Effect of Eccentric Exercise of Quadriceps on Spasticity in Post Ischemic Stroke Patients

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ABSTRACT

Background: Stroke patients encounter spasticity and marked motor impairment.
Objective: The primary purpose of this research is to discover the effectiveness of eccentric resistance training in reducing spasticity of quadriceps muscles of paretic limb and thereby reduce disability after stroke.
Methods: A total of 30 patients of adult post ischemic stroke participated in the study. They were given eccentric resistance training for a period of 6 weeks with initial resistance 20% of their respective 1RM. A 10% increment of resistance was done at the end of every 2 weeks by isokinetic dynamometer. Resistive Positional peak torque (RPT), Average Peak Torque (APT), Modified Asworth Score (MAS) and Modified Rankin Score (MRS) was assessed before and after 6weeks of interventions.
Results: In response to eccentric resistance training all the patients showed significant reduction in MAS score, after 6 weeks of interventions. There was significant difference in resistive peak torque and average peak torque (dependent $t \leq 0.05$). Similarly Modified Rankin Score and Modified Asworth score showed significant improvement ($p \leq 0.05$). Overall improvement in motor function was noted in all patients after the completion of intervention.
Conclusion-: Eccentric resistance training can be useful in reducing spasticity of quadriceps in ischemic stroke patients. It is quite safe and can be added to post stroke intervention protocol to improve gait in persons with ischemic stroke.

Keywords: *Persons with Ischemic Stroke, Eccentric Training, Resistive Peak Torque, Average Peak Torque.*

INTRODUCTION

Stroke is described as "Rapidly developed clinical signs of focal or global disturbances of cerebral function lasting more than 24 hours or leading to death with no apparent cause other than vascular origin" by World Health Organisation in 1970.[1] It is of 2 types, i.e. ischemic and haemorrhagic. Ischemic strokes account for approximately 62% of all strokes, followed by Intra cranial haemorrhage (28%) and Subarachnoid haemorrhage (10%).[2-4] The annual incidence rate in India is 46 per 100000 in young population (age 18-49). According to JOS journal (journal if stroke) the cumulative incidence of stroke is 105-152 per 100000 per year. The crude

prevalence of ischemic stroke is 44.29-559/ 100000 indifferent parts of the country (Feigin et al., 2021). Stroke is the third greatest cause of premature death and disability, as measured by Disability Adjusted Life Years (DALY).[5] and the second leading cause of mortality worldwide, according to the 2010 Global Burden of Disease Study.[6]

Stroke leads to abnormally increased muscle tone and stiffness due to hypertonia, atrophy and paresis (Joongsan wang et al, 2016).[4] Spasticity is defined as "an imbalance of sensory-motor system that demonstrates an increased response to a velocity dependant stretch of muscle tendon resulting from hyper excitable muscle stretch reflexes".[7] More recently spasticity is defined as "disordered sensory-motor control, resulting from an upper motor neuron lesion (UMN), presenting as an intermittent or sustained involuntary activation of muscle".[8] Amidst the diverse complication of stroke spasticity is the most common. The prevalence ranges from 30%-80%. The Incidence of spasticity among stroke patient is 27 at 1 month 28% at 3-month, 23%-43% at 6 month and 34% at 18 months. The onset of spasticity is variable among stroke patient but studies suggest that spasticity develops and peaks 1-3 months after stroke.[9] Although neural component peaks at 3 months after stroke but musculoskeletal component peaks very slowly over time contributing to spasticity at 6months post stroke. In Upper limb and lower limb flexor and extensor synergy predominates respectively.[9]

Pathophysiology of spasticity: The stretch response in healthy person is mediated by stimulatory connection between I a afferent fibres and α- motor neurons of the same muscle. Muscular spindles are excited by passive muscular stretching, which causes transmission of information from Ia fibres to α-motoneurons via monosynaptic as well as oligosynaptic pathways.

With respect to any damage from acute events like stroke, the delay between the emergence of spasticity and neurological insults suggests some sort of plastic changes occurring in central nervous system (CNS) rather than simple release phenomena.

