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ORIGINAL ARTICLE



Extracorporeal Shockwave Therapy on Spasticity after Central Nervous System Injury: A Systemic Review and Meta-Analysis

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Background: Spasticity is a disorder characterized by velocity dependently increasing in the tonic stretch reflexes (muscle tone). There were variable managements for spasticity. Treatment of spasticity depends on the severity, involved part, and patient's and families' preference. However, there were more trials completed in studying the efficacy of extracorporeal shockwave therapy (ESWT) in treating spasticity in different disease. Aim: The goal of our study is to evaluate the efficacy of ESWT in treating of spasticity after central nervous system lesions and to analyze the influences of related factors; we performed a systemic review to survey the effect. Methods: We performed a thoroughly systematic review and meta-analysis. Results: Totally 9 studies were included 4 studies examined the spasticity in stroke group, 4 studies were in cerebral palsy, and 1 study in multiple sclerosis. Regarding the effect of spasticity reduction in overall populations, the pooled effect showed that the modified Ashworth scale grade reduction compared with the baseline values were standardized mean difference (SMD): -4.07 (95% confidence interval (CI), -5.37 - 2.76; P < 0.001) immediately after ESWT, SMD: -2.51 (95% CI, -3.40 - 1.62; P < 0.001) after 4 weeks, and SMD: -1.44 (95% CI, -1.92-0.95; P < 0.001) after 12 weeks. In terms of the disease types, the SMD in stroke patients was SMD: -4.03 (95% CI, -5.44–-2.61; Z = 5.57; P < 0.001) immediately after ESWT, SMD: -2.34 (95% CI, -3.01–-1.66; Z = 6.80; P < 0.001) after 4 weeks and SMD: -1.50 (95% CI, -2.06 - 0.93; Z = 5.20; P < 0.001) after 12 weeks. No significant adverse events were found. Conclusion: The present meta-analysis revealed that ESWT effectively alleviates spasticity in patients after upper motor neuron lesions, regardless of disease type and parts treated. Both radial and focus ESWTs could decrease spasticity, regardless of the treatment session. The result could last for 12 weeks after treatment. Moreover, no serious side effects were observed after ESWT. Further studies with randomization and more parameters of ESWT were advised to setup to improve the clinical effectiveness.

Key words: Extracorporeal shock wave therapy, spasticity, meta-analysis

INTRODUCTION

Spasticity is a disorder characterized by velocity dependently increasing in the tonic stretch reflexes (muscle tone), along with exaggerated tendon jerks, which were resulting from the hyperexcitability of the stretch reflex.^{1,2} It is a presentation of upper motor neuron syndrome. There are multiple mechanisms to explain the development of spasticity after upper motor neuron lesions, and the core concept is that

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the imbalance of supraspinal inhibitory and excitatory inputs directed to the spinal cord, which leads to the hyperexcitability of the stretch reflex.³

Spasticity is a common complication following central nerve system lesions, such as in stroke, cerebral palsy (CP), traumatic brain injury, multiple sclerosis, and spinal cord injury. In stroke, the prevalence of spasticity has a wide range, which from 19% to 92% and is variable at different timing after

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stroke.⁴⁻⁶ Spasticity has both positive and negative effects. In the recovery phase, spasticity may provide the weight-bearing support during standing, ambulation, or even functional movements. However, it is also a major cause of disability and affecting health-related quality of life in patient with central nervous system (CNS) lesions.^{7,8} The disabled factors from motor impairment and spasticity after stroke include paresis, muscle overactivity, and soft-tissue contracture.^{9,10}

There were variable managements for spasticity, ranged from the conservative treatment to invasive procedure, including physical therapy, positioning, oral medications, chemical neurolysis, and surgical interventions.¹¹ Nevertheless, the treatment goals are to improve function, decrease symptoms and care burden and improve health-related quality of life.¹¹

The treatment of spasticity depends on the severity, involved part, and patient's and families' preference. Although several managements existed for treating spasticity after CNS lesions, the adverse effects may be a consideration in developing the new methods, especially for those with chronic spasticity and need repeated treatments. The oral antispastic medications may have a systemic side effect such as lethargy or drowsy, while it indeed reduces the muscle tone after use.12 Prolonged use of medication may develop drug tolerance. The use of chemodenervation, for instance, the phenol blocks that may cause sensory loss and dysesthesia.¹³ Botulinum toxin is a widely used treatment in managing spasticity, however, repeated injection of the toxin may induce the formation of neutralizing antibodies, 14-16 which could profound reduce the therapeutic effect after injection. Extracorporeal shockwave therapy (ESWT) was initially applied for treating urolithiasis, ¹⁷ and currently be a newly developed noninvasive modality in treating spasticity. It also had been used and studied in recent decade.

