Comparison of Therapeutic Ultrasound and Radial Shock Wave Therapy in the Treatment of Plantar Flexor Spasticity After Stroke: A Prospective, Single-blind, Randomized Clinical Trial

Hojjat Radinmehr, PhD, PT,* Noureddin Nakhostin Ansari, PhD, PT,†‡§ Soofia Naghdi, PhD, PT,†‡§ Azade Tabatabaei, MSc, PT,† and Ehsan Moghimi, MD‡

Background: This study aimed to compare the effects of therapeutic ultrasound (US) and radial extracorporeal shock wave therapy (rESWT) in the treatment of plantar flexor spasticity after stroke. *Materials and Methods:* In this prospective, single-blind, randomized clinical trial, 32 patients (age range 42-78 years; male 19) with stroke were randomly divided into two groups: The US group (n = 16) received the continuous ultrasound, intensity 1.5 w/cm^2, frequency 1 MHz, and duration 10 minutes. The rESWT group (n = 16) was treated with rESWT, 0.340 mJ/mm^2, 2000 shots. Both groups received the treatments for 1 session. The H-reflex tests of H_{max}/M_{max} ratio and H-reflex latency, the Modified Modified Ashworth Scale (MMAS), active range of motion (AROM), passive range of motion (PROM), passive plantar flexor torque (PPFT), and the timed "up and go" test (TUG) were blinded assessed at baseline (T0), immediately post-treatment (T1), and one hour follow-up (T2). *Results:* The H-reflex tests did not improve across the groups. However, the MMAS spasticity scores, AROM and PROM, PPFT, and TUG improved significantly within groups. The results found no significant differences between groups for all outcome measures. *Conclusions:* The US and rESWT had similar effects, and the rESWT was not more effective than the US in improving ankle plantar flexor spasticity after stroke.

Key Words: Stroke—therapeutic ultrasound—shock wave therapy—spasticity

Introduction

Spasticity is a common and disabling problem in patients with stroke observed approximately in 50% of patients.[1-3] Classically, spasticity has been defined by Lance (1980) as a motor disorder characterized by a velocity-dependent increase in muscle tone due to hyperexcitability of the stretch reflex.[4] The alterations in neural networks as well as abnormality in the mechanical properties of the muscles have been suggested as possible mechanisms underlying spasticity.[5-7] Severe spasticity must be managed as it may cause secondary long-term complications such as pain, soft tissue contractures, functional limitations and reduced quality of life.[8-11] Muscle spasticity after stroke may be treated using oral medicine, botulinum toxin injection, intrathecal therapy, and physiotherapy. Physiotherapy is the main treatment of the multimodal strategy for the management of spasticity.[7,8,12-15]

Physiotherapy uses several strategies to treat spasticity among them are modalities such as therapeutic ultrasound (US) and extracorporeal shock wave therapy (ESWT). Therapeutic ultrasound is a kind of mechanical

From the *Department of Physiotherapy, School of Rehabilitation, Hamadan University of Medical Science, Hamadan, Iran; †Department of Physiotherapy, School of Rehabilitation, Tehran University of Medical Science, Tehran, Iran; ‡Sports Medicine Research Center, Neuroscience Institute, Tehran University of Medical Sciences, Tehran, Iran; and §Neuromusculoskeletal Research Center, Iran University of Medical Sciences, Tehran, Iran.

Received June 3, 2018; revision received February 16, 2019; accepted March 1, 2019.

Address correspondence to Hojjat Radinmehr, PhD, PT, Department of Physiotherapy, School of Rehabilitation, Hamadan University of Medical Sciences, Fahmide ST, Hamadan, Iran. E-mails: h.radinmehr@umsha.ac.ir, hradinmehr@yahoo.com.

1052-3057/$ - see front matter

https://doi.org/10.1016/j.jstrokecerebrovasdis.2019.03.008

energy with a frequency typically between 1.0 and 3.0 MHz. The US can be used in continuous or pulsed mode. Continuous US is primarily indicated where tissue heating is preferred.

The shock wave is an acoustic, compression wave with high amplitude, and rapid pressure changes of a high followed by a low pressure phase. The typical conventional focal type of shock wave is recognized with high peak pressure (100 MPa), fast pressure rise (<10 ns), and short duration (10 μs). The shock wave can be transmitted to the area with an energy density ranging between 0.003 and 0.89 mJ/mm^2. The radial extracorporeal shock wave therapy (rESWT) is distinguished from the focused type with lower peak pressure (0.1 MPa), longer rise time (50 μs), and duration between 200 and 2000 μs.[16] Therapeutic US and rESWT have been used in previous investigations in people with various neurological conditions.[17-20] However, there is no study comparing the effects of therapeutic US and rESWT in people with stroke. Therefore, the aim of this study was to compare the effects of therapeutic US and rESWT in the treatment of plantar flexor spasticity after stroke.

Methods

This study used a prospective, single-blind randomized design. The study protocol and methods were approved by the Review Board, School of Rehabilitation, and the Ethical Committee of Tehran University of Medical Sciences (TUMS), Iran.