In the CNS, Denervation hypersensitivity of muscle membrane receptors to neuromuscular transmitter has been firmly established.[10] This may be resulted by either the genesis of novel receptors or by morphogenic alteration of denervated receptors. This denervation super-sensitivity could be embroiled in the escalated responsiveness of α-motor neuron deprived of their regular descending excitation from the corticospinal

pathways.[11] Furthermore, α-motor neurons after an UMN injury are acknowledged to release growth factors provincially.[11] These events foster local burgeoning from neighbouring interneurons, thus facilitating generation of new abnormal synapse between these interneurons and somatic membrane of the deprived motor neuron. This leads to branching of new inter-neuronal ending onto the membrane of α-motor neurons and inhabitation of the vacant space left by missing descending fibres.[12], resulting in the creation of new abnormal reflex pathways.[13]

Normally, muscle tone is critically balanced by the prohibitive drives of Cortico Spinal Tract (CST) and dorsal reticulo spinal tract and facilitatory drive from Rubro spinal tract and to some extent Vestibulo spinal tract.[14] When the brain and spinal cord are unable to communicate due to injury of the upper motor neurones, the spinal reflexes become disinhibited.[9]

Brain stem descending pathways including reticulospinal, vestibulospinal, tectospinal and rubrospinal tract, may become more engaged in executing motor commands after CST disruption. However, the excitatory inputs this pathway provides to spinal motor neuron are less likely precise than those of CST potentially resulting in increased muscle activity.[15]

Isolated involvement of CST is inadequate to produce spasticity.[15,16] Cortical damage causes spasticity due to associated involvement of Cortico–Reticular fibres, which link the premotor cortex to medullary reticular formation, from where the dorsal reticulospinal tract originates.

Eccentric Muscle Contraction: "An eccentric muscle contraction occurs when a force applied to muscle exceeds the momentary force produced by the muscle itself, resulting in the forced lengthening of muscle-tendon system while contracting".[17] Eccentric contraction possesses several unique features like greater force production, less oxygen consumption and less motor unit activation requirement. Due to these unique features eccentric exercises are now being given more importance in rehabilitation.

A study conducted by Andrea Manca *et al.* In 2020 on "Effect of eccentric strength training on elbow flexor spasticity and muscle weakness in people with multiple sclerosis: A proof-of-concept single system case series" showed following intervention RPT decreased by at least 2 SD in all participants and with significant reduction at group level, person with the disease reported reduction in perceived level spasticity.[18]

Another study on "eccentric exercise to enhance neuromuscular control" (Lindsey

K.Leply and Aadam S. Leply) suggests that hamstring torque angle has been shifted to more working length following eccentric contraction.[19]

Trans cranial magnetic stimulating study suggests that eccentric contraction utilise unique neural mechanism compared to other muscle contraction i.e. Eccentric contraction employs pronounced excitability of motor cortex compared to concentric contraction that uses spinal level reflexive excitability.

Greater cortical excitability exercised during eccentric contraction acts as a redemptory mechanism to account for inhibition at spinal level.

Blood oxygen dependant signal- a functional MRI correlating neural activity has shown that eccentric exercise increases neural activity of cerebellum. Thus, it improves motor function by producing controlled motor contraction.[19]

Aim of The Study: This experimentation aims to determine whether eccentric resistance training can be effective in reducing spasticity of quadriceps muscles of paretic limb and reduce disability of post ischemic stroke patient.

METHODOLOGY:

Study Design:

Progressive observational study with pre and post analysis.

Study set up: Department of physiotherapy, SVNIRTAR, OLATPUR.

Subjects: 30 medically diagnosed sub-acute and chronic ischemic stroke patients.

Inclusion Criteria:

1. Sub-acute, early chronic stage stroke up to 3 years from onset.

2. Modified Rankin Scale score>/=3

3. Spasticity of the lower limb with Modified Asworth scale (MAS) score less than or equal to 3 of quadriceps.

4. Both genders are considered.

5. Age 35 y -65 y

6. Medically stable (vitals)

7. MCA, ACA territory involvement.

8. *Lower extremity Brunstrom stage 3 and4*

Exclusion Criteria:

1. Persons with medical conditions that contraindicates participation in resistance training exercises

2. Fixed contracture / profound atrophy of the affected limb

3. Previous or ongoing use of spasticity reducing pharmacological and or surgical intervention in the past 6 months

4. ongoing casting for spasticity of the limb of interest

5. Clinically relevant cognitive deficits (MMSE score>/=24)

6. Impaired sensation and proprioception.

7. Joint pain of hemi paretic limb.

8. ≥1 episode of stroke, seizure disorder

Variables:

1. Independent variable- eccentric exercise of quadriceps at a rate of 20 % of participant specific 1RM given by isokinetic dynamometer.

