International Journal of Sport Studies for Health

Journal Homepage

Comparison of Motor Planning in the Cerebral Cortex During Gait Initiation Between Athletes with Chronic Ankle Instability and Healthy Individuals

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A r t i c l e   I n f o

Article type:
Original Research

How to cite this article:
Yousefi, M., Solgi, B., Mohammadnia Ahmadi, M., Seghatoleslami, A., & Zivari, S. (2025). Comparison of Motor Planning in the Cerebral Cortex During Gait Initiation Between Athletes with Chronic Ankle Instability and Healthy Individuals. *International Journal of Sport Studies for Health, 8*(3), 1-9.
http://dx.doi.org/10.61838/kman.intjssh.8.3.9

A B S T R A C T

Objective: This study aimed to investigate motor cortex activity during gait initiation in athletes with CAI compared to healthy individuals.

Methods and Materials: Twenty-six participants were enrolled, divided into two groups: CAI and control (CON). After informed consent, participants completed the FAAM and Cumberland questionnaires. Following a warm-up, they stood on a force plate while wearing an EEG cap. Gait initiation was triggered by an auditory cue, and EEG data were collected using EEGLAB and analyzed with MATLAB.

Findings: Analysis of motor planning parameters, including theta (4–8 Hz), alpha (8–12 Hz), beta (14–25 Hz), and gamma (35–50 Hz) frequency bands, revealed significant differences between the CAI and CON groups ($p < 0.05$). Theta activity, associated with attention and focus, showed significant differences at channels Fcz, Cz, and Cp4. Alpha band activity, linked to the activation and inhibition of sensorimotor functions, also showed significant differences at F4, Fz, Fc3, and Cp4.

Conclusion: These findings indicate that athletes with CAI exhibit altered motor planning strategies, as demonstrated by changes in theta, alpha, beta, and gamma frequency bands, particularly in the frontal and parietal regions of the brain. These changes were characterized by reduced power in all examined frequency bands in the CAI group, suggesting disruptions in cortical motor planning compared to healthy individuals.

Keywords: *Motor Planning, Chronic Ankle Instability, Gait Initiation, Electroencephalography*

1. Introduction

Lateral ankle sprains (LAS) are the most common musculoskeletal injuries, typically caused by excessive inversion and internal rotation, with or without plantarflexion (1). Epidemiological studies suggest that approximately 80% of individuals will experience an ankle sprain in their lifetime (2). Among those with a history of

Article history:
Received 25 January 2025
Revised 17 May 2025
Accepted 24 June 2025
Published online 01 July 2025

LAS, at least 73% develop chronic ankle instability (CAI), a condition characterized by recurrent sprains, pain, a sense of instability, and functional limitations. As a result, 20–40% of individuals with CAI reduce or discontinue physical activity (3). Recurrent injuries to the ankle joint complex often disrupt sensorimotor function (4-7), and despite evolving theories, there is still no consensus on the exact mechanisms underlying CAI (8).

Research indicates that individuals with CAI tend to adopt more cautious motor planning strategies. This is evident in reduced center of pressure (COP) displacement during gait initiation (GI) and altered neuromuscular activation patterns during GI tasks (9). Gait initiation marks the transition from quiet standing to steady-state walking and is a demanding functional task that requires precise postural control and feedforward motor planning (10). Anticipatory postural adjustments (APAs), which occur before voluntary movement, play a central role during GI. These adjustments are manifested as a backward and lateral shift of the COP toward the swing leg, facilitating forward propulsion of the center of mass (COM) and its shift toward the stance leg (11). Quantitative assessments of APAs and muscle activity during GI can offer valuable insights into supraspinal motor control mechanisms (12).

Previous studies have revealed altered neuromuscular control in individuals with CAI, including changes in muscle activation patterns during activities such as walking, landing, and cutting (13). It has been further suggested that those with CAI display reduced COP displacement during GI (5) and modified neuromuscular activity during planned gait termination (14). For example, Van Deun et al. reported that individuals with CAI activated lower leg muscles—such as the fibularis longus, tibialis anterior, and gastrocnemius—only after movement onset (6). Additionally, changes in corticomotor excitability in muscles like the fibularis longus and soleus point to long-term cortical adaptations in individuals with CAI (15, 16). Even in healthy individuals, motor cortex excitability has been shown to be closely related to cortical sensorimotor activity (4).

