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Review

Pathogenetic Mechanisms, Epidemiology and Modern Treatment Strategies for Spasticity Using Extracorporeal Shock Wave Therapy: A Narrative Review

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Abstract

Post-stroke spasticity is a common complication of stroke that limits motor activity, functional independence, and the effectiveness of rehabilitation. Despite the availability of various therapeutic approaches, there remains a need to optimize treatment strategies targeting both the central and peripheral mechanisms underlying spasticity.

Objective. To conduct a narrative review of current evidence on the epidemiology and pathophysiological mechanisms of post-stroke spasticity, summarize existing therapeutic approaches, evaluate the efficacy of extracorporeal shock wave therapy (ESWT), and synthesize current evidence regarding its proposed mechanisms of action.

Materials and Methods. A literature search was conducted in the PubMed/MEDLINE, Cochrane Library, Scopus, Web of Science, and Google Scholar databases. The primary search period covered publications from January 2020 to July 2026; earlier sources were included when necessary. The review included randomized controlled trials, prospective, cohort, and observational studies, as well as systematic reviews and meta-analyses.

Results. The prevalence of post-stroke spasticity varies substantially depending on the follow-up period, severity of motor impairment, and diagnostic criteria. A higher risk of spasticity is observed in patients with severe paresis and marked functional impairment. The analyzed studies demonstrated that ESWT is associated with reductions in muscle tone and spasticity severity, as well as improvements in selected measures of motor function. In some studies, these effects persisted for several months. The proposed mechanisms of action include effects on the mechanical properties of muscles and connective tissue, neuromuscular transmission, local blood circulation, and neuroplasticity.

Conclusions. ESWT is a promising adjunctive modality in the comprehensive rehabilitation of patients with post-stroke spasticity. However, heterogeneity in treatment parameters, small sample sizes, and limited follow-up duration preclude the determination of an optimal treatment protocol. Multicenter randomized controlled trials with standardized treatment parameters and long-term follow-up are warranted.

Keywords: spasticity, post-stroke spasticity, stroke, pathogenesis, epidemiology, extracorporeal shock wave therapy, ESWT, rehabilitation.

1. Introduction

Spasticity is a motor disorder characterized by an exaggerated tonic stretch reflex, accompanied by hyperreflexia and increased muscle tone. Individuals with spasticity may exhibit multijoint spasms, co-contraction of agonist and antagonist muscles, and dystonia or abnormal postures. Spasticity occurs in various neurological conditions, including stroke, multiple sclerosis, cerebral palsy, and spinal cord injury.

Stroke survivors frequently experience functional impairments and neurological deficits. The principal factors limiting activities of daily living after stroke include motor impairments, such as contractures, spasticity, muscle weakness, paresis of central origin, ataxia, and postural and gait disturbances, as well as emotional and cognitive impairments. Altered muscle tone is one of the clinical factors that adversely affect motor activity. Changes in muscle tone reflect both dysfunction within motor control systems and alterations in sensorimotor control. These changes often follow a sequential pattern, with hypotonia giving way to hypertonia and the development of early post-stroke spasticity. Regardless of its severity, post-stroke spasticity (PSS) further restricts functional activity and reduces the effectiveness of rehabilitation, as spastic muscles tend to remain in a shortened position for prolonged periods, thereby limiting voluntary activation of antagonist muscles [1].

Coordinated voluntary movements of the upper and lower limbs, which enable object manipulation and locomotion, are complex, multicomponent processes governed by a multilevel system of neurophysiological regulation. Recovery of upper- and lower-limb motor function requires prolonged, intensive rehabilitation programmes involving repetitive practice to promote sensorimotor learning, motor cortex reorganization, and neuroplasticity. However, conventional approaches relying primarily on therapeutic exercise and passive interventions are often insufficient to achieve sustained clinical benefits.

Despite the wide range of contemporary treatment options, the principles of spasticity management remain a subject of debate. Furthermore, adequate control of spasticity is not always achieved. The limited duration of therapeutic benefits and the adverse effects of pharmacological treatment underscore the need for further research into highly effective interventions, particularly those targeting peripheral muscular factors that contribute to spasticity [2–5].

Developing effective treatments requires an understanding of the specific mechanisms underlying increased muscle tone, as these mechanisms should guide the selection of therapeutic targets.