Participants

Eligible participants were included in the study if they met the following inclusion criteria: (1) first-ever stroke; (2) stroke duration of at least 1month; (3) age range between 40 and 70 years old; (4) a score ≥1on the Modified Modified Ashworth Scale (MMAS) for ankle plantar flexor spasticity; (5) able to walk independently with or without assistive aids; (6) able to follow instructions. Patients were excluded from participating in the present study if they had the following criteria: (1) more than 10% reduction in the affected ankle dorsiflexion range of motion (ROM) compared to the unaffected side; (2) taking antispastic drugs; (3) currently being under treatment for spasticity; (4) participating in other study concurrently; (5) having diabetes; (6) wound or infection in the affected lower limb;(7) presence of sensory disturbances; (8) presence of any other neurological disorder; (9) contraindications to US or rESWT.

Procedure

Patients referred to the Physiotherapy Clinic for Stroke, School of Rehabilitation, TUMS, were evaluated for the inclusion and exclusion criteria. First, oral, and written informed consent was obtained after describing the procedure to the patients. Subsequently, patients were randomized using a blocked randomization method to either US or rESWT group.

The outcome variables were measured three times at baseline (T0), immediately post-treatment (T1), and 1 hour post-treatment (T2).[20] All assessments were carried out and interventions were given in the same Physiotherapy Clinic. Measurements were performed in a random order by a physiotherapist who was not involved in the administration of the interventions and was blinded to treatment allocation of the patients.

Intervention

Various treatment protocols (Intensity 0.03 to 0.63 mJ/mm^2, Frequency 4-10 Hz) were previously used for rESWT. Patients in the rESWT group received a single session of rESWT (enPuls Version 2.0, Zimmer Medizin System, Germany) in the gastrocnemius bulk with the following protocol: head applicator 15 mm, 2000 shots, frequency 5 Hz, energy density of 60 mJ (1 bar), energy applied 0.340 mJ/mm^2.[20]

Patients in the US group received a single session continuous ultrasound (Shrewsbury Medical, model SM 3371, United Kingdom) applied to the gastrocnemius bulk with the following protocol: frequency 1 MHz, intensity 1.5 W/cm^2, duration 10 minutes.[17] Participants in both groups received the treatments in the prone position.

Outcome Measures

Measurements described and used previously were followed.[20]

MMAS

Plantarflexor spasticity was assessed using the Persian MMAS[21] from 0 meaning no increase in muscle tone to 4 meaning affected part rigid in flexion or extension[22-25] with the knee flexed 30° for soleus spasticity or the knee in the extended position for gastrocnemius spasticity.

Ankle ROM

Patients were positioned supine on a bed, and the active ROM (AROM) and passive ROM (PROM) were measured using an ankle biplane goniometer (Bissell Health Care, model 7524) with the knee flexed 30° for soleus extensibility or extended for gastrocnemius extensibility. Evaluating ankle AROM and PROM was started at rest position regardless of ankle plantarflexion or dorsiflexion position, and motion performed to dorsiflexion end range.

Passive Plantar flexor Torque (PPFT)

To measure PPFT (Nm), the physiotherapist followed the method similar to that used for clinical assessment of plantarflexor spasticity using a hand-held dynamometer

(North Coast Medical dynamometer, model 2845). Measurements were taken with the knee in flexed or extended position at low (approximately 3 seconds) as well as high (approximately 1 second) velocities with the orders determined randomly. A one-minute interval was employed between these tests. The high reliability measurement of resistive torque using the hand-held dynamometer have been shown previously.[26-29]

Timed Up and Go (TUG) Test

The TUG test was used to assess the mobility and dynamic balance in our sample of patients with stroke.[30,31] The patients were instructed to sit on a standard height chair, stand-up and walk for 3 meters, then turn around, and walk back and sit again on the chair. A stopwatch was used to record the time in seconds as the TUG test score.

H-reflex Tests

The Medelec electromyography machine, TD50 TEKA model, United Kingdom, was used to record the maximal H-reflex, the maximal M-wave, and the H-reflex latency. To record the H-reflex parameters, the patients were in a prone position with their head turned to one side, and their feet suspended over the end of the bed. The surface, self-adhesive recording, and reference electrodes were placed on the skin over the soleus muscle and the Achilles tendon, respectively. The ground electrode was placed over the lateral gastrocnemius muscle between the stimulation and the recording electrodes. Once the placements of the electrodes were adjusted, the tibial nerve was stimulated at the popliteal fossa (a single, 1-ms rectangular electrical pulse, stimulus frequency 1 per 5 seconds) with the bipolar silver-silver chloride surface electrode, 5 mm in diameter. The signals were recorded with band-pass filter set at 5 Hz to 3 kHz, sweep speed at 5 msec/div, and sensitivity at 500 μV-2 mV/div. The H_{max} and M_{max} with largest amplitudes were achieved; the H_{max} divided by the M_{max} was calculated as the H_{max}/M_{max} ratio.

