

# Metatarsalgia in a patient with chronic hemiparetic stroke managed with alcohol block of the tibial nerve: A case report

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## Abstract

We report the case of a patient with a hemiparetic stroke whose metatarsalgia was successfully managed by reducing the spasticity of plantar flexor using neurolysis with 20% ethyl alcohol. A 57-year-old female patient with left hemiparesis following an infarct of the right corona radiata and basal ganglia one year previously presented with pain (numeric rating scale: 7) in the forefoot under the second to fourth metatarsal heads for six months. We diagnosed her with metatarsalgia and considered that the forefoot pain was associated with mechanical stress around the metatarsal head due to the spasticity (Modified Ashworth Scale: 1+) of the ankle plantar flexor. We performed neurolysis of the medial and lateral motor branches of the tibial nerve to the gastrocnemius muscle with 20% ethyl alcohol. After the alcohol block, foot pain and spasticity significantly reduced (numeric rating scale: 1; Modified Ashworth Scale: 0). Moreover, this effect persisted for at least three months. Clinicians should consider the possibility that spasticity can contribute to the development of foot pain in a patient with stroke.

**Keywords:** Metatarsalgia; spasticity; alcohol block, tibial nerve; 20% ethyl alcohol; stroke

## INTRODUCTION

Various types of pain occur after stroke.<sup>1,2</sup> Altered musculoskeletal position/alignment secondary to motor weakness or spasticity after stroke can cause musculoskeletal pain.<sup>3</sup> In the lower leg, spasticity usually occurs in the ankle plantar flexor muscles<sup>4</sup>, causing mechanical stress to the forefoot area. This may cause joints or soft tissues to be inflamed and painful.<sup>3</sup> Foot pain causes difficulty in standing and walking, thus limiting daily living activities. Moreover, decreased physical activity due to foot pain hinders recovery after stroke. Therefore, management of pain in patients with stroke is necessary.

Metatarsalgia is forefoot pain associated with increased stress on one or more metatarsal heads.<sup>5</sup> In many cases of metatarsalgia, excess force to the forefoot is caused by disturbed biomechanics of the lower extremity.<sup>5</sup> We think spasticity in the ankle plantar flexor muscles in patients with stroke increases metatarsal loading, which can lead to metatarsalgia in the affected foot. Therefore, we proposed that proper management of spasticity could reduce abnormal mechanical pressure in the forefoot, which would reduce foot pain.

We report the successful management of foot pain by reducing ankle spasticity through neurolysis of the tibial nerve motor branches to the gastrocnemius muscle, using 20% alcohol.

## CASE REPORT

A 57-year-old woman with a one-year history of cerebral infarct of the right corona radiata and basal ganglia visited the rehabilitation department of a university hospital due to piercing pain (numeric rating scale [NRS]: 7) in the left forefoot under the second to fourth metatarsal heads for 6 months (Figure 1). The pain was aggravated when standing or walking. She had no previous history of foot pain. In the local pain clinic, she received physical therapy modalities, such as hot pack and ultrasound, and oral medication (meloxicam and acetaminophen/tramadol hydrochloride) without a reduction in her foot pain. Physical examination revealed motor weakness of the left extremities (Medical Research Council:<sup>6</sup> 1 to 2- in the upper extremity, 4+ in the lower extremity). She had 1+ spasticity on the Modified Ashworth Scale (MAS)<sup>7</sup> in the left ankle plantar flexor muscle. However, there was no spasticity in the left toe

