanic current [3].
- Treatment plan was for 7 days.
- All the data was documented and analysed.

DATA ANALYSIS

The study was conducted to compare the effectiveness of ultrasound and ischaemic compression on upper trapezius latent trigger point, using direct current to assess thresholds (TF, TP, and TT). 30 subjects were randomly divided in 2 groups, Group A treated with ultrasound therapy and Group B with Ischaemic compression therapy.

Table1: Comparison of mean value of pre and post treatment with ultrasound on TF (mA).

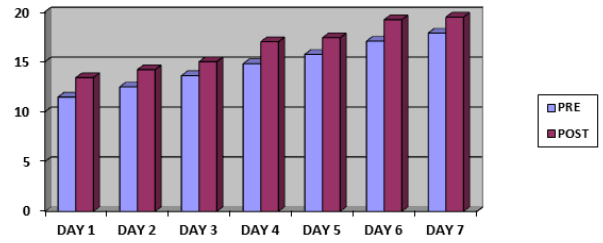
DAYS	1	2	3	4	5	6	7
PRE	3.13	3.4	3.66	3.8	3.86	4.13	4.46
POST	3.66	4	4.26	4.73	4.8	4.93	5.2



Graph1: Comparison of mean value of pre and post treatment with ultrasound on TF (mA).

Table2: Comparison of mean value of pre and post treatment with ultrasound on TP (mA).

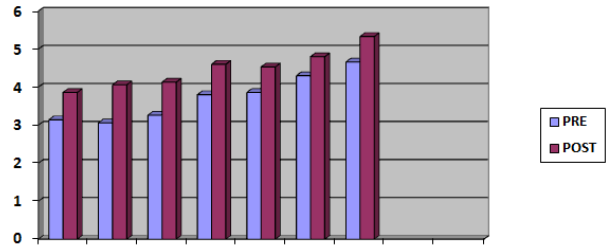
DAYS	1	2	3	4	5	6	7
PRE	7.66	8.4	10.2	10.13	10.93	11.66	12.4
POST	9	10	11	12.06	12.53	13.53	14.2



Graph2: Comparison of mean value of pre and post treatment with ultrasound on TP (mA).

Table3: Comparison of mean value of pre and post treatment with ultrasound on TT (mA).

DAYS	1	2	3	4	5	6	7
PRE	11.46	12.48	13.6	14.8	15.73	17.06	17.86
POST	13.4	14.2	15	17	17.4	19.2	19.46

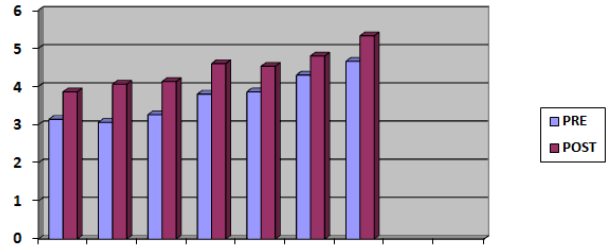


Graph3: Comparison of mean value of pre and post treatment with ultrasound on TT (mA).

Table and Graph 1, 2, 3 show pre and post mean values of TF, TP and TT of each day from day 1 to day 7 for subjects treated with ultrasound therapy shows that there is an immediate effect on TF, TP, TT on each day following treatment with Ultrasound. Also the mean values of pretreatment on the next day are reduced than the post treatment mean value of the previous day. This indicate that the effect of ultrasound treatment is not maintained on the next day and the threshold is reduced but is more than the threshold of the pretreatment mean value of previous day.

Table4: Comparison of mean value of pre and post treatment wi th Ischaemic compression therapy on TF (mA).

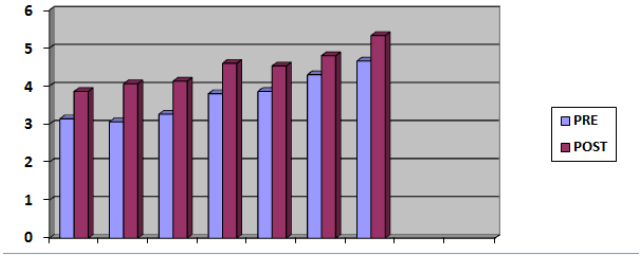
DAYS	1	2	3	4	5	6	7
PRE	3.14	3.06	3.26	3.8	3.86	4.3	4.66
POST	3.86	4.06	4.13	4.6	4.53	4.8	5.33



Graph 4: Comparison of mean value of pre and post treatment with Ischaemic compression therapy on TF (mA).

Table5: Comparison of mean value of pre and post treatment with Ischaemic compression therapy on TP (mA).

