European Journal of Physical and Rehabilitation Medicine
EDIZIONI MINERVA MEDICA

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European Journal of Physical and Rehabilitation Medicine 2017 Oct 25
DOI: 10.23736/S1973-9087.17.04801-8

Article type: Original Article

Article first published online: October 25, 2017
Manuscript accepted: October 25, 2017
Manuscript revised: October 13, 2017
Manuscript received: May 4, 2017

Comparison of the effect of focused and radial extracorporeal shock waves on spastic equinus in patients with stroke – A randomized controlled trial

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Abstract:

BACKGROUND: Recent studies have suggested that either focused or radial shock wave therapy is an effect method for the treatment of spasticity in patients with stroke. However, no previous study compared these two types of extracorporeal shock wave on spasticity in patients with stroke. This study aimed to compare the effect of focused and radial shock wave therapy for the treatment of spastic equinus in patients with stroke.

DESIGN: Randomized control trial.

SETTING: Outpatient rehabilitation center in a medical center

POPULATION: 32 stroke patients with spastic equinus (18 males and 14 women; mean age, 60.1±10.6 years).

METHODS**:** Patients were randomly assigned to receive three sessions of either focused or radial shock wave therapy at 1-week intervals. The intensities that were used during focused shock wave therapy (0.12 mJ/mm^2) and radial shock wave therapy (2.4 bar) were comparable. The patients were evaluated at baseline and at 1, 4, and 8 weeks after the final shockwave treatment. The primary outcome measure was change of modified Ashworth scale score of gastrocnemius muscle. The secondary outcome measures were Tardieu scale, ankle passive range of motion, dynamic foot contact area and gait speed. A

linear mixed model with repeated measures was used to compare each outcome measure between the two groups.

RESULTS: Both groups improved significantly in terms of modified Ashworth scale score and Tardieu scale, and no differences were found between the two groups. In terms of ankle passive range of motion and plantar contact area during gait, the radial shock wave therapy yielded a significantly greater improvement than the focused shock wave therapy. No significant changes were observed in gait speed in either group.

CONCLUSIONS: Our study suggested that focused and radial shock wave therapy resulted in similar significant improvements in the modified Ashworth scale score and Tardieu scale, but those in the radial shock wave therapy group experienced greater improvements in the ankle passive range of motion and plantar contact area during gait.

CLINICAL REHABILITATION IMPACT: Both focused and radial shock wave therapy yielded similar improve the spasticity of gastrocnemius muscle. Radial shock wave therapy is superior to focused shock wave therapy in terms of improving the ankle passive range of motion and plantar contact area during gait in patients with stroke.

Keyword: High Energy Shock Waves, Muscle Spasticity, Equinus Deformity, Stoke

Introduction

Stroke is one of the leading causes of adult disability in most countries.[1, 2] Spasticity is a common condition in patients with stroke, characterized as velocity-dependent increase in muscle tone with exaggerated tendon reflexes.[3, 4] Spastic equinus, a common pattern of spasticity in patients with stroke, is primarily due to triceps surae spasticity (gastrocnemius and soleus muscles) and can reduce mobility and change gait pattern with increased risk of falls.[5]

Current management of spastic equinus includes physiotherapy, anti-spastic medication, chemical neurolysis, botulinum toxin injections, selective neurotomy, and orthopedic surgery. [6] Physical modalities are the first step in the treatment of spastic equinus because of availability and fewer side effects.[7] Extracorporeal shock wave therapy, a novel physical modality, involves a sequence of single sonic pulses with high peak pressure, fast pressure rise, and short duration conveyed through a generator to the target area, providing effective treatment. [8] Extracorporeal shock wave therapy has been reported to be valuable in the treatment regimen of various neurological and musculoskeletal conditions. Several studies have reported a favorable effect of extracorporeal shock wave therapy on spasticity in patients with cerebral palsy, [9, 10] multiple sclerosis[11] and stroke.[12-16] Some studies have also found that

extracorporeal shock wave therapy can enhance the effect of botulinum toxin type-A injection treatment on post-stroke spasticity.[17] A meta-analysis reported that extracorporeal shock wave therapy had a significant effect on improving spasticity 4 weeks after treatment in patients with brain injury.[18]

Based on the propagation pattern of the wave, extracorporeal shock wave therapy can be classified into two main modalities: focused and radial shock wave therapy.[19] The waves from focused shock wave therapy are generated from the probe and converge to the target area. In contrast, radial shock wave devices develop their maximum energy at the probe tip and distribute it radially into the tissue. Recent studies have suggested that either focused or radial shock wave therapy is effective in reduction of spasticity in patients with stroke.[12-15] To date, no research has compared the effect of these two types of extracorporeal shock wave on spasticity in patients with stroke.

