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Original Research

The Effects of Additional Radial Shock Wave Therapy on Spasticity of Upper Extremity Muscle

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ABSTRACT

Background: Spasticity is one of the most common problems and greatly interferes with the functional capacity of chronic stroke patients. The most commonly used treatments are infrared therapy and stretching exercises, but they have not reduced spasticity effectively. The addition of Radial Shock Wave Therapy (RSWT) was expected to be more effective in reducing spasticity in chronic stroke patients.

Aim: The purpose of this study is to prove that the addition of RSWT is more effective reducing spasticity of chronic stroke patients.

Material and methods: This study was a simple randomized controlled pre- and post-experimental design. The total sample of 30 chronic spastic stroke patients with the Modified Asworth Scale 2-3 was divided into 2 groups: the study group and the control group. RSWT was provided once a week to the muscle belly of the flexor wist muscle on the ventral aspect of the forearm, the intrinsic muscle of the hand, and flexor digitorum tendon. They were added to infrared therapy and stretching exercises in the upper extremities which are provided three times a week for six weeks consecutively. The level of spasticity was measured by the Tardieu Scale, which measures quality and angle of resistance at the beginning and end of the study.

Results: A significantly greater reduction was obtained ($p<0.05$) from the level of spasticity measured by the Tardieu Scale, both on the quality and angle of resistance, in the study group.

Conclusion: The addition of RSWT has been shown to have a greater reduction in spasticity in upper extremity muscle in chronic stroke patients.

Keywords: *Chronic stroke, Infrared, Muscle, Radial shock wave therapy, Spasticity, Stretching, Tardieu scale.*

INTRODUCTION

The World Health Organization defined stroke as rapidly developing clinical signs of focal (or global) disturbance of cerebral function, lasting more than 24 hours or leading to death with no apparent cause other than vascular origin. Although still widely used, the World Health Organization definition relies heavily on clinical symptoms and is now considered outdated by the American Heart Association and American Stroke Association due to significant advances in the nature, timing, clinical recognition of stroke and its mimics, and imaging findings that require an updated definition.[1]

Based on the 2013 National Riset Kesehatan Dasar (Ritkesdas), the prevalence of stroke in Indonesia based on diagnosis by health workers was 7 ‰; the highest was North Sulawesi (10.8 ‰), followed by DI Yogyakarta (10.3 ‰), Bangka Belitung, and DKI Jakarta, each was 9.7 ‰. Whereas, based on diagnosis by symptoms, the prevalence of stroke in Indonesia was 12.1 ‰ , the highest was in South Sulawesi (17.9 ‰), DI Yogyakarta (16.9 ‰), Central Sulawesi (16.6 ‰), and East Java (16 ‰).[2]

Lance defined spasticity as a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of UMN syndrome.[3]

The theory of spasticity suggests that the emergence of spasticity is the result of an imbalance between the excitatory and inhibitory systems in the midbrain and brainstem reticularis formation. In stroke patients with lesions in the brainstem or cerebral cortex (primary, secondary, and additional motor areas), there is an increase in muscle tone due to the following reasons:

1. Damage to the corticospinal pathway,
2. Termination of impulses superior to the reticular inhibitor area in the lower brainstem, or
3. Reinforcement of excitability from areas of reticular facilitation or excitation in the midbrain and brainstem.[4,5]

The situations above will cause an overflow of facilitation to the spinal cord, which is spread through the reticulospinal, vestibulospinal, and other pathways and results in a change in the balance between the motor neuron systems α and γ (α and ɤ coactivation). Failure or loss of the central inhibitory effect that normally suppresses or reduces the spinal stretch reflex (spinal stretch reflex) will be followed by excessive muscle contraction (motor neuron α hyperexcitability) and increased resistance when stretched. α motor neurons and muscles are the common pathways for expressing motor function, including spasticity. Impulses on the α motor neuron arriving at the terminal end open Ca2+ ion channels and cause the release of acetylcholine from the synaptic vesicles into the synaptic cleft. Acetylcholine diffuses to nicotinic acetylcholine receptors on the motor end plate. Acetylcholine binding to receptors increases membrane permeability to Na+ and K+ ions, causes Na+ ions to enter, produces muscle membrane depolarization, and triggers action potentials in the muscles, resulting in extrafusal muscle contractions. Continuing loss of inhibition causes extrafusal muscle contraction to become continous. This is the main reason for the occurrence of spasticity.[4,5]