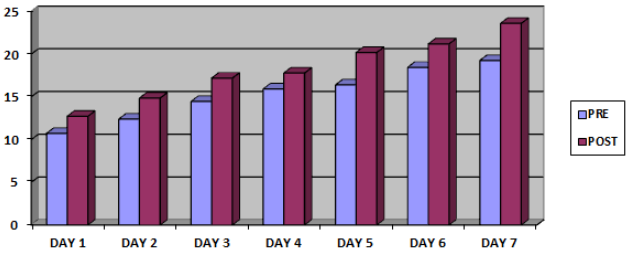
DAYS	1	2	3	4	5	6	7
PRE	7.53	7.93	9.33	10.93	12.6	13.6	14
POST	8.73	10.33	11.33	12.4	14.53	14.3	15.86



Graph5: Comparison of mean value of pre and post treatment with Ischaemic compression therapy on TP (mA).

Table6: Comparison of mean value of pre and post treatment with Ischaemic compression therapy on TT (mA).

DAYS	1	2	3	4	5	6	7
PRE	10.73	12.4	14.46	15.93	16.4	18.46	19.26
POST	12.73	14.86	17.2	17.8	20.2	21.2	23.6



Graph6: Comparison of mean value of pre and post treatment with Ischaemic compression therapy on TT (mA).

Similarly, table and graph 4, 5, 6 also show an immediate effect on TF, TP and TT on each day following ischaemic compression therapy. Also the mean values of pretreatment on the next day are reduced than the post treatment mean value of the previous day. This indicate that the effect of ischaemic compression treatment is also not maintained on the next day and the threshold is reduced but is more than the threshold of the pretreatment mean value of previous day.

Table7: Pre (day 1) and post (day 7) treatment statistical analysis of TF between Ultrasound therapy and Ischaemic compression therapy.

	MEAN (D1)	MEAN (D7)	MD (D1-D7)	SD	t- VALUE	P VALUE
UST	3.13	5.2	2.066	1.1	7.3	S at 0.001
ICT	3.13	5.33	2.2	1.146	7.58	S at 0.001

Table 7 shows mean value, mean difference, standard deviation, t- value and probability of day 1 and day 7 of TF for Ultasound

therapy and Ischaemic compression therapy, Using paired t-test, the result is significant at P<0.001 for both ultrasound and ischaemic compression therapy independently suggesting that there is increase in TF in subjects treated with ultrasound as well as ischaemic compression.

Table8: Pre (day 1) and post (day 7) treatment statistical analysis of TP between ultrasound and ischaemic compression therapy.

	MEAN (D1)	MEAN (D7)	MD (D1-D7)	SD	t-VALUE	P VALUE
UST	7.66	14.2	6.53	2.64	8.91	S at 0.001
ICT	7.53	15.86	8.26	3.4	9.17	S at 0.001

Table 8 shows mean value, mean difference, standard deviation, t- value and probability of day 1 and day 7 of TP for ultrasound and ischaemic compression therapy.

Using paired t-test, the result is significant at P<0.001 for ultrasound and ischaemic compression independently suggesting that there is increase in TP in those treated with ultrasound as well as ischaemic compression. This infers that there is decrease in pain sensitivity of latent trigger point.

Table9: Pre (day 1) and post (day 7) treatment statistical analysis of TT between ultrasound therapy and Ischaemic compression therapy.

	MEAN (D1)	MEAN (D7)	MD (D1-D7)	SD	t-VALUE	P VALUE
UST	11.46	19.46	8.06	4.07	7.098	S at 0.001
ICT	10.73	23	12.26	4.30	11.046	S at 0.001

Table 9 shows mean value, mean difference, standard deviation,t- value and probability of day 1 and day 7 of TT for ultrasound and ischaemic compression.

Using paired t-test on the data, the result are significant at P<0.001 for both ultrasound and ischaemic compression independently suggesting that there is increase in TT in subjects treated with ultrasound as well as ischaemic compression.

Table10: Comparison of efficacy of ultrasound and ischaemic compression therapy on TF.

UST	ICT	MEAN DIFF	t-VALUE	P VALUE
2.066	2.2	0.134	0.292	NS at 0.001

Table11: Comparison of efficacy of ultrasound and ischaemic compression therapy on TP.

UST	ICT	MEAN DIFF	t-VALUE	P VALUE
6.53	8.26	1.73	0.494	NS at 0.001

Table12: Comparison of efficacy of ultrasound and ischaemic compression therapy on TT.

UST	ICT	MEAN DIFF	t-VALUE	P VALUE
8.06	12.26	4.20	0.656	NS at 0.001

Table 10, 11,12 show mean difference ,standard deviation, t-value of D1 and D7 for comparing effect between ultrasound and ischaemic compression therapy on TF, TP, TT.

Using paired t-test on the data, the results are not significant at P<0.001 indicating that there is no difference in TF, TP, TT for subject treated with ultrasound and ischaemic compression therapy. The above analysis of data indicates slight increase in TF, TP and TT in subjects treated with ischaemic compression than that with ultrasound but is statistically insignificant.

