요족 변형 환자에서의 얇은종아리신경병증: 증례 보고

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Superficial Peroneal Neuropathy Associated with Cavus Foot: A Case Report

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Cavus foot is a deformity marked by an abnormally high medial longitudinal arch. It may be an inherited condition (accompanying Charcot-Marie-Tooth disease or congenital clubfoot), part of a central nervous system (CNS) disorder (ie, cerebral palsy, poliomyelitis), or the result of traumatic injury. There is usually an antecedent neuropathy. Hindfoot varus and forefoot valgus are ultimately produced, inciting foot pain and lateral ankle instability. Long-term complications of cavus foot are metatarsalgia, fifth metatarsal stress fracture, plantar fasciitis, and iliotibial band syndrome. Peripheral neuropathy due to cavus foot is uncommon. In fact, no instances of peripheral neuropathy stemming from long-term cavus foot have been reported to date. Described herein is the first documented case of superficial peroneal neuropathy in conjunction with cavus foot. Importantly, this neuropathy may be improved by correcting the cavus foot deformity.

Key Words: cavus foot, pain, peroneal neuropathy

Introduction

Cavus foot is a deformity marked by an abnormally high medial longitudinal arch, rendering the foot inflexible in severe cases. Inherent defects include pro-

nated forefoot, first metatarsal valgus, and hindfoot varus. Hindfoot varus and forefoot valgus produce foot pain and lateral ankle instability. Long-term complications of cavus foot are metatarsalgia, fifth metatarsal stress fracture, plantar fasciitis, and iliotibial band syndrome.¹ In the general population, the prevalence of bilateral cavus foot is 8~15%.² This condition is attributable to peripheral polyneuropathies that are inherited (Charcot-Marie-Tooth disease) or acquired, imposed by central nervous system disorders (poliomyelitis) or traumatic injuries.^{2,3} However, most cases appear to be idiopathic.²

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Some studies have indicated that inversion of a plantar-flexed foot exerts traction on the peroneal nerve at fibular head.⁴ However, an association between cavus foot and superficial peroneal neuropathy has yet to be reported. Herein, we describe a patient with superficial peroneal neuropathy related to cavus foot.

Case Report

A 45 year-old man (height, 178 cm; weight, 105 kg; body mass index, 33.14 kg/m²) presented with lengthy (7-year) history of tingling and pain involving the dorsa of both feet. He had no problems walking, but

was prone to exercise-related sprains (due to ankle inversion), occurring on multiple occasions in his twenties. He also suffered from hypertension. On physical exam, high-arched feet were evident, with hindfoot varus and forefoot valgus. Muscle strength of both lower extremities was normal, but dorsal foot surfaces showed hypesthesia. Along the lateral, upper one-third of each lower leg, “pins and needles” sensations were elicited. Knee jerk and ankle jerk reflexes were hypoactive (1+).

Plain radiography disclosed typical features of cavus foot (Fig. 1). By bone scan, focal proximal tibial uptake was noted bilaterally, consistent with enthesopathies (Fig. 2). Low-amplitude action potentials

Fig. 1. Plain radiographs (lateral views) of feet showing calcaneal pitch of (A) 35.1° on right and (B) 33.5° on left (calcaneocavus, > 30°), with Meary's angle of (C) 9.5° on right and (D) 18° on left (normal, 0~5°).

Fig. 2. Bone scan images of knees: (A) anterior and (B) posterior views of focal uptake at proximal tibias (greater on left), indicative of enthesopathies.

Table 1. Motor and Sensory Nerve Conduction Studies

Motor nerve	Stimulation	Latency (ms)	Amplitude (mV)	CV (m/s)
Right deep peroneal	Ankle	3.9	7.5	–
	Fibular head	10.7	7.2	49
Left deep peroneal	Ankle	4.1	6.0	–
	Pop fossa	10.8	5.1	47
Right tibial	Ankle	4.9	20.5	–
	Pop fossa	13.1	20.0	48
Left tibial	Ankle	4.8	21.1	–
	Pop fossa	13.0	16.8	49
Sensory nerve	Stimulation	Latency (ms)	Amplitude (uV)	CV (m/s)
Right sural	Calf	3.5	17	–
Left sural	Calf	4.0	13	–
Right superficial peroneal	Lateral leg (14 cm)	3.5	15	–
	Lateral leg (7 cm)	1.9	24	–
Left superficial peroneal	Lateral leg (14 cm)	3.4	9	–
	Lateral leg (7 cm)	1.7	8	–

CV: conduction velocity, pop fossa: popliteal fossa

Table 2. Needle Electromyography

Muscle	IA	PSW	MUAP	Interferential pattern
Both L3–S1 Paraspinal muscle	Normal	None		
Right Gluteus maximus	Normal	None	Normal	Full
Right Gluteus medius	Normal	None	Normal	Full
Right Vastus medialis	Normal	None	Normal	Full
Right Tibialis anterior	Normal	None	Normal	Full
Right Peroneus longus	Normal	None	Normal	Full
Right Peroneus brevis	Normal	None	Normal	Reduced
Right GCM (medial)	Normal	None	Normal	Full
Left Tibialis anterior	Normal	None	Normal	Full
Left Peroneus longus	Normal	None	Normal	Reduced
Left Peroneus brevis	Normal	None	Normal	Full
Left GCM (medial)	Normal	None	Normal	Full

IA: insertional activity, PSW: positive sharp wave, MUAP: motor unit action potential, GCM: gastrocnemius

of left superficial peroneal nerve were demonstrated by conduction study of sensory nerves (Table 1). During needle electromyography, there were no positive sharp waves in any of the muscles tested, but interferential patterns of right peroneus brevis and left peroneus longus muscles were reduced (Table 2). According to surface electromyography (while walking), root mean square (RMS) values of peroneus longus muscles (right, 8.93 uV; left, 5.30 uV) exceeded those of tibialis anterior muscles (right, 4.72 uV; left, 5.13 uV).