The purpose of our study was to compare the effects of focused and radial shock wave therapy on spastic equinus foot in patients with chronic stroke as measured by the change in muscle spasticity and gait pattern.

Methods

Between January 2015 and January 2016, 32 chronic stroke patients with spastic equinus foot were recruited from the rehabilitation department of a

tertiary hospital in Taiwan. Eligible subjects met the following criteria: (1) aged 18 years and older; (2) time from stroke onset of at least 6 months; (3) with stable spasticity in the triceps surae muscle group (muscle tone grade at least 1+ on the Modified Ashworth Scale (MAS) score); and (4) ability to walk alone, with or without an orthosis. Exclusion criteria included participation in other trials, fixed ankle joint contracture (MAS grade 4), previous treatment of the affected leg with phenol, alcohol, botulinum toxin injection or surgical procedures, concomitant progressive central nervous system diseases, skin breakdown, sensation deficits and vascular disease in the affected leg. The regimens and dosages of anti-spasticity medication were not adjusted and the rehabilitation program of the target area remained unchanged from 2 months prior to participation to the end of the follow-up period. All patients were outpatients and provided their informed consent to participate in the study. The study protocol was reviewed and approved by the Institutional Review Board of Mackay Memorial Hospital (No.15MMHIS194e) and was registered on ClinicalTrial.gov (identifier: NCT03129529).

After completing the baseline screening and evaluation, eligible patients were randomly assigned to either focused or radial shock wave treatment group in a 1:1 ratio. A permuted-block randomization procedure with block size 4 was

used to generate an allocation sequence. An investigator not involved in the subject recruitment developed a computer-generated random allocation schedule and placed the assignments in sealed, sequentially numbered envelopes. As each patient entered the trial, the next envelope in the sequence was opened, indicating which treatment the patient would undergo. Treatment allocation was concealed from the subjects and the outcome assessors during the trial.

The extracorporeal shock wave therapy was performed by the same physiatrist, who was not involved in baseline evaluation and further follow-up assessment. Shock wave treatment was applied with the Duolith SD1 device (Storz medical, Tagerwilen, Switzerland) that can deliver both electromagnetically generated focused or pneumatically driven radial extracorporeal shock waves. Treatment was administered directly to the middle of the muscle bellies of the spastic triceps surae muscle in three sessions, with one-week interval. During each session, 3000 pulses (1500 shots in the gastrocnemius muscle and 1500 shots in the soleus muscle) were delivered at 5 Hz. The intensities that were used during focused shock wave therapy (0.10 mJ/mm^2) and radial shock wave therapy (2.0 bar) were comparable.[20]

Clinical assessments were performed before the first shock wave treatment and at 1, 4, and 8 weeks after the last session of treatment by a different investigator who was blinded to the treatment allocation. Adverse events were monitored during the study and documented during treatment and at follow-up visits.

The primary outcome measure was the change of MAS scores at the gastrocnemius muscle from baseline to week 8. The secondary outcome measures included the Tardieu angles, ankle passive range of motion (PROM), dynamic foot plantar contact area on the affected foot, gait speed, and adverse events.

For the MAS and Tardieu angles measurements, patients lay supine with the knee in full extension and the subtalar joint stabilized. The MAS is a six-point ordinal scale of muscle tone, measuring resistance during passive muscle stretching. Scores range from 0 (no increase in muscle tone) to 4 (rigid) and include a rating of 1+.[21] For the convenience of statistical analysis, a MAS grade 1+ was matched to 2 points; and grade 2, 3, and 4 were matched to 3, 4, and 5 points, respectively. The Tardieu angles measure muscle spasticity by two stretch maneuvers: performed as slow and as fast as possible. The Tardieu angle is the difference between the arrest angle at slow speed and the

catch angle at fast speed.[22] Ankle PROM was measured using a hand-held goniometry. The degrees of dorsiflexion from the joint's neutral position were recorded as a positive number and the degrees of plantar flexion as a negative number. Dynamic plantar contact area on the affected foot was recorded during level barefoot walking using the Tekscan platform (Boston, MA, USA). Patients were instructed to walk at their own comfortable walking speed and to continue walking past the platform. The mean plantar contact area on the affected foot was calculated from the Tekscan software.[23] The gait speed with or without aids was measured using the 10-m walk test. Patients were instructed to walk 10 m as quickly and safely as possible. The time taken to walk was timed with a stopwatch and was expressed as meters per second.