Accordingly, this narrative review aims to examine current evidence on the epidemiology and pathophysiological mechanisms of post-stroke spasticity, summarize contemporary treatment approaches, evaluate the effectiveness of extracorporeal shock wave therapy (ESWT), and synthesize current understanding of its proposed mechanisms of action.

2. Materials and methods

This study was conducted as a narrative literature review. The literature search was performed using PubMed/MEDLINE, the Cochrane Library, Scopus, Web of Science, and Google Scholar. The primary search period covered publications from 1 January 2020 to July 2026, allowing the review to focus on contemporary research into the pathogenesis, prevalence, and treatment of post-stroke spasticity. Earlier publications were also consulted where necessary to describe fundamental principles, the historical development of knowledge about spasticity, and previously established concepts.

The search strategy used combinations of keywords, terms, and synonyms relevant to the main topics of the review. Search queries included the following terms: spasticity, post-stroke spasticity, stroke, pathogenesis, epidemiology, extracorporeal shock wave therapy, ESWT, rehabilitation.

The search strategy comprised several interrelated thematic components. The first focused on identifying studies addressing the epidemiology and prevalence of post-stroke spasticity, including its incidence at different time points after stroke and associated risk factors. The second covered publications on the pathophysiological and neurophysiological mechanisms underlying spasticity, including alterations in the central and peripheral regulation of muscle tone, sensorimotor control, and the mechanical properties of muscle tissue. The third focused on identifying studies investigating the treatment of post-stroke spasticity.

The review included original clinical studies, randomized controlled trials, prospective and cohort studies, observational studies, systematic reviews, and meta-analyses reporting data directly relevant to its thematic sections. The epidemiological assessment drew on studies evaluating the prevalence or incidence of post-stroke spasticity, including at different time points after stroke. The assessment of ESWT effectiveness included studies measuring spasticity severity, muscle tone, motor function, range of motion, functional independence, and instrument-based

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outcomes, including electrophysiological and ultrasound assessments.

Publications were selected according to their relevance to the review topic, scientific rigour, methodological quality, study design, patient characteristics, and the clinical relevance of their findings. Priority was given to recent studies and publications providing high levels of evidence, including systematic reviews, meta-analyses, and randomized controlled trials.

As this study was a narrative review rather than a systematic review or meta-analysis, no formal systematic study selection process involving the sequential quantification of excluded studies was undertaken. Accordingly, a PRISMA flow diagram was not used. Sources were selected through targeted, topic-based searches, followed by expert assessment of their relevance and scientific rigour.

3. Results

Epidemiology

A comprehensive review of the literature indicated that 30–80% of stroke survivors develop post-stroke spasticity [4,5]. European and Asian studies have reported prevalence estimates ranging from 7% to 38% over an 18-month follow-up period [6,7].

Among patients with paresis, spasticity has been reported in 4–27% during the early post-stroke period, from weeks 1 to 4, in 19–27% during the subacute phase, and in 17–43% during the chronic phase [8]. In two studies conducted in Germany and Sweden, the prevalence of spasticity among patients with initial limb paresis after stroke was 42% at six months and 46% at 12 months, respectively [9,10]. Meanwhile, complete recovery at six months after stroke is achieved in only 20% of cases [11]. Patients with post-stroke spasticity generally require long-term care and therapeutic interventions, highlighting substantial unmet needs for healthcare and social support. The high prevalence of spasticity underscores the need to investigate its pathophysiology and treatment.

Several authors have identified severe paresis, greater disability, and greater stroke severity at baseline as the principal predictors of subsequent spasticity, which develops in more than one-third of patients [12]. The volume of intracerebral haemorrhage or cerebral infarction and scores on neurological assessment scales also play a significant role in the development of increased muscle tone [13]. Clinical assessment tools used in stroke units, such as the National Institutes of Health Stroke Scale (NIHSS), the modified Rankin Scale (mRS), and the Mini-Mental State Examination (MMSE), may facilitate the early identification of patients at high risk of post-stroke spasticity [14]. Evidence suggests that severe spasticity is more common among patients with left-sided paresis, smokers, patients with depression, and those with more severe upper-limb motor impairment [15].

