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CASE REPORT / OLGU SUNUMU

Peroneal and tibial nerve paralysis after prolonged knee flexion

Dizin uzun süre fleksiyonda kalması nedeniyle görülen peroneal ve tibial sinir paralizisi

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ABSTRACT

Concurrent paralysis of tibial and peroneal nerves due to acute compression is a rare incident. Here, a case of combined peroneal and tibial nerve lesion is reported for the first time ever in the literature which developed acutely due to forced, long-term flexion of the knee joint and failed to show adequate clinical or electrophysiological improvement despite eighteen months of follow-up. A 38-year-old female patient was kept in a fixed position in her bed for about 10 hours in such a way that her left knee was in full flexion. Only partial improvement could be achieved in her neurological symptoms 18 months after the incident and electrophysiological assessment showed limited regeneration. Severe axonal degeneration of both nerves was considered to occur in this patient particularly due to extreme flexion of the knee together with long-term compression. Knee flexion coexisting with long-term compressions may severely damage the peroneal nerve and also the tibial nerve, which is more resistant to compression neuropathy compared to the peroneal nerve. Knee flexion for about 10 hours of compression might have resulted in severe axonal degeneration and affected the prognosis unfavorably.

Keywords: Electromyography, Peripheral nerve injuries, Peroneal neuropathy, Tibial neuropathy

ÖZ

Tibial ve peroneal sinirlerin akut kompresyona bağlı olarak birlikte paralizisi nadir görülen bir durumdur. Bu olgu raporunda, literatürde ilk olarak diz ekleminin uzun süreli zorlu fleksiyonda kalmasına bağlı olarak akut gelişen ve 18 aylık takip süresine rağmen klinik ve elektrofizyolojik olarak yeterli düzelme gösteremeyen bir kombine peroneal ve tibial sinir lezyonu bildirildi. Otuzsekiz yaşında kadın hasta yaklaşık 10 saat süreyle sol dizi tam fleksiyonda olacak şekilde yatağında sabit pozisyonda kalmıştı. Hastada olayın üzerinden 18 ay geçmesine rağmen nörolojik tabloda sadece kısmi bir düzelme oldu, elektrofizyolojik incelemede ise sınırlı bir rejenerasyon gösterilebildi. Hastada basının uzun süreli olması ile birlikte özellikle dizin aşırı fleksiyonda kalması nedeniyle her iki sinirde ağır aksonal dejenerasyon geliştiği anlaşılmaktadır. Diz fleksiyonu ile birlikte olan uzun süreli kompresyonlar peroneal sinirle birlikte, kompresyon nöropatisine ondan daha dayanıklı olan tibial siniri de ağır biçimde hasarlayabilmektedir. Diz fleksiyonda iken yaklaşık 10 saat süreli kompresyon ağır aksonal dejenerasyona yol açabilmekte ve prognozu olumsuz etkilemektedir.

Anahtar kelimeler: Elektromyografi, Periferik sinir paralizisi, Peroneal nöropati, Tibial nöropati

Introduction

Peroneal neuropathy and paralysis at the knee (fibular head) level is seen due to mechanic, rheumatologic, vascular or traumatic causes. Isolated lesion of the tibial nerve around knee region occurs much less frequently compared to the peroneal nerve. However, concurrent damage in both nerves has been reported very rarely. Whereas, previously reported combined nerve lesions generally resulted from trauma or chronic compression, concurrent acute paralysis of both nerves due to external compression as observed in our case was reported in only one other case in the literature [1]. What is remarkable in our case is that it is not only a rare event which affected both nerves, but also it was associated with

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severe axonal destruction and limited nerve regeneration in both nerves which persisted despite a rather long follow-up period of 18 months.

Case Report

A 38-year-old female patient admitted to Physical Medicine and Rehabilitation outpatient clinic with inability of moving her left foot. In her history, 4 months ago, she was found at a semi-conscious state in her bed the next morning after ingesting 25 tablets of 25 mg amitriptyline at evening hours for suicide purpose. As reported by her relatives, when she was found she was lying in the bed with her left knee in full flexion and her lower left extremity (the part under the knee) was entrapped under her left leg.

During her physical examination, her left leg circumference was found to be decreased by 3 centimeters compared to the right leg. Left ankle dorsiflexion and plantar flexion were 0/5. Severe hypoesthesia was present in the left leg, beginning from the knee. Left achilles reflex was absent.

During the first electroneuromyography (ENMG) assessment performed at 4 months after the event, no motor and sensory responses could be obtained from left peroneal and tibial nerves and there were no sensory action potentials at the left sural nerve and no H reflex on the left side. Needle electromyography (EMG) showed fibrillation potentials and positive sharp waves in peroneal (tibialis anterior, extensor hallucis longus) and tibial innervated muscles (gastrocnemius) (Table I).