Data analysis

Sample size calculations were based on detecting 1 point in the MAS score between the two types of extracorporeal shock wave therapy, assuming a standard deviation of 0.7, a two-tailed test, an alpha level of .05, a drop-out rate of 20%, and a desired power of 90%.[14, 24] Based on these calculations, the minimum sample size was estimated to be 16 patients per group.

Data were expressed as mean ± standard deviation (SD) or median with interquartile range for continuous variables as appropriate and as proportions

for categorical variables. Baseline demographic variables were compared between treatment groups using independent *t* tests or Mann-Whitney U test for continuous data and chi-square tests of independence for categorical data. Linear mixed model analyses for repeated measures were performed for each of the outcome measures to evaluate changes over time and between groups. The mixed model included group (focused vs. radial shock wave therapy) and time (four time points of measurement) as fixed effects and subjects as random effect. The difference between two groups over time was studied by an interaction term of group with the time variable. Planned contrast tests were used to compare the difference in outcome variables at each follow-up assessment. The changes in outcome variables between baseline and each follow up were analyzed using Wilcoxon signed-rank test. All statistical analyses were performed using SAS, version 9.2 (SAS, Cary, NC, USA). A P-value of 0.05 was considered statistically significant.

Results

Figure 1 shows the study flowchart. Of the 37 patients screened, 32 patients were randomly assigned with 16 patients each in the focused and radial shock wave therapy groups. One patient in the focused shock wave therapy group refused treatment and was excluded from the analysis. The demographic and

clinical characteristics of the remaining 31 patients are summarized in Table 1. No significant difference was found between the focused and radial shock wave therapy groups with regard to age, sex, weight, height, stroke type, affected side, and onset duration before treatment. No adverse events, such as skin petechiae, muscle hematoma, and focal edema were reported during the study period.

Table 2 presents the primary and secondary outcome variables by point of measurement for both groups. For the MAS score and Tardieu angle, no significant group-by-time interaction was observed. Neither were significant differences in the MAS scores nor spastic angles found between two groups at any of the measured time points from one to eight weeks after the treatment sessions. However, the MAS score and spastic angle were significantly lower at one, four, and eight weeks after treatment sessions than at baseline in both groups.

Statistical analysis showed significant group-by-time interactions for Ankle PROM ($P<0.001$) and the dynamic foot plantar contact area ($P=0.003$), with patients who underwent radial shock wave therapy, experiencing greater improvement on ankle PROM and dynamic plantar contact area compared with those in the focused shock wave therapy group. Contrast testing indicated

that radial shock wave therapy yielded greater increase in ankle PROM and dynamic foot plantar contact area at one and four weeks post-treatment. Compared with baseline, both groups resulted in significantly greater ankle PROM and dynamic foot contact area at one, four, and eight weeks after treatment sessions (Table 3). With regard to the 10-m walk test, no significant change was found between baseline and post-treatment in the two groups.

Discussion

The current study found that both focused and radial shock wave therapy can yield significant reduction of spasticity, improvement in ankle PROM and dynamic plantar contact area on the affected foot in stroke patients with spastic equinus foot. However, no significant difference was observed in changes for either MAS scores or Tardieu angles between the two groups. Our study also suggested that radial shock wave therapy seems to yield greater improvement in ankle PROM and dynamic plantar contact area on the affected foot. No significant improvements were observed in gait speed in two groups.