Pathophysiological Mechanisms of Post-Stroke Spasticity

In 1980, J. W. Lance defined spasticity as a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes, accompanied by exaggerated tendon reflexes resulting from stretch reflex hyperexcitability, as a component of the upper motor neuron syndrome [11]. However, this definition did not explicitly address the sensory component. In 2005, the SPASM project group proposed a definition emphasizing impaired sensorimotor control resulting from an upper motor neuron lesion and manifesting as intermittent or sustained involuntary muscle activation. In 2018, a broader definition was proposed, describing spasticity as involuntary muscle overactivity in the presence of paresis of central origin.

The literature identifies two interrelated levels of regulation involved in the development of spasticity: spinal and supraspinal. Brain regions involved in motor control—including the primary motor cortex, premotor cortex, supplementary motor area, posterior parietal cortex, and primary somatosensory cortex—contribute to sensorimotor processing and the generation and transmission of motor commands. However, isolated lesions in these cortical regions are insufficient to produce spasticity [16].

Spasticity is associated not only with increased muscle tone due to neurally mediated hyperreflexia but also with alterations in the mechanical properties of muscles. Changes in muscle mechanics and in the properties of collagen and tendons develop gradually and may contribute to increased resistance to movement as an adaptive response secondary to paresis. Changes in motor units and their contractile properties may reflect adaptation to alterations in the intrinsic properties of muscle tissue. Following the onset of post-stroke paresis, muscles may remain in a shortened position, and their fibres may exhibit increased stiffness compared with those of unaffected individuals. These adaptations may facilitate force production at shorter muscle lengths, partially compensating for motor weakness and enabling movement at a simpler level of motor organization [17].

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Treatment Approaches for Spasticity

When muscle spasticity is identified, particularly when it progresses, interventions aimed at reducing it should be incorporated into the rehabilitation programme [18]. However, not all cases of spasticity require treatment. Treatment is appropriate when spasticity causes substantial motor impairment, marked difficulty maintaining posture, pain, or difficulties with patient care. In addition to reducing muscle tone and improving passive mobility, treatment may reveal residual voluntary motor activity that is masked by increased muscle tone or spasticity of antagonist muscles.

The management of patients with spasticity requires a comprehensive approach delivered by a multidisciplinary team in collaboration with the patient and their family. Rehabilitation interventions should be combined to maximize functional improvement [19]. Regardless of the clinical presentation of post-stroke spasticity, treatment should be initiated when clear indications are present and intervention is necessary to address its harmful consequences.

The primary treatment goal in all cases is to reduce abnormal sensory input, thereby decreasing excessive α-motoneuron activity—a principle based on the underlying pathophysiology.

The main treatment approaches include:

– physical interventions, including patient care, physiotherapy, occupational therapy, therapeutic positioning, therapeutic exercise, stretching, orthotic management, and splinting;

– pharmacological therapy;

– surgical interventions;

– botulinum toxin therapy [20].

Combining different treatment modalities generally yields the greatest therapeutic benefit [21–23].

Pharmacological therapy primarily aims to reduce muscle hypertonia, whereas non-pharmacological interventions focus on improving functional activity by addressing soft tissues, joints, and movement patterns. These interventions contribute to the prevention of contractures, deformities, pain, and secondary complications.

Pharmacological treatment includes muscle relaxants and antispastic agents. However, their effectiveness is limited, and prolonged use may require dose escalation to maintain clinical benefits. This may increase the frequency and severity of adverse effects. Centrally acting muscle relaxants may cause sedation and hypotension, which often limit their use in clinical practice [24,25]. When spasticity is focal and systemic muscle relaxants are undesirable, local interventions are preferred. Botulinum toxin therapy offers an alternative to systemic antispastic medication. Although botulinum toxin is an exceptionally potent neurotoxin, it is a safe and effective treatment for post-stroke spasticity in adults when administered at appropriate therapeutic doses [26].

The effectiveness of botulinum toxin therapy for spasticity has been demonstrated in several randomized, double-blind, placebo-controlled trials. Treatment is generally well tolerated, with a low risk of adverse effects, and allows targeted injections into one or more muscles, with the dose individually adjusted for each muscle. Botulinum toxin therapy may also modulate cortical excitability, potentially mitigating maladaptive plasticity and helping to prevent contractures. Ultrasound guidance improves injection accuracy and procedural safety. Adherence to ergonomic principles during ultrasound-guided injections promotes patient comfort and reduces the risk of work-related injuries among clinicians [27]. However, the use of botulinum toxin therapy is limited by the high cost of the drug, the need for appropriately trained healthcare professionals, and the requirement for repeated injections to maintain therapeutic benefits.

