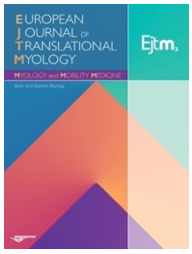
European Journal of Translational Myology



pISSN: 2037-7452 eISSN: 2037-7460

https://www.pagepressjournals.org/index.php/bam/index

Eur J Transl Myol 2024 [Online ahead of print]

To cite this Article:

Li W, Liu X, Wen Y, et al. **Evaluation of diagnostic value of Doppler ultrasound in the diagnosis of malignant breast masses.** *Eur J Transl Myol* doi: 10.4081/ejtm.2024.12172

The effect of fascial manipulation therapy on lower limb spasticity and ankle clonus in stroke patients

Wenyan Li,[1] Xin Liu,[1] Yinghua Wen,[1] Junying Wu,[1] Federico Giordani,[2] Carla Stecco[3]

[1]First Hospital of Shanxi Medical University, TaiYuan, China; [2]Villa Rosa Neurological Rehabilitation Hospital, APSS Trento, Italy; [3]Neuroscience Department, University of Padova, Padova, Italy

Abstract

Lower limb spasticity and clonus are common sequelae after cerebral stroke. An important part of their etiopathogenesis has been related to the peripheral component of spasticity. Rheological properties of the tissues seem to be involved. Several studies highlighted anatomical and functional changes in the connective structures. The fasciae might be implicated in the pathological process. Thus, this study intends to investigate the effect of the Fascial Manipulation (FM) technique on triceps surae in stroke patients through a clinical randomized controlled trial, to provide a reference for clinical treatment of lower limb spasticity and ankle clonus. A total of 40 patients with post-stroke ankle clonus were selected and divided into a control group and an observation group by random number table method, with 20 cases in each group. Both groups received conventional rehabilitation therapy, while the FM group received Fascial Manipulation based on conventional rehabilitation therapy. Before the first treatment and after 3 weeks of treatment, the Comprehensive Spasticity Scale (CSS), the Passive Range Of Motion (PROM), the simplified Fugl-Meyer motor function score (FMA), and the Modified Ashworth Scale (MAS) were used to assess the degree of ankle clonus, ankle passive range of motion, and lower limb motor function of the two groups of patients. Before treatment, there was no statistically significant difference between the control group and the FM group in terms of CSS, PROM, FMA, and MAS of the affected lower limbs ($P>0.05$). After 3 weeks of treatment, the CSS and MAS of the affected lower limbs in the control group and FM group decreased, while PROM and FMA increased compared to pre-treatment evaluation,

with statistically significant differences (P<0.05). Moreover, the FM group showed a statistically significant decrease in CSS and MAS, as well as an increase in PROM and FMA, compared to the control group (P<0.05). Conclusions: Fascial manipulation in addition to conventional therapy can effectively reduce spasticity and ankle clonus in stroke patients in a short time, and improve the passive range of motion of the ankle joint and the function of lower limbs.

Key words: stroke; spasticity; fascia; peripheral component; clonus; triceps spasm.

Cerebral stroke, also known as cerebrovascular accident, includes cerebral infarction, cerebral hemorrhage, and subarachnoid hemorrhage. With the development of an older society and the improvement of medical treatments, the death rate of stroke has gradually decreased, instead, the disability rate has increased year by year. Clonus is a form of hypertonia, often manifested as velocity-dependent stretch hyperreflexes accompanied by tendon twitching, and is one of the manifestations of upper motor neuron syndrome.[1] The prevalence of post-stroke spasticity is 25.3%, and as high as 39.5% in hemiplegic patients, among which 9.4% of hemiplegic patients have severe or disabling symptoms.[2] The moderation of the spasticity is conducive to posture maintenance and a certain extent beneficial to rehabilitation. Whereas, excessive spasticity will lead to the occurrence of abnormal movement patterns and pain, which seriously limit the improvement of patients' daily living activities and prognosis.

