

Research Article



Effects of Reactive and Proactive Exercises on Ankle Spasticity in Patients with Chronic Stroke: A Randomized Clinical Trial

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ABSTRACT

Introduction: Stroke is a leading cause of adult disability. Spasticity is a neurological condition that occurs secondary to stroke and affects the patient's quality of life. This study aimed to compare the effects of reactive and proactive exercises on spasticity in people with stroke.

Materials and Methods: In this single-blind clinical trial, 30 individuals with stroke were randomly divided into three groups: Control, reactive, and proactive, of which 27 patients completed the study. These individuals were treated for 12 sessions for 4 weeks (three times a week). The control group received conventional exercises, the reactive group received conventional exercises plus reactive exercises, and the proactive group received conventional exercises plus proactive exercises. The outcomes were clinical measures of spasticity at baseline and neural properties, evaluated using the Modified Modified Ashworth Scale and H-reflex latency and Hmax/Mmax ratio. All statistical analyses were performed using SPSS software, version 20.

Results: No significant differences were observed between the groups in H-reflex latency ($F=0.404$, $P=0.672$, $\eta_p^2=0.033$) and Hmax/Mmax ratio ($F=0.878$, $P=0.429$, $\eta_p^2=0.071$).

Conclusion: Intergroup comparisons did not show a significant advantage of proactive and reactive exercises over conventional exercises alone in improving neural properties of spasticity.

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Introduction

Stroke remains the principal cause of long-term disability in adults and the second most common cause of death worldwide [1]. Spasticity, a frequent consequence of upper motor neuron lesions, exerts marked effects on skeletal muscle function [2]. Lance originally defined spasticity as a velocity-dependent enhancement of stretch reflex activity in the absence of voluntary contraction [3]. More recently, Dressler has described it as involuntary muscle overactivity in central paresis, triggered by either slow or rapid passive joint motion or by sensory input [4]. The reported prevalence after stroke varies widely, ranging from 4–46% within the first month, 4.16–48% at 1–3 months, 6.9–63% at 3–6 months, and 7.6–49% beyond 6 months [5].

Clinically, spasticity is associated with increased muscle tone and stiffness, exaggerated tendon reflexes, and contracture formation. These abnormalities contribute to reduced voluntary strength, impaired balance, and deficits in motor control, ultimately limiting functional independence [6–8]. Spastic overactivity of ankle plantar flexors is an important predictor of post-stroke falls [9, 10] and is closely linked to reduced quality of life [11].

Current management strategies include removal of noxious stimuli, use of antispastic positioning, active and passive range-of-motion and stretching exercises, night and standing ankle splints, chemo-denervation with botulinum toxin, and oral antispastic agents, such as tizanidine, baclofen, and benzodiazepines [12, 13]. More recent rehabilitation concepts emphasize task-specific, goal-directed, engaging, progressive, and adaptive training approaches to enhance motor recovery after stroke [14].

In recent decades, evidence-based frameworks have been proposed to optimize posture and movement in this population. These frameworks rely on principles of motor learning and exploit both feedback and feedforward mechanisms to improve motor control [15]. Neurophysiological studies have shown that proactive motor control relies on feedforward mechanisms while reactive motor control relies on feedback mechanisms [16].

Anticipatory postural adjustments, often referred to as proactive exercises, are mediated through feedforward mechanisms and represent self-initiated, predictable reactions [17]. They stabilize the trunk and stabilizing musculature before voluntary limb motion or predictable perturbations [18]. In contrast, compensatory postural

responses, or reactive exercises, are driven by feedback mechanisms and are elicited in response to unexpected perturbation [17]. Reactive postural control therefore reflects the ability to respond rapidly after a perturbation [19]. While anticipatory and compensatory strategies are related, they play distinct roles; efficient anticipatory control reduces reliance on compensatory reactions [18]. Neurophysiological studies have shown that these responses engage spinal and subcortical pathways that enable precise action at the shortest possible latencies [20]. In older adults and in individuals with neurological conditions such as stroke, anticipatory mechanisms are often delayed or impaired, resulting in a greater dependence on compensatory responses to maintain stability [18].