2. Dependant variable- Resistive peak torque, Average peak torque, MAS, modified Rankin score.

Instrumentation:

HUMAC isokinetic dynamometer

Data Collection:

Entirely 113 patients were screened. Initial data was collected by carrying out multiple pre-tests based on inclusion and exclusion criteria. 30 subjects were chosen. Informed consents were collected from all participant.

Method:

Dynamometric tests were performed using an isokinetic device (HUMAC). Interventions were carried out in the affected limb only. Participants were made to sit on the dynamometer seat, positioned according to the joint and muscle group under test, and the body parts that needed fixation were stabilized by custom straps and harnesses. The axis of rotation of the dynamometer shaft was aligned with the centre of rotation of the knee joint for testing knee joint.

The equation used for calculation of 1RM was-1RM =21.38+(0.24×peak torque) +(0.18×body weight).[20]

Base Line Assessments:

Severity of the spasticity was assessed by the clinician-reported MAS.

MRS of participants was noted as a self-reported disability score resulting from spasticity. The variations in patient-reported perception of Delayed onset muscle soreness (DOMS) in the trained muscles, was measured using numerical rating scale (NRS), with a score from 0 to 10 with higher score indicating higher DOMS.

Intervention:

Before starting intervention, a 10 minute of warming up exercise was performed for all individuals. It included gentle passive stretching of quadriceps and plantar flexors, gluteus maximus bridges, good morning (hams stretching self), calf raise etc. The intervention was carried out on the same isokinetic device. Subjects underwent an eccentric training program for quadriceps after positioning knee joint axis, aligned with the lever arm axis of rotation of dynamometer and the patient was asked to break the resistance offered by machine by actively flexing the knee from 0º to 80˚. The machine was set at 60˚/s angular velocity.

After calculating 1RM the work load was set at 20% of 1RM and each week the work load was increased at a rate of 10%. The angular torque per time was taken at the end of 6 week at predetermined angular velocity. DOMS following the interventions was noted using NRS scale.

Post intervention Assessments:

It was done within 1 week from the end of intervention.

Statistical Analysis

Statistical analysis was done using SPSS software (version 25.0). Paired t- test was performed to compare pre and post of RPT and APT. For MAS and MRS, Wilcoxon signed rank test was performed. The alpha critical level of 0.05 was considered statistically significant results.

RESULTS

All individuals i.e.6 women with mean age (52.66± 8.47 year) and 24 men with mean age (47.25±10.39 year) had definite diagnosis of ischemic stroke.

The overall result of this study after 6 weeks of treatment consisting of 3 days per week showed significant decrease in spasticity of quadriceps and considerable improvement in spasticity induced disability. The outcome measures which were intended to measure spasticity i.e. resistive peak torque and Modified Asworth score showed significant reduction following intervention. Average peak torque which was intended to measure strength gain showed improvement in strength and there was also reduction in modified rankin score which was selected to measure spasticity induced disability at the conclusion of intervention.

Resistive Peak Torque of Quadriceps (Affected Side):

Paired t- test suggests there is significant reduction in resistive peak torque from pre to post. Figure 1 showed remarkable reduction of mean of resistive peak torque from pre to post.

t (29) =8.012, p=0.000

Average Peak Torque of Quadriceps (Affected Side):

Paired t- test suggests there is significant improvement in Average peak torque from pre to post. Figure 2 showed noteworthy increase of mean of Average peak torque from pre to post

t (29) =-13.579, p=0.000

Modified Asworth Score of Quadriceps (Affected Side):

Wilcoxon signed rank test for Modified Asworth score suggests there was significant improvement from pre to post with reduction in MAS score. Figure 3 showed remarkable reduction of mean of MAS from pre to post

Z= -4.976, p=0.000

Table 1: Statistical analysis result for the outcome measures

OUTCOME MESURE	STATISTICAL TEST	RESULT
Resistive peak torque	Paired *t*- test	$t(29)= 8.012, p=0.000$
Average Peak torque	Paired *t*- test	$t(29)= -13.579, p=0.000$
MAS	Wilcoxon signed rank test	$Z= -4.976, p=0.000$
Modified Rankin Score	Wilcoxon signed rank test	$Z=-4.736, p=0.000$