The common clinical presentation of spasticity in stroke patients with hemiplegia is the lower extremity extensor pattern. The manifestations are straightening of the knee joint, foot drop, and pronation. The triceps surae is formed by the medical before lateral gastrocnemius and the soleus muscles. The gastrocnemius and soleus muscles work together to flex the foot. Stroke patients with hemiplegia have leg triceps hypertonia, clonus, and abnormal gait, which increases the risk of falling[3] and seriously affects the walking function. Therefore, effective treatment of calf triceps spasticity is essential. At present, the treatment methods for triceps spasticity of the lower leg mainly include stretch, radial or focused extracorporeal shock wave therapy,

neuromuscular electrical stimulation and other physical factor therapy, wearing ankle and foot orthotics, oral drug therapy, local and intrasheath drug injection therapy, and surgical treatment.[4] Therefore, treatments targeting spasticity that are more economical, effective, and noninvasive with fewer side effects still need to be studied. Recently, some papers have highlighted the possible involvement of the muscular fascia in spasticity, and some clinical trials suggest that a change in the fascial viscosity can decrease the muscular symptoms in post-stroke patients. The hypothesis is consistent with recent literature that proposes a role for peripheral tissue in the development of spasticity, in particular, the increase in viscoelastic properties of tissue.[5,6]

This study intends to explore the effectiveness of Fascial Manipulation on the triceps muscle of the calf in stroke patients to reduce spasticity. It aims to provide a new approach for lower limb spasticity.

Materials and Methods

Participants

Patients enrollment

A total of 40 patients with post-stroke triceps spasms were selected from October 2020 to December 2021 in the Department of Rehabilitation Medicine, the First Hospital of Shanxi Medical University. All the participants were in line with the stroke diagnosis points formulated by the Fourth Conference on Cerebrovascular Diseases of the Chinese Medical Association.[7] This study was approved by the Ethics Committee of the First Hospital of Shanxi Medical University, and all the patients and their families agreed to join the study and signed informed consent.

Inclusion criteria

i) Cerebral infarction or cerebral hemorrhage was diagnosed by head CT or MRI; ii) First onset, stable condition, understanding and cooperative treatment, course of disease 2 weeks to 6 months; iii) There was significant triceps surae spasticity, the Composite Spasticity Scale (CSS) was ≥7 and the Modified Ashworth Scale(MAS)≤level 3.

Exclusion criteria

i) Unstable vital signs; ii) Severe cognitive dysfunction; iii) Limited ankle joint activity, or local skin damage, infection; iv) Have coagulation dysfunction or thrombosis; iv) Have received other antispasmodic treatment in addition to conventional rehabilitation treatment.

Experimental grouping

This blinded randomized controlled trial was approved by the First Affiliated Hospital of Shanxi Medical University ethics committee. Forty patients with triceps spasms after stroke were selected and divided into two groups by random number table method: control group and FM group, with 20 cases in each group. Both groups received conventional rehabilitation therapy, and the FM group was treated with Stecco Fascia Manipulation^{R} based on conventional rehabilitation therapy. Stecco Fascia Manipulation^{R} for 3 weeks, twice a week, 30 minutes a time. In addition to blinding the subjects, we blinded the clinicians and the scale evaluators.

Treatment methods

i) Conventional rehabilitation therapy includes good limb placement, stretching training, physical factor therapy, balance function training, sitting and standing transfer training, muscle strength training, range of motion training, walking training, etc. The treatment duration is 3 weeks, 5 times per week, 40 minutes per time. The conventional rehabilitation therapy group was treated by the same physician. ii) Stecco Fascia Manipulation^{R}: According to the Stecco Fascia Manipulation^{R} assessment, diagnosis, and treatment system including movement and palpation examination, comprehensive evaluation, and scoring were performed. Finally, the selected myofascial chains were treated. Under the premise of fully exposing the skin of the calf and foot on the affected side, the therapist pressed and rubbed CC through the knuckles, finger abdomen, elbow joints, etc., and ended the treatment at this point when the patient reported that the pain was halved or the tissues gliding restored. Initial myofascial points were selected by a Fascia specialized Rehabilitation physician using a specific assessment methodology—Fascial Manipulation[8] involving clinical examination by movement and palpatory verifications of specific points

termed Centers of Coordination (CC). The rehabilitation specialist is certified as a Fascial Manipulation Specialist. The experts need training to obtain a fascia certification. A CC corresponded to the convergence of vectorial forces, into the deep fascia, generated by mono and biarticular motor units moving a joint in a specific direction. Palpation evaluation of these points included patient pain rate, radiation, and the presence of tissue stiffness.[8] The stiffness perceived by the physician. Dysfunctional segments were identified based on palpation evaluation and a hypothesis-driven differential by clinical history. The CC selected for treatment and belonging to the selected dysfunctional segments were compared to the muscles localized by standard dystonia assessment (Figure 1).

