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Plantar flexor muscle weakness may cause stiff-knee gait☆

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ABSTRACT

Aim: The iterative simulation studies proclaim that plantar flexor (PF) muscle weakness is one of the contributors of stiff knee gait (SKG), although, whether isolated PF weakness generates SKG has not been validated in able-bodied people or individuals with neuromuscular disorders. The aim of the study was to investigate the effects of isolated PF muscle weakness on knee flexion velocity and SKG in healthy individuals.
Method: Twenty able-bodied young adults (23 ± 3 years) participated in this study. Passive stretch (PS) protocol was applied until the PF muscle strength dropped 33.1% according to the hand-held dynamometric measurement. Seven additional age-matched able-bodies were compared with participants' to discriminate the influence of slow-walking. All participants underwent 3D gait analysis before and after the PS. Peak knee flexion angle, range of knee flexion between toe-off and peak knee flexion, total range of knee-flexion, and time of peak knee flexion in swing were selected to describe SKG pattern.
Results: After PS, the reduction of plantar flexor muscle strength (33.14%) caused knee flexion velocity drop at toe-off ($p = 0.008$) and developed SKG pattern by decreasing peak knee flexion ($p = 0.0001$), range of knee flexion in early swing ($p = 0.006$), and total knee flexion range ($p = 0.002$). These parameters were significantly correlated with decreased PF velocity at toe-off ($p = 0.015$, $p = 0.0001$, $p = 0.005$, respectively). The time of peak knee flexion was not significantly different between before and after stretch conditions ($p = 0.130$).
Conclusions: These findings verified that plantar flexor weakness cause SKG pattern by completing three of SKG parameters. Any treatment protocol that weakens the plantar flexor muscle might impact the SKG pattern.

1. Introduction

Spastic paretic stiff-knee gait (SKG) is among the most common gait abnormalities, which is characterized by a diminished and delayed peak knee flexion (PKF) angle during the swing phase [1–3]. Reduced knee flexion (KF) during swing is common in individuals with spastic paretic gait as a result of upper motor neuron injuries due to cerebral palsy (CP), stroke, and traumatic brain injuries [3–5]. It causes an increased energy expenditure [7], which leads to an inefficient gait pattern [8]. Several potential causes have been proposed in the literature [3,8–10] for SKG in which excessive activity of the rectus-femoris during swing or pre-swing is the major treatment target [1,6,11]. It is surgically treated by transferring of the distal rectus-femoris, during which the insertion of the muscle is relocated to a new site [1,3], intramuscular lengthening [12], neurologically treated by injecting neuromuscular blocking agents [11], in physiotherapy by stretching rectus-femoris

☆ Can plantar flexor muscle weakness, itself, generate stiff knee gait (SKG) pattern?: Yes, it completes three of the four stiff knee gait parameters except the one about the timing of peak knee flexion in swing which is surprisingly significantly earlier instead of expectedly delay for unimpaired individuals.

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and/or strengthening antagonist muscles. Although these procedures are widely used in the clinic, inconsistent outcomes have been reported [2,13,14]. Additionally, to our knowledge there is no study that examines the plantar flexor weakness-related SKG pattern for able-bodied individuals in the literature.

Goldberg et al., observed that many subjects with SKG and CP did not exhibit excessive knee extension moment during the swing phase, instead they walked with low KF velocity at toe-off [15]. Computerized simulation studies have shown that KF velocity in pre-swing is an important factor for SKG [13,16], and hip flexors and PF muscles have potential influence on KF velocity during double support [9]. Although the lack of ankle push-off power causes an insufficient KF velocity at toe-off according to these simulation studies [15,16], these findings have not been proved and validated in able-bodied participants and/or children with CP or stroke survivors.

Calf muscle tightness in gait is common as a result of increased plantar flexion among patients with spastic diplegia and hemiplegia [17,18]. Therapeutic approaches commonly focus on PF muscles shortness, to restore the muscle length by surgical (achilles tendon lengthening and gastrocnemius recession) and non-surgical (radical muscle stretching exercises, neural blockage injections, or serial casting, etc.) procedures, which are also may reduce the PF muscle strength [19,20]. Therefore, before formulating an effective treatment protocol, it is a requisite to understand the mechanisms that underlie the SKG pattern in these patients. Besides for children with CP, the influences of isolated PF muscle weakness (especially gastrocnemius and soleus) on lower extremity kinematics and its potential of causing SKG even for able-bodied individuals have not been fully investigated in the literature yet. According to simulation studies, clinicians should be aware of the possible relation between decreasing PF muscle strength and its secondary effects, such as SKG. Therefore, our aim was to detect the influences of isolated PF muscle weakness on knee kinematics in able-bodied subjects. We hypothesized that weak PF muscles have potential for reducing knee and hip flexion velocity at toe-off and contributes SKG gait pattern.