The first investigation into cortical plasticity following joint injury in humans used electroencephalography (EEG) to assess somatosensory cortex changes in individuals with anterior cruciate ligament (ACL) injuries (17). EEG records electrophysiological signals from the cerebral cortex using surface electrodes, capturing excitatory and inhibitory neural activity across cortical regions. This method provides valuable insights into central nervous system (CNS) reorganization and helps identify brain areas involved in functional deficits following sports injuries (3). EEG has also been widely used to study cortical adaptations following motor skill and balance training. By measuring the synchronized electrical activity of large populations of neurons, EEG enables time-frequency analysis to explore motor planning processes. Evidence suggests that rehabilitation interventions for CAI may promote beneficial neuroplasticity, as reflected by changes in EEG spectral power (4). Recent findings confirm that both acute and chronic ligament injuries can lead to CNS changes involving multiple cortical regions, including the somatosensory and motor cortices (3, 8). Continued investigation of cortical activity is crucial for understanding the neuromechanical consequences of ankle sprains. Such insights are vital for the secondary prevention of long-term impairments and the development of targeted therapeutic strategies (3). Therefore, the aim of this study was to assess cortical activation characteristics during gait initiation in athletes with and without CAI.

2. Methods and Materials

2.1 Study Design

Twenty-six male participants voluntarily took part in this study and were assigned to either the chronic ankle instability (CAI) group (n = 13; age: 24.31 ± 0.81 years; height: 175.12 ± 4.28 cm; weight: 71.15 ± 7.21 kg) or the control group (n = 13; age: 23.40 ± 1.70 years; height: 176.32 ± 6.41 cm; weight: 72.25 ± 6.14 kg). Inclusion criteria for the CAI group followed the standards set by Gribble et al. (2013) and required participants to meet specific cutoff scores on the Cumberland Ankle Instability Tool and the Foot and Ankle Ability Measure (FAAM) questionnaires . Additionally, CAI participants must have experienced at least one ankle sprain requiring protected weight-bearing or immobilization for a minimum of three days within 12 to 3 months prior to testing.

Control group participants were required to have no history of lateral ankle sprains. Exclusion criteria for both groups included bilateral ankle sprains, lower extremity fractures, knee injuries, history of lower extremity musculoskeletal surgery, known balance or vision impairments, and any neurological condition that could affect postural control (e.g., diabetes) or interfere with EEG data interpretation (e.g., epilepsy). All participants provided written informed consent before participation in the study. The study protocol was approved by the research ethical and approval committee of University of Birjand (IR.BIRJAND.REC.1404.008)

2.1.1 Ensuring cultural, linguistic, and geographical diversity

Given YouTube's global and multilingual reach, several steps were taken to enhance the diversity and comprehensiveness of the analysis:

- While initial searches were conducted in English, additional keywords in other languages were used to gather content from various cultural backgrounds.
- Video selection prioritized geographic diversity by examining creators' locations or profiles.
- During analysis, cultural elements such as symbols, body language, values, dietary habits, and types of physical activity were carefully observed to ensure representation across different contexts.
- Supplementary data—including subtitle languages, tagging locations, and comment languages—were also used to assess cultural and regional diversity.

These measures ensured that the final analysis offered a broad and culturally diverse perspective on Generation Z's health behaviors, reflecting YouTube's global context.

2.2 Data Collection

Electroencephalography (EEG) data were recorded using a 64-channel Quick-Cap system connected to a NuAmps digital EEG amplifier. To ensure optimal signal quality and low electrode impedance, conductive gel was applied to each electrode site using a syringe after the cap was securely positioned on the participant's scalp (Figure 1).