Physiotherapy interventions aim to reduce spasticity and improve muscle control. In recent decades, non-invasive interventions have gained increasing attention in the management of spasticity. One such intervention is extracorporeal shock wave therapy (ESWT). ESWT involves the delivery of a series of acoustic pulses, characterized by high peak pressure and a rapid rise in pressure, to a specific target area using a shock wave generator [28].

The literature describes several proposed mechanisms underlying the effects of shock wave therapy on spastic muscles. These mechanisms are multifactorial and encompass both mechanical and biological effects. At the muscle tissue level, disruption of actin–myosin interactions has been proposed, alongside a reduction in connective tissue stiffness. The physical effects of ESWT include cavitation, tissue microtrauma, and mechanotransduction, which may initiate a cascade of biological responses, including changes in neurotransmitter expression [29,30].

ESWT has also been reported to influence angiogenesis, inflammation, and connective tissue remodelling, potentially improving the functional properties of muscles and surrounding structures. Increased nitric oxide synthesis may promote the formation of new blood vessels and capillary networks in muscles and tendons. Effects on synaptic plasticity have also been proposed, potentially contributing to the formation of new neuromuscular connections [31].

All studies reviewed reported reductions in spasticity severity and muscle tone. In a systematic review and meta-analysis published in 2023, Ou-Yang

et al. concluded that ESWT was effective in reducing post-stroke spasticity, with therapeutic benefits lasting up to approximately three months and associated improvements in limb motor function. The analysis included 13 studies involving 677 participants [32].

Several studies have reported benefits from combining ESWT with other interventions, including therapeutic exercise, botulinum toxin therapy, functional electrical stimulation (FES), and other device-based physiotherapy modalities. Bumbea et al. conducted a retrospective observational study involving the medical records of 52 patients. The control group received galvanic stimulation and electrical stimulation of flaccid upper-limb muscles, including the finger extensors, extensor carpi radialis longus, and extensor carpi ulnaris. The intervention group underwent a rehabilitation programme combining galvanic stimulation of the paretic upper limb, FES of flaccid upper-limb extensors, and ESWT applied to spastic upper-limb flexors.

Treatment effectiveness was assessed using dynamometry, goniometry, the Ashworth and Tardieu scales, and a handgrip test. Patients received ten treatment sessions using the same parameter progression. ESWT was initiated at a pressure of 0.5 bar and a frequency of 5 Hz, with 300 pulses per session. These parameters were progressively increased to 1.5 bar, 10 Hz, and 1,000 pulses per session applied to the target muscle groups. The intervention group showed greater improvements than the control group. Following combined ESWT and FES, handgrip performance increased from 0.28 ± 0.28 to 0.99 ± 0.36 ($p < 0.001$). The treatment effect remained evident after accounting for the control group's more favourable baseline clinical status. Muscle strength increased from 0.39 ± 0.54 to 7.67 ± 3.89 ($p < 0.001$) [33].

Lee et al. reported greater reductions in spasticity and significantly lower Ashworth Scale scores among patients receiving botulinum toxin injections combined with ESWT than among those receiving botulinum toxin injections alone. However, between-group differences in effectiveness were no longer evident over time. Therapeutic benefits persisted for approximately three months [34].

Tabra et al. (2021), Fan et al. (2024), and Yang et al. (2024) used electrophysiological assessments based on the ratio of maximal H-reflex amplitude to maximal M-wave amplitude (Hmax/Mmax). A reduction in this ratio following treatment suggests changes in α-motoneuron excitability and may reflect modulation of spinal mechanisms regulating muscle tone [35–37].

A randomized controlled trial published in January 2026 investigated the effects of ESWT combined with a neuroplasticity-based training protocol on lower-limb spasticity after stroke, incorporating ultrasonographic assessment. Participants received six weekly ESWT sessions, each delivering 1,500 pulses at 5 Hz with an energy flux density of 0.10 mJ/mm². Outcomes were assessed at weeks 6 and 12. Improvements in Ashworth Scale scores, lower-extremity Fugl–Meyer Assessment scores, and ultrasonographic measures supported the effectiveness of the combined intervention [38].