One commonly used method for quantifying spasticity is the Hoffmann (H) reflex. This electrophysiological measure reflects the excitability of α -motor neurons and correlates with spastic hypertonia [21]. Enhancement of the H reflex may arise from diminished inhibitory modulation of lower motor neurons or increased stretch reflex excitability due to reduced presynaptic inhibition [22]. Several studies have shown that H-reflex and M-wave parameters provide relatively objective indices of spasticity and can complement clinical assessment [23, 24].

Although several studies have explored the influence of exercise programs on spasticity in stroke [2, 14], there is limited evidence regarding training paradigms that explicitly engage feedback- and feedforward-based postural control. Therefore, the present clinical trial aimed to compare the effects of reactive and proactive exercises, designed to activate feedback and feedforward mechanisms, on ankle spasticity in patients with stroke.

Materials and Methods

Study setting

The trial was conducted between 2022 and 2024 in the physiotherapy department of Ayatollah Kashani Hospital, Isfahan, Iran.

Participants and experimental design

This study was a parallel, single-blind, randomized clinical trial. Individuals experiencing a first unilateral stroke were identified through referrals from neurology outpatient services. Patients who expressed willingness to participate provided written informed consent. Eligible participants were then randomly allocated to one of three arms: A reactive exercise group, a proactive exercise group, or a control group receiving conventional

therapy. In this study, a block randomization method was used for random allocation. Using statistical software, the statistician designed 10 blocks of three, because there were three groups. Randomization was performed by the clinic secretary, who was unaware of the study.

Inclusion and exclusion criteria

The inclusion criteria were:

first-ever unilateral stroke confirmed by CT scan or MRI and neurologist report [2]; time since stroke ≥ 6 months [19]; sufficient cognitive ability to comprehend and execute basic verbal instructions, as determined by a Persian version of the Mini-Mental State Examination (score ≥ 24) [2]; ability to stand and walk independently without assistive devices [25]; presence of gastrocnemius spasticity graded ≥ 1 on the modified modified ashworth scale (MMAS ≥ 1) [2].

The exclusion criteria comprised:

additional neurological conditions such as Parkinsonism or diabetic neuropathy [2]; severe visual, depth perception, or vestibular disorders [25]; serious musculoskeletal problems in the lower limbs such as fixed contractures [2]; intense lower-limb pain rated 75–100 mm on the visual analog scale [26]; participation in other concurrent physiotherapy interventions [2]; use of medications affecting muscle tone, sensation, or strength [27]; body weight >150 kg or height >2.1 m [19].

Sample size

Based on a previous study [19] and using a significance level of $\alpha=0.05$ ($Z=1.96$), power of 80% ($Z_{1-\beta}=0.84$), correlation $\rho=0.8$, and one pre-test ($v=1$) and two post-intervention measurements ($\omega=2$), yielding $R=0.8$, the required sample size was calculated as 10 participants per group (Equations 1 and 2).

$$n_{repeated} = R \left\lceil \left[\frac{2(Z_{1-\alpha/2} + Z_{1-\beta})}{\Delta^2} + \frac{Z_{1-\alpha}^2}{4} \right] \right\rceil \quad (1)$$

$$R = \left\lceil \frac{1 + (\omega - 1)\rho}{\omega} \right\rceil - \frac{v\rho^2}{1 + (v - 1)\rho} \quad (2)$$

Patient recruitment

A total of 68 individuals with stroke were screened. Thirty-eight did not meet the inclusion criteria, met at least one exclusion criterion, or declined participation. The remaining 30 participants were randomly assigned to the control, reactive, or proactive groups (10 per group) (Figure 1).

Treatment protocol

Following baseline assessment, all groups received 12 treatment sessions over four weeks (three sessions per week on alternate days) [2].

Control group (conventional therapy): Each session lasted 35 minutes: A 5-minute warm-up period, followed by 25 minutes of conventional balance-focused physiotherapy, and concluding with a 5-minute cool-down phase. Conventional therapy consisted of lower-limb range-of-motion activities, weight-bearing tasks on the affected side, gait training on parallel bars with forward, backward, and sideways steps, and bridging exercises and muscle-stretching routines [28].