The location and operation methods of CC: i) IR-TA-CC: inside 1/3 of the middle leg, on the fascia of the tibial posterior muscle: the patient was supine with the inner leg facing upward; the therapist is located on the same side of the treatment point and uses the knuckles or elbows; ii) ER-TA-CC: peroneal longus and brevis: the patient was in a lateral position; the therapist is located on the same side of the treatment point and uses the elbow joint, or is located opposite the treatment point and uses the elbow joint; iii) RE-TA-CC: on the fascia of the triceps surae: the patient was placed in the prone position; the therapist is located on the same side of the treatment site and uses the elbow joint; iv) ME-TA-CC: the medial head of the gastrocnemius muscle is close to the tendon: the patient was placed in the prone position; the therapist is located on the same side of the treatment site and uses the elbow joint; v) AN-PE-CC: between the first and second phalanges, on the fascia of the extensor hallucis brevis muscle; the Patient was placed in the prone position and bent his knee; the therapist is located on the same side of the treatment point and uses the finger joint; vi) LA-PE-CC: dorsal side of the 2nd and 3rd interosseous muscles; the Patient was placed in the prone position and bent his knee; the therapist is located on the same side of the treatment point and uses the finger joint; each point was treated for 5 minutes, the treatment lasted 30 minutes and was performed twice a week for a total of 3 weeks before conventional rehabilitation therapy.

Evaluation method

The same rehabilitation physician evaluated the two groups of patients before the first treatment and 3 weeks after treatment, respectively. The physician does not know the different groups. The specific evaluation methods were as follows.

CSS

CSS[9] was used to reflect the changes in the degree of triceps spasm before and after treatment in the two groups. CSS includes three aspects of evaluation: muscle tension of calf triceps: 0, 2, 4, 6, and 8 points are assigned according to the size of muscle tension. The greater the muscle tension, the higher the score; Achilles tendon reflexes: from no reflexes to hyperreflexes, rated 0 to 4; Ankle clonus: On a scale of 1 to 4, the larger the score, the more severe the ankle clonus. The sum of the three rating scores is the final CSS score, CSS≥7 points is spasticity, the higher the CSS score, the more severe the spasticity.

Passive range of motion

Passive Range of Motion (PROM)[10] was used to measure the passive range of motion of the ankle. The patient was in the supine or seated position, and the rehabilitation physician measured the maximum passive dorsiflexion and plantarflexion Angle of the affected ankle joint with the help of a protractor. The sum of the two was PROM. A larger PROM indicates better ankle motion and less limitation of motion.

Fugl-Meyer motor function score

Simplified Fugl-Meyer motor function score (FMA)[11,12] was used to evaluate the motor function of the affected lower extremity. The total score was 34 points. The higher the score was, the better the motor function of the affected lower extremity was.

Modified Ashworth Scale (MAS)

Level 0: No increase in muscle tone, scored 0; Level I: Mild increase in muscle tension, with the affected part passively flexing and extending, the end of the range of motion suddenly getting stuck and showing minimal resistance, scored 1; Level I+: Mild increase in muscle tension. During passive flexion and extension, 50% of the joint's range of motion suddenly gets stuck. When continuing to conduct joint motion examination to the end, there is always a small resistance, scored 2; Level II: Muscle

tension increases significantly, and resistance increases significantly when moving through most of the range of motion of the joint. However, the affected part can still move more easily, scored 3; Level III: Severe increase in muscle tone, difficulty in passive activity examination, scored 4; Level IV: Stiffness, inability to bend or extend the affected part, scored 5.[12]

Statistical analyses

SPSS 25.0 software was used to analyze the data. Counting data were tested by chi-square test. Measurement data were expressed as mean ± standard deviation (X±S), paired sample t-test was used for intra-group comparison, and independent sample t-test was used for inter-group comparison. $P<0.05$ indicated a statistically significant difference.

Results

General information

The gender, age, course of disease, and lesion nature of the two groups were compared, and the difference was not statistically significant (P>0.05), which was comparable, as shown in Table 1.

Evaluation of clinical efficacy

Comparison of CSS before and after treatment

Before treatment, there was no significant difference in CSS between the two groups (P>0.05). After treatment, the CSS in the two groups was lower than that before treatment, the difference was statistically significant ($P < 0.05$). The reduction in the FM group was higher, compared with the control group, and the difference was statistically significant ($P < 0.05$), (Table 2).

PROM comparison before and after treatment

Before treatment, there was no significant difference in PROM between the two groups (P>0.05). After treatment, the PROM of the two groups was increased

compared with that before treatment, and the difference was statistically significant ($P<0.05$). The increase of the FM group was more obvious, compared with the control group, and the difference was statistically significant ($P<0.05$; Table 3).