The time measured from the beginning of the stimulation to the start of the initial deflection of the H-reflex wave was recorded as the H-reflex latency. The H-reflex tests are reliable measures for assessing motor neuron excitability after stroke.[32]

Sample Size

A power analysis was conducted using G*power 3.1.9.2 with the H_{max}/M_{max} ratio as main outcome measure and data provided previously.[17] The analysis produced a sample size of 15 per group indicating sufficient to achieve 80% power at an alpha of 5%.

Statistical Analysis

The statistical analyses were performed with SPSS version 21(SPSS, Chicago, IL). The normal distribution of data for all variables was demonstrated by the Kolmogorov-Smirnov test. A general linear model, two-way mixed analysis of variance (ANOVA) was performed for continuous data. The Bonferroni adjustments were used to determine the differences between testing time points. The Greenhouse-Geisser estimate of sphericity was applied to correct the degrees of freedom when the Mauchley's test revealed nonhomogeneity of variances. The Friedman's test was used to test for the effects of interventions on spasticity assessed by an ordinal MMAS. A post-hoc Wilcoxon Signed-Rank Test was applied for paired differences between the testing time points within groups. The differences between groups on the MMAS spasticity outcome were investigated using the Mann-Whitney U test. The statistical significance was set at $P \leq .05$.

Results

A total of 32 patients equally assigned to the groups completed the study. The demographic data of the two groups are shown in Table 1. There were no significant differences between groups on age, stroke duration, and body mass index ($P > .05$). Repeated Measure ANOVA showed no significant difference between groups at T0 in all quantitative variables, but Man-Whitney test showed significant difference between groups in MMAS at T0; two subjects had MMAS score 3 in the rESWT group that contributed to the significant difference between groups at baseline.

MMAS Score

The median scores in both groups are demonstrated in Table 2. The MMAS scores improved in both groups regardless of knee positions (Friedman's test, $P < .001$), and the MMAS scores at T1 and T2 improved compared to T0 (Wilcoxon Signed-Rank Test, $P < .05$). The MMAS scores were not statistically different between groups in the knee flexed or extended position (Mann-Whitney U test: $P > .05$).

Table 1. *Demographics of patients in both groups presented as mean ±SD or frequency (n)*

	US Group (n = 16)	rESWT Group (n = 16)
Age, years	56.2 ± 8.4	56.0 ± 12.3
BMI	28.0 ± 4.9	29.2 ± 3.8
Stroke onset, months	36.8 ± 15.1	34.4 ± 20.5
Gender (male/female), n	10/6	9/7
Affected side(right/left), n	5/11	7/9

US, ultrasound; rESWT, radial extracorporeal shock wave therapy.

Table 2. *The Modified Modified Ashworth Scale (MMAS) scores in the knee flexed and extended positions in both groups presented as median (Interquartile range) at baseline (T0), immediately after treatment (T1), and 1hour after treatment (T2),p value presented for Friedman and Mann-Whitney tests*

	US Group (n = 16)						rESWT Group (n = 16)					
	Knee flexed			Knee extended			Knee flexed			Knee extended		
	T0	T1*	T2*	T0	T1*	T2*	T0	T1*	T2*	T0	T1*	T2*
MMAS scores	1(1-1.75)	1(0-1)	1(0-1)	1(1-2)	1(0-1)	1(0-1)	2(1-2)	1(0-1)	1(0-1)	2(2-2)	1(0.25-1)	1(0.25-1)

Between groups comparison.
Mann-Whitney test (Knee flexed) T0 $P = .06$, T1 $P = .74$, T2 $P = .74$, (Knee extended) T0 $P = .04$, T1 $P = .53$, T2 $P = .53$.
*Significant compared to T0.

Ankle ROM

The both active and passive ankle range of motion are demonstrated in Table 3. The ankle AROM ($F_{1.27,\ 38.25} = 24.50$, $P < .001$) and PROM ($F_{1.40,\ 42.08} = 45.42$, $P < .001$) with knee flexed or extended significantly improved in the both groups at T1 and T2 compared to T0. There were no main effects of group ($F_{1,\ 30} = 0.31$, $P = .58$) or knee position on AROM ($F_{1,\ 60} = 1.44$, $P = .23$). However, there was a main effect of knee position for PROM, and the PROM in the knee flexed position was greater than that in the knee extended position (Greenhouse-Geisser: $F_{1,\ 60} = 5.19$, $P = .03$). The ankle AROM (Group × Time interaction, $F_{1.24,\ 74.19} = 0.32$, $P = .62$) was not different over time between groups and PROM (Group × Time interaction; $F_{1.41,\ 84.32} = 4.81$, $P = .02$) was different over time between groups. Besides, no Time and Position interaction was revealed for AROM ($F_{1.24,\ 74.19} = 1.16$, $P = .3$) and PROM ($F_{1.41,\ 84.32} = 0.37$, $P = .62$).Three factor ANOVA revealed time × position × group interaction for PROM ($F_{1.4,\ 84.32} = 4.88$, $P = .019$), and no interaction between time, position, and group for AROM ($F_{1.23,\ 74.19} = 0.66$ $P = .44$).