Yang et al. (2024) reported a dose-dependent effect of ESWT. In their study involving 39 patients with spasticity, the group receiving twice the number of pulses achieved better functional outcomes than the control group [37]. Furthermore, Ke et al. (2026) reported a potential advantage of treatment at 4 Hz over 10 Hz [39].

Senarath et al. demonstrated the effectiveness of ESWT in the chronic phase of stroke, whereas Brunelli et al. suggested that ESWT is most effective when applied during the early stages of post-stroke spasticity [40,41].

The key studies are summarized in Table 1.

Table 1 - Potential benefits of shock wave therapy for upper- and lower-limb motor function

Study authors, year	Study design	Treatment site and parameters	Outcome measures	Main findings
Leng Y, Lo WLA, Hu C, Bian R, Xu Z, Shan X, Huang D, Li L., 2021 [4]	RCT, n = 27	1,500 pulses; 1.5 bar; 0.038 mJ/mm²; 4 Hz; 15 mm Forearm flexor muscles	MAS, FMA	ESWT is effective in addressing the peripheral component of spasticity through changes in muscle mechanical properties. The optimal intervention protocol should account for both neural and non-neural factors.
Tabra, S.A.A., Zaghloul, M.I. &Alashkar, D.S., 2021 [35]	RCT, n = 40	2,000–3,000 pulses at 0.25–0.84 mJ/mm²; 2.8 bar; 15 Hz Upper-limb muscles	MAS; electromyographic assessment of Hmax/ Mmax	ESWT is a valuable adjunctive treatment for post-stroke hand and wrist spasticity, with improvements in functional activity.

Table 1 (Continuation) -- Potential benefits of shock wave therapy for upper- and lower-limb motor function

Study authors, year	Study design	Treatment site and parameters	Outcome measures	Main findings
Brunelli, S., Gentileschi, N., Spanò, B.,et al,2022 [41]	RCT, n = 40	2,000 pulses; 1.5 bar; 10 Hz Upper-limb muscles: biceps and forearm muscles	MAS	Early ESWT for post-stroke spasticity prevents its progression and reduces the use of oral antispastic medication.
Senarath, I. D., Thalwathte, R. D., Pathirage, M., & Kularatne, S. A. 2023 [40]	Blinded RCT, n = 95	1,500 pulses; 0.03 mJ/mm²; 5 Hz Upper-limb muscles: upper-arm muscle and forearm flexors	MAS, FMA-UE, ARAT	ESWT is effective for chronic post-stroke upper-limb spasticity. Patients showed improved hand function compared with TENS therapy.
Yuan, Z.; Luo, J.; Cheng, Q.-F.; Zhang, Q., 2023 [32]	RCT, n = 60	3,000 pulses; 1.1–1.3 bar; 8–14 Hz Upper limb	MBI, FMA	ESWT reduces spasticity, enhances upper-limb motor function, and improves patients' functional capacity.
Fan T, Chen R, Wei M, et al. 2024 [36]	RCT, n = 95	Treatment parameters not reported Upper-limb flexors	MAS; Hmax/ Mmax ratio; root mean square; co-contraction coefficient; muscle mechanical parameters and temperature	ESWT may alleviate upper-limb spasticity in stroke survivors.
Lee, J.; Yang, S.N., 2024 [34]	RCT, n = 16	4 Hz; energy flux density of 0.030 mJ/mm²; 1,000 pulses Upper-limb muscles	MAS, UE-FMA, MBI, ARAT	Botulinum toxin A injection with adjunctive ESWT produced marked improvements in distal upper-limb flexor spasticity.
Yang, SM., Chen, YH., Lu, YL. et al, 2024 [37]	Double-blind RCT, n = 39	4 Hz; energy flux density of 0.10–0.134 mJ/mm² 2,000 pulses: control group 4,000 pulses: intervention group Lower-limb muscles: gastrocnemius	MAS, MTS, ankle ROM, TUG test, MBI, and strain elastography of the plantar flexor muscles	A double dose of ESWT is more effective than a single dose in improving functional outcomes and elastographic measures. ESWT demonstrates dose-dependent effectiveness in managing spasticity in stroke survivors.
Ke M., Zhou, P., Li, D. et al. 2026 [39]	RCT, n = 30	4 and 10 Hz; 2 bar; 2,000 pulses Lower-limb muscles	MAS, passive ROM, ankle clonus scale, MGF, LGF, MGS, LGS, FMA-LE, VAS	Both a single ESWT session and a three-week treatment course safely reduced ankle plantar flexor spasticity. The findings suggest a potential advantage of 4 Hz over 10 Hz.
Bumbea, A.M.; Trăistaru, R.; Târtea, E.-A.; Dan, A.O.; Turcu-Stiolica, A.; Matei, D.; Pătru, S.; Bumbea, B.S.; Daia, C.O., 2026 [33]	Retrospective observational study, n = 52	Initially 0.5 bar and 5 Hz, applied longitudinally, with 300 pulses per session; progressively increased to 1,000 pulses per session at 1.5 bar and 10 Hz Upper-limb muscles	mRS, MAS, MTS, VAS	Combined FES and ESWT provided additional benefits for upper-limb rehabilitation compared with conventional electrical stimulation. Targeting spastic flexors with ESWT while activating weakened extensors with FES may provide additional functional benefits after stroke.