Reactive and proactive groups: Each session lasted 60 minutes, comprising a 5-minute warm-up, 25 minutes of general balance exercises performed by the control group, an additional 25 minutes of group-specific balance training (reactive or proactive), and a final 5-minute cool-down.

For each group, five specific exercises were used, with 5 minutes allocated per exercise. Within each 5-minute block, the exercise was repeated two to five times depending on the patient's capacity. All training sessions were performed under the direct oversight of a physiotherapist.

Reactive exercise examples: Examples include seated and standing perturbation exercises, therapist-applied push-and-release challenges, balance tasks on unstable surfaces, and "cowboy-style" movements performed on a therapy ball.

Proactive exercise examples: Catching and throwing a ball, rotational movements, reaching tasks, single-leg stance, and tandem walking.

Outcome measurements

All measurements were performed by an assessor who was not involved in treatment delivery and remained blinded to group allocation. Spasticity of the ankle plan-

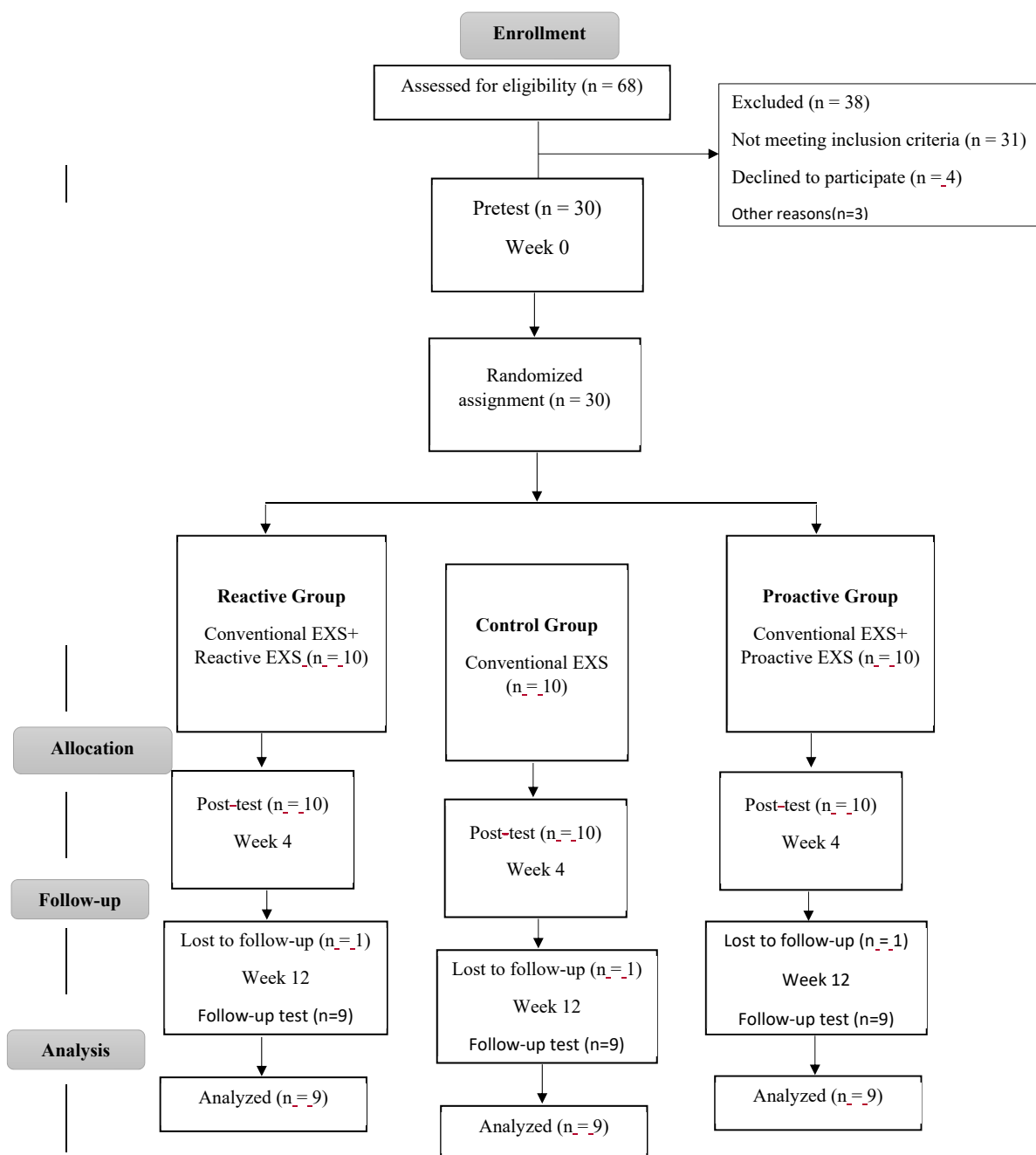


Figure 1. The CONSORT flowchart

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tar flexors was evaluated clinically at baseline and electrophysiologically at three time points: pre-intervention, immediately after the 4-week intervention, and two months after treatment completion.

Clinical assessment of spasticity

Resistance of the hemiplegic ankle plantar flexors to passive stretching was graded using the MMAS, a widely used and reliable instrument for quantifying spasticity

[29]. Using the validated Persian version of the MMAS, the blinded examiner rated resistance to passive movement on a 0–4 scale [30].

Electrophysiological assessment of spasticity

H-reflex and M-wave responses of the soleus muscle were recorded using an electrodiagnostic device. Participants lay prone on the treatment bed with their feet extending beyond the edge. After shaving and cleans-

Table 1. Demographic features of participants in the proactive, reactive, and control groups (n=10)

Variables	Mean±SD			95% CI for Mean		P
	Proactive Group	Reactive Group	Control Group	Lower Bound	Upper Bound	
Age (y)	57.9±8.47	63.2±10.64	52.9±10.33	54.1	61.9	0.083
BMI (Kg/m ²)	25.54±3.37	22.73±2.4	25.54±3.57	23.36	25.85	0.090
Post stroke duration (m)	26.5±17.04	14.7±14.33	25.9±22.79	15.43	29.31	0.288

BMI: Body mass index.