ESWT produces high-energy shock from the sequence of single acoustic pulses, with high peak pressure, short duration, rapid increasing of pressure and transmission.¹⁸ The types of ESWT can be divided into focus or radial, according to the physical characteristic and generating technique. It has been widely used in musculoskeletal diseases, such as calcified tendinitis of shoulder,¹⁹ plantar fasciitis,²⁰ lateral epicondylitis of elbow,²¹ and nonunion bone fracture.²² The effects from ESWT were believed to be a result of mechanotransduction, which is the process that mechanical stimuli transformed into downstream chemical signals.²³

In the previous review article, there have been two meta-analyses that discuss the use of ESWT in treating spasticity after brain injury or limited to stroke.^{24,25} These articles suggested the ESWT had a significant improvement in spasticity when using modified Ashworth scale (MAS) as an evaluation tool. ESWT was considered to be a safe treatment

for cases of spasticity after stroke. However, there were more trials completed in studying the efficacy of ESWT in treating spasticity in different diseases. The goal of our study is to evaluate the efficacy of ESWT in treating of spasticity after CNS lesions and to analyze the influences of related factors; we performed a systemic review to survey the effect.

MATERIALS AND METHODS

Search strategy

We searched two electronic databases, PubMed and Embase, from the earliest record to October 2022 for relevant articles. We also searched the Cochrane Collaboration Central Register of Controlled Clinical Trials, Cochrane Systematic Reviews, ClinicalTrials.gov for suitable references. Moreover, we manually searched the reference lists of the included studies for possible relevant trials. The key terms extracorporeal shockwave therapy, spasticity, and muscle hypertonia were entered as the medical subject heading and text words for searching.

The complete search strategy is visualized in Appendix 1.

Study selection

The detailed inclusion criteria were: (1) the study enrolled patients with spasticity development after CNS injury, including stroke, CP, multiple sclerosis, traumatic brain injury, or spinal cord injury, (2) study design with a controlled group, including randomized controlled trial (RCT) and non-RCT, (3) the outcome measurements included the MAS and the follow-up timing at least 4 weeks or 1 month, and (4) publications in English with full-length text available. Studies with comparing to any treatment other than sham or studied on animal were not included in the present meta-analysis.

Data extraction and quality assessment

The eligible articles were evaluated by two authors independently. Data of patients' demographic information (age, female/male ratio, onset time), disease types, type and regimen of ESWT, treat site, and MAS were recorded by two authors in dual using the same form.

The methodological quality of the enrolled studies was assessed using the Cochrane risk-of-bias tool in RCT. The risk of bias was classified into high, low, or unclear²⁶ and using Newcastle–Ottawa Scale for evaluation nonrandomized trial.²⁷

Quality assessment was based on the following aspects: sequence generation (selection bias), allocation concealment (selection bias), blinding of patients and personnel (performance bias), blinding of outcome assessment (detection bias), incomplete outcome data (attrition bias), selective outcome reporting (reporting bias), and other

sources of bias. Disagreements between the two evaluators during the assessment of the selected trials were solved by discussions or judgment by the third author.

Data synthesis and analysis

The grading of MAS is the only outcome used to evaluate the spasticity. The MAS grading was extracted in the following point: baseline, immediate, 4-week, and 12-week after ESWT, and was presented in mean \pm standard.

Due to relative small studies could be obtained in RCTs; the pooling of RCTs and non-RCTs was to maximize the study participants. Since the evaluation method in non-RCTs used self-control with before and after design, we used the standardized mean difference (SMD) of MAS between pretreatment and posttreatment. Whether the effect was influenced by the disease type, type of ESWT, treatment session, or treatment site was evaluated by subgroup analysis. A random effects model was applied to the statistical method as we observed the significant heterogeneity existed (P < 0.1 and an $I^2 > 50\%$).

