Cavus foot is a deformity marked by an abnormally high medial longitudinal arch, rendering the foot inflexible in severe cases. Inherent defects include pronated 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. 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 pronated 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. In the general population, the prevalence of bilateral cavus foot is 8~15%. 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. However, most cases appear to be idiopathic.
Some studies have indicated that inversion of a plantar-flexed foot exerts traction on the peroneal nerve at fibular head. 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

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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.
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 intereferential 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

| 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 |
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<td>Latency (ms)</td>
<td>Amplitude (uV)</td>
<td>CV (m/s)</td>
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<td>Lateral leg (7 cm)</td>
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CV: conduction velocity, pop fossa: popliteal fossa

| Table 2. Needle Electromyography |
|------------------|------------------|-----------------|----------------|----------------|
| Muscle           | IA               | PSW             | MUAP           | Interferential pattern |
| Both L3–S1Paraspinal muscle | Normal           | None            | Normal         | Full            |
| 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
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%). 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. 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. 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. 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.

As determined by surface electromyography,
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. 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 weak-
ened tendon may become chronically inflamed or
develop tears, undermining lateral ankle instability.
In this case, the sequence of events is unclear. It may
be that constant overexertion of the peroneus muscle
couraged enthesopathy or that cavus foot deformity
incited peroneal tendinosis.

By ultrasonography, CSA of superficial peroneal
nerve was measured at the fibular head, on the fibu-
lar 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 ± 2.44 mm² at the
fibular head in age 40 to 59. Compared with the val-
ue 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 super-
ficial 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 or-
thotics, which are the standard means of correcting
cavus foot. Lateral-wedge insoles and heel lifts helped
to relieve the pain inflicted.

In summary, patients with cavus foot should un-
dergo screening (ie, electrodiagnostic studies or ultra-
sonography) for early signs of peripheral neuropathy,
even if asymptomatic. Proactive treatment of any ex-
isting symptoms is then essential.

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