PPFT

The Table 3 demonstrates the PPFT for both groups. There was a main effect of time regardless of knee position (flexed vs extended) or velocity (low vs high) ($P < .001$). Regardless of knee position (flexed versus extended), the PPFT decreased in the both groups when low ($F_{2,\ 60} = 12.23$, $P < .001$) or high velocity ($F_{2,\ 60} = 14.37$, $P < .001$) was applied at T1 and T2 compared to T0. But no significant differences existed between groups ($P > .05$).

Further ANOVA with position and velocity as between-subject factors and time as the within-subject factor demonstrated a main effect for time ($F_{2,\ 240} = 5.16$, $P = .006$), velocity ($F_{1,\ 120} = 149.64$, $P < .001$) and position ($F_{1,\ 120} = 7.49$, $P = .007$). The PPFT in the knee extended position and the high velocity was greater than those in the knee flexed position and the low velocity, respectively. The Time × Position interaction was significant ($F_{2,\ 240} = 3.40$, $P = .04$).

The Time × Group ($F_{2,\ 240} = 0.47$, $P = .62$), Time × Velocity ($F_{2,\ 240} = 1.20$, $P = .30$), or Time × Position × Velocity interaction ($F_{2,\ 240} = 0.04$, $P = .96$), or Time × Position × Group ($F_{2,240} = 0.07$, $P = .93$), or Time × Velocity × Group ($F_{2,240} = 0.35$, $P = .7$), or Time × Position × Velocity × Group interaction ($F_{2,240} = 0.68$, $P = .51$) was not statistically significant. However, the Time × Position interaction was significant ($F_{2,\ 240} = 3.40$, $P = .04$).

TUG

The TUG scores decreased in the two groups (ANOVA time-effect: test $F1.64, 49.35 = 10.40$, $P < .001$) at T1 compared to T0. Difference between T1 and T2 was not statistically significant. There was no significant difference between groups ($F_{1,30} = 0.02$, $P = .89$). The Time × Group interaction was not significant ($F_{1.64,\ 49.35} = 1.29$, $P = .28$) (Table 4).

Table 3. *The Mean ± standard deviation (range) of ankle active range of motion (AROM), passive range of motion (PROM), and passive plantarflexor torque (PPFT) at knee flexed and extended positions before treatment (T0), immediately after treatment (T1), and 1hour after the end of treatment (T2)*

	US Group (n = 16)						rESWT Group (n = 16)					
	Knee flexed			Knee extended			Knee flexed			Knee extended		
	T0	T1	T2	T0	T1	T2	T0	T1	T2	T0	T1	T2
AROM (degree)	11.9 ± 9.3 (0.0-30.0)	16.3 ± 11.4* (0.0-36.0)	16.9 ± 11.6* (0.0-36.0)	9.1 ± 7.5 (0.0-23.0)	12.2 ± 9.1* (0.0-31.0)	11.9 ± 9.0* (0.0-30.0)	10.5 ± 11.0 (0.0-32.0)	14.1 ± 12.4* (0.0-35.0)	13.8 ± 12.7* (0.0-37.0)	8. 2 ± 10.8 (0.0-32.0)	11.7 ± 12.5* (0.0-37.0)	11.3 ± 12.4* (0.0-37.0)
PROM (degree)	27.4 ± 4.9 (18.0-35.0)	32.0 ± 6.4* (21.0-45.0)	32.3 ± 5.0* (21.0-40.0)	24.9 ± 4.3 (17.0-32.0)	28.5 ± 5.6* (18.0-43.0)	27.6 ± 4.9* (19.0-37.0)	26.1 ± 8.7 (10.0-45.0)	31.2 ± 7.9* (15.0-45.0)	30.6 ± 8.4* (12.0-45.0)	20.6 ± 8.0 (8.0-33.0)	28.1 ± 7.2* (15.0-40.0)	28.1 ± 7.6* (12.0-38.0)
PPFT (slow velocity) (N.m)	12.4 ± 3.1 (6.8-16.5)	11.2 ± 3.0* (5.8-15.8)	11.1 ± 2.9* (5.2-16.0)	14.6 ± 4.3 (8.0-23.1)	13.1 ± 4.3* (5.6-20.4)	13.2 ± 3.8* (5.4-119.3)	13.3 ± 2.8 (8.1-17.5)	12.5 ± 2.3* (7.8-15.6)	12.5 ± 2.6* (7.8-17.1)	15.5 ± 3.4 (10.5-24.7)	13.5 ± 3.2* (8.1-21.9)	13.2 ± 3.5* (7.9-22.4)
PPFT (high velocity) (N.m)	20.7 ± 4.9 (10.9-32.9)	19.4 ± 4.2* (10.6-25.8)	19.5 ± 3.6* (12.7-26.0)	21.9 ± 4.6 (11.5-28.7)	21.2 ± 4.5* (10.9-28.2)	20.9 ± 4.3* (11.9-28.3)	20.8±4.6 (14.1-32.5)	18.5±3.5* (13.3-25.4)	18.9 ± 4.5* (13.1-31.3)	22.5 ± 4.8 (14.7-34.1)	21.0 ± 5.0* (13.6-32.6)	20.9 ± 5.0* (13.3-33.6)

*Significant compared to T0.