Comparison of lower limb FMA before and after treatment

Before treatment, there was no statistically significant difference in lower limb FMA between the two groups ($P>0.05$); after treatment, in intra-group comparison, the lower limb FMA of the two groups was higher than that before treatment, the difference was statistically significant($P<0.05$), and the increase was more obvious in the FM group, compared with the control group, the difference was statistically significant ($P<0.05$; Table 4).

Comparison of MAS before and after treatment

Before treatment, there was no significant difference in MAS between the two groups ($P>0.05$). After treatment, the MAS in the two groups was lower than that before treatment, the difference was statistically significant ($P<0.05$), and the reduction in the FM group was more obvious, compared with the control group, the difference was statistically significant ($P<0.01$; Table 5).

Discussion

Spasticity is a form of hypertonia, often occurring in stroke patients. Although there is much research on the mechanism of spasticity after stroke, the specific pathophysiological mechanism is still not completely clear. At present, it is believed that the mechanism of spasticity after stroke mainly includes neural mechanisms and peripheral mechanisms. The neural mechanism is mainly manifested in abnormal descending regulation and abnormal intraspinal processing function. Over-excitation of α-motor neurons is the main manifestation of spinal cord changes in stroke patients with spasticity.[13] The peripheral mechanism is mainly the change of muscle mechanical properties,[14] that is, the inherent properties and muscle metabolism and function of the tissues that make up muscles, tendons, joints, and other structures. To further explore the mechanism of spasticity after stroke, Mirbagheri *et al.*[15] found in

an observation of the mechanical properties of elbows of patients with muscle spasms after stroke that the spasticity mechanism gradually transitioned over time from neurological factors to peripheral mediated factors. In a study on the number of motor units of the hypothenar muscle in the hands of patients with cerebral infarction, Arasaki *et al.*[16] found that the tissue structure of the muscle changed as early as 4 hours after cerebral infarction. Compared with normal muscles, spastic muscles showed increased stiffness after stroke.[17] At the same time, the increased stiffness of the muscle will further aggravate the spasticity of the limb. As for endomysium and perimysium, the collagen densities in connective tissue increases. Thus, injured muscles trended to become stiffer with a more linear behavior and a larger viscous component.[18]

The primary lesion leading to spasticity lies within the central nervous system, but the connective tissue in patients with spasticity is also dramatically altered because of paralysis and the ensuing immobilization. Antonio Stecco[19] argues that connective tissue alterations begin a vicious circle composed of three phases: i) an increase in the viscosity of the extra-cellular matrix leading to active muscle stiffness; ii) exacerbation of neurally mediated reflex mechanisms due to subclinical contractures affecting the threshold of muscle spindle activation; iii) fibrosis due to collagen deposition and an increase in passive muscle stiffness. Fibrosis leads to a further increase in extracellular matrix viscosity in the surrounding areas re-starting the circle. These peripheral mechanisms contribute to abnormal postural adaptation, and further disuse and disability. Thus, restoring normal connective tissue architecture and tissue gliding mechanisms might help interrupt the vicious circle.

Fascia is a dense, irregular, and malleable connective tissue that penetrates the human body to form a continuous three-dimensional structural support matrix of the whole body, which can adjust mechanical, thermal, and metabolic stress, and can be restored to its physiological state through external manipulative treatment.[20] The deep fascia refers to all the ordered, dense, fibrous layers that interact with the muscles, connecting different structures of the musculoskeletal system and transmitting muscle power far away.[7] Located at the junction of the deep fascia and the muscle surface, Hyaluronic Acid (HA) is a lubricant that enables normal sliding between the deep fascia and the epimysium.[21] Excessive accumulation of HA in the Extracellular Matrix (ECM) of muscle can dramatically increase its viscosity and alter its lubricating properties. Viscosity of the ECM has not been traditionally considered to

contribute to passive resistance in muscles. Muscle overactivity due to spasticity has been associated with hyperviscous ECM. The resulting increase in passive resistance to movement and reduction in force transmission can lead to muscle stiffness.[22,23] It is not just the accumulation of hyaluronic acid, but also the polymerization of HA that increases the viscosity of the ECM. The polymerization of HA has been affiliated with cites of CC's as well as increase in HA itself.[24] The role polymerization of HA plays beyond just volume increases of HA is further elaborated by many other scientists and the thought is offered for consideration. In a controlled clinical study, 3D-T1P magnetic resonance imaging was used to compare HA quantity in muscles of five healthy participants to that of five post-stroke patients with stiffness. It was found that HA concentration in patients with post-stroke muscle stiffness is higher compared to controls.[25,26] Other small clinical trials showed that after treating patients with post-stroke muscle stiffness with intramuscular hyaluronidase injection, there was a significant improvement in stiffness and an increase in passive and active movement.[25-28] Increased viscosity of hyaluronic acid and acidification of extracellular matrix lead to dysfunction of fascia.