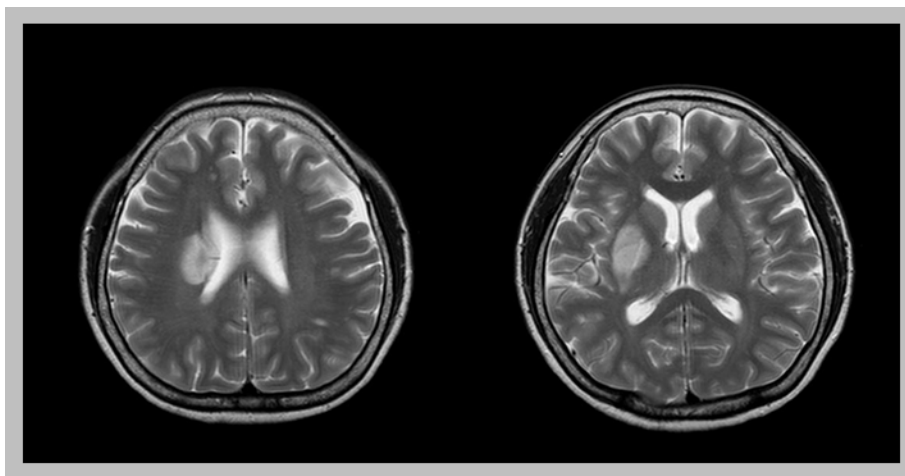


Figure 1. T-2 weighted brain images performed 10 days after onset show cerebral infarction on the right corona radiata and basal ganglia

flexor muscle. Ankle clonus was 4 to 5 beats. There was no sensory and cognitive deficits. In the left foot anteroposterior and lateral radiographs, there was no foot deformity found other than the plantar calcaneal heel spur (Figure 2). The calcaneal pitch angle was  $24.8^\circ$  (normal range:  $20\text{--}25^\circ$ ), and the lateral talocalcaneal angle was  $44.7^\circ$  (normal range:  $25\text{--}55^\circ$ ). Also, the 1<sup>st</sup>-2<sup>nd</sup> intermetatarsal and 1<sup>st</sup> metatarsophalangeal angles were measured to be  $8.1^\circ$  (cutoff  $> 9^\circ$ ) and  $10.2^\circ$  (cutoff  $> 15^\circ$ ), respectively. Tenderness on the sole of the foot under the left second to fourth metatarsal heads was checked. We diagnosed her with metatarsalgia.

We thought the ankle plantar flexor muscle

spasticity caused repetitive mechanical pressure on the forefoot, inducing metatarsalgia. Therefore, we decided to manage this spasticity by reducing the pressure on the forefoot. To manage the spasticity, we blocked the medial and lateral motor branches of the gastrocnemius muscle of the left tibial nerve with 5 mL of 20% ethyl alcohol. The block of the nerve branches was performed based on the method described by Jang *et al.*<sup>8</sup> We located the tibial nerve, using the nerve stimulator in the popliteal area. The cathode stimulating needle (Teflon-coated, 23-gauge needle) was slowly advanced toward the nerve. The needle hub was connected by injection tubing to a syringe containing 20% ethyl alcohol. Contractions were



Figure 2. The left foot anteroposterior (left) and lateral (right) radiographs show no deformity other than the plantar calcaneal heel spur.

seen and palpated as the tip approached the tibial nerve branch. At this stage, the intensity of the current was between 3 mA and 5 mA. After the needle was placed close to the targeted nerve or motor point, the needle was rotated to reach the end-point. The tip was assumed to be in contact with the nerve when maximum contraction was obtained with minimum current. The needle was finally positioned at a current of 1 mA with the nerve block. We did not apply other treatments, such as metatarsal pad, insole, or physiotherapy, for the management of metatarsalgia.

To evaluate dynamic foot pressure, the foot pressure measurement system (FPMS) (TP Scan; BioMechanics, Goyang, Korea)<sup>9</sup> was used. Dynamic foot contact pressure data during gait were recorded on the FPMS floor mat (40.5 x 40.5 cm<sup>2</sup>) at the middle of the gait test. Foot pressure was color-coded: red was the highest pressure, followed by orange, yellow, green, and blue. Foot contact pressure was evaluated twice before and one month after the nerve block. Before the procedure, contact pressure was widely distributed in the forefoot; however, reduced distribution was observed in the hindfoot (Figure 3). After the procedure, the distribution of foot contact pressure was similar to that of a normal control (57-year-old female) (Figure 3). One month after neurolysis, the left ankle plantar flexor spasticity nearly completely disappeared (MAS: 0), and ankle clonus was not checked. Additionally, the

foot pain related to metatarsalgia was reduced to NRS 1 (pre-treatment: NRS 7). At two and three months after treatment, the spasticity and pain had not recurred.