The Tardieu scale (TS) and the Modified Tardieu scale (MTS) measure the quality and angle of spasticity. First introduced by Tardieu et al. in 1954 and then developed by Held and Pierrot Deseilligny in 1999. Spasticity is measured at 3 different

speeds: at the slowest possible speed (V1), the speed at which the extremity falls due to gravity (V2), and as soon as possible (V3). The angle at which muscle resistance occurs is measured with a goniometer. R1 is the angle at which resistance occurs when the extremity is moved as fast as possible (V1), and R2 is the angle at which the greatest passive range of motion can be achieved when the extremity is moved as slowly as possible (V3). The difference between R1 and R2 shows the dynamic tone component of the muscle.[6]

The following scale used to evaluate the quality of muscle resistance was:
0: there is no resistance during passive movement.
1: slight resistance throughout passive movement, absent clear clasp.
2: real clamp at a certain angle in a passive movement, followed by resistance release.
3: fatigable clonus (< 10 seconds) appearing at an angle certain.
4: infatigable clonus (> 10 seconds) appearing at an angle certain.[6]

By moving the extremity at different speeds, the response to stretching can be more easily measured because the stretch reflex response differs with speed. The difference in the speed of stretching shows the velocity dependence of the spasticity. Therefore, the Tardieu scale (TS) and the Modified Tardieu scale (MTS) are said to measure spasticity, which better supports the definition of spasticity according to Lance.[6]

The prevalence of post-stroke spasticity varies greatly, ranging from 30% to 80%, with the prevalence of disability ranging from 2% to 13%, especially in the upper extremities.[7,8] Existing treatment methods for controlling spasticity include physical therapy and modality, oral anti-spasticity drugs, nerve blocks and motor points with phenol or ethyl alcohol, botulinum toxin injections, and surgical procedures.[9]

Heating and stretching are the most common treatment techniques used in spasticity management, as they are easy to do and relatively low-cost. The most commonly used heating method and almost always available in medical rehabilitation services is infrared. However, because the warming effect of infrared has a short duration, to get maximum results in lowering spasticity, it should be combined with stretching exercises. Pin et al. identified ten studies on the effectiveness of passive stretching exercises against muscle spasticity in children with cerebral palsy measured by goniometers, of which four were randomized controlled trials. Overall, researchers reported an average increase in the range of motion of the joints of less than 10° and no lasting effect.[10,11]

Extracorporeal shock wave therapy (ESWT) is a new technology that uses a ballistic source to generate pressure and shock waves. ESWT has been used in a variety of musculoskeletal disorders, such as plantar fasciitis, patellar tensinopathy, and calcifying and non clacifying tendonitis of the shoulder. Recently, ESWT has been suggested as a non-invasive alternative treatment for spasticity. Yah Ting Wu et al. reported that 3 ESWT sessions with intervals of 1 week gave noninferior results against botulinum toxin injection for spasticity. Even Yah Ting Wu et al. reported greater increases in the passive range of motion (PROM) wrists and elbows, as well as upper extremity scores of the Fugl-Meyer Assessment (UE-FMA), in the ESWT group.[12]

Tara and colleagues[13] conducted a study on the application of low-energy radial shock wave therapy in patients with peripheral arterial disease who were receiving antiplatelet or anticoagulant

medication and reported side effects of skin redness, petechiae, and hematomas that disappeared within 1 week. No fatal side effects were reported in this study.

Considering that stroke is the first cause of disability both in developed and developing countries, the high prevalence of stroke in Indonesia, the small effectiveness of infrared therapy and stretching exercises, and the safety of RSWT application for stroke patients, it is hypothesized that the addition of RSWT can more effectively decrease the spasticity of chronic stroke patients who have received infrared therapy and stretching exercises.