Spasticity is a common and disabling condition in patients with stroke. Current managements of post-stroke spasticity can often be challenging and unsatisfactory for some patients and clinicians. Extracorporeal shock wave therapy has been suggested to be a novel and effective method for improving

spasticity in patients with stroke. The possible mechanism accounting for this effect might be related to a direct effect of shock wave on the rheological properties of the spastic muscle in patients with stroke. Shock wave pressure can break the functional link between actin and myosin, reducing the intrinsic stiffness of connective tissue in spastic muscle.[25, 26] The soft tissue change cause the pulling forces to be transmitted more readily to the muscle spindles that can decrease spinal cord excitability and reduce spasticity. In addition, focal vibration through the shock wave on a tendon or muscle preferentially activates primary spindle afferent fibers, resulting in inhibition of the monosynaptic reflex. Several studies have demonstrated a reduced inhibition of the H-reflex and a decrease in spasticity due to vibratory stimuli in stroke patients.[27, 28]

Based on the propagation pattern of the wave, extracorporeal shock wave therapy can be classified as focused and radial shock wave therapy. Previous studies have demonstrated that either focused or radial shock wave therapy has a benefit effect on spasticity in patients with stroke. Manganotti and Amelio[13] found that wrist and finger spasticity were significantly decreased after one session of focused shock wave therapy. The therapeutic effect of focused shock wave therapy on upper limb spasticity could last at least 4

weeks, maximally at 12 weeks. Moon et al. conducted a cross-over study and found significant improvement in the spasticity and ankle PROM after three sessions of focused shock wave therapy for stroke patients.[14] For studies regarding radial shock wave therapy on post-stroke spasticity, Daliri et al.[12] stimulated the flexor carpi ulnaris and flexor carpi radialis muscles with one session of radial shock wave therapy. The authors concluded that radial shock wave therapy improved post-stroke spasticity of the wrist flexor muscles and motor neuron excitability. Similarly, Li et al. found a significant reduction in spasticity of the hand and wrist after radial shock wave therapy. Three sessions of radial shock wave therapy had a more long-lasting effect than one session.[15] Radial extracorporeal shock waves differ from focused extracorporeal shock waves by penetration depth and certain physical properties. Radial shock waves lack the characteristic features of shock waves, such as short rise-time, high peak pressure, and non-linearity.[29] In addition, radial shock wave had a more superficial effect compared with focused shock waves that penetrated and can focus their energies much deeper into the tissue.[30] The differences between radial and focused shockwaves related to clinical difference are unclear. Although, both focused and radial shock wave therapy had a significant decrease in spasticity in patients with stroke in the present study, no

between-group differences were observed.

Previous studies had showed that either focused or radial shock wave therapy could increase in ankle passive range of motion, and the plantar contact area on the affected side. This may be due to continuous or intermittent shock wave pressure breaking the functional link between actin and myosin, reducing intrinsic stiffness of connective tissue, and increasing extensibility of the gastrocnemius muscle. Moreover, shock wave may lead to local release of angiogenetic factors and growth factors, resulting in the addition of sarcomere and increased muscle length.[31, 32] Both change the stiffness of the periarticular tissue and increased muscle length can result in increased ankle passive range of motion and the plantar contact area on the affected side. Our results are in accordance with the results of previous studies and found that both focused and radial shock wave therapy were beneficial in increasing ankle passive range of motion and foot contact area,[33, 34] but the radial shock wave therapy produced a greater benefit. A possible explanation for this might be that radial shock wave therapy is characterized by having a broader therapeutic area and high energy in the superficial tissue as opposed to focused shock wave therapy.[19] Hence, radial shock wave therapy might affect the mechanical properties of the whole muscle belly rather than a small spot in

the muscle and yield greater increased in ankle passive range of motion and plantar contact area.[35]

With regard to walking speed, some studies have found that extracorporeal shock wave therapy can improve walking speed in patients with cerebral palsy.[10] In contrast, neither focused nor radial shock wave therapy improved walking speed in our study. However, previous studies suggested that lower limb muscle strength and coordination pattern rather than spasticity are major contributing factors to walking speed in patients with stroke.[36, 37] Further larger studies are warranted to investigate the effects of extracorporeal shock wave therapy on the walking speed in patients with stroke.