A sensitivity test was used to investigate the influence of individual study, by excluding one of the studies each time and reconducting a meta-analysis to explore the individual bias. The visualization of the funnel plot and Begg's test were used to assess the potential publication biases.

The analyses were conducted using Review Manager 5.3 (Cochrane Collaboration, Oxford, UK), and a P < 0.05 was considered statistically significant. Begg's test and sensitivity analysis were estimated by STATA 12.0 (StataCorp LP, College Station, TX).

RESULTS

Study search

The primary search screened 58 studies by database searches. After the exclusion of duplicated studies and screening by titles and abstracts, 11 studies were eligible. Nine studies were included for final detailed evaluations. Two studies were excluded additionally: one study used the inconsistent evaluation scale for spasticity, while another study provided insufficient data in the article [Figure 1].

In the 9 studies, 4 studies examined the spasticity in the stroke group, 4 studies were in CP, and 1 study in multiple sclerosis. The type and related parameters of ESWT, intervention site, follow-up time, and adverse effects are listed in Table 1.

The studies enrolled in this meta-analysis included RCT and non-RCT, therefore the quality assessment was performed using the Cochrane risk of bias tool in the RCT and Newcastle–Ottawa Scale in non-RCT [Table 2].

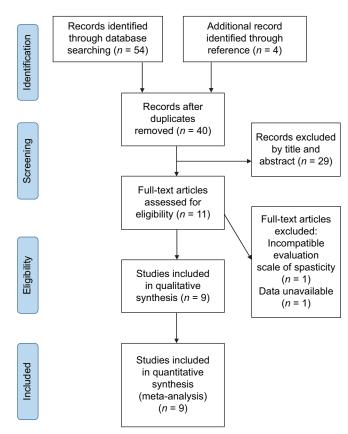


Figure 1: PRISMA flowchart

Standardized mean differences in overall population and different disease

The pooled SMDs of spasticity reduction in overall populations and according to the disease types immediately, after 4 and 12 weeks are detailed in Figure 2. Regarding the effect of spasticity reduction in overall populations, the pooled effect showed that the MAS grade reduction compared with the baseline values were SMD: -4.07 [95% confidence interval (CI), -5.37–-2.76; Z = 6.11; P < 0.001, Figure 2a] immediately after ESWT, SMD: -2.51 [95% CI, -3.40–-1.62; Z = 5.52; P < 0.001, Figure 2b] after 4 weeks, and SMD: -1.44 [95% CI, -1.92–-0.95; Z = 5.84; P < 0.001, Figure 2c] after 12 weeks.

Figure 3 represents the results of different disease types. In terms of the disease types, the SMD in stroke patients was SMD: -4.03 (95% CI, -5.44—-2.61; Z=5.57; P<0.001) immediately after ESWT, SMD: -2.34 (95% CI, -3.01—1.66; Z=6.80; P<0.001) after 4 weeks and SMD: -1.50 (95% CI, -2.06—0.93; Z=5.20; P<0.001) after 12 weeks. While in the CP populations, the pooled SMD was -6.06 (95% CI, -11.54—-0.57; Z=2.16; P<0.001) immediately after ESWT, SMD: -2.44 (95% CI, -4.22—-0.66; Z=2.69; P<0.001) after 4 weeks and SMD: -1.12 (95% CI, -1.51—-0.73; Z=5.63; P<0.001) after 12 weeks. The multiple sclerosis included only one study.