RESULT

There is an immediate effect on TF, TP and TT on each day following treatment with ultrasound therapy and Ischaemic compression therapy. Using paired t-test, pre and post treatment for Group A and Group B the results are statistically significant at P<0.001. However when compared for the difference between ultrasonic therapy and Ischaemic compression therapy using paired t-test on the data, the results are not significant at P<0.001 indicating that there is no statistical significance in TF,TP and TT between ultrasound and ischaemic compression therapy.

CONCLUSION

Both ultrasound and ischaemic compression are equally effective in treating trigger point. Both groups showed increase in TF, TP, and TT and thus subsequently there is reduction in pain sensitivity in trigger point. However Ischeamic compression may be a preferred therapy for myofascial trigger point in a physcial therapy setup as it is easily avaliable, accesible, cost effective, non-dependent on any machines.

DISCUSSION

Study demonstrated for the first time on lateral gastrocnemius muscle that a single bout of static myofascial release is effective in reducing MTrP sensitivity. The mechanism inducing the pain relief may be analogous to manual ischaemic compression. Previous research found that the sustained pressure applied in related techniques leads to reactive hyperaemia [42], which, in turn, causes a release of muscle fibre tension. Furthermore, increased blood flow accelerates removal of biochemicals such as bradykinin, CGRP, IL-6, IL-8 or TNF-α, which have been shown to be accumulated in myofascial trigger points [43]. Another

hypothesis potentially explaining the analgesic treatment effect relates to neurological rather than biochemical mechanisms. It has been suggested that tactile stimulation of a painful area leads to a pre-synaptic inhibition of slow, pain-transmitting nerve fibres [44].

Kostopoulos et al. [45] compared efficacy of ischemic compression, passive stretching, and the combination of ischemic compression and passive stretching for the first time and reported that the combination was significantly more effective for pain reliefs than the others. Lake [46] evaluated the efficacy of ischemic compression on 13 patients with 40 myofascial trigger points and reported that ischemic compression was significantly efficient for treatment in comparison with control group, but did not define the optimal level of ischemic compression.

Effect of physical therapy modalities (hot packs, active exercise, ischaemic compression, TENs, stretch spray, IFT, myofascial release) on MTrP in upper trapezius. Results state that Ischemic compression therapy using either low pressure (pain threshold) and a long duration (90s) or high pressure (the average of pain threshold and pain tolerance) and short duration (30s) for immediate pain relief and MTrP sensitivity suppression. Others were effective only for pain relief [39].

One of the manual techniques used to treat trigger points is ischemic compression. Based on the evidence, the taut band of trigger points and nodules is due to a shortage of sarcomeres in muscle fibers that produce contraction nodules and disks [47]. Presumably, the manual pressure applied to these contraction knobs reduces the height and thus increases the length of the sarcomeres in the involved muscle [48]. J. Edwards, in his study explained the simple posture corrections that make significant differences in the outcome of physical therapy. It is important to evaluate common faulty sitting, standing, and sleeping postures, work postures as this Postural Habits Perpetuate myofascial Trigger Point Pain. This may be one of the cause why ultrasound effect was not maintained next day as the subject were not given for any postural and ergonomic advice [36].

The result of Kannan P study is consistent with the present study. He studied the effect of therapeutic ultrasound, laser, and ischemic compression in reducing pain and improving the cervical range of motion in patients with myofascial trigger points [49]. Results showed beneficial effects of ultrasound therapy, low-power laser therapy and ischemic compression in tissue healing and also play an important therapeutic role in pain modulation by **reducing myofascial trigger point sensitivity.** Ischemic compression is one of the common manual therapy techniques, but this treatment is somewhat painful. One of the problems is the pain created by this technique [50].

Thus it is evident that the studied therapeutic modalities may have a similar, but not identical mechanism of action at neuromuscular level. Indeed In our observation ultra sound and ischemic compression technique requires additional training and experience for the treating therapist. It requires good communication and concentration for both patient and therapist. Whereas ICT has the advantage over these factors which makes it a handy tool in the management of MTrPs.

There were few limitations in our study which includes lack of a control group, limited study sample and short term follow up of therapeutic benefits. This study could be repeated with an additional group undergoing placebo therapy to rule out the fact that pain relief could not have been due to time factor.

LIMITATIONS

The limitations of the study comprised a low sample size, no long-term follow up of the patients, and the absence of the control group. Future studies could compare the effectiveness of ischemic compression, ultrasound therapy and laser therapy with other advanced techniques.

We only examined the intervention effects immediately after treatment. It is possible that the soft tissue adapts after a latency period, which cannot be covered without further measurements. Future studies might hence include a longer time frame. Finally, in our study, we focused on the feel, pain and tolerance threshold only, which is a limitation. It would be interesting to examine additional outcome parameters in future studies. The assessment of subjective activity-related pain (e.g. visual analogue scale), viscoelastic tissue properties (stiffness and elasticity), or imaging characteristics (diagnostic ultrasound, elastography) could provide valuable information helping to explain the treatment effects.

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