When dysfunction occurs, FM can reduce viscosity in loose connective tissue, and this result can be reflected by ultrasound.[29] Stecco FM has been widely used in the treatment of musculoskeletal diseases in recent years, which can effectively reduce pain and improve disability.[30]

Although the exact mechanism has not been explained, we argue that the mechanism of Fascial Manipulation may relieve spasticity by modulation of the following factors: i) it stimulates the central nervous system and autonomic nervous system at the same time; the regulation of the central nervous system reduces the overall muscle tension, while the autonomic nervous system reduces the tension by dilating blood vessels and reducing tissue viscosity to relieve spasms; ii) it can effectively reduce the viscoelasticity of extracellular matrix, and tissue stiffness and increase the sliding between collagen fiber layers of deep fascia caused by the accumulation of hyaluronic acid, relieving spasm; iii) fascia can actively contract has abundant innervation, and is rich in proprioceptors such as Ruffini and Pacini corpuscles, which can sense changes in tension.[31] Restoring the physiologic state of the fascia can improve nerve response and stimulate proprioception at the same time. In a study on ankle spasms in stroke patients with hemiplegia, Mirbagheri *et al.*[32] observed changes in neuromuscular characteristics throughout the entire range of

ankle motion. They found that nerve reflexes combined with peripheral factors such as muscle and connective tissue change to limit ankle movement. Among them, the movement limitation caused by nerve reflexes was most obvious in the neutral ankle position, while the changes of peripheral factors such as muscle characteristics influenced the angle of ankle dorsiflexion to a greater extent. In the present study, after a course of treatment, the PROM of the ankle joint was significantly enlarged in the observation group, which we can assume is the result of FM. Therefore, stroke patients with hemiplegia, due to tibial anterior muscle weakness and triceps spasm of the calf, are mostly manifested as foot drop, foot varus, limited dorsiflexion of the ankle, and even the development of Achilles tendon contracture retraction. The effect of Fascial Manipulation is to reduce the degree of leg spasticity by affecting the peripheral mechanism. The reduction of spasticity can improve the strength of the tibial anterior muscle on the hemiplegic side by influencing the reciprocal inhibition and combining it with routine rehabilitation training,[33] thus greatly increasing the range of motion of the ankle joint and improving the lower limb function (expressed as FMA score) on the hemiplegic side, which is consistent with the results of this study.

In the treatment of patients with hand spasms after stroke, Zhang Zengqiao *et al.*[34] found that acupuncture of Feng's fascia point could effectively relieve spasms. Liu Baoguo *et al.*[35] applied the theory of myofascial injury to stroke patients to relieve spasms through acupuncture, massage, and other ways. The efficacy of Chinese medicine acupuncture fascia points to relieve spasmodic has been proven. Compared with acupuncture on the fascia point, the Stecco FM adopted in this study has the advantages of being non-invasive, having less pain, having an immediate effect, and having higher tolerance. However, there are still some limitations in this study. First of all, the sample size included in this study is insufficient to completely exclude the influence of chance. Second, patients in the observation group were not followed up in this study to evaluate the long-term effect of FM. Third, FM should be investigated compared to actual therapy options such as shockwave therapy or botulinum toxin injection therapy. Finally, the mechanism of FM to relieve spasticity is still unclear and needs to be confirmed by a large number of studies. Therefore, the specific mechanism of FM to improve spasticity, as well as the medium- and long-term efficacy needs further study.

Conclusions

Fascial Manipulation can effectively relieve spasticity of the lower limb and ankle clonus in stroke patients, and improve the passive range of motion of the ankle joint, and the motor function of the affected lower limb in the short term. It acts on the peripheral component of spasticity partially restoring physiological viscoelastic properties of the tissues and tissue gliding. As a new anti-spasmodic method, Fascial Manipulation has the advantages of being non-invasive, repeatable, and highly effective. It enhances the effect of conventional physiotherapy and can be proposed as an alternative or complementary to other therapies such as botulinum toxin.