Note. ARAT, Action Research Arm Test; FES, functional electrical stimulation; FMA, Fugl–Meyer Assessment; MAS, Modified Ashworth Scale; MBI, Modified Barthel Index; MGF, medial gastrocnemius oscillation frequency; MGS, medial

gastrocnemius dynamic stiffness; mRS, modified Rankin Scale; MTS, Modified Tardieu Scale; LGF, lateral gastrocnemius oscillation frequency; LGS, lateral gastrocnemius dynamic stiffness; n, number of participants; RCT, randomized controlled trial; ROM, range of motion; TUG, Timed Up and Go; UE, upper extremity; VAS, visual analogue scale for pain. Additional abbreviations used in the table: LE, lower extremity; TENS, transcutaneous electrical nerve stimulation; Hmax/Mmax, ratio of maximal H-reflex amplitude to maximal M-wave amplitude; ESWT, extracorporeal shock wave therapy.

4. Discussion

The findings confirm that post-stroke spasticity is a common and clinically significant complication of stroke. Its reported frequency varies according to the duration of follow-up, the severity of motor impairment, and the diagnostic criteria used. Patients with severe paresis and substantial functional impairment are at greater risk of developing spasticity. These findings underscore the need for early identification of patients at risk and timely implementation of interventions aimed at controlling spasticity.

The literature reviewed supports the effectiveness of extracorporeal shock wave therapy and its potential for further use in the management of post-stroke spasticity. In most studies, ESWT was associated with reductions in muscle tone and improvements in motor function. All studies reported improvements in Modified Ashworth Scale scores. Furthermore, most studies demonstrated improvements in motor function and functional independence, as assessed using the upper- and lower-extremity Fugl–Meyer Assessment, the Action Research Arm Test (ARAT), the Timed Up and Go (TUG) test, and the Modified Barthel Index. However, these findings should be interpreted in light of the substantial heterogeneity of the study protocols.

One of the principal challenges is the absence of a standardized ESWT protocol for post-stroke spasticity. The studies reviewed differed in treatment frequency, energy flux density, pressure, number of pulses, number of sessions, intervals between sessions, and treatment sites. This variability limits direct comparisons across studies and makes it difficult to determine optimal treatment parameters.

The available evidence also suggests that the clinical benefits of ESWT may be associated not only with changes in the mechanical properties of spastic muscles but also with effects on the neurophysiological mechanisms regulating muscle tone. Reductions in the Hmax/Mmax ratio reported in several studies suggest possible changes in α-motoneuron excitability and spinal regulatory mechanisms. However, these findings require further confirmation in studies using standardized electrophysiological protocols.

The use of ESWT within comprehensive rehabilitation programmes warrants particular attention. Studies combining ESWT with functional electrical stimulation, therapeutic exercise, botulinum toxin therapy, or neuroplasticity-based training suggest potential additional functional benefits. Nevertheless, the designs and sample sizes of most such studies do not allow the independent contribution of ESWT to the overall treatment effect to be clearly established.