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ing the skin with soap-water-soaked cotton, a line was drawn from the midpoint of the popliteal fossa to the Achilles tendon and divided into eight equal segments to optimize electrode placement. The active electrode was positioned over the soleus muscle at the fifth or sixth distal segment, while the reference electrode was placed on the Achilles tendon. A ground electrode was located between the active electrode and the stimulation anode. The tibial nerve was stimulated in the popliteal fossa with the cathode proximal to the anode. Recording settings were: gain 500 μ V, sweep speed 10 ms/cm, stimulus duration 1 ms, and band-pass filter 2–10,000 Hz [31]. Supramaximal stimuli were used to determine the maximal M-wave amplitude. The recorded variables included M- and H-wave amplitudes, H-reflex latency, and the Hmax/Mmax ratio. All electrophysiological measurements were obtained by an experienced neurologist blinded to group allocation.

Data analysis

Data were analyzed using IBM SPSS software, version 20. The normality of the distributions was checked using the Shapiro–Wilk test. For normally distributed variables, one-way analysis of variance (ANOVA) and repeated-measures ANOVA were applied. Non-normally distributed variables were evaluated using the Kruskal–Wallis test. A $P<0.05$ was considered statistically significant.

Results

Baseline characteristics

The Mean±SD values of age, body mass index (BMI), and post-stroke duration for the three groups are summarized in Table 1. One-way ANOVA revealed no significant between-group differences in age or BMI ($P=0.09$), and the Kruskal–Wallis test showed no significant group differences in stroke duration ($P=0.29$).

Table 2 presents the distribution of qualitative variables: Sex, affected side, stroke type, use of assistive devices, and baseline spasticity scores. According to the Kruskal–Wallis test, none of these variables differed significantly among the groups ($P>0.05$).

Electrophysiological outcomes

Table 3 presents the detailed electrophysiological data. H-reflex latency: Repeated-measures analysis showed no significant main effect of time across the three groups ($F=1.467$, $P=0.241$, $\eta_p^2=0.058$). The time×group interaction was also non-significant ($F=0.589$, $P=0.673$, $\eta_p^2=0.047$), and there were no significant differences between groups overall ($F=0.404$, $P=0.672$, $\eta_p^2=0.033$). Based on the effect size index, neither treatment method had a significant advantage over the change in H-reflex delay time, and the magnitude of the difference between groups for this outcome was not significant.