Study	Design	Enroll participants (n)		Population Average age (year)	Site	Onset (months)	Type	Energy/ pressure	Dosage and frequency	Session/interval	Follow up timing	Adverse effect
Amelio and Manganotti ²⁸ 2005	Self- control	20	Stroke	63 (38-76)	Forearm and hand	>9.00	Focused 0.030 (mJ/m	(mJ/mm²)	Forearm: 1500 shots/ NA Hand: 3200 shots/NA (800 for each interossei muscle)	1 session	Immediate, 1, 4, 12 weeks	Nil
Amelio <i>et al.</i> ²⁹ 2010	Self- control	12	Cerebral palsy	8±0.31	Gastrocnemius NA muscle	NA	Focused 0.030 (mJ/m	(mJ/mm^2)	1500 shots/NA	1 session	Immediate, 1, 4, 12 weeks	Not mentioned
Moon <i>et al</i> .30 2013	Self- control	30	Stroke	52.6±14.9	Gastrocnemius muscle	2.68±1.55	Focus	0.089 (mJ/mm²)	1500 shots/4 Hz	1 session	Baseline, immediate, 1, 4 weeks	Not mentioned
Gonkova et al.³¹ 2013	Self- control	25	Cerebral palsy	4.84±3.11	Gastrocnemius and soleus muscle	NA	Radial	1.5 (bar)	1500 shots/5 Hz	1 session	Immediate, 2, 4 weeks	Not mentioned
Marinelli et al. 32 2014	RCT	68 ESWT, 34 Control, 34	Multiple sclerosis	ESWT: 51.74±11.29 Control: 51.00±13.17	Gastrocnemius muscle and Achilles tendon	NA	Radial	1.5 (bar)	2000 shots/4 Hz	4 session/1 week	Baseline, 1, 4 weeks	II.
El-Shamy et al. ³³ 2014	RCT	30 ESWT, 15 Control, 15	Cerebral palsy	ESWT: 6.8±0.7 Control: 6.93±0.8	ESWT: 6.8±0.7 gastrocnemius Control: and soleus 6.93±0.8 muscles	NA	Focus	0.030 (mJ/mm ²)	1500 shots/5 Hz	12 session/1 week 12 weeks	12 weeks	Not mentioned
Li <i>et al.</i> ³⁴ 2016 RCT	RCT	1 session: 20 3 session 20 Control, 2	Stroke	1 session: 55.35±3.05 3 session: 56.80±3.00 Control: 55.95±2.64	Forearm and hand	1 session: 61.70±9.73 3 sessions: 66.65±9.56 Control: 66.95±10.04	Radial	Forearm: 3.5 (bar) Hand: 3 (bar)	Forearm: 4000 shots/5 Forearm:3 Hz Rand: 4000 shots/5 Hz Hand: Forearm	Forearm :3 session/1 week Hand: Forearm	Baseline 1, 4, 8, 12, 16 weeks	N:I
Wang <i>et al.</i> ³⁵ 2016	Case- control	66 ESWT, 34 Control, 32	Cerebral palsy	ESWT: 56.5±11.6 Control: 54.9±9.4	Gastrocnemius muscle	ESWT: 26.9±13.1 Control: 27.0±14.2	Radial	0.6 (bar)	1500 shots/8 Hz	12 sessions/1 week	Baseline, 1 month, 3 months	II.
Taheri <i>et al.</i> ³6 2017	RCT	25 ESWT,13 Control, 12	Stroke	ESWT: 56.5±11.6 Control: 54.9±9.4	Gastrocnemius muscle	ESWT: 33±21.4 Control: 25.8±9.9	Focus	0.1 (mJ/mm ²)	1500 shots/4 Hz	3 sessions/1 week Baseline, 1, 4, 12 weeks	Baseline, 1, 4, 12 weeks	Not mentioned

Table 2: Cochrane risk of bias for randomized controlled trials

			A. Cochrane risk o	A. Cochrane risk of bias for randomized controlled trials	controlled trials				
Study	Random sequence generation (selection bias)	Allocation concealment (selection bias)	Blinding of participants and personnel (performance bias)	Blinding of outcome assessment (detection bias)	Incomplete outcome data (attrition bias)	Selectiv (repor	Selective reporting (reporting bias)	Other bias	bias
El-Shamy et al., 2014	Unclear	Low risk	High risk	Low risk	Low risk	Lo	Low risk	Unclear	ear
Marinelli et al., 2014	Low risk	Unclear	Low risk	Low risk	Low risk	Lo	Low risk	Unclear	ear
Li et al., 2016	Low risk	Unclear	Low risk	Low risk	Low risk	Lo	Low risk	Unclear	ear
Taheri et al., 2017	Low risk	Unclear	High risk	Unclear	Low risk	Lo	Low risk	Unclear	ear
		B. New	vcastle-Ottawa quality	B. Newcastle-Ottawa quality assessment of in nonrandomized controlled trials	ndomized controlled	1 trials			
Study	Adequate definition of case	Representativeness of cases	Selection of control	Definition of control	Control for Exposure important factor or assessment additional factor		Same method of Nonresponse ascertainment rate for cases and controls	Nonresponse	Total quality scores
Gonkova et al., 2013	*	*	*	*	*	*	*	*	8
Amelio et al., 2010	*	*	*	*	*	*	*	*	6
Manganotti <i>et al.</i> , 2005	*	*	*	*	*	*	*	*	∞
Moon et al., 2013	*	*	*	*	*	*	*	*	6
Wang et al., 2016	*	*	*		*	*	*	*	8