## DISCUSSION

We reported a patient with reduced foot pain related to metatarsalgia after spasticity control using neurolysis with 20% ethyl alcohol. For the treatment of metatarsalgia, identifying the etiology and analyzing the involved biomechanical factors are essential.<sup>5</sup> In this patient, we believed that ankle plantar flexor spasticity caused repeated mechanical stress and chronic irritation in the metatarsal head area, which seemed to induce metatarsalgia. Over half of patients with stroke experience spasticity.<sup>10</sup> To manage spasticity after stroke, clinicians have performed neurolysis on various nerves, such as the musculocutaneous, obturator, and tibial nerves.<sup>8,11,12</sup> We performed neurolysis on the motor branches of the tibial nerve to the gastrocnemius muscle. After treatment, her spasticity and forefoot pain were significantly reduced. Moreover, the mechanical stress abnormally concentrated on the forefoot during gait was corrected to nearly normal distribution. We demonstrated this change using FPMS. Correcting foot contact pressure distribution seemed to correct deteriorated musculoskeletal position or alignment induced by ankle spasticity,

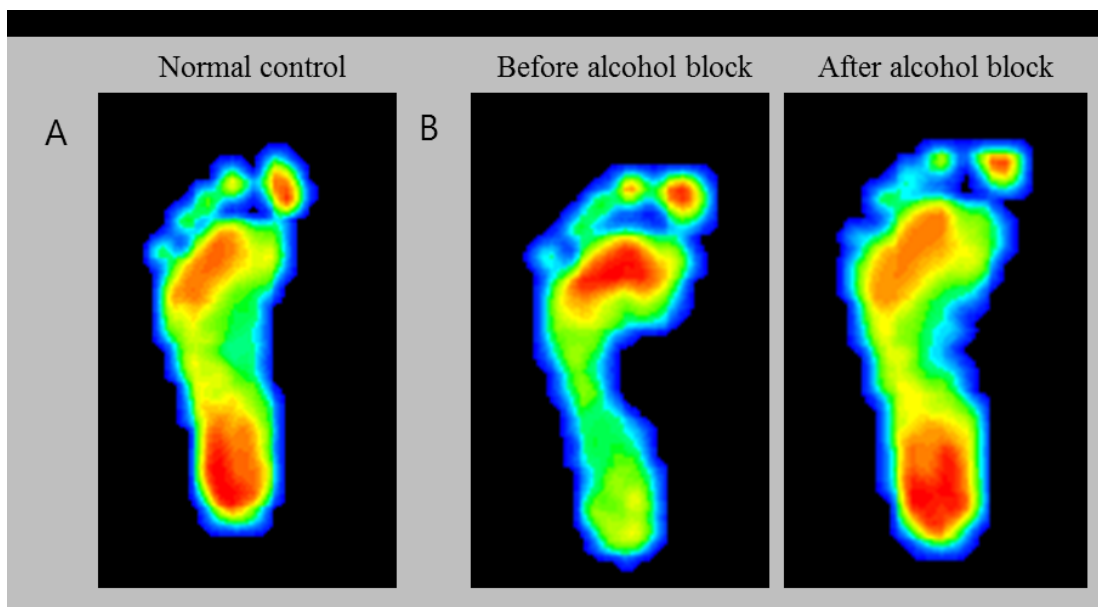


Figure 3. Foot pressure measurement image. (A) The foot pressure image of the normal control and (B) the changed foot pressure image between pre-alcohol block (left) and post-alcohol block (right)

thus reducing pain in the forefoot.

In summary, we presented a patient with stroke whose metatarsalgia was effectively controlled by neurolysis of the medial and lateral motor branches of the tibial nerve to the gastrocnemius muscle with 20% ethyl alcohol. Malposition or malalignment due to lower extremity spasticity after stroke can cause abnormal mechanical load distribution on the affected foot, and proper management of the spasticity can successfully control the foot pain. Therefore, when patients with stroke complain of foot pain, clinicians should consider that spasticity could contribute to foot pain.

## DISCLOSURE

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Conflict of interest: None

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