Table I. Repeated electroneuromyography (ENMG) findings of the patient and their comparison.

		1st ENMG at 4th Month	2nd ENMG at 7th Month	3rd ENMG at 12th Month	4th ENMG at 18th Month
Nerve conduction studies					
L	Peroneal CMAP (EDB)	NR	NR	NR	NR
L	Peroneal CMAP (PL)	NR	NR	Low amplitude	Low amplitude
L	Tibial CMAP (AH)	NR	NR	NR	NR
L	Tibial CMAP (Soleus)	NR	NR	Low amplitude	Low amplitude
L	Sural SAP	NR	NR	NR	NR
L	Superficial peroneal SAP	NR	NR	NR	NR
H-reflex, left soleus		NR	NR	NR	NR
Needle EMG					
L	Extensor hallucis longus	PSWs, Fibs	PSWs, Fibs	No potentials	No potentials
L	Peroneus longus	PSWs, Fibs	PSWs, Fibs	Nascent MUPs	Nascent MUPs
L	Tibialis anterior	PSWs, Fibs	No potentials	No potentials	No potentials
L	Gastrocnemius	PSWs, Fibs	PSWs, Fibs, CRDs	PSWs, Fibs, Nascent MUPs	Nascent MUPs

CMAP: compound muscle action potential; EDB: extensor digitorum brevis; NR: No response; PL: peroneus longus; AH: abductor hallucis; SAP: sensory action potential; PSWs, positive sharp waves; Fibs: fibrillation potentials; MUPs: motor unit action potentials; CRDs: complex repetitive discharges.

At the follow-up visit three months later, no improvement was observed in the strength of muscles innervated by the tibial and peroneal nerves. Sensory examination showed persistence of advanced hypoesthesia. Achilles reflex could not be obtained. At that time, pes cavus was seen (Figure 1).

In the second ENMG assessment at 7th month, no left tibial and peroneal motor, and sural sensory responses could be obtained. Soleus H-reflex was absent. Complex repetitive discharges (CRDs) were recorded from the left gastrocnemius muscle. Abnormal spontaneous activity was observed in the left gastrocnemius and extensor hallucis longus (Table I). Voluntary motor unit activity could not be recorded from these muscles. These findings demonstrated ongoing severe axonal damage with no regeneration.

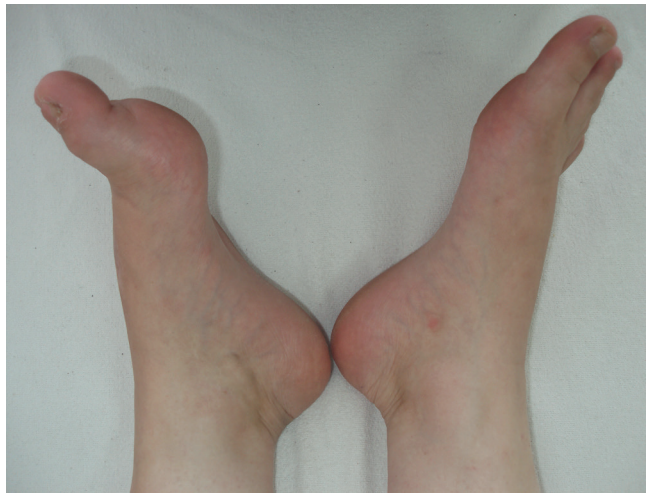

Figure 1. Image of pes cavus at the left side.

Repeated evaluation of the patient at 1 year revealed reduced hypoesthesia in her left leg for the last month. Physical examination showed no improvement in the left ankle dorsiflexion and big toe extension. However, left ankle plantar flexion muscle strength was 3/5. A reduced left Achilles reflex was obtained. In the 3rd ENMG at 1 year, although no motor responses could be obtained from peroneal or tibial nerves by recording from the extensor digitorum brevis and abductor hallucis muscles, very low amplitude motor responses were obtained from the peroneus longus and soleus muscles. Sensory responses were absent, together with the H-reflex. In the needle EMG, no voluntary motor activity could be obtained from the tibialis anterior and extensor hallucis longus muscles. Nascent motor unit potentials (MUPs) were recorded from the peroneus longus muscle, indicating reinnervation. Positive sharp waves, fibrillation potentials and CRDs were observed in the gastrocnemius muscle and also nascent MUPs were recorded during voluntary contraction (Table I). Hence, there was an evidence of persistent severe axonal damage and signs of regeneration in the peroneal and tibial nerves.