Our study has some potential limitations. First, our study design lacked a sham or non-intervention control group. Therefore, the beneficial effect of either focused or radial shock wave therapy simply due to natural recovery cannot be ruled out. However, the magnitude of improvement makes spontaneous recovery unlikely. In addition, other studies have already shown that either focused or radial shock wave therapy had the advantage over placebo for the treatment of spasticity in patients with stroke. Second, the treatment intensity and sessions were based on previous relevant studies. We do not know if a greater number of sessions or treatment intensity would have revealed greater

changes in outcomes or differences between the two groups. Third, our study was adequately powered for the primary outcome (the MAS score) but underpowered for several secondary outcomes. It is therefore doubtful that the effect of shock wave therapy on walking speed might have been observed even with larger sample studies. Fourth, we did not evaluate the structural and mechanical alterations in the spastic muscle. Some studies found that patients with higher spastic muscle echo-intensity may have a reduced response to anti-spastic treatment. [15, 38] Further studies are needed to investigate this issue. Finally, the generalizability of the study may be limited by the data from a single institution.

Despite these limitations, our study is the first to directly compare the effect on post-stroke equinus following focused and radial shock wave therapy applied with identical energy flux densities. The current results add to the growing evidence that focused and radial shock wave therapy are effective and safe modalities in treating post-stroke spasticity. These results may also assist authors of future studies in providing specific recommendations regarding the use of extracorporeal shock wave therapy in patients affected by spasticity.

In summary, both focused and radial shock wave therapy may effectively improve muscle spasticity, ankle PROM, and plantar contact area in chronic

stroke patients with equinus. The radial shock wave therapy is likely associated with greater improvement in ankle PROM and plantar contact area.

Funding

This study was supported by research grants from the Ministry of Science and Technology, in Taiwan. (MOST-105-2314-B-195-001)

Declaration of interests

The authors report no conflicts of interests.

Acknowledgements

The authors express thanks to the participants in the study.

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Legends:

Figure 1 CONSORT flow diagram of recruitment, allocation, and participation of study

Table I. Demographic and Clinical Characteristics at Baseline

Table II. Outcome variables at baseline and each follow-up assessment in both groups

Table III. Outcome variables change from baseline (mean, 95% CIs) to each follow-up assessment in both groups

Table I. Demographic and Clinical Characteristics at Baseline

	FSWT group	RSWT group	P-value
Variables	N, 15	N,16	
Age (years)	60.3± 9.9	59.6± 11.3	0.65
Weight (Kg)	63.3± 13.6	64.2± 12.4	0.85
Height(cm)	161.7± 5.6	162.7± 7.4	0.67
Time since stroke onset (months)	53.2± 26.7	55.7± 26.1	0.79
Gender			0.83
Female	6 (40%)	7 (43.8%)	
Male	9 (60%)	9 (56.2%)	
Stroke subtype			0.81
Hemorrhage stroke	5 (33.3%)	6 (37.5%)	
Ischemic stroke	10 (66.7)	10 (62.5%)	
Affected site			0.59
Left	8 (53.3%)	7 (46.7%)	
Right	7 (43.7%)	9 (56.3%)	

Continuous data are presented as mean± standard deviations and categorical data as number (percent).

Abbreviation, FSWT, focused shock wave therapy; RSWT, radial shock wave therapy

Table II Outcome variables at baseline and each follow-up assessment in both groups