Correspondence: Federico Giordani, Villa Rosa Neurological Rehabilitation Hospital, APSS Trento, Trento, Italy.
ORCID ID: 0000-0001-7598-8189
E-mail: federico.giordi@gmail.com

Co-authors:

Wenyan Li,

Orcid ID:0009-0006-2402-9037

E-mail: wen198102@163.com

Xin Liu,

Orcid ID:0009-0001-9764-7278

E-mail: robbie234@163.com

Yinghua Wen,

Orcid ID:0009-0000-0423-3758

E-mail: 164932433@qq.com

Junying Wu,

Orcid ID:0009-0004-9841-6537

E-mail: wujunying777@126.com

Carla Stecco

Orcid ID: 0000-0002-8767-4555

E-mail: carla.stecco@unipd.it

Contributions: conceptualization, CS and JW; methodology, WL; software, XL; validation, WL, XL, and YW; data curation, XL; writing—original draft preparation, WL; writing—review and editing, FG, CS; supervision, JW; project administration, CS. All authors have read and agreed to the published version of the manuscript.

Funding: this research received no external funding.

Institutional review board statement: the study was conducted in accordance with the Declaration of Helsinki, and approved by the Ethics Committee of the First Hospital of Shanxi Medical University (2020 K-K088).

Informed consent statement: informed consent was obtained from all subjects involved in the study. Written informed consent has been obtained from the patients to publish this paper.

Availability of data and materials: the article does not publicly share research data.

Acknowledgments: the article does not accept any support given which is not covered by the author's contribution or funding sections.

Conflicts of interest: the authors declare no conflict of interest.

The list of abbreviations

Abbreviations	Complete spelling
CC	Centers of Coordination
CSS	comprehensive spasticity scale
ECM	Extracellular Matrix
FM	Fascial Manipulation
FMA	Fugl-Meyer motor function score
HA	Hyaluronic Acid
MAS	Modified Ashworth Scale
PROM	passive range of motion

References

[1] Lance JW. The control of muscle tone, reflexes, and movement: Robert Wartenbeg Lecture. Neurology 1980;30:1303-13.

[2] Zeng H, Chen J, Guo Y, Tan S. Prevalence and risk factors for spasticity after stroke: a systematic review and meta-analysis. Front Neurol 2021;11:616097.

[3] Luo WY, Tang YM. Expert consensus on fall risk assessment and comprehensive intervention after stroke. J Clin Intern Med 2022;39:63-8.

[4] Bethoux F. Spasticity management after stroke. Phys Med Rehabil Clin 2015;26:625-39.

[5] Trompetto C, Marinelli L, Mori L, et al. Pathophysiology of spasticity: implications for neurorehabilitation. Biomed Res Int 2014;2014:354906.

[6] Azzollini V, Dalise S, Chisari C. How does stroke affect skeletal muscle? State of the art and rehabilitation perspective. Front Neurol 2021;12:797559.

[7] Chinese Medical Association. Diagnostic points of various cerebrovascular diseases. Chin J Neurol 1996;6:60-1.

[8] Stecco C, Day JA. The fascial manipulation technique and its biomechanical model: a guide to the human fascial system. Int J Ther Massage Bodywork 2010;3:38.

[9] Yan TB. Study on the reliability of comprehensive spasm scale. Chin J Rehabil

Med 2002;5:263-5.
[10] Yan GB. Range of motion, ROM. Chin J Joint Surg Electron Ed 2014;8:409.
[11] Lu ZY. Application of Fugl-Meyer scale in stroke rehabilitation assessment. J Clin Med 2016;3:2032-4.
[12] Blackburn M, Van Vliet P, Mockett SP. Reliability of measurements obtained with the modified Ashworth scale in the lower extremities of people with stroke. Phys Ther 2002;82:25-34.
[13] Katz RT, Rymer WZ. Spastic hypertonia: mechanisms and measurement. Arch Phys Med Rehabil 1989;70:144-55.
[14] Li S, Francisco GE. New insights into the pathophysiology of post-stroke spasticity. Front Hum Neurosci 2015;9:192.
[15] Mirbagheri MM, Tsao C, Settle K, et al. Time course of changes in neuromuscular properties following stroke. In: 30th Annual International Conference of the IEEE Engineering in Medicine and Biology Society. Piscataway: IEEE; 2008, pp 5097-5100.
[16] Arasaki K, Igarashi O, Ichikawa Y, et al. Reduction in the motor unit number estimate (MUNE) after cerebral infarction. J Neurol Sci 2006;250:27-32.
[17] Kesikburun S, Yaşar E, Adıgüzel E, et al. Assessment of spasticity with sonoelastography following stroke: a feasibility study. PM R 2015;7:1254-60.
[18] Jalal NIM, Gracies JM, Zidi M. Mechanical and microstructural changes of skeletal muscle following immobilization and/or stroke. Biomech Model Mechanobiol 2020;19:61-80.
[19] Stecco A, Stecco C, Raghavan P. Peripheral mechanisms contributing to spasticity and implications for treatment. Curr Phys Med Rehabil Rep 2014;2:121-7.
[20] Adstrum S, Hedley G, Schleip R, Stecco C, Yucesoy CA. Defining the fascial system. J Bodyw Mov Ther 2017;21:173-7.
[21] McCombe D, Brown T, Slavin J, Morrison W. The histochemical structure of the deep fascia and its structural response to surgery. J Hand Surg 2001;26:89-97.
[22] Rasool G, Wang AB, Rymer WZ, Lee SS. Shear waves reveal viscoelastic changes in skeletal muscles after hemispheric stroke. IEEE Trans Neural Syst Rehabil Eng 2018;26:2006-14.
[23] Wang AB, Perreault EJ, Royston TJ, Lee SS. Changes in shear wave propagation within skeletal muscle during active and passive force generation. J Biomech