MATERIAL AND METHODS

This study was a simple randomized controlled pre- and post-experimental design in chronic spastic stroke patients conducted at KRMT Wongsonegoro Hospital in Semarang from November to December 2018 and has been approved by the research ethics committee.

For inclusion criteria: the subjects of the study were stroke patients for the first time with spastic paresis for ≥ 1 year, aged between 45-65 years, with Modified Ashwort Scale score 2 – 3, muscle strength ≥ 3, no pain in the upper extremities of the paresis side, normal Mini Mental State Examination (MMSE) Score (≥24), cooperative and willing to participate in the study by signing informed consent.

Subjects who have recently used antispasticity medications, had open wounds, bleeding, inflammation, malignancy, and implants in the ventral area of the forearm and palms; received injections of botulinum on the ventral forearm and palm within the last six months; underwent surgical procedures within the previous six months to treat upper-limb spasticity; received RSWT treatment throughout the past two weeks on the ventral portion of the forearm and palm; less than one week before the study, received heat therapy or used an upper mobility device for exercise therapy; Hematomas can form spontaneously or with little trauma, which are clinical indicators of blood coagulation abnormalities. Participants in other studies were excluded from the study. The subjects who did not complete the study were not presented ≥ 1 time in RSWT therapy and/or ≥ 4 times at stretching exercises; did not come at the time of the initial and final assesment of the study, or experienced unexpected side effects in the form of extensive hematoma in the area of therapy would be excluded from the study.

The subjects of this study was 30 participants, calculated using the mean difference test formula for each group, divided randomly into 2 groups: the treatment group and the control group. The treatment group received RSWT interventions in the muscle belly of the wrist flexor muscles in the ventral forearm, intrinsic muscles of the hands, and tendon flexors digitorum 1 time per week, infrared therapy, and stretching exercises in the upper extremities three times per week for six consecutive weeks. The control group received infrared therapy and stretching exercises in the upper extremities three times per week, for six consecutive weeks.

1500 shots of RSWT were used at the midbelly of the wrist flexor muscles in the ventral forearm at 3.5 bar and 5 Hz. Additionally, the intrinsic hand muscles and the tendon flexor digitorum the intrinsic hand muscles received 4000 shocks at 3 bar and 5 Hz. RSWT used gel as a coupling agent between the skin and the probe. Infrared therapy was given at the upper extremity with a light distance to the muscles of 50 cm for 15 minutes. Static stretching was given with the stretch maintained for 30 econds and 10 repetitions for each muscle group of the upper extremity. Assessment of the level of spasticity in this study was

measured using the Tardieu Scale, which measures the quality and angle of the resistance, was conducted at the beginning and end of the study. Statistical analysis using SPSS 20 with a value of p<0.05 is a significant value.

RESULT

Based on the International Physical Activity Questionnaire (IPAQ) questionnaire, mild physical activity levels have been assessed for all participants in both groups. There was no meaningful difference between the treatment group and the control group with a value of p>0.05 (Table 1).

Table 1. Subject Characteristics

Variable	X ± SD; median; min-max		P
	Intervention (n=15)	Control (n=15)	
Age (years)	56,4 ± 6,03; 58; 48-65	54,9 ± 4,50; 55; 48-63	0,437§
Gender:			
Male	7 (46,7%)	6 (40,0%)	1.000¥
Female	8 (53,3%)	9 (60,0%)	
Type of Stroke:			
Ischemic	14 (93,3%)	15 (100%)	1,000¥
Hemorrhagic	1 (6,7%)	0 (0,0%)	
Stroke Duration (years)	1,9±0,78; 1,50; 1,00-3,33	1,8±0,79;1,58;1,00-3,25	0,901‡
Parese			
Left	5 (33,3%)	6 (40,0%)	1,000¥
Right	10 (66,7%)	9 (60,0%)	

¥ *Chi square;* § *Independent t;* ‡ *Mann whitney*

No subjects who dropped out were ever acknowledged before the study's conclusion. Additionally, neither the patients' nor the researchers' examinations revealed any side effects that were reported during or after the administration of RSWT. The effect of adding radial shock wave therapy to spasticity was measured by the Tardieu Scale, which consists of an examination of the resistance quality and the resistance angle.