H-reflex Tests

The H-reflex latency increased in the both groups ($F_{2,60} = 1.46$, $P < 0.001$), but no difference in the H-reflex latency was found between groups ($F_{1,30} = 0.78$, $P = .38$). There was no significant main effects of time ($F_{1.67, 50.04} = 1.20$, $P = .3$) or group ($F_{1,30} = 1.76$, $P = .19$) for H_{max}/M_{max} ratio. No Time × Group interaction was found for H_{max}/M_{max} ratio ($F_{2, 60} = 1.46$, $P = .24$) and H-reflex latency ($F_{2, 60} = 0.82$, $P = .45$) (Table 4).

Discussion

This comparative study found that (1) within group, both US and rESWT improved all the outcomes with the exception of H_{max}/M_{max} ratio, and this effect persisted for 1 hour, and (2) improvements were not different between groups. To our knowledge, this study is the first prospective, randomized, single-blinded, clinical trial to compare the benefits of physiotherapeutic ultrasound and rESWT for plantar flexor spasticity after stroke.

Impact on Spasticity

At the end of treatment, patients in the both groups improved the plantar flexor spasticity. Clinical effects of US[17,33-35] and rESWT[20,36-38] on spasticity in patients with plantar flexor spasticity has been assessed by previous studies. Two studies assessed the effects of US alone. One pilot, pretest-post-test study in four adult patients with first stroke showed a nonsignificant decrease of plantar flexor spasticity assessed by the original Ashworth scale.[33] Interestingly, in another randomized, single-blind, placebo-controlled study, investigators showed that 15 treatment sessions of real US, 3 days per week, every other day significantly improved the Ashworth measure of ankle plantar flexor spasticity poststroke.[17] One more study compared the immediate effects of therapeutic US and infrared (IR) in patients with plantar flexor muscle spasticity and found neither infrared nor US improved the spasticity.[34] Further study compared the effects on spasticity between 2 groups: 10-minute continuous US + passive stretching exercise versus passive stretching exercise alone, and found that while the within group improvement of plantar flexor spasticity in terms of MMAS was statistically significant, but the combined US with passive stretching exercise was not more effective on the spasticity improvement than passive stretching exercise alone.[35] Inconsistencies observed across studies on the effects of US on spasticity could be due to the differences in methodology, treatment protocol, and number of subjects included. In the rESWT group, patients improved the ankle plantar flexor spasticity in agreement with previous investigations in which beneficial effects of rESWT on spasticity were found.[20,36-39]

After the end of treatments, the improvement of spasticity observed following either US or rESWT treatment was not statistically different between the two groups,

Table 4. *The Mean ± standard deviation (range) of Time up and go (TUG) test and H-reflex measures before treatment (T0), immediately after treatment (T1), and 1 hour after the end of treatment (T2)*

	US Group (n = 16)			rESWT Group (n = 16)		
	T0	T1	T2	T0	T1	T2
TUG (second)	20.8 ± 10.2 (8.2-39.5)	19.8 ± 9.7* (8.4-40.3)	19.8 ± 9.8* (7.5-37.6)	21.9 ± 10.8 (12.0-48.2)	20.4 ± 11.0* (10.5-48.6)	19.8 ± 10.8* (9.8-48.3)
H_{max}/M_{max} ratio	0.37 ± 0.21 (0.07-0.92)	0.38 ± 0.21 (0.06-0.86)	0.36 ± 0.19 (0.09-0.85)	0.49 ± 0.23 (0.14-0.88)	0.46 ± 0.24 (0.14-0.90)	0.47 ± 0.22 (0.12-0.88)
H-reflex latency(msec)	29.96 ± 3.61 (22.9-37.7)	30.29 ± 3.35* (24.8-37.3)	30.7 ± 3.67* (24.8-39.9)	30.71 ± 3.07 (26.6-38.0)	31.69 ± 3.57* (27.2-40.8)	31.72 ± 3.31* (26.90-39.2)

*Significant compared to T0.

suggesting that the US and the rESWT have similar effects on ankle plantar spasticity in terms of MMAS scores. In this study, only one single session of treatment was offered. Longer treatment protocols with the US or the rESWT may depict the differences between the two treatments. Hence, it would be interesting to compare the effects of US versus the rESWT on spasticity with longer treatment protocols.

Ankle ROM

The AROM of ankle dorsiflexion, in line with previous reports (US[17,34]; rESWT[20]) increased after treatment regardless of group and knee position. These findings indicate the similar effects of US and rESWT on the ankle active dorsiflexion, and knee positions, flexed or extended, had no additional effects. However, the gain in AROM was low (maximum ~ mean 5°). The minimally clinically important changes in AROM have not been evaluated in patients after stroke. Hence, it is important to estimate the minimally clinically important changes for AROM in patients after stroke, and relate the results to treatments and patients' satisfaction.

