

Medial calcaneal neuropathy is associated with plantar fasciitis

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Abstract

Objective: To demonstrate a method of sensory nerve conduction study (NCS) for the medial calcaneal nerve (MCN) and confirm the medial calcaneal neuropathy in patients with plantar fasciitis (PF).

Methods: Twenty-six patients with clinical and ultrasonographic diagnosis of PF participated in the present study. An antidromic method for sensory NCS of MCN was performed in each patient and in 30 controls. The conduction latency, sensory nerve conduction velocity (SNCV) and amplitude of the sensory nerve action potential (SNAP) were measured and the correlation of the SNCV of MCN with both body weight and body mass index (BMI) was studied.

Results: The mean conduction latency obtained in the MCN was greater in the PF patients than in the normal controls. Mean SNCV and SNAP amplitude of the MCN were significantly less in the PF patients than in the normal controls. Body weight and BMI were greater in PF patients than in controls. Six patients were identified as having a medial calcaneal neuropathy by using the criteria of the lowest normal values of the NCS of MCN from the normal controls.

Conclusions: Medial calcaneal neuropathy is associated with PF. The present method of sensory NCS is useful and objective in the diagnosis of the medial calcaneal neuropathy.

Significance: Medial calcaneal neuropathy was confirmed by the sensory NCS of MCN and shown to be associated with PF.

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Keywords: Medial calcaneal neuropathy; Plantar fasciitis; Heel pain; Nerve conduction; Electrophysiology

1. Introduction

Plantar fasciitis (PF) is one of the most common soft tissue disorders of the foot and the most common cause of heel pain. Previous studies have reported that heel pain accompanied with PF might occur due to heel contusion, plantar fascia rupture, heel pad atrophy (Cole et al., 2005), calcaneal epiphysitis (Gill, 1997), infection, vascular insufficiency (Dillavou and Kahn, 2003), sickle cell disease (Stuart and Nagel, 2004) and metabolic disorders (Buchbinder, 2004). However, the exact etiology of PF remains unclear. Heel pain in PF patients is frequently considered as a soft tissue disorder. Nevertheless, it can be implicated with neuropathic factors such as an entrapment of the cal-

canal nerve or a peripheral neuropathy. Heel pain caused by nerve entrapment has been previously reported (Johnston, 1994). Some researchers have mentioned that problems with neuropathy, especially involving the medial calcaneal branch of the posterior tibial nerve, might present as an important factor causing heel pain (Govindarajan et al., 2003; Rose et al., 2003), although this has not been clearly confirmed by the electrophysiological studies.

The aim of the present study is to propose a method of sensory NCS for MCN, and to demonstrate that the medial calcaneal neuropathy is associated with the PF.

2. Subjects and methods

2.1. Patients profile

Twenty-six patients consecutively admitted to the outpatient clinic in a university hospital from 2003 through

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2004 participated in the present study. All of the patients were under the clinical and ultrasonographic diagnosis of PF. They were informed and provided consent. These patients include 11 men and 15 women, ranging in age from 38 to 67 years with a mean of 53.2 years. Patients with heel trauma, ankle and foot deformity, lumbosacral radiculopathy or underlying systemic diseases such as diabetes, uremia or blood dyscrasia were excluded in the present study.

Diagnosis of PF is based on the patient's history, results of physical examination and ultrasonographic examination. Three diagnostic criteria of PF include: (1) heel pain presents in postures with weight bearing such as in standing, walking and climbing. (2) Physical examination shows tenderness around the medial calcaneal tuberosity at the plantar aponeurosis. (3) Ultrasonographic examination shows two or more of the following indications: local inflammatory change of swelling or edema, thicker aponeurosis, thickening plantar fascia (>3.5 mm), fibrous tissues or calcified tissues around the medial calcaneal tuberosity.