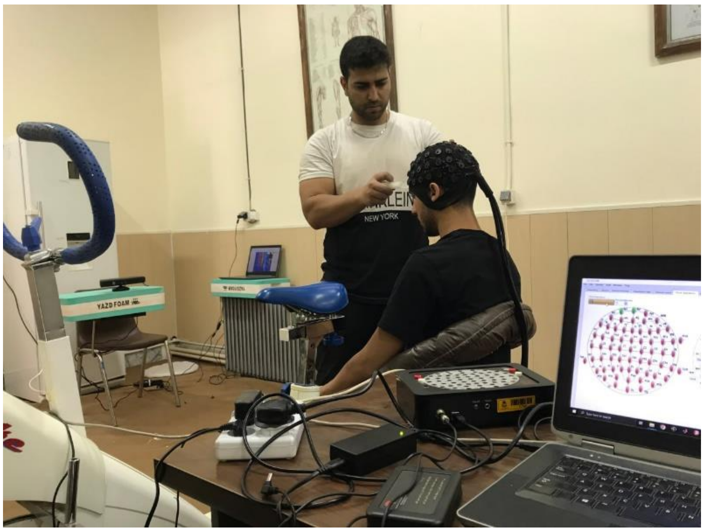

Figure 1. Application of conductive gel and participant preparation for EEG recording.

The research procedure was first explained to all participants, after which their height and weight were recorded. Participants then completed a 15-minute walking session as a warm-up. Following the warm-up, they were fitted with an EEG cap and instructed to initiate gait naturally from a standing position on a force plate upon hearing an auditory cue (Figure 2). Cortical activity was recorded during the execution of a single-step gait initiation task. This task was chosen for its safety and its capacity to activate a broad range of neuromuscular systems, making it suitable for detecting potential functional deficits (18).

Figure 2. Gait initiation task setup.

2.3 Data Processing

The step initiation phase was first identified using force plate data. The anticipatory postural adjustment (APA) phase was defined as the interval between the auditory cue and the first peak in the center of pressure (COP) signal. This definition is consistent with that proposed by Yiou et al., who described the APA phase as the period extending from the onset of the auditory stimulus to the first peak observed in the COP trajectory recorded by the force plate (Figure 3).

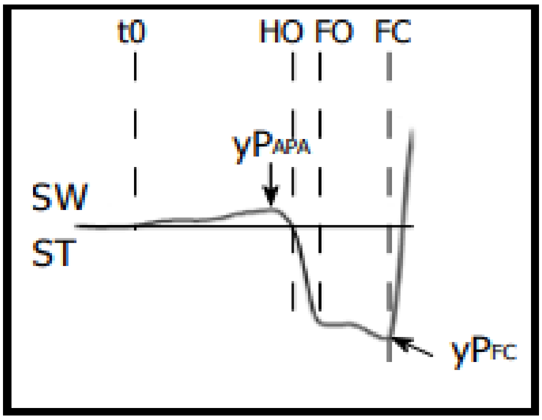

Figure 3. Example of biomechanical traces during ipsilateral isolated stepping.

Cortical activity was recorded during the postural phase of gait initiation (GI). EEG signals were preprocessed using a 50 Hz notch filter to eliminate powerline noise, followed by a zero-phase shift Butterworth band-pass filter with a slope of 4 and a frequency range of 0.1 to 50 Hz. Signal analysis was performed using EEGLAB version 7.1.4, and relevant epochs were extracted using a custom MATLAB 2019 script. The extracted signals were then subjected to Fast Fourier Transform (FFT) analysis to convert time-domain data into their corresponding frequency-domain representations. The resulting spectral output was visualized as plots with frequency on the horizontal axis and amplitude on the vertical axis. Variations in EEG signal power were interpreted as changes in the synchronized activity of large neuronal populations, reflecting underlying cortical dynamics. The EEG signals were analyzed across four frequency bands: theta (4–8 Hz), alpha (8–12 Hz), beta (14–25 Hz), and gamma (30–50 Hz). These bands were selected due to their established roles in sensorimotor integration and movement preparation (4).

2.4 Data Analysis

The normality of data distribution was assessed using the Kolmogorov–Smirnov (K–S) test. All statistical analyses were conducted using SPSS software. A Multivariate Analysis of Variance (MANOVA) was performed to examine differences between groups across the variables of interest. A significance level of $p < 0.05$ was considered statistically significant for all tests.

3. Results

To investigate cortical activity, 15 EEG channels were used: FP1, FPz, FP2, F3, Fz, F4, FC3, FCz, FC4, C3, Cz, C4, CP3, CPz, and CP4 (Figure 4). Channels FP1, FPz, and FP2 correspond to the anterior region of the frontal lobe; channels F3, Fz, and F4 correspond to the posterior region of the frontal lobe; and channels FC3, FCz, and FC4 are located at the junction between the frontal lobe and the central sulcus, which separates the frontal lobe from the parietal lobe. Finally, channels C3, Cz, and C4 are positioned along the central sulcus, while channels CP3, CPz, and CP4 correspond to the parietal lobe.