The very small improvements observed in AROM after both treatments could be due to the spasticity reductions. This may imply that the more ankle AROM might be achieved if the plantar flexor spasticity was improved more considerably (median changes 1 grade). We evaluated the active ankle dorsiflexion ROM as a potential marker for improvements in motor control and cortical networks as it is an essential component for normal walking. Individuals with stroke who have impairments in initiation of swing phase[40] and lack sufficient active ankle dorsiflexion use compensatory movements with the knee flexion, hip lateral rotation, and pelvic tilt to obtain toe clearance. Nonetheless, one possible reason for small improvements in ankle AROM could be the impairments in neural drive, descending pathways, and integrated sensorimotor network.[41,42] The movement training was not included in the treatment protocol used in the current study. Combination of US or rESWT with dorsiflexion movement exercises might improve the outcome. One more possible reason might be the dosage and parameters used in the current study. Further works are needed to determine the optimal technical parameters and dose of US and rESWT to observe clinically relevant improvements in AROM.

The PROM of ankle dorsiflexion, in line with previous reports increased after treatment with US[17,34] or rESWT.[20,36,37] Further significant increases at post T2 was not visible in the both groups, and the improvements obtain at post T1 remained for 1 hour after the conclusion of treatment. However, no difference was observed on ankle PROM between both the US and rESWT groups. No significant difference between the both groups suggests that improvement in ankle PROM is not associated with type of treatment applied particularly US or rESWT. The decrease in spasticity along with soft tissue biomechanical changes induced by US and rESWT might have increased the ankle dorsiflexion PROM in the both groups.[17,20,34,43]

The ankle PROM was significantly affected by knee position, and the PROM, pre-and post-treatment, was greater with the knee flexed position than the knee extended position. However, a recent report to examine the effects of rESWT on plantar flexor spasticity after stroke (n = 12) showed that the knee position had no significant on the ankle PROM.[20] The difference between the two studies might be from the larger sample of patients included in this study. Following stroke, the affected lower limb is in the extended pattern, and the gastrocnemius muscle is typically involved.[44] The synergic pattern is associated with higher spasticity.[45] Therefore, the higher spasticity of ankle plantar flexors in particular gastrocnemius muscle could increase the resistance to passive movement in the knee extended position and subsequently reduced the ankle PROM.

PPFT

This study found that the PPFT, regardless of knee position and velocity, decreased in the both groups, suggesting that both US and rESWT altered the PPFT and improved the resistive passive torque for 1 hour after treatment. A similar effect has been reported in a previous study evidencing the improvement of PPFT in the patients treated with the rESWT.[20] No previous studies have

evidenced the PPFT as an outcome in studies of therapeutic ultrasound on spasticity in stroke population. The improvements of spasticity and ankle PROM may account for the improvements in the PPFT observed in the both groups. This study demonstrated no significant differences between the US and rESWT on the PPFT after stroke.

We observed that the PPFT in the knee extended position, regardless of group, is greater than the knee flexed position. The possible reason could be that the hemiplegic lower limb poststroke is restricted to the extension limb synergy,[44] and the extensor synergy in the lower limb is associated with more spasticity in the extensor muscle groups.[45] As such, the lower limb extensors after stroke present more resistance than the flexors during passive stretch when assessed in the lower limb extended position.

The higher PPFT in the knee extended versus knee flexed position could be due to the more resistance offered by the gastrocnemius, as well. One study used the rESWT to treat the plantar flexors in patients after stroke and found no significant effects of knee position on the PPFT.[20] The significant Time × Position interaction indicates the greater PPFT over time in the knee extended position. The significant greater PPFT encountered with high velocity corroborate the velocity dependent of spasticity in the upper motor neuron syndrome including stroke.[4]

TUG Test

The TUG test was used to assess functional mobility and balance abilities.[46] The TUG improved in both groups after treatment. However, in line with previous report,[20] the amount of improvement was small (<5% in US group, <10% in rESWT group). Indeed, the minimal change in the TUG in patients with stroke is 23% defined as clinically important and meaningful change.[47] The small improvements in TUG score occurred in both groups could be from the fact that the subjects had ankle plantar flexor spasticity along with decreased ankle ROM after treatment, which could have a negative influence on the walking ability and speed. In this study, patients received only one treatment session, and the intensive physiotherapy was not provided in the groups. The US and rESWT combined with task-specific gait training might improve the TUG score greatly. Further study with more treatment sessions and in combination with task-specific gait training is newsworthy. The improvements of TUG after treatment were not statistically different between groups which indicate similar effects of US and rESWT on mobility.

H-reflex Tests

The small significant increase of H-reflex latency was observed after treatment in both groups. Although the increase in latency were statistically significant, the changes observed were not clinically relevant as changes were in the normal range as found in previous reports in patients with spasticity.[20,38,48] Despite meaningful improvements occurred in the MMAS in both groups after treatment, the H-reflex latency did not change concordantly which may indicates that the H-reflex latency is not sensitive enough for detecting changes in spasticity. The effects of US and rESWT on the H-reflex latency were not different between groups.

In the current study, the H_{max}/M_{max} ratio, in line with previous reports,[20,36,38,49] did not decrease over time after US or rESWT. However, one study found decreasing in the H_{max}/M_{max} ratio when investigating the effects of rESWT on poststroke wrist flexor spasticity.[19] The both US and rESWT had no significant effect on the H_{max}/M_{max} ratio as a measure of reflex hyperexcitability. The lack of significant changes in H-reflex measures found in the current study may suggest a mechanical effect on spastic muscles and shortened soft tissues for US and rESWT.