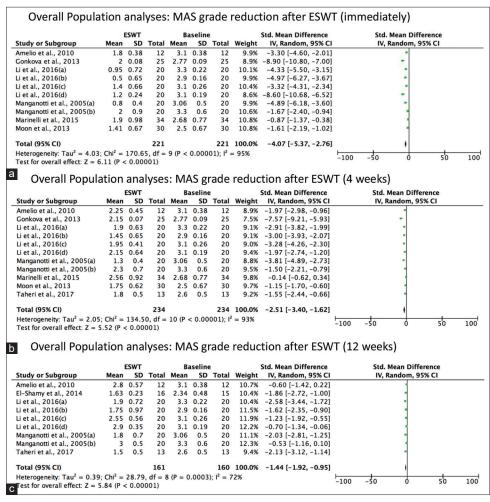


Figure 2: Forest plot of Overall Population analyses: MAS grade reduction after ESWT (a)immediately, (b) 4 weeks, (c) 12 weeks. MAS: Modified ashworth scale, ESWT: Extracorporeal shock-wave therapy

Standardized mean differences in subgroup analysis

Subgroup analyses were conducted if the pooling data revealed high heterogeneity [$I^2 > 50\%$, Table 3]. Several variable factors, which had clinically significant meaning or significant heterogeneity from meta-regression, were chosen for subgroup analyses.

In the subgroups analysis, which factors including the session numbers, type of ESWT, and treatment part of body, showed no significant intergroup difference in each evaluation timing. The pooled SMD in MAS grade reduction among one treatment session and more than 3 treatment sessions was -4.42 (95% CI: -6.09-2.76) and -3.35 (95%CI: -6.25-0.44) immediate after ESWT, -2.89 (95%CI: -4.03-1.74) and -1.7 (95% CI: -2.24-0.51) after 4 weeks, while at 12 weeks the results was -0.97 (95%CI: -1.28-0.66) and -1.63 (95%CI: -2.11-1.15).

The focus and radial ESWT both revealed significant result. The pooled SMD of focus and radial ESWT was -2.76 (95% CI:

Table 3: Subgroup analysis of the pooled standardized mean difference in spasticity reduction immediate, 4th and 12th weeks after extracorporeal shockwave therapy

Pooled SMD immediately	Pooled SMD at week 4	Pooled SMD at week 12
-4.42 (-6.092.76)	-2.89 (-4.031.75)	-0.97 (-1.280.66)
-3.35 (-6.250.44)	-1.7 (-2.240.511)	-1.63 (-2.111.15)
-2.76 (-4.061.45)	-1.92 (-2.721.12)	-1.29 (-2.150.43)
-5.05 (-7.462.65)	-2.34 (-3.451.23)	-1.32 (-1.750.88)
-4.48 (-6.092.88)	-2.69 (-3.401.98)	-1.41 (-2.020.81)
-3.43 (-5.451.41)	-1.66 (-2.580.73)	-1.26 (-1.710.82)
	immediately -4.42 (-6.092.76) -3.35 (-6.250.44) -2.76 (-4.061.45) -5.05 (-7.462.65) -4.48 (-6.092.88)	

Values are expressed by their point estimate with a 95% CI. CI=Confidence interval; SMD=Standardized mean difference; ESWT=Extracorporeal shockwave therapy