The reported persistence of therapeutic benefits for several months is clinically relevant; however, the duration of these benefits remains insufficiently investigated. Short follow-up periods in some studies and differences in the timing of outcome assessments make it difficult to determine the durability of treatment effects.

Overall, the available evidence supports further investigation of ESWT for post-stroke spasticity. Larger randomized controlled trials with standardized protocols and extended follow-up are needed to establish optimal treatment parameters, treatment course duration, session frequency, and the duration of therapeutic benefits.

5. Conclusions

This literature review indicates that post-stroke spasticity is a common complication of stroke. However, prevalence estimates vary considerably depending on the timing of post-stroke follow-up, the severity of neurological deficits, and the diagnostic criteria used. Patients with substantial motor deficits and severe functional impairment are at greater risk of developing spasticity.

Post-stroke spasticity is a multifactorial condition involving impaired central regulation of muscle tone, alterations in spinal reflex mechanisms, and secondary peripheral changes in muscles and soft tissues. These mechanisms underscore the need for a comprehensive, individualized approach to treatment.

The principal treatment goals are to reduce pathological muscle tone and pain, preserve range of motion, prevent contractures and deformities, improve motor activity, and facilitate patient care. Pharmacological treatment, botulinum toxin therapy, physiotherapy, and rehabilitation interventions are selected according to each patient's clinical characteristics.

The published evidence suggests that ESWT may be effective in reducing spasticity severity and

improving selected measures of motor function after stroke. Nevertheless, study findings are heterogeneous, and ESWT is primarily regarded as an adjunct to comprehensive rehabilitation.

Proposed mechanisms of action include effects on neuromuscular transmission, the mechanical properties of muscles and connective tissue, local blood circulation, and neuroplasticity. However, the contribution of each mechanism has yet to be established and requires further experimental and clinical investigation.

Existing studies are limited by small sample sizes and differences in ESWT protocols, treatment parameters, outcome measures, and follow-up duration. Multicentre randomized controlled trials using standardized ESWT protocols, consistent clinical and instrument-based outcome measures, and extended follow-up are needed to establish the durability of therapeutic benefits and the independent contribution of ESWT to rehabilitation outcomes.

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All authors have read and approved the final version of the manuscript and signed the copyright transfer agreement.

AI Declaration. AI was used for language editing, stylistic refinement, and translation of the manuscript from Russian into English. All AI-assisted content was reviewed and edited by the authors, who take full responsibility for the accuracy and integrity of the final manuscript.

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Спастиканың патогенетикалық механизмі, эпидемиологиясы және оны экстракорпоралды соққы толқыны терапиясын қолдана отырып емдеудің заманауи стратегиялары: Нарративтік шолу

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Түйіндеме

Инсульттен кейінгі спастикалық синдром инсульттің кең таралған асқынуларының бірі болып табылады және қозғалыс белсенділігін, функционалдық тәуелсіздікті және оңалтудың тиімділігін шектейді. Әртүрлі емдеу әдістерінің болуына қарамастан, спастикалық синдромның орталық және перифериялық механизмдеріне бағытталған емдеу тәсілдерін оңтайландыру қажеттілігі сақталуда.

Шолудың мақсаты: инсульттен кейінгі спастикалық синдромның эпидемиологиясы мен патофизиологиялық механизмдері жөніндегі заманауи деректерге нарративті шолу жүргізу, оны емдеудің қазіргі тәсілдерін жинақтау, экстракорпоралды соққы-толқындық терапияның (ЭСТТ) тиімділігін бағалау және оның ықтимал әсер ету механизмдері туралы қазіргі түсініктерді жүйелеу.

Әдістері. Әдебиеттерді іздеу PubMed/MEDLINE, Cochrane Library, Scopus, Web of Science және Google Scholar деректер базаларында жүргізілді. Негізгі іздеу кезеңі 2020 жылғы қаңтардан 2026 жылғы шілдеге дейінгі жарияланымдарды қамтыды. Қажет болған жағдайда бұдан ертерек жарияланған дереккөздер де пайдаланылды. Шолуға рандомизацияланған бақыланатын зерттеулер, проспективті, когорттық және бақылаулық зерттеулер, сондай-ақ жүйелі шолулар мен мета-талдаулар енгізілді.