After both US and rESWT treatment, has been not recorded any side effect in subjects.

Limitations of the study must be noted. First, the follow-up was limited to 1 hour, and the possible differences between two interventions could need longer period to appear. Second, despite power analysis, our cohort was small and the differences between groups might have been statistically significant with a larger sample size. Third, patients were not blinded to the type of treatment.

Conclusions

Both therapeutic ultrasound and rESWT improved spasticity, but neither improved the reflex hyperexcitability. Improvements in spasticity did not translate into meaningful improvements in functional mobility. There were no statistically significant differences between groups in any of the outcome measures used in this study.

Acknowledgments

We would like to thank patients for participation in this study. We greatly appreciate the Research Deputy, Tehran University of Medical Sciences. The present study did not receive financial support for the study, authorship and/or publication of this article.

References

1. Watkins CL, Leathley MJ, Gregson JM, et al. Prevalence of spasticity post stroke. Clin Rehabil 2002;16:515-522.
2. Sommerfeld DK, Gripenstedt U, Welmer AK. Spasticity after stroke: an overview of prevalence, test instruments, and treatments. Am J Phys Med Rehabil 2012;91:814-820.
3. Wissel J, Manack A, Brainin M. Toward an epidemiology of poststroke spasticity. Neurology 2013;80:S13-S19.
4. Lance JW. The control of muscle tone, reflexes, and movement: Robert WartenbeRg Lecture. Neurology 1980; 30:1303-1313.
5. Sheean G, McGuire JR. Spastic hypertonia and movement disorders: pathophysiology, clinical presentation, and quantification. PMR 2009;1:827-833.
6. Mukherjee A, Chakravarty A. Spasticity mechanisms for the clinician. Front Neurol 2010;1:149.