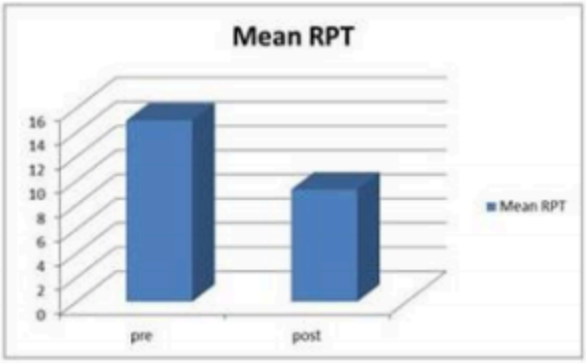

Figure 1: Showes there is significant reduction in Mean Resistive Peak Torque from pre to post.

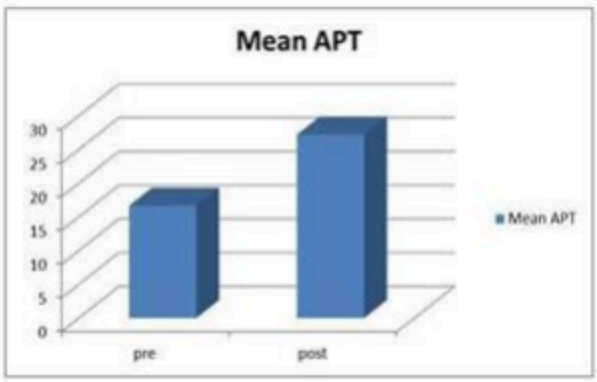

Figure 2: Shows there is significant increase in Mean Avarage Peak Torque from pre to post.

Modified Rankin Score:

Wilcoxon signed rank test for Modified Rankin Score suggests there was significant improvement from pre to post with reduction in MRS score. Figure 4 showed appreciable reduction of mean of MRS from pre to post.

Z=-4.736, p=0.000

DISCUSSION

Persons with Stroke experience substantial detriment in their daily life due to spasticity. Reduction in spasticity induced disability is a major functional goal of the present study. We investigated an eccentric training protocol that was carried out in HUMAC isokinetic dynamometer and the

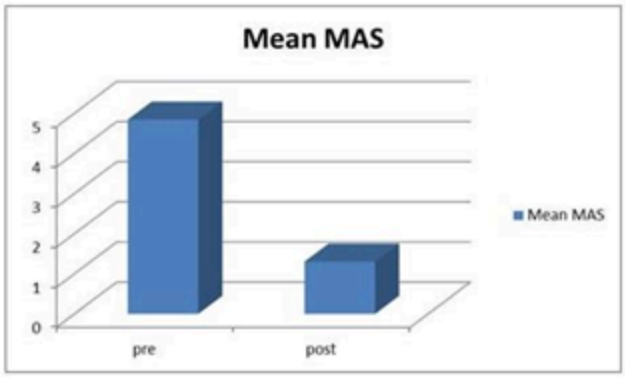

Figure 3: Shows there is significant reduction in Mean Modified Asworth Score from pre to post.

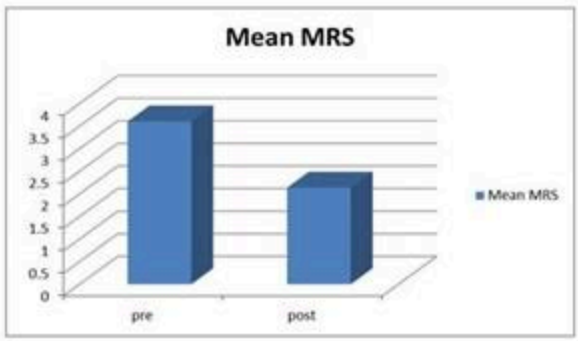

Figure 4: Shows there is significant reduction in Mean Modified Rankin Score from pre to post.

results showed marked reduction in spasticity.