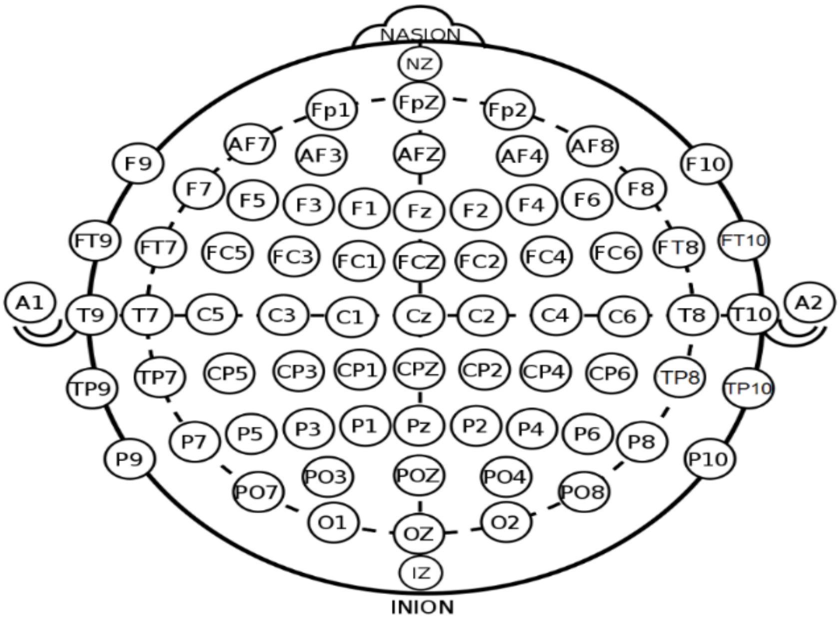

Figure 4. Location of EEG electrodes across cortical regions.

The results show significant differences between the two groups in the Fcz, Cz, and Cp4 channels for the theta frequency band (Table 1), in the F4, Fz, Fc3, and Cp4 channels for the alpha frequency band (Table 2), and in the C4 channel for both the beta (Table 3) and gamma (Table 4) frequency bands.

Table 1. MANOVA Theta Frequency Band Results

Channel	Group	Mean (SD)	P
Fz	CAI	0.13(0.05)	0.11
	Control	0.18(0.10)	
F4	CAI	0.14(0.06)	0.29
	Control	0.17(0.09)	
Fc3	CAI	0.13(0.06)	0.32
	Control	0.13(0.06)	
Fcz	CAI	0.11(0.03)	0.04*
	Control	0.16(0.07)	
Cz	CAI	0.10(0.03)	0.03*
	Control	0.15(0.07)	
C4	CAI	0.09(0.02)	0.28
	Control	0.12(0.06)	
Cp4	CAI	0.09(0.02)	0.009*
	Control	0.22(0.16)	

CAI: Chronic Ankle Instability, Fz and F4: channels of the posterior part of the frontal lobe, Fc3 and Fcz: channels which located at the junction between the frontal lobe and the central sulcus, Cz and C4: channels which located along the central sulcus, Cp4: One of the channels of the parietal lobe, *: Indicates a significant difference (P<0.05).

Table 2. Results of MANOVA for the Alpha Frequency Band

Channel	Group	Mean (SD)	P
Fz	CAI	0.07(0.03)	0.05*
	Control	0.13(0.08)	
F4	CAI	0.06(0.02)	0.04*
	Control	0.11(0.07)	
Fc3	CAI	0.05(0.02)	0.04*
	Control	0.10(0.06)	
Fcz	CAI	0.06(0.03)	0.08
	Control	0.10(0.06)	
Cz	CAI	0.05(0.03)	0.06
	Control	0.11(0.05)	
C4	CAI	0.06(0.05)	0.15
	Control	0.09(0.07)	
Cp4	CAI	0.11(0.23)	0.01*
	Control	0.32(0.18)	

CAI: Chronic Ankle Instability; Fz and F4: Channels located in the posterior region of the frontal lobe; Fc3 and Fcz: Channels positioned at the junction between the frontal lobe and the central sulcus; Cz and C4: Channels located along the central sulcus; Cp4: A channel in the parietal lobe; *: Indicates a significant difference ($p < 0.05$).