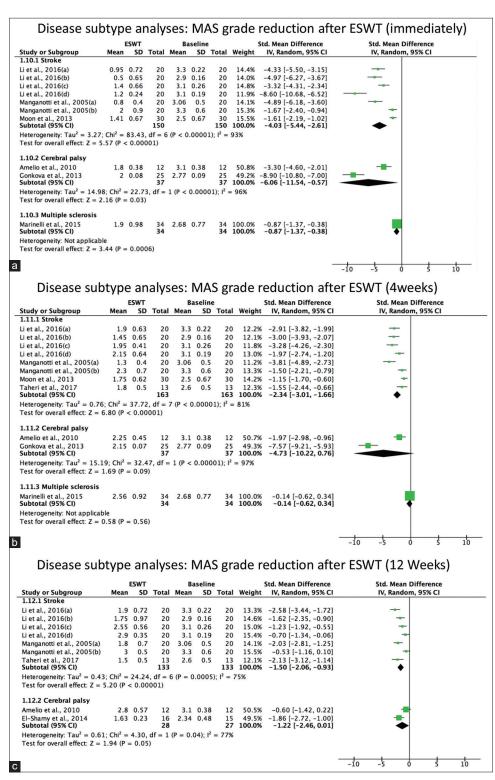


Figure 3: Forest plot of Disease subtype analyses: MAS grade reduction after ESWT (a)immediately, (b) 4 weeks, (c) 12 weeks. MAS: Modified Ashworth scale, ESWT: Extracorporeal shockwave therapy

-4.06—1.45) and - 5.05 (95% CI: -7.46—2.65) immediate after -1.23) after 4 weeks, while at 12 weeks the results was - 1.29 ESWT, -1.92 (95% CI: -2.72—1.12) and -2.34 (95% CI: -3.45— (95% CI: -2.15—0.43) and -1.32 (95% CI: -1.75—0.88).

In terms of the treatment part of spastic muscles, the pooled SMD of upper limb and lower limb was -4.48 (95% CI: -6.09-2.88) and -3.43 (95% CI: -5.45-1.41) immediate after ESWT, -2.69 (95% CI: -3.40 - -1.98) and -1.66 (95% CI: -2.58-0.73) after 4 weeks, while at 12 weeks, the results were -1.41 (95% CI: -2.02-0.81) and -1.26 (95% CI: -1.71-0.82). Besides, there was no significant intersubgroup differences in these three factors.

Adverse events

Among the 9 studies, 4 studies reported no adverse events, while the other 5 studies did not mention the adverse events [Table 1].

DISCUSSION

In the present study, we examined the effectiveness of ESWT in the treatment of spasticity after central nerve systems injury using the data assembled from the controlled trials. The result suggested that ESWT effectively alleviates spasticity in patients after upper motor neuron lesions, regardless of disease type and parts treated. The effect of spasticity reduction could last for 12 weeks, but with the tendency of attenuation within time. The effect of decreasing spasticity was shown in both radial and focus ESWT and regardless the treatment session number. Moreover, no serious side effects were observed after ESWT.

The mechanism of ESWT that alleviating the spasticity remains unclear. However, previous studies had examined the hypothesis that might modulate the spasticity. As we know, the development of spasticity was the combination of impaired reflex and secondary changes in rheological muscle properties, such as fibrosis, stiffness, and atrophy.³⁷ In terms of the neurophysiological survey, the ESWT did not change the F-wave response or H-reflex (Hmax) to maximum M-response ratio, suggesting that the alpha motor neuron excitability was not the factor to explain the therapeutic effect.^{38,39}

Changes in the rheological components in spastic muscle are more likely to be the explanation. A hypothesis is that the reduced muscle extensibility in hypertonic muscle might increase the spasticity by generating a pulling force to the muscle spindles, which increased the afferent signal input. Recently, the sonoelastography is a developing measurement tool in evaluation and possible quantification of spasticity. A study evaluated the change of sonoelastographic outcome in CP patients by comparing botulinum toxin type A (BoNT-A) and extracorporeal shockwave therapy (ESWT) versus BoNT-A alone. The report found that a significant improvement of hardness of muscle tissue in group BoNT-A with ESWT, but not in BoNT-A alone, suggesting that ESWT

could change muscle elasticity.⁴¹ As for the underlying molecular mechanism to change the rheology of spastic muscle remained unclear.

Regarding the effectiveness of ESWT in treatment session, subtype or target muscles, the subgroup analysis provided the clinical implications. First, the treatment session was not related to the therapeutic effect event in each follow-up timing. In Li *et al.*, a dose-dependent effect was comparing single and three sessions.²⁸ However, the effect was not seen in the pooled SMD.