2019;94:115-22.

[24] Pavan PG, Stecco A, Stern R, Stecco C. Painful connections: densification versus fibrosis of fascia. Curr Pain Headache Rep 2014;18:441.

[25] Menon RG, Raghavan P, Regatte RR. Quantifying muscle glycosaminoglycan levels in patients with post-stroke muscle stiffness using T1ρ MRI. Sci Rep 2019;9:14513.

[26] Menon RG, Oswald SF, Raghavan P, Regatte RR, Stecco A. T1ρ-mapping for musculoskeletal pain diagnosis: Case series of variation of water bound glycosaminoglycans quantification before and after fascial manipulation® in subjects with elbow pain. Int J Environ Res Public Health 2020;17:708.

[27] Raghavan P, Lu Y, Mirchandani M, Stecco A. Human recombinant hyaluronidase injections for upper limb muscle stiffness in individuals with cerebral injury: a case series. EBioMedicine 2016;9:306-13.

[28] Raghavan P. Emerging therapies for spastic movement disorders. Phys Med Rehabil Clin 2018;29:633-44.

[29] Hughes E, Koenig JM, Lee RS, McDermott K, Freilicher T, Pitcher MH. Pilot study assessing the effect of Fascial Manipulation on fascial densifications and associated pain. Eur J Transl Myol 2022;32:10369.

[30] Arumugam K, Harikesavan K. Effectiveness of fascial manipulation on pain and disability in musculoskeletal conditions. A systematic review. J Bodyw Mov Ther 2021;25:230-9.

[31] Morley J, Fan C, McDermott K, Fede C, Hughes E, Stecco C. The crural interosseous membrane re-visited: a histological and microscopic study. Eur J Transl Myol 2019;29:8340.

[32] Mirbagheri MM, Alibiglou L, Thajchayapong M, Rymer WZ. Muscle and reflex changes with varying joint angle in hemiparetic stroke. J Neuroeng Rehabil 2008;5:6.

[33] Hara T, Abo M, Hara H, Sasaki N, Yamada N, Niimi M, Shimamoto Y. The effect of repeated botulinum toxin A therapy combined with intensive rehabilitation on lower limb spasticity in post-stroke patients. Toxins 2018;10:349.

[34] Zhang ZQ, Li KP, Zhu Y, Zhou P, Hu CD, Hu XS, Feng W. Preliminary observation on clinical efficacy of Feng's fascia point on relieving hand spasm after stroke. Shanghai J Tradit Chin Med 2018;52:66-8.

[35] Liu BG, Wu J, Li ZY, You QZ, Le S. Current situation and treatment of myofascial injury in stroke patients. Med Forum 2021;25:2375-7.

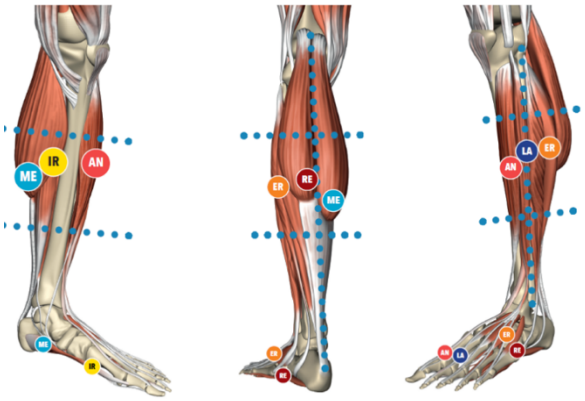


Figure 1. The CCs were selected for treatment under standard dystonia assessment.