Table 3. Results of MANOVA beta frequency band

Channel	Group	Mean (SD)	P
Fz	CAI	0.04(0.02)	0.55
	Control	0.08(0.07)	
F4	CAI	0.09(0.21)	0.75
	Control	0.07(0.06)	
Fc3	CAI	0.03(0.01)	0.05
	Control	0.07(0.05)	
Fcz	CAI	0.03(0.01)	0.06
	Control	0.08(0.07)	
Cz	CAI	0.05(0.04)	0.13
	Control	0.08(0.09)	
C4	CAI	0.02(0.01)	0.03*
	Control	0.08(0.06)	
Cp4	CAI	0.21(0.28)	0.41
	Control	0.29(0.21)	

CAI refers to Chronic Ankle Instability. The Fz and F4 channels are located in the posterior region of the frontal lobe, while Fc3 and Fcz are positioned at the junction between the frontal lobe and the central sulcus. Cz and C4 are channels along the central sulcus, and Cp4 is a channel in the parietal lobe. The symbol * indicates a significant difference ($p < 0.05$).

Table 4. Results of MANOVA gamma frequency band

Channel	Group	Mean (SD)	P
Fz	CAI	0.01(0.02)	0.19
	Control	0.03(0.03)	
F4	CAI	0.02(0.04)	0.90
	Control	0.02(0.02)	
Fc3	CAI	0.01(0.03)	0.98
	Control	0.03(0.02)	
Fcz	CAI	0.01(0.09)	0.10
	Control	0.02(0.03)	
Cz	CAI	0.04(0.09)	0.66
	Control	0.02(0.01)	
C4	CAI	0.01(0.01)	0.03*
	Control	0.02(0.03)	
Cp4	CAI	0.10(0.13)	0.07
	Control	0.27(0.32)	

CAI: Chronic Ankle Instability; Fz and F4: Channels located in the posterior part of the frontal lobe; Fc3 and Fcz: Channels located at the junction between the frontal lobe and the central sulcus; Cz and C4: Channels located along the central sulcus; Cp4: A channel in the parietal lobe; *: Indicates a significant difference ($p < 0.05$).

4. Discussion and Conclusion

To investigate motor planning in the cortical regions of athletes with chronic ankle instability (CAI) and compare it with healthy individuals, electroencephalography (EEG) was employed. The frequency power across various bands (theta, alpha, beta, and gamma) was analyzed and compared during the anticipatory postural adjustment (APA) phase of gait initiation (GI). Functional instability differs fundamentally from mechanical instability, which is characterized by excessive joint movement exceeding the natural mechanical limits of the joint (19). This mechanical constraint is influenced by proprioceptive signals transmitted from the joint to the central nervous system (CNS) (20). During an ankle sprain, both ligaments and muscles are vulnerable to injury, and the joint and muscle receptors may also be compromised (21). Proprioception, a complex sensory mechanism, enables the perception of movement and body position and is often assessed through joint position sense, movement sense, and force sense evaluations (22, 23). Proprioceptive information plays a crucial role in the CNS's regulation of both basic and complex movements, as it relies on signals from various receptors for coordinated motor execution (20, 23). Damage to these receptors impairs the body's ability to generate appropriate movement responses, leading to joint instability (22).

Deficits in proprioception and primary afferent signaling, combined with pain and swelling, can significantly impact sensorimotor responses (20). Impaired sensory afferent transmission is likely to alter sensorimotor processing, and proprioceptive deficits reduce the contribution of sensory information to movement control. As a result, motor efferent pathways may undergo modifications, requiring more extensive planning and greater reliance on alternative sensory inputs. Under these conditions, the central nervous system (CNS) may adopt compensatory strategies, such as increased reliance on visual information to supplement sensory input for movement control (24). Disruptions in sensory input lead to impairments in postural control and modifications in movement control patterns, potentially contributing to adaptations in motor control (20). Research by Hess et al. demonstrated that these changes occur bilaterally, with alterations observed during gait initiation (GI) in the unaffected limb of individuals with CAI. This suggests that bilateral motor control alterations may result from modifications in supraspinal control mechanisms (5). Studies indicate that individuals with CAI experience proprioceptive deficits and disruptions in sensory afferent transmission (21, 25-27). Therefore, individuals with CAI not only face mechanical instability due to injury but also undergo maladaptive neural changes that increase their susceptibility to recurrent sprains. This indicates that ankle injuries lead to a cascade of neural adaptations and neuromuscular alterations, potentially heightening reliance on feedback mechanisms, such as vision, to control ankle joint movements.