	Time point	FSWT group		ESWT group		*P-value		
		Mean ± SD	Median (IQR)	Mean ± SD	Median (IQR)	Time	Group	Interaction
MAS	Baseline	3.0± 0.7	3 (3 to 4)	3.1± 0.7	3 (3 to 4)	<0.001	0.9	0.38
	1-week follow-up	2.5 ± 0.7	2 (2 to 3)	2.3± 0.5	2 (2 to 3)			
	4-week follow-up	1.9 ± 0.5	2 (2 to 2)	1.8 ± 0.5	2 (2 to 2)			
	8-week follow-up	2.1 ± 0.7	2 (2 to 3)	2.2 ± 0.7	2 (2 to 3)			
Tardieu angle (degrees)	Baseline	21.6± 8.3	19.5 (16.0 to 23.0)	20.0 ± 2.7	19.5 (17.0 to 22.0)	<0.001	0.55	0.24
	1-week follow-up	16.1 ± 8.1	16.0 (9.0 to 21.0)	13.0 ± 5.5	11.5 (8.5 to 16.5)			
	4-week follow-up	14.0 ± 6.5	17.0 (8.5 to 18.0)	13.9 ± 4.8	14.5 (10.0 to 17.0)			
	8-week follow-up	14.2 ± 5.2	14.0 (10.5 to 18.0)	14.8 ± 4.8	15.0 (11.5 to 18.0)			
Ankle PROM (degrees)	Baseline	25.6 ± 8.9	24.0 (20.5 to 35.0)	25.2 ± 7.3	23.0 (20.0 to 29.5)	<0.001	0.15	<0.001
	1-week follow-up	31.4 ± 8.6	29.5 (25.0 to 39.0)	38.7 ± 8.9	39.0 (33.0 to 44.0)			
	4-week follow-up	32.9 ± 9.9	29.0 (26.0 to 39.0)	40.0 ± 8.4	34.0 (28.5 to 42.0)			
	8-week follow-up	29.3 ± 9.4	30.0 (24.0 to 35.0)	31.3 ± 5.9	32.0 (27.5 to 34.0)			
Dynamic foot contact area (cm^2)	Baseline	47.1 ± 17.1	41.5 (35.4 to 59.5)	50.0 ± 10.9	52.7 (45.5 to 57.1)	<0.001	0.22	0.003
	1-week follow-up	55.5 ± 18.5	46.1 (43.3 to 74.3)	62.9 ± 12.8	63.1 (54.3 to 71.3)			
	4-week follow-up	53.8 ± 17.3	50.0 (42.5 to 74.8)	64.2 ± 13.2	68.6 (52.5 to 73.6)			
	8-week follow-up	54.0 ± 18.3	50.0 (42.0 to 72.1)	59.5 ± 11.8	63.0 (56.0 to 69.0)			
10-meter walk test (second)	Baseline	24.3 ± 19.8	22.5 (13.3 to 35.0)	22.7± 14.6	21.7 (11.6 to 32.5)	0.7	0.07	0.55
	8-week follow-up	23.1 ± 18.2	21.0 (11.5 to 33.5)	20.2 ± 12.8	19.5 (12.5 to 31.5)			

Abbreviation, FSWT, focused shock wave therapy; RSWT, radial shock wave therapy, SD, standard deviation, IQR, interquartile range, MAS, Modified Ashworth Scale, PROM, passive range of motion.

*P-value was obtained by linear mixed model

†The 6-point MAS was modified so that a MAS grade 1+ was matched to 2 points; and grade 2, 3, and 4 were matched to 3, 4, and 5 points, respectively.

Table III. Outcome variables change from baseline (mean, 95% CIs) to each follow-up assessment in both groups

	Time interval	FSWT group	P-value*	ESWT group	P-value*
Modified Ashworth Scale					
	Week 1 - Baseline	-0.53 (-0.99, -0.07)	0.05	-0.87 (-1.20, -0.54)	<0.001
	Week 4 - Baseline	-1.13 (-1.48, -0.77)	<0.001	-1.31 (-1.56, -1.05)	<0.001
	Week 8 - Baseline	-0.87 (-1.26, -0.46)	<0.001	-0.93 (-1.17, -0.71)	<0.001
Tardieu angle (degrees)					
	Week 1 - Baseline	-5.45 (-8.29, -2.62)	0.002	-6.98 (-10.19, -3.77)	<0.001
	Week 4 - Baseline	-7.59 (-12.17, -3.00)	<0.001	-6.11 (-9.02, -3.20)	<0.001
	Week 8 - Baseline	-7.35 (-11.25, -3.46)	<0.001	-5.15 (-8.17, -2.14)	0.004
Ankle PROM (degrees)					
	Week 1 - Baseline	5.87 (3.28, 8.47)	<0.001	13.53 (10.75, 16.31)	<0.001
	Week 4 – Baseline	7.36 (4.07, 10.66)	<0.001	9.80 (6.75, 12.85)	<0.001
	Week 8 - Baseline	3.78 (0.16, 7.41)	0.01	6.16 (3.61, 8.07)	<0.001
Dynamic plantar contact area (cm^2)					
	Week 1 - Baseline	8.35 (6.14, 10.56)	<0.001	12.77 (9.45, 16.09)	<0.001
	Week 4 - Baseline	6.68 (3.83, 9.54)	<0.001	14.10 (10.18, 18.02)	<0.001
	Week 8 - Baseline	6.84 (4.52, 9.17)	<0.001	12.48 (9.49, 15.48)	<0.001
10-meter walk test (second)					
	Week 8 - Baseline	-1.25 (-2.73, 0.22)	0.15	-2.45 (-6.37, 1.47)	0.25

Abbreviation, FSWT, focused shock wave therapy; RSWT, radial shock wave therapy; CI, confidence interval, PROM, passive range of motion