Hmax/Mmax ratio: For the ratio of maximum H-reflex amplitude to M-wave amplitude, there was no significant main effect of time ($F=1.491$, $P=0.236$, $\eta_p^2=0.061$). The interaction between time and group was non-significant ($F=0.267$, $P=0.898$, $\eta_p^2=0.023$), and no significant group differences were observed ($F=0.878$, $P=0.429$, $\eta_p^2=0.071$). According to the effect size index, the magnitude of the difference between groups for this outcome was not significant.

Discussion

This randomized trial did not demonstrate significant changes in H-reflex latency or Hmax/Mmax ratio within or between the reactive, proactive, and conventional therapy groups.

Post-stroke spasticity can cause considerable complications, including pain, impaired upper- and lower-limb function, reduced range of motion, gait disturbances, limitations in daily activities, and substantial deterior-

Table 2. The frequency of qualitative background variables in proactive, reactive, and control groups (n=10)

Variables	Proactive Group	Reactive Group	Control Group	P
Gender	F:2, M:8	F:4, M:6	F:2, M:8	0.517
Affected side	L:6, R:4	L:8, R:2	L:7, R:3	0.631
Stroke type	I:5, H:5	I:7, H:3	I:4, H:6	0.404
Assistive device	Yes:5, No:5	Yes:5, No:5	Yes:7, No:3	0.592
Spasticity score	1:0, 2:7, 3:3, 4:0	1:2, 2:3, 3:5, 4:0	1:2, 2:5, 3:3, 4:0	0.857

Abbreviations: F: Female; M: Male; L: Left; R, right; I: Ischemic; H: Hemorrhagic.

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Table 3. Electrophysiological measurements of the study population (n=9)

Variables	Mean±SD								
	Baseline			Post Training			Follow-up		
	Proactive Group	Reactive Group	Control Group	Proactive Group	Reactive Group	Control Group	Proactive Group	Reactive Group	Control Group
H-RL (ms)	33.15±4.88	32.08±5.42	32.9±3.61	32.74±4.15	31.45±5.12	32.5±3.53	33.4±4.5	31.62±5.42	32.96±3.17
H/M R (mv)	0.369±0.056	0.325±0.035	0.367±0.054	0.376±0.064	0.34±0.039	0.374±0.058	0.366±0.062	0.334±0.035	0.364±0.059

Note: H/M R: H max /M max Ratio; H-RL: H-reflex latency.

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ration in quality of life [32, 33]. Both pharmacological and non-pharmacological modalities are used to address spasticity and improve functional outcomes, including conservative interventions, acupuncture, and dry needling [34]. Exercise-based rehabilitation remains a central, low-cost strategy for reducing abnormal muscle tone and enhancing function in individuals after stroke. Zhang et al. reported that aquatic exercise resulted in better functional outcomes than land-based therapy, despite a similar degree of spasticity in both groups [35]. Among conservative interventions, stretching is one of the most frequently prescribed components to increase joint range of motion and manage spasticity in rehabilitation programs [36].

Previous work has shown that proactive and reactive training approaches can improve voluntary movement control, enhance rapid postural responses, reduce fall incidence, and augment reactive balance in individuals with stroke [15, 37, 38]. Specific reactive exercises, such as perturbation-based balance training (PBT), tar-

get these mechanisms and may therefore be particularly useful for fall prevention [38]. Mansfield et al. evaluated body-weight-supported PBT in comparison with traditional balance exercises in individuals with chronic stroke and reported that PBT resulted in superior gains in reactive balance control [19]. There is increasing evidence that the cortex becomes engaged during reactive balance control as balance task difficulty increases [20]. Recent work has demonstrated cortical involvement in reactive balance responses [39]. It seems that the same situation occurred in our study, meaning that these exercises affected more areas of the cortex; therefore, our measurement methods, which measured subcortical parameters, were unable to track these changes.