Foot pressure analysis showed increased peak pressures at first-fifth metatarsal heads and the heels, with inordinate midfoot pressure-point relief (Fig. 3). By ultrasonography, the cross sectional area (CSA) values

were: at the right fibular head, 7.5 mm²; at the left fibular head, 8.1 mm²; at the right fibular neck, 6.0 mm²; at the left fibular neck, 6.9 mm²; on right 12 cm above upper lateral malleoli, 11.1 mm²; and on left 12 cm above upper lateral malleoli, 9.6 mm². Focal swellings of both superficial peroneal nerves were observed on 12 cm above upper reaches of both lateral malleoli (Fig. 4). Based on above clinical manifestations and test results, a diagnosis of left superficial peroneal neuropathy was established. The presumptive diagnosis was chronic overexertion of peroneus muscles due to cavus foot deformities. Barton's lateral-wedge insoles were initiated thereafter and brought some improvement within 2 weeks, although intermittent

tingling of both lower legs persisted. Heel lifts (5 mm) were further instituted in follow-up.

Discussion

Peroneal neuropathy is the most common peripheral neuropathy of the lower extremities. It is linked to prolonged posturing (23.1%), surgery (20.3%), weight loss (14.5%), or trauma (11.6%) and may be idiopathic (16%).⁵ The clinical symptom is acute foot drop, with sensory loss in dorsal foot and lateral calf. Our patient experienced dorsal foot hypesthesia and neuropathic pain of lateral calf, qualifying as superficial peroneal

neuropathy.

This patient had endured ankle inversion sprains repeatedly, in his twenties. Superficial peroneal neuropathy may occur due to repetitive ankle sprains, especially ankle inversion injuries.⁶ It is suspected that peroneal neuropathy may have been caused by ankle sprain. However, he had no real history of trauma in the last ten year. Also, previous pain due to repetitive ankle sprain was improved by exercise. After improvement, the pain reoccurred 6 to 7 years ago without ankle sprain. Cavus foot can continuously involves stretching of peroneus muscles.⁶ Thus, it is assume that multiple ankle sprain was caused by cavus foot. Presumably, the cavus foot deformities prompted inversion of plantarflexed ankles, placing traction force on the fibular heads. The peroneal nerves eventually were overextended. In an earlier study, peroneal neuropathy was found to weaken the peroneus muscle, leading to hindfoot varus and forefoot valgus through subsequent imbalance between peroneus and tibialis anterior muscles.³ Unfortunately, there are no reports of this happening in reverse.

In cavus foot, the elevated medial longitudinal arch reduces surface of midfoot contact, thereby increasing pressures in areas where contact occurs. The heightened metatarsal verticality also transmits greater force to the forefoot, so there is excessive lateral and forefoot pressure on foot scan (especially at fifth metatarsal base), and pressure exerted on the great toe is significantly less than otherwise expected.⁷

As determined by surface electromyography,

Fig. 3. Excessive forefoot and hindfoot pressure and prominent lateral weight-bearing on foot scan, consistent with hindfoot rotation and forefoot cavus.

Fig. 4. Ultrasonographic assessment of superficial peroneal nerves (12 cm above upper reaches of lateral malleoli), with areas (A) 11.1 mm² on right and (B) 9.6 mm² on left constituting focal nerve swellings.

chronic ankle instability results in a lower RMS value for tibialis anterior muscle but distinctly higher RMS values for peroneus longus, medial gastrocnemius, and gluteus medius muscles in the pre-initial contact period of gait phase.⁸ These findings are aligned with our patient's test results, confirming his likely proclivity for ankle instability (while walking) due to cavus feet. Enthesopathies were showed as well. Peroneal tendinosis occur due to repetitive stretching. A weakened tendon may become chronically inflamed or develop tears, undermining lateral ankle instability.⁹ In this case, the sequence of events is unclear. It may be that constant overexertion of the peroneus muscle encouraged enthesopathy or that cavus foot deformity incited peroneal tendinosis.

By ultrasonography, CSA of superficial peroneal nerve was measured at the fibular head, on the fibular neck, and on 12 cm above upper lateral malleoli, bilaterally. In the previous study, the reference value of CSA on peroneal nerve was $7.8 \pm 2.44 \text{ mm}^2$ at the fibular head in age 40 to 59.¹⁰ Compared with the value at the fibular head, the CSA values increase about 1.2 to 1.5 times on 12 cm above upper lateral malleoli, bilaterally. Therefore, the assessment of CSA in the patient shows focal nerve swellings.

To our knowledge, this is the first report of superficial peroneal neuropathy in conjunction with cavus foot. Fundamentally, there is tensile nerve injury over time, imposed by peroneus longus muscular stress. Such outcomes may be prevented or alleviated by orthotics, which are the standard means of correcting cavus foot.² Lateral-wedge insoles and heel lifts helped to relieve the pain inflicted.

In summary, patients with cavus foot should undergo screening (ie, electrodiagnostic studies or ultrasonography) for early signs of peripheral neuropathy,

even if asymptomatic. Proactive treatment of any existing symptoms is then essential.

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