* P-value was obtained by Wilcoxon rank-sum test

CONSORT 2010 checklist of information to include when reporting a randomised trial*

Section/Topic	Item No	Checklist item	Reported on page No
Title and abstract			
	1a	Identification as a randomised trial in the title	Page.1
	1b	Structured summary of trial design, methods, results, and conclusions (for specific guidance see CONSORT for abstracts)	Page.2-3
Introduction			
Background and objectives	2a	Scientific background and explanation of rationale	Page.5-6
	2b	Specific objectives or hypotheses	Page 6, Line 14
Methods			
Trial design	3a	Description of trial design (such as parallel, factorial) including allocation ratio	Page 7, Line 19
	3b	Important changes to methods after trial commencement (such as eligibility criteria), with reasons	No change
Participants	4a	Eligibility criteria for participants	Page 7, Line 1-9
	4b	Settings and locations where the data were collected	Page 6, Line 19
Interventions	5	The interventions for each group with sufficient details to allow replication, including how and when they were actually administered	Page 8, Line 8-19
Outcomes	6a	Completely defined pre-specified primary and secondary outcome measures, including how and when they were assessed	Page 9, Line 6-19 Page 10, Line 1-11
	6b	Any changes to trial outcomes after the trial commenced, with reasons	No change
Sample size	7a	How sample size was determined	Page 10, Line 13-17
	7b	When applicable, explanation of any interim analyses and stopping guidelines	No interim analyses
Randomisation:			
Sequence generation	8a	Method used to generate the random allocation sequence	Page 8, Line 2
	8b	Type of randomisation; details of any restriction (such as blocking and block size)	Page 7, Line 19
Allocation concealment mechanism	9	Mechanism used to implement the random allocation sequence (such as sequentially numbered containers), describing any steps taken to conceal the sequence until interventions were assigned	Page 8, Line 1-7
Implementation	10	Who generated the random allocation sequence, who enrolled participants, and who assigned participants to interventions	Page 8, Line1,

Blinding	11a	If done, who was blinded after assignment to interventions (for example, participants, care providers, those assessing outcomes) and how	Page 9, Line 2-3
	11b	If relevant, description of the similarity of interventions	Page 8, Line 16
Statistical methods	12a	Statistical methods used to compare groups for primary and secondary outcomes	Page 11, Line 1-11
	12b	Methods for additional analyses, such as subgroup analyses and adjusted analyses	No subgroup or adjusted analysis
Results			
Participant flow (a diagram is strongly recommended)	13a	For each group, the numbers of participants who were randomly assigned, received intended treatment, and were analysed for the primary outcome	Figure I
	13b	For each group, losses and exclusions after randomisation, together with reasons	Page 11, Line 19
Recruitment	14a	Dates defining the periods of recruitment and follow-up	Page 6, Line 18
	14b	Why the trial ended or was stopped	No stopped
Baseline data	15	A table showing baseline demographic and clinical characteristics for each group	Table 1
Numbers analysed	16	For each group, number of participants (denominator) included in each analysis and whether the analysis was by original assigned groups	Page 11, Line 18-19
Outcomes and estimation	17a	For each primary and secondary outcome, results for each group, and the estimated effect size and its precision (such as 95% confidence interval)	Table II
	17b	For binary outcomes, presentation of both absolute and relative effect sizes is recommended	Nil
Ancillary analyses	18	Results of any other analyses performed, including subgroup analyses and adjusted analyses, distinguishing pre-specified from exploratory	Nil
Harms	19	All important harms or unintended effects in each group (for specific guidance see CONSORT for harms)	Page
Discussion			
Limitations	20	Trial limitations, addressing sources of potential bias, imprecision, and, if relevant, multiplicity of analyses	Page 17, Line 11-19 Page 18, Line 1-10
Generalisability	21	Generalisability (external validity, applicability) of the trial findings	Page 18, Line 9-10
Interpretation	22	Interpretation consistent with results, balancing benefits and harms, and considering other relevant evidence	Page 13 Line 8-19
Other information			
Registration	23	Registration number and name of trial registry	Page 7, Line 16
Protocol	24	Where the full trial protocol can be accessed, if available	https://clinicaltrials.gov
Funding	25	Sources of funding and other support (such as supply of drugs), role of funders	Page 19, Line 4-5