Нәтижелері. Инсульттен кейінгі спастикалық синдромның таралу жиілігі бақылау кезеңіне, қозғалыс бұзылыстарының ауырлығына және диагностикалық критерийлерге байланысты айтарлықтай өзгереді. Айқын парезі және ауыр функционалдық бұзылыстары бар науқастарда спастикалық синдромның даму қаупі жоғары болады. Зерттеулерді талдау ЭСТТ бұлшықет тонусы мен спастикалық синдромның ауырлығын төмендетумен қатар, қозғалыс қызметінің жекелеген көрсеткіштерін жақсартуға ықпал ететінін көрсетті. Бірқатар зерттеулерде бұл әсер бірнеше айға дейін сақталған. ЭСТТ-ның ықтимал әсер ету механизмдеріне бұлшықеттер мен дәнекер тіндердің механикалық қасиеттеріне, жүйке-бұлшықет берілісіне, жергілікті қан айналымына және нейропластикалық үдерістерге әсер ету жатады.

Қорытынды. ЭСТТ инсульттен кейінгі спастикалық синдромы бар науқастарды кешенді оңалтудың болашағы қосымша әдісі болып табылады. Алайда емдеу параметрлерінің әркелкілігі, зерттеу үлгілерінің шағын болуы және бақылау кезеңінің шектеулі ұзақтығы оңтайлы емдеу хаттамасын анықтауға мүмкіндік бермейді. Стандартталған емдеу параметрлері және ұзақ мерзімді бақылауы бар көпорталықты рандомизацияланған бақыланатын зерттеулер жүргізу қажет.

Түйін сөздер: спастикалық синдром, инсульттен кейінгі спастикалық синдром, инсульт, патогенез, эпидемиология, экстракорпоралды соққы-толқындық терапия, ЭСТТ, оңалту.

Патогенетические механизмы, эпидемиология и современные стратегии лечения спастичности с применением экстракорпоральной ударно-волновой терапии: Нарративный обзор

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Резюме

Постинсультная спастичность является распространенным осложнением инсульта, ограничивающим двигательную активность, функциональную независимость и эффективность реабилитации. Несмотря на наличие различных методов лечения, сохраняется необходимость оптимизации подходов, направленных как на центральные, так и на периферические механизмы спастичности.

Цель обзора: проанализировать современные данные об эпидемиологии и патофизиологических механизмах постинсультной спастичности, обобщить подходы к ее лечению, оценить эффективность экстракорпоральной ударно-волновой терапии (ЭУВТ) и систематизировать представления о предполагаемых механизмах ее действия.

Методы. Поиск литературы проводился в базах PubMed/MEDLINE, Cochrane Library, Scopus, Web of Science и Google Scholar. Основной период поиска включал публикации с января 2020 года по июль 2026 года.

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При необходимости использовались более ранние источники. В обзор включались рандомизированные контролируемые, проспективные, когортные и наблюдательные исследования, а также систематические обзоры и метаанализы.

Результаты. Распространенность постинсультной спастичности существенно варьирует в зависимости от периода наблюдения, тяжести двигательного дефицита и диагностических критериев. Более высокий риск отмечается при выраженном парезе и тяжелых функциональных нарушениях. Анализ исследований показал, что ЭУВТ сопровождается снижением мышечного тонуса и выраженности спастичности, а также улучшением отдельных показателей двигательной функции. Эффект в ряде исследований сохранялся до нескольких месяцев. Предполагаемые механизмы включают воздействие на механические свойства мышц и соединительной ткани, нервно-мышечную передачу, локальное кровообращение и нейропластичность.

Выводы. ЭУВТ является перспективным дополнительным методом комплексной реабилитации пациентов с постинсультной спастичностью. Однако неоднородность параметров лечения, небольшие выборки и ограниченная длительность наблюдения не позволяют определить оптимальный протокол. Необходимы многоцентровые рандомизированные исследования со стандартизированными параметрами и длительным наблюдением.

Ключевые слова: спастичность, постинсультная спастичность, инсульт, патогенез, эпидемиология, экстракорпоральная ударно-волновая терапия, ЭУВТ, реабилитация.

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