The timing of intervention appears critical. PBT applied during the subacute phase of stroke may yield greater benefits than during the chronic stage, because substantial spontaneous recovery and neuroplastic changes occur in the early weeks after stroke, plateauing around three months and declining thereafter; beyond six

months, neurological recovery is typically limited [38]. In the present study, all participants had chronic stroke with a duration >6 months, which may have constrained the potential for neurophysiological change.

In addition to clinical outcomes, the current trial incorporated electrophysiological indices to evaluate potential neural adaptations induced by reactive and proactive training. Among reflex responses evoked by electrical stimulation, the H-reflex is widely used. H-reflex latency and the Hmax/Mmax ratio are considered reliable markers of α -motor neuron excitability [40]. Prior research has documented increased H/M ratios in stroke survivors compared to healthy controls [41, 42]. This ratio reflects the excitability level of motor neuron pools, which tends to rise in the presence of spasticity; typically, the H-reflex amplitude is elevated, whereas the M-wave amplitude may be reduced [43]. Such changes may result from augmented loop gain within the monosynaptic reflex arc, enhanced postsynaptic excitation from descending pathways, and/or diminished presynaptic inhibition [41, 44].

In contrast to some earlier reports, we did not observe significant modifications in H-reflex latency or the H/M ratio following either conventional therapy or the reactive and proactive protocols. Nor were there significant differences among groups. Another randomized trial has shown that functional stretching exercises can alter neural properties in chronic stroke patients [2], whereas some studies, such as that by Bakheit et al., have similarly reported no change in Hmax/Mmax ratio or H-reflex latency after a single session of isotonic or isokinetic stretching in spastic stroke [31].

A likely explanation for the absence of neurophysiological changes in our study is the participants' chronic stage. When a long time has elapsed since stroke, neural recovery is limited, and plasticity is reduced. Furthermore, evidence suggests that stroke survivors can be categorized into two groups with low or high reflex torque based on neurological characteristics [45]. In patients with spastic hemiplegia, facilitation of Ia afferents during soleus H-reflex testing may be comparable to that in healthy individuals, suggesting preserved presynaptic inhibition. Consequently, reflex stiffness may be similar to that of controls, and mechanical alterations in muscle rather than changes in reflex pathways may account for much of the spasticity observed in chronic stroke.

This study has several limitations. The sample size was relatively small, which may have limited statistical power to detect subtle effects, and there is a possibility of a Type II error. The exercise time was not the same among

the three groups. We also did not systematically measure adherence to the exercise protocols or participants' perceptions of acceptability and tolerability. Future research should consider larger cohorts and incorporate additional neurophysiological markers. As suggested by Heng-Yi Shen et al., the Hslp/Mslp ratio may provide a more sensitive indicator of spasticity in both upper and lower limbs in hemiplegic patients [19].

Conclusion

In this randomized controlled trial, reactive and proactive exercises did not show statistically significant differences compared with conventional exercises alone in electrophysiological measures of ankle spasticity in patients with chronic stroke. The sensitivity of the measurements in this study was low, and more advanced methods are needed to monitor the effects of these exercises at the cortical and subcortical levels in future studies.

Ethical Considerations

Compliance with ethical guidelines

The study protocol was approved by the Ethics Committee of [Isfahan University of Medical Sciences](#), Isfahan, Iran (Code:IR.MUI.RESEARCH.REC.1399.302) and was prospectively registered in the [Iranian Registry of Clinical Trials \(IRCT\)](#) (Code: IRCT20200101045970N4). All participants were fully informed about the study objectives and procedures prior to enrolment.

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Authors' contributions

Conceptualization, funding acquisition and study design: Ehsan Ghasemi; Administrative, technical, or logistic support: Ehsan Ghasemi, Faezeh Kouhestani Dehaghi, and Majid Ghasemi; Resources: Majid Ghasemi; Data collection: Ehsan Ghasemi and Majid Ghasemi; Data analysis and interpretation: Ehsan Ghasemi and Faezeh Kouhestani Dehaghi; Final approval: Ehsan Ghasemi and Tayebbeh Roghani; Critical revision of the article for important intellectual content: All authors.

Conflict of interest

The authors declared no conflict of interest.

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