Resistive Peak Torque:

This study showed that eccentric exercise can effectively and safely reduce spasticity-induced resistance to passive motion in post ischemic stroke patients. Furthermore, as assessed objectively by isokinetic dynamometry, the eccentric training leads to decreases in the muscles' resistance to passive motion. Lastly, just as predicted, the intervention also markedly increased the spastic muscles' maximal strength. The results are in accordance with the previous studies where effect of eccentric training was studied on elbow flexor spasticity that showed "positive result of reduction of mechanically computed spasticity through isokinetic dynamometer and self-reported spasticity".[18]

The reduction in spasticity might be due to increased cortical activation along with increased blood flow to the devitalized area due to ischemia that resulted in improved supra spinal control over the lower spinal centres reducing spasticity. This is in accordance with the study where it has been found that eccentric contraction is capable of improving blood circulation to both cortex and cerebellum which improves control of higher centres along with imparting supra-spinal inhibition.[19]

Eccentric training affects the overall length range and the length-tension relationship of

muscles via this lengthening action. It also sustains shift in the angle of peak torque and the curve width, so that the range over which torque can be sustained above 50% of maximum (curve width) is significantly wider than that obtained with the conventionally employed concentric actions.[21,22] As eccentric contraction results in sarcomere lengthening and collagen extensibility it targets the non-neural components of spasticity that is characterised by soft tissue stiffness and reduced viscoelasticity of muscles.[23]

Modified Asworth Score:

The MAS testing for quadriceps revealed a remarkable change in the clinician-rated impression of spasticity. Statistical analysis shows there is significant reduction MAS score.

There is very less evidence where MAS has been found to be having significant changes following eccentric exercise. Since MAS has significant degree of heterogeneity among research that has made the responsiveness of MAS scores questionable; the findings typically range from poor to marked responsiveness to change after rehabilitation.[18] But Resistive peak torque and average peak torque in this study showed improvement in statistical analysis along with MAS which provides supportive evidence in for of reduction of spasticity despite of less specificity of MAS.

Average Peak Torque of Quadriceps:

APT measured by dynamometer to find out any strength gain in terms of increase in peak torque showed significant improvement. The possible reasons for this result is eccentric exercise improves strength by higher force production, increased muscle cross section area in the exercising muscle so there is expected strength gain in the quadriceps muscle.

A study conducted on "Effect of eccentric strength training on elbow flexor spasticity and muscle weakness in people with multiple sclerosis: proof of concept single system case series" by Andrea Manca, Gianlinca Martinez, Elena Ailleo, Lucia Ventura, Franca Deriu involved 6 patients showed significant improvement in average peak torque in all the patients suggesting similar finding to our study.[18]

Another study suggests eccentric exercise produce Sarcomerogenesis in animal model there by improve muscle strength.[24]

Modified Rankin Score:

There was significant change in the MRS in statistical analysis. Till now no study is available where modified ranking score is so far used as an outcome measure in an eccentric or concentric training programme to see its effect on disability. It is a

subjective scale and there is high variability in rankin score.

This study also proved that eccentric resistance training when applied at a submaximal rate can be safe and feasible without DOMS. Though the patients reported DOMS after 2-3 sessions of intervention but it got subsided within 2 weeks without any worsening which was reflected in NPRS score.

Anatomically and physiologically, the lower leg muscles that were studied in this investigation were extremely capable of recovering and highly functional. Considering our findings, it's challenging to say whether the advancement shown resulted from a modification of motor control brought about by a cerebral plasticity or that was muscular gain i.e. merely peripheral. It's possible that the adaptation had a mixed origin and was hence neuromuscular. When engaging in muscular training, there are two categories of modifications: An extremely significant neurologic adjustment in the initial 1 month that results in an increase in the number and size of motor units, enabling improved inter and intra-muscle coordination followed by a structural adaptation that comes as hypertrophy. Since our protocol consisted of 6 weeks protocol these changes observed is expected to be mainly due to neuromuscular adaptation.[25]

CONCLUSION

It is concluded from the above study that both eccentric exercises reduce spasticity and is safe to incorporate in stroke patient's rehabilitation protocol. However further research in this field is required to establish more concrete findings regarding the use of eccentric and concentric exercise in management of spasticity.

Study Limitations:

There was no control group to limit the effect of extraneous variable. There is no trace of the retention of the training effect.

Future Suggestions:

1. Control group can be taken to limit the effect of extraneous factors.

2. Follow up can be taken to find out the carry over effect of the intervention.

Conflict of Interest: No conflict of interest is there.

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