7. Ghai A, Garg N, Hooda S, et al. Spasticity - Pathogenesis, prevention and treatment strategies. Saudi J Anaesth 2013;7:453-460.
8. Francisco GE, McGuire JR. Poststroke spasticity management. Stroke 2012;43:3132-3136.
9. Graham LA. Management of spasticity revisited. Age Ageing 2013;42:435-441.
10. Thompson AJ, Jarrett L, Lockley L, et al. Clinical management of spasticity. J Neurol Neurosurg Psychiatry 2005; 76:459-463.
11. Zorowitz RD, Gillard PJ, Brainin M. Poststroke spasticity: sequelae and burden on stroke survivors and caregivers. Neurology 2013;80:S45-S52.
12. Bhakta BB. Management of spasticity in stroke. Br Med Bull 2000;56:476-485.
13. Franek A, Turczynski B, Opara J. Treatment of spinal spasticity by electrical stimulation. J Biomed Eng 1988; 10:266-270.
14. Thibaut A, Chatelle C, Ziegler E, et al. Spasticity after stroke: physiology, assessment and treatment. Brain Inj 2013;27:1093-1105.
15. Watanabe T. The role of therapy in spasticity management. Am J Phys Med Rehabil 2004;83:S45-S49.
16. Gonkova MI, Ilieva EM, Ferriero G, et al. Effect of radial shock wave therapy on muscle spasticity in children with cerebral palsy. Int J Rehabil Res 2013;36:284-290.
17. Ansari NN, Naghdi S, Bagheri H, et al. Therapeutic ultrasound in the treatment of ankle plantarflexor spasticity in a unilateral stroke population: a randomized, single-blind, placebo-controlled trial. Electromyogr Clin Neurophysiol 2007;47:137-143.
18. Amelio E, Manganotti P. Effect of shock wave stimulation on hypertonic plantar flexor muscles in patients with cerebral palsy: a placebo-controlled study. J Rehabil Med 2010;42:339-343.
19. Daliri SS, Forogh B, Emami Razavi ZS, et al. A single blind, clinical trial to investigate the effects of a single session extracorporeal shock wave therapy on wrist flexor spasticity after stroke. NeuroRehabilitation 2015;36:67-72.
20. Radinmehr H, Nakhostin Ansari N, Naghdi S, et al. Effects of one session radial extracorporeal shockwave therapy on post-stroke plantarflexor spasticity: a single-blind clinical trial. Disabil Rehabil 2017;39:483-490.
21. Nakhostin Ansari N, Naghdi S, Forogh B, et al. Development of the Persian version of the Modified Modified Ashworth Scale: translation, adaptation, and examination of interrater and intrarater reliability in patients with poststroke elbow flexor spasticity. Disabil Rehabil 2012; 34:1843-1847.
22. Ansari NN, Naghdi S, Moammeri H, et al. Ashworth Scales are unreliable for the assessment of muscle spasticity. Physiother Theory Pract 2006;22:119-125.
23. Naghdi S, Ansari NN, Mansouri K, et al. Neurophysiological examination of the Modified Modified Ashworth Scale (MMAS) in patients with wrist flexor spasticity after stroke. Electromyogr Clin Neurophysiol 2008;48:35-41.
24. Ghotbi N, Ansari NN, Naghdi S, et al. Inter-rater reliability of the Modified Modified Ashworth Scale in assessing lower limb muscle spasticity. Brain Inj 2009;23:815-819.
25. Ghotbi N, Nakhostin Ansari N, Naghdi S, et al. Measurement of lower-limb muscle spasticity: intrarater reliability of Modified Modified Ashworth Scale. J Rehabil Res Dev 2011;48:83-88.
26. Dvir Z, Arbel N, Bar-Haim S. The use of hand-held dynamometry for measuring the effect of short-leg tone reducing casts on the passive compliance of calf muscles in children with cerebral palsy. Neurorehabil Neural Repair 1991;5:229-234.
27. Boiteau M, Malouin F, Richards CL. Use of a hand-held dynamometer and a Kin-Com dynamometer for evaluating spastic hypertonia in children: a reliability study. Phys Ther 1995;75:796-802.
28. Lamontagne A, Malouin F, Richards CL, et al. Evaluation of reflex- and nonreflex-induced muscle resistance to stretch in adults with spinal cord injury using hand-held and isokinetic dynamometry. Phys Ther 1998;78:964-975. discussion 76-8.
29. Lorentzen J, Grey MJ, Geertsen SS, et al. Assessment of a portable device for the quantitative measurement of ankle joint stiffness in spastic individuals. Clin Neurophysiol 2012;123:1371-1382.
30. Herman T, Giladi N, Hausdorff JM. Properties of the 'timed up and go' test: more than meets the eye. Gerontol 2011;57:203-210.
31. Flansbjer UB, Holmback AM, Downham D, et al. Reliability of gait performance tests in men and women with hemiparesis after stroke. J Rehabil Med 2005;37:75-82.
32. Misiaszek JE. The H-reflex as a tool in neurophysiology: its limitations and uses in understanding nervous system function. Muscle Nerve 2003;28:144-160.
33. Ansari NN, Adelmanesh F, Naghdi S, et al. The effect of physiotherapeutic ultrasound on muscle spasticity in patients with hemiplegia: a pilot study. Electromyogr Clin Neurophysiol 2006;46:247-252.
34. Nakhostin Ansari N, Naghdi S, Hasson S, et al. Efficacy of therapeutic ultrasound and infrared in the management of muscle spasticity. Brain Inj 2009;23:632-638.
35. Sahin N, Ugurlu H, Karahan AY. Efficacy of therapeutic ultrasound in the treatment of spasticity: a randomized controlled study. NeuroRehabilitation 2011;29:61-66.
36. Santamato A, Micello MF, Panza F, et al. Extracorporeal shock wave therapy for the treatment of poststroke plantar-flexor muscles spasticity: a prospective open-label study. Top Stroke Rehabil 2014;21:S17-S24.
37. Moon SW, Kim JH, Jung MJ, et al. The effect of extracorporeal shock wave therapy on lower limb spasticity in subacute stroke patients. Ann Rehabil Med 2013; 37:461-470.
38. Sohn MK, Cho KH, Kim YJ, et al. Spasticity and electrophysiologic changes after extracorporeal shock wave therapy on gastrocnemius. Ann Rehabil Med 2011; 35:599-604.
39. Lee JY, Kim SN, Lee IS, et al. Effects of extracorporeal shock wave therapy on spasticity in patients after brain injury: a meta-analysis. J Phys Ther Sci 2014; 26:1641-1647.
40. Patterson KK, Parafianowicz I, Danells CJ, et al. Gait asymmetry in community-ambulating stroke survivors. Arch Phys Med Rehabil 2008;89:304-310.
41. Bowden JL, Taylor JL, McNulty PA. Voluntary activation is reduced in both the more- and less-affected upper limbs after unilateral stroke. Front Neurol 2014;5:239.
42. Dobkin BH, Firestine A, West M, et al. Ankle dorsiflexion as an fMRI paradigm to assay motor control for walking during rehabilitation. Neuroimage 2004;23:370-381.
43. Mori L, Marinelli L, Pelosin E, et al. Shock waves in the treatment of muscle hypertonia and dystonia. Biomed Res Int 2014;2014:637450.

44. Brunnstrom S. Movement therapy in hemiplegia: A neurophysiological approach. New York: Harper & Row; 1970.
45. Welmer AK, Holmqvist LW, Sommerfeld DK. Hemiplegic Limb synergies in stroke patients. Am J Phys Med Rehabil 2006;85:112-119.
46. Podsiadle D, Richardson S. The timed "up & go": a test of basic functional mobility for frail elderly persons. J Am Geriatr Soc 1991;39:142-148.
47. Flansbjer UB, Holmbback AM, Downham D, et al. Reliability of gait performance tests in men and women with hemiparesis after stroke. J Rehabi Med 2005;37:75-82.
48. Brouwer B, Sousa de Andrade V. The effects of slow stroking on spasticity in patients with multiple sclerosis; a pilot study. Physiother Theory Pract 1995;11:13-21.
49. Manganotti P, Amelio E. Long-term effect of shock wave therapy on upper limb hypertonia in patients affected by stroke. Stroke 2005;36:1967-1971.