Clinical symptoms of the heel pain in the PF patients frequently occurs with the first few steps in the morning or after periods of inactivity. Walking barefoot, on the heels or upstairs may exacerbate the pain. The heel pain may localize at the point of the calcaneal tuberosity, or it may extend to the base or the margins of the heel. The experience of the most painful intensity was determined and measured by a visual analog scale (VAS) in each PF patient. Standardized neurological examination was performed in every patient. Sensory impairment with the findings of hypoesthesia, hypoalgesia or hyperesthesia might present over the skin of the heel. Tinel's sign is routinely tested over the MCN. Radiographic examinations for the feet as well as the ultrasonographic examinations for the heel were performed in each PF patient. In addition, body weight and height were measured and body mass index (BMI) was calculated as $\text{weight}/\text{height}^2$ (kg/m^2) in all PF patients and in 30 age-matched normal controls. In the control group, persons with BMI greater than $24 \text{ kg}/\text{m}^2$ are categorized as overweight, while BMI values between the ranges of 18 and $24 \text{ kg}/\text{m}^2$ are categorized as normal weight. In the same control group, persons are divided into two subgroups according to their estimated duration of the daily walking and standing activities. Twelve persons with daily walking and standing activities longer than 4 h are categorized as long-heel loading, whereas 14 persons with the activities less than 4 h are categorized as regular-heel loading.

2.2. Sensory NCS of the MCN

An EMG/NCV system of Synergy T2 System (Medelec, Surrey, UK) was used in the present study. The electrode used for recording was Neuroline 710 (Ambu Medicotest, Olsykke, Denmark) with a self adhesive ring and Ag/AgCl content. To exclude other neuropathy or tarsal tunnel

syndrome in the PF patients, screening nerve conduction studies were performed in the sural nerve, medial and lateral plantar nerves and the posterior tibial motor nerve. Sensory NCS of the MCN was performed by an antidromic method by placing the active recording electrode one-third of the way from the apex of the heel to the prominence of the medial malleolus. This point is the common location through which the MCN passes. The reference recording electrode was placed 3 cm distal to the active recording electrode. Stimulating electrodes were placed on the tibial nerve at the medial border of the Achilles tendon 10 cm proximal to the point of the active recording electrode. The grounding electrode was placed on the medial ankle between the stimulating and recording electrodes. Landmarks for the positions of stimulation and recording electrodes are illustrated in Fig. 1. By setting the filter band at 10–2 kHz, sweep velocity at 1–2 ms/division and sensitivity at 5–10 μV , a supramaximal stimulation with a duration of 0.1–0.2 ms at a stimulation frequency of 1 Hz was repeated more than 20 times. During the NCS, averaging technique was required and the skin temperature of the medial ankle was kept constantly above 30°C with an infrared lamp if necessary. Fig. 2 demonstrates an example of sensory nerve action potential obtained from the MCN in a PF patient.

2.3. Statistics

The study results are presented as mean values \pm SD. Statistical analysis of the comparisons between the patient groups and controls were performed with Wilcoxon rank-sum test. Logistic regression tests were used to study the relationship between the body weight, BMI, pain intensity, duration of daily heel loading and the SNCV of the MCN. Significance was set at p level of 0.05.

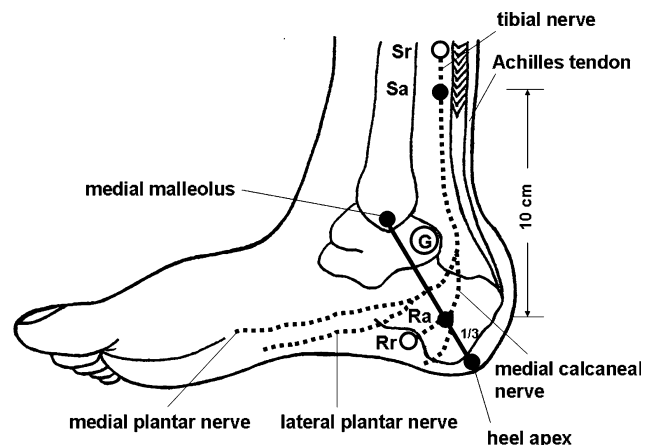


Fig. 1. The landmarks for placing electrodes for the nerve conduction study of the medial calcaneal nerve. Sa, active stimulation electrode; Sr, reference stimulation electrode; Ra, active recording electrode; Rr, reference recording electrode and G, grounding electrode.

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