The average quality of wrist and finger resistance in both groups at the beginning of the study was the same. At the end of the study, the average quality of wrist and finger resistance in the treatment group and the control group differed significantly. However, the average decrease in wrist and finger resistance quality in the treatment group was significantly greater than in the control group with a value of p<0.05 (Table 2).

Table 2. Average quality of wrist and fingers resistance

Resistance	Quality	Intervention	Control	P
Pre	Wrist			
	1	0 (0%)	0 (0%)	-
	2	0 (0%)	0 (0%)	
	3	15 (100%)	15 (100%)	
Post	Wrist			
	1	4 (26,7%)	0 (0%)	0,002ϕ*
	2	11 (73,3%)	7 (46,7%)	
	3	0 (0%)	8 (53,3%)	
Pre	Fingers			
	1	0 (0%)	0 (0%)	-
	2	0 (0%)	0 (0%)	
	3	15 (100%)	15 (100%)	
Post	Fingers			
	1	0 (0%)	0 (0%)	
	2	15 (100%)	7 (46,7%)	0,002ϕ*
	3	0 (0%)	8 (53,3%)	

** Significant (p < 0,05); ϕ Chi square*

From Tables 3-6, it can be seen that at the beginning of the study, there was no meaningful difference in the average resistance angle of the wrist and fingers treatment group when compared to the control group, with a value of p>0.05. At the end of the study, the average angle of wrist and finger resistance of the treatment group and the control group differed significantly compared to the beginning of screening. However, the average decrease in the angle of the wrist and fingers in the treatment group was greater when compared to the control group, with a value of p<0.05.

Table 3. Average of wrist resistance angle

Wrist resistance angle	Group		p
	Intervention (n=15)	Control (n=15)	
Pre	30,13 ± 5,03; 30,0; 20,0-40,0	28,53 ± 8,33; 30,0; 15,0-40,0	0,531§
Post	9,53 ± 6,56; 10,0; 0,0-18,0	18,47 ± 7,52; 20,0; 5,0-30,0	0,004‡*
p	0,001†*	<0,001*	
Δ Wrist resistance angle	20,60 ± 6,22; 21,0; 10-30	10,07 ± 3,28; 10,0; 4-15	0,000§*

** Significant (p < 0,05) ; § Independent t; ‡ Mann whitney; ¶ Paired t; † Wilcoxon*

Table 4. Average of metacarpophalangeal (MCP) resistance angle

MCP resistance angle	Group		P
	Intervention (n=15)	Control (n=15)	
Pre	46,72 ± 13,38; 45,6; 24,0-68,8	46,57 ± 10,42; 44,4; 31,6-68,4	0,974§
Post	18,11 ± 7,45; 16,2; 6,4-33,6	36,89 ± 11,51; 30,8; 22,2-61,4	0,000§*
p	0,000¶*	0,000¶*	
Δ MCP resistance angle	28,61 ± 7,07; 29,4; 16,6-40,0	9,68 ± 2,87; 9,4; 5,6-13,6	0,000§*

** Significant (p < 0,05) ; § Independent t; ¶ Paired t*

Table 5. Average interphalangeal proximal (PIP/IP) resistance angle

PIP/IP resistance angle	Group		P
	Intervention (n=15)	Control (n=15)	
Pre	64,04 ± 10,71; 65,0; 48,0-83,0	61,52 ± 9,86; 66,98; 42,0-78,80	0,508§
Post	32,81 ± 6,66; 6,67; 23,40-45,40	50,76 ± 9,68; 9,68; 36,8-71,0	<0,001§*
p	< 0,001¶*	0,000¶*	
Δ PIP/IP resistance angle	31,23 ± 6,64; 31,6; 18,8-43,6	10,76 ± 3,58; 10,8; 5,2-19,6	0,000§*