We have evaluated the patient at 18 months after the incident. There was not any substantial change in her examination findings compared to those observed during the previous visit (Figure 2). The ENMG was similar to the previous one and there were findings of only minimal reinnervation in the left peroneus longus and gastrocnemius muscles (Table I).

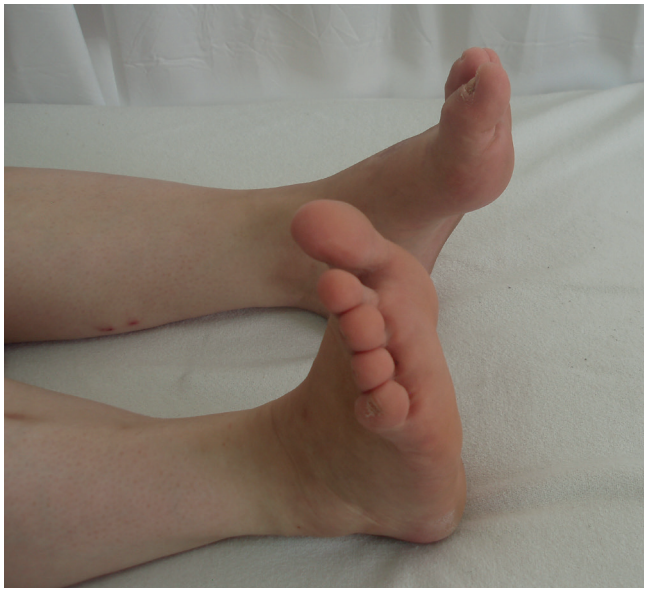

Figure 2. At the end of the 18 months physical examination showed no significant improvement in the left ankle dorsiflexion and big toe extension muscle strengths.

Discussion

Peripheral nerves may be damaged indirectly due to ischemia resulting from acute or chronic compression or directly from causes including trauma or infection. Vasa nervorum of the peroneal nerve are asymmetrical and less abundant compared to those of the tibial nerve. In addition, close localization of peroneal nerve to the fibular head makes it more vulnerable to compressive events. Tibial nerve is more resistant to peripheral neuropathic events versus peroneal nerve owing both to its more deep-seated localization and its vascularization containing symmetrical and abundant collaterals [2]. In a study on individuals trapped under earthquake rubble, no tibial nerve lesion was found among 55 peripheral nerve lesions [3].

A review of the existing literature showed only a limited number of combined peroneal and tibial nerve lesions [1,4-7]. A review of the literature revealed only one case, which was reported by Tacconi et al. who described an acute combined nerve lesion that resulted from external compression of the extremity [1].

Peripheral nerves are known to be vulnerable to compression and ischemia because of their long axons. However, the magnitude and duration of pressure that would cause damage in a nerve and the extent of resulting damage are not totally clear. This could only be investigated in animal experiments. In a study conducted on the tibial nerve in rabbits, it was shown that external compression with 20mmHg pressure impaired blood circulation in epineural venules [8]. In another study, circulation impairment in the sciatic nerve of rabbits began to occur at pressures of about 15-20mmHg and circulation was totally blocked at a mean pressure of 48.7mmHg [9]. In rats, substantial amount of endoneural edema occurs only when the sciatic nerve is exposed to 30mmHg for eight hours [10].

In humans, the relationship between the magnitude of pressure exerted on peripheral nerves and lesion could be elucidated only to some extent by performing pressure studies in carpal tunnel. It was observed that epineural blood flow was impaired with approximately 20mmHg of pressure and axonal transport was depressed and endoneural edema and nerve dysfunction occurred at 40mmHg pressure; whereas myelin sheath structure was destroyed at 50mmHg pressure [11].

In a study by Gündüz et al., peripheral nerve pathologies resulting from trapping under earthquake rubble after 1999 Marmara earthquake were explored and it was reported that, when duration of compression increased, the severity

of ENMG findings increased and regeneration period was prolonged. In that study, mean duration of compression was 7.3±4.1 (2-18) hours [3]. Data on extremities exposed to compression shows a critical period of about 8 hours [12].

The fact that no adequate clinical or electrophysiological improvement was observed in our patient after 18 months suggests that prolonged compression of the knee in full flexion for about 10 hours lead to severe and permanent axonal degeneration. Substantial improvement was observed in the case described by Tacconi et al. with duration of compression comparable to our case [1]. This suggests that both direct mechanical effects and resulting neural ischemic damage were significantly increased in our patient due to prolonged full flexion of the knee.

In conclusion, we would like to emphasize the impact of the extremity position and the duration of compression on the severity of the nerve lesion when extremities are exposed to external pressure. Prolonged compression for more than 8 hours and particularly compression of certain joints such as elbow or knee when they are in extreme flexion may worsen the prognosis by increasing axonal damage.

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