In other words, after the initial injury, alterations in sensory feedback interact with compensatory movement patterns, ultimately modifying CNS mechanisms and movement control strategies. Disruptions in motor control programs suggest changes at both the cortical and spinal levels, leading to postural impairments (23, 28). EEG studies investigating individuals with CAI have reported increased brain activity in sensory and attentional regions (29). These changes are likely driven by neuroplastic adaptations, as well as mechanical and biological alterations in the ankle joint, accompanied by reduced proprioceptive accuracy. The increased brain activity in sensory and attentional regions may reflect decreased neural efficiency or a higher neural load required to perform similar motor tasks.

The results of this study revealed significant differences in theta band power in the premotor and motor cortex areas (Fcz and Cz) during the APA phase between the two groups. Additionally, notable differences in alpha band power were observed in central frontal lobe areas (Fz, F4, Fc3, and Cp4). Since theta power tends to increase in response to task complexity, attentional demands, and cognitive load (30), this suggests that the observed differences may reflect the additional mental effort required by individuals with CAI during motor tasks. Zhang et al. found significant differences in frontal theta power (Fz) between healthy individuals and soccer players with CAI during jumping and landing activities. This difference in theta power was linked to variations in cortical function related to attention and focus, suggesting that jumping and landing tasks are more complex and demanding for individuals with CAI (3). In the present study, no significant difference in frontal theta power was observed between the two groups, which is consistent with the findings of Giesche and colleagues. This outcome may be attributed to the fact that EEG measurements were taken before movement initiation, when participants remained in a stable position that required minimal sensory input from the limb (31). Uzlaşır et al. also found changes in theta and alpha power at the Cz channel in athletes with CAI after 6 weeks of balance training with stroboscopic glasses. However, no significant changes were observed in a control group that did not use the glasses (32). Similar results were reported by another study, where no changes in theta, alpha, or beta waves were observed at the Cz channel following four weeks of balance training with open eyes in CAI participants (4). These findings highlight the important role of vision in the sensorimotor system and suggest that athletes may need to exert greater concentration to maintain balance when visual input is restricted. In this study, significant differences in alpha band power were found in central frontal lobe regions, specifically at the Fz, F4, Fc3, and Cp4 channels. Alpha waves are associated with cortical deactivation and inhibition, which are vital for sensorimotor functions (33). Consequently, differences in alpha power may reflect variations in motor activity between the groups. Beta waves play a crucial role in analytical problem-solving, alertness, processing external information, and decision-making (34). The lack of significant changes in beta waves may be due to the absence of heightened alert conditions or atypical situations requiring external information processing during balance tasks (32). Changes in theta and alpha band power in motor, premotor, and central frontal lobe indicate altered brain activity in these regions. These findings indicate that cortical motor planning during gait initiation is significantly altered in athletes with CAI compared to healthy individuals. Therefore, this impairment is not solely a result of a local musculoskeletal condition but reflects altered neuromuscular activation patterns within the CNS. These findings enhance our understanding of CAI by providing neurophysiological evidence that motor planning and cortical control are disrupted even before movement begins. This underscores the need to address central mechanisms—alongside peripheral deficits—when designing rehabilitation strategies for individuals with CAI.

Authors' Contributions

All authors made equal contributions to the development, preparation, and revision of this manuscript.

Declaration

In order to correct and improve the academic writing of our paper, we have used the language model ChatGPT.

Transparency Statement

Data are available for research purposes upon reasonable request to the corresponding author.

Acknowledgments

The authors sincerely appreciate the invaluable support provided by the Laboratory of Gait Biomechanics. The advanced facilities and expertise offered by the laboratory significantly contributed to the progress and quality of this research.

Declaration of Interest

The authors report no conflict of interest.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Ethical Considerations

The study was conducted in accordance with the Declaration of Helsinki, which provides ethical guidelines for medical research involving human participants. All participants provided written informed consent prior to participation. The ethical approval for the study was obtained from https://ethics.research.ac.ir/EthicsProposalView.php?&code=IR.BIRJAND.REC.1404.008

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