** Significant (p < 0,05) ; § Independent t; ¶ Paired t*

Table 6. Average interphalangeal distal (DIP) resistance angle

DIP resistance angle	Group		P
	Intervention (n=15)	Control (n=15)	
Pre	58,47 ± 11,07; 61,25; 37,50-78,75	56,13 ± 7,51; 58,75; 40,75-60,50	0,505§
Post	26,27 ± 8,63; 23,5; 15,75-47,25	46,60 ± 6,25; 48,25; 35,5-56,25	0,000§*
p	0,000¶*	0,000¶*	
Δ DIP resistance angle	32,20 ± 8,34; 31,75; 18,75-48,00	9,53 ± 3,72; 8,5; 4-16,5	0,000§*

** Significant (p < 0,05) ; § Independent t; ¶ Paired t*

DISCUSSION

Infrared as a heat modality with a peak intensity of 1000 nm can increase the temperature of superficial tissues to above 40°C. An increase in tissue temperature up to 42°C leads to a decrease in the excitation rate of muscle spindles, as well as an increase in the excitation rate of the golgi tendon organ. In addition, heating increases the extensibility of connective tissue, peaking at 40°C to 45°C for 5 to 10 minutes. The above mechanism explains how heating plays an important role in lowering spasticity. [14,15]

However, the heating effect will immediately decrease as the tissue

temperature decreases; therefore, it is recommended that the heating be continued immediately by stretching so that the maximum length of tissue can be achieved. In addition, heating before stretching is useful to reduce the risk of tissue injury and decrease the power required to stretch. Decreased tissue temperature will be followed by a loss of influence on excitation of the muscle spindle and golgi tendon organs, as well as decreased extensibility of connective tissue. This results in a degree of quality and angle of resistance that can not be maintained to the maximum only by heating.[3,14]

Continuous stretching exercises result in changes in the organization of collagen fibers and changes in the viscoelasticity of collagen fibers, thus maintaining a decrease in quality and angle resistance.[15]

In the treatment group that received the addition of radial shock wave therapy, the delta of the average Tardieu score decreased significantly when compared to the control group.

This result is obtained from the direct mechanical effect of RSWT through the resulting shock waves. Shock waves did not only break the actin-myosine bonds in spastic muscles but also break the fibrosis tissue that occurs in the muscles as a result of spasticity occurring over a long period of time, resulting in greater tissue extensibility.[16] Therefore, delta decreased the average resistance angle obtained with the addition of RSWT more significantly.

The stiffness of connective tissue causes the strain to be immediately transmitted to the muscle spindle, increasing resting discharge and increasing the sensitivity of the muscle spindle to the strain. With reduced stiffness of connective tissue by shock waves, resting discharge and muscle spindle sensitivity to strain will be reduced. In addition, shock waves cause temporary dysfunction of nerve conduction in neuromuscular junctions caused by acetylcholine receptor degeneration. Shock waves also have a direct effect on organ tendon golgi in the form of compressive exitability of motor nerves. All of the above underlies a more significant decrease in delta resistance quality in the group that received RSWT.[11,16]

Subjectively, the study subjects also reported that the feeling of stiffness on the affected side had significantly decreased, even almost disasspearing after they had RSWT.

In this study, it was also found that the use of RSWT was proven safe for patients with chronic spastic stroke. No pain or side effects were noted in the literature that could occur. The high effectiveness and minimal side effects of RSWT can be the basis for considering RSWT as an additional therapy in the management of stroke spasticity.

The limitation of this study was that researchers were unable to rule out the influence of physical activity involving the movement of hand joints due to the absence of measuring instruments that can provide restrictions on joint movement that can affect spasticity. The physical activity level measurement tool in the form of the International Physical Activity Questionnaire (IPAQ) in this study only groups the level of physical activity based on the effort required by the subject to perform the activity. Other than that, the researchers also did not assess how long the reduction in spasticity produced by the addition of RSWT lasted. Further research ought to consider this limitation into consideration about the effects of additional RSWT to reduce spasticity, especially in stroke patients.

CONCLUSION

We draw the conclusion from this study that the addition of RSWT has been proven to have a greater impact on the reduction of upper extremity muscle spasticity in chronic stroke patients.

DISCLOSURES

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Conflict of interest

All authors have no conflict of interest.

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Author Contribution

All authors have contributed to all processes in this research, including preparation, data gathering and analysis, drafting and approval for publication of this manuscript.

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