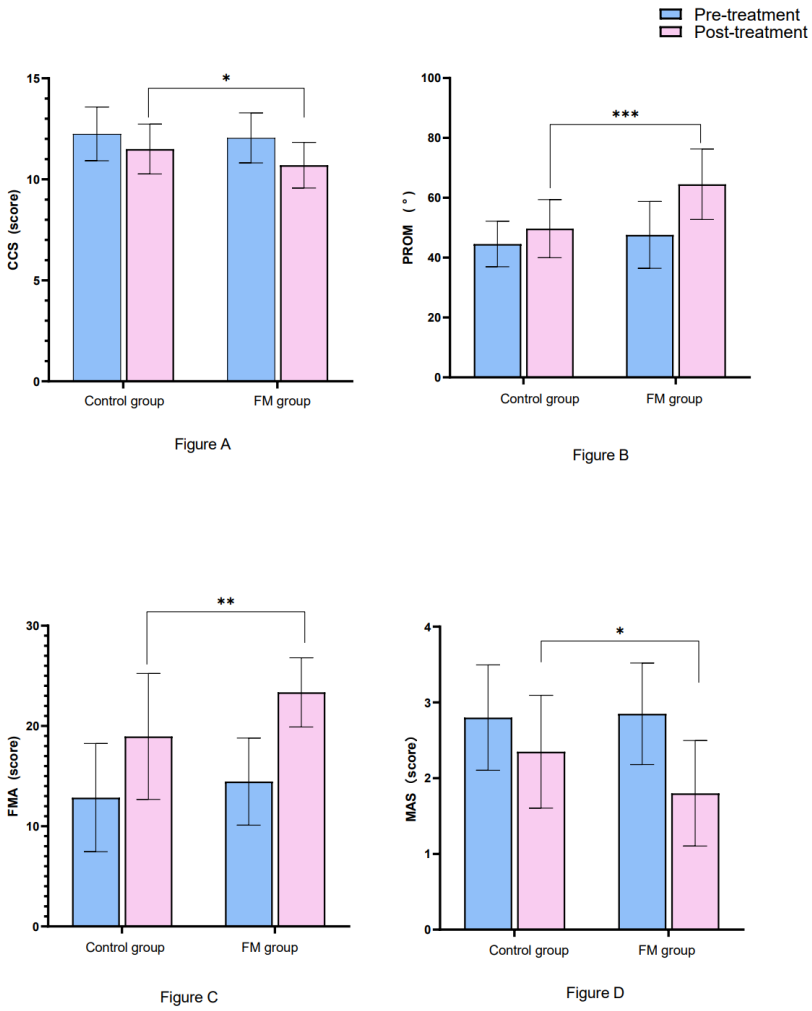


Figure 2. Comparison of various indexes before and after treatment between 2 groups.

Table 1 Comparison of general data between the two groups (X±S)

Group	Total Patients	Sex		Age (year) (`X±S)	Duration of stroke (days) (`X±S)	Stroke type	
		male	female			Infarct	hemorrhage
Control group	20	12	8	55.75±13.08	53.21±18.22	11	9
FM Group	20	10	10	52.63±13.73	50.80±13.32	13	7

Table 2. Comparison of CSS before and after treatment between the two groups

(X±S).

Group	Number	Pre-treatment	Post-treatment	*t*	*P*
Control group	20	12.25±1.33	11.50±1.23[a]	3.290	0.004
FM Group	20	12.05±1.23	10.70±1.12[ab]	10.283	0.000
t		0.492	2.138		
P		0.625	0.039		

Table 3. Comparison of PROM before and after treatment between 2 groups (X±S).

Group	Number	Pre-treatment	Post-treatment	*t*	*P*
Control group	20	44.56±7.59	49.68±9.69[a]	-3.104	0.006
FM Group	20	47.59±11.16	64.51±11.77[ab]	-5.140	0.000
t		-1.005	-4.347		
P		0.321	0.000		

Table 4. Comparison of lower limb FMA before and after treatment between 2 groups （X±S ）

Group	Number	Pre-treatment	Post-treatment	*t*	*P*
Control group	20	12.85±5.39	18.95±6.27[a]	-6.118	0.000
FM Group	20	14.45±4.34	23.35±3.45[ab]	-10.682	0.000

t		-1.033	-2.746		
P		0.308	0.010		

Table 5. Comparison of MAS before and after treatment between 2 groups（X±S）

Group	Number	Pre-treatment	Post-treatment	*t*	*P*
Control group	20	2.80±0.70	2.35±0.75[a]	2.932	0.009
FM Group	20	2.85±0.67	1.80±0.70[ab]	5.294	0.000
t		-0.231	2.971		
P		0.818	0.005		