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Evaluation of Etiologic Factors and Electrophysiologic Findings in Patients with Peroneal Neuropathy

Peroneal Nöropatisi Olan Hastalarda Etiyolojik Faktörlerin ve Elektrofizyolojik Bulguların Değerlendirilmesi

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Abstract

Objective: Peroneal neuropathy is the most common entrapment mononeuropathy in the lower extremity. The common site of injury is around the fibular head where the nerve becomes superficial. Compressive pathologies are the most frequently seen etiologies. The treatment plan is designed according to the etiology. Electrophysiologic investigations are accepted as the gold standard for the diagnosis of peroneal neuropathy. In this study, we aimed to evaluate the etiologic factors and electrodiagnostic findings in peroneal neuropathy.

Method: We retrospectively analyzed the etiological and electrodiagnostic test findings of patients with clinical features compatible with peroneal neuropathy, who presented to the Electromyography Laboratory of İstanbul Medipol University Hospital between January 2016 and December 2019. Patients with polyneuropathy or a disease that may cause polyneuropathy such as diabetes mellitus, those with lumbosacral radiculopathy or plexopathy, and those with neurodegenerative diseases were excluded.

Results: A total of 30 patients with clinical features compatible with peroneal neuropathy (19 males, 11 females, median age 30 years; range 21 to 66) were enrolled in the study. Four (13.3%) patients had a comorbid disease. The median duration (minimum-maximum) of the symptoms was 20.5 (2-140) weeks. The affected side of the peroneal nerve was 43.3% right, 43.3% left, and 13.3% bilateral. The common cause of peroneal nerve injuries was due to compression (40%). Potential causes of compression in five out of 12 cases were iatrogenic. Weight loss was found in 10% of patients and one patient (3.3%) had a history of a recurrent ganglion cyst. Approximately 23% of lesions were due to traction injury and 23%

Öz

Amaç: Peroneal nöropati, alt ekstremitede en sık görülen tuzak mononöropatidir. Yaygın yaralanma bölgesi, sinirin yüzeysel olduğu fibula başındadır. En sık görülen etiyolojiler kompresif patolojilerdir. Tedavi planı etiyolojiye göre tasarlanır. Elektrofizyolojik incelemeler peroneal nöropati tanısında altın standart olarak kabul edilmektedir. Bu çalışmada peroneal nöropatide etiyolojik faktörleri ve elektrodiagnostik bulguları değerlendirmeyi amaçladık.

Yöntem: İstanbul Medipol Üniversite Hastanesi Elektromiyografi Laboratuvarı'na Ocak 2016-Aralık 2019 tarihleri arasında başvuran peroneal nöropati ile uyumlu klinik özellikleri olan hastaların etiyolojik ve elektrodiagnostik test bulgularını retrospektif olarak inceledik. Polinöropatili veya diabetes mellitus gibi polinöropatiye neden olabilecek bir hastalığı olan hastalar, lumbosakral radikülopati veya pleksopatisi olanlar ve nörodejeneratif hastalığı olanlar çalışma dışı bırakıldı.

Bulgular: Çalışmaya peroneal nöropati ile uyumlu klinik özelliklere sahip 30 hasta (19 erkek, 11 kadın, medyan yaş 30 yıl, 21-66 aralıkta) alındı. Dört (%13,3) hastada ek hastalık vardı. Medyan (minimum