In the disease type, we included three disease types together including stroke, CP, and multiple sclerosis patients. The clinical manifestations of spasticity in stroke cases and CP cases are different (mostly unilateral in stroke, and bilateral in CP). We performed subgroup analyses trying to analyze the possible factors that would influence the results.

The results showed that ESWT had a significant effect in patients with stroke or CP immediately, however, the effect evaluated at 4 and 12 weeks revealed a significant effect in stroke patients but without significant effect in CP patients. This may be explained by the high heterogeneity and small study numbers. Only one study targeting multiple sclerosis could be obtained in the current searching, therefore, to make a conclusion is insufficient in the current evidence. Moreover, the evidence of ESWT in other population, such as spinal cord injury or traumatic brain injury, was lack in the current survey.

There was a similar effect regarding the targeting region. The ESWT had no significant difference in spasticity reduction effect between upper limb muscles and lower limbs muscle, though the absolute value in MAS changed seemed to have a greater effect in upper limb muscles. When reviewing the protocol of ESWT, the energy flux density/pressure and dosage were heterogeneous. We may assume that the volume and area of muscle in lower limbs were larger than upper limbs, and the energy transmitted into the target area may decrease within the similar dose of ESWT. However, the dosage in the upper limb muscles, for example in each interosseous muscle, was lesser than other lower limb muscle. Therefore, the overall effect seemed to have no significant different in the spasticity reduction effect in upper and lower limb muscles.

In comparing the types of ESWT, the result suggested both types had therapeutic effects. The result reflected that the spasticity of muscle was a diffuse rheological change in essence, instead of the concentrated lesion-like calcified tendinitis of the shoulder.

The energy flux density of ESWT was not discussed as a subgroup analysis in the current study was mainly due to the enrolled study were belonged in low-to-middle intensity (defined as (0.030–0.100 mJ/mm²)), with narrow

range of energy distribution.⁴² Therefore, we were unable to propose an optimum energy in these patients.

Significant heterogeneity existed in the present study, even after using the random effect in survey and performed the subgroup analysis. Furthermore, the outcomes from sensitivity analysis indicated the consistency of the results, all within the range of high heterogeneity. Possibly, the high heterogeneity is related to the variability in ESWT protocols, the relative small number of participants in each trial and if the combination of medication that were not mentioned in the articles. Another consideration if that the widely used MAS grades extent may have contribution. Six grades were used in this grading system and considered to be a continuous variable, while subtle change lesser than the one-grade difference could not be defined, and thus affected the pooling result. Few studies enrolled tried to expand the outcome measurement to evaluate the spasticity, however, MAS was still the universal measurement tool in all studied and currently applicate in clinical practice.

Overall, the pooled data and subgroup demonstrate the spasticity reduction effect immediately, 4 weeks and 12 weeks after ESWT compared with baseline. To use ESWT in treating spasticity is a new field. It could be a complement treatment for those without positive response to previous treatment.

Study limitation

The strength of our study is to evaluate the available recent studies designed with controlled study in patient with spasticity after different CNS injury and concluded that the ESWT is effective. However, there was several limitations to mentioned in the present study. First, the studies we enrolled were not limited to the RCTs, which were the preferred choice in minimizing the bias in the essence of study design. Due to the limited RCTs study number available during searching, the current meta-analysis was set to enroll study with controlled group.

Second, the important limitation is the presence of significant heterogeneity.

Further researches designed in RCT and defined the protocol of ESWT were necessary to clarify the effectiveness and set up the recommendation in clinical use. Third, if the change of spasticity could be linked to the functional change or improvement of health-related quality of life were not evaluated due to insufficient data in the obtained articles.

Clinical implications

Spasticity is a common complication developed after upper motor neuron injury, and is a factor that cause disability and influence quality of life. ESWT could be a safe and effective modality in treating spasticity after upper motor neuron lesion.

CONCLUSION

The present meta-analysis revealed that ESWT effectively alleviates spasticity in patients after upper motor neuron lesions, regardless of disease type and parts treated. Both radial and focus ESWTs could decrease spasticity, regardless of the treatment session. The result could last for 12 weeks after treatment. Moreover, no serious side effects were observed after ESWT. Further studies with randomization and more parameters of ESWT were advised to be set up to improve the clinical effectiveness.

Data availability statement

The data that support the findings of this study are available from the corresponding author, Lee CH, upon reasonable request.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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