2. Method

The biomechanical effects of weakened ankle PF muscles by using a specific stretching protocol were analyzed utilizing 3D gait analyses.

A total of 20 able-bodied participants were included into the study. Their mean age was 23.4 ± 3.2 years (range, 19–30 years), height: 175.1 ± 9.3 cm and weight: 63.3 ± 13.3 kg. The participants had no prior history of neurologic and musculoskeletal disorders and had not participated in any physical sporting activities in the three days prior to undergoing our tests. The subjects in this study underwent computerized 3D gait analysis at Istanbul Medical Faculty, Gait Analysis Laboratory, from April through November 2014. Gait analysis was conducted using an optoelectronic system (ELITE2002; BTS, Milan, Italy) and two force-plates (Kistler, MS, USA). Passive markers were placed as described by Davis [21]. All participants were volunteers and they read and signed the consent form to participate in this study, in accordance with the requirements of the local ethics committee.

Participants were analyzed over two walking sessions in (1) pre-stretching period (*N*) and (2) post-stretching period (A–S). In the N condition, the gait analysis was undertaken at a self-selected speed. PF muscle strength was then assessed using a hand-held dynamometer (HHD) in a sitting position. Participants were allowed to sit on the examination table with hips in 45° flexion, their knees were fully extended and their ankles in a neutral position, as described in the literature [22]. Afterwards, to weaken the PF muscle strength by 30% of maximal voluntary contraction (MVC), a cyclic stretching protocol (P_S) was conducted to the participant's non-dominant PF while they were in a standing position on a 25-cm high step, as illustrated in Fig. 1. P_S protocol, which consists of 135-s stretch with 13 repetitions and 5-s rest periods, was strictly applied for all the participants [23]. Stretching intensity was 8/10 in severity according to visual analogue scale (VAS) and pain onset was determined as 10 points. Entire stretching procedure was supervised by the same physical therapist and the participants were verbally acknowledged the stretching intensity that was under the pain threshold in every 30 s. As soon as the stretching protocol ended, PF muscle strength was measured within 2 min as performed in the pre-stretching period. The stretching and testing protocols were completed in approximately 34 min. For some of the participants whose muscle strength was not reduced as desired (30% after the 13 repetitions P_S), 5 more repetitions were added to the stretching protocol and muscle strength test was repeated. Immediately after the stretching period and muscle strength test, gait acquisition was recorded within 40 s for A–S. None of the participants noted pain or discomfort during the walking after the stretching period.

Four gait parameters were selected to determine SKG pattern: (P1) PKF angle; (P2) range of KF between toe-off to PKF; (P3) total range of KF; and (P4) duration from toe-off to PKF in swing. A limb was considered as "stiff" if 3 or more parameters were observed significantly reduced in A–S, and as "not-stiff" if one or none of the criteria were observed. The limb was classified as a borderline case if two of the indications of SKG were seen [2].

The 20 participants were above the minimum sample size needed to ensure a power of 90% confidence level and to detect statistical significance at a two-sided significance level of 0.05 ($\beta = 0.2$) by considering the effects of size 17% in average between two conditions and by using the calculation method for the basis of descriptive studies [24].

Wilcoxon test was used to calculate significance of differences between interested gait parameters on both stretched and unstretched sides. The relationship between related parameters was also analyzed using Spearman's correlation coefficient test. In order to clearly understand the influence of gait veloicty on kinetic and kinematic prarmeters for both sides in A–S, mean walking velocity was taken as a covariate parameter in statistical analysis by ANCOVA for the Bonferroni test to eliminate its effect on other parameters. Moreover, additional seven more participants who are similar age (24.7 ± 1.4), height (173 ± 13.8), weight (172.8 ± 8.8), and walk naturally (not intentionally) as slowly (1.06 ± 0.06) and at low pace (cadence: 103.2 ± 11.6) as the participants in A–S were also analyzed and the SKG parameters were compared by student *t*-test ($p < 0.05$).

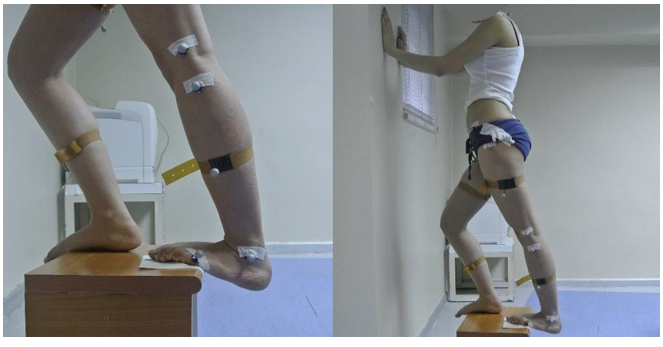

Fig. 1. Passive stretching procedure (PS). Fig. 1 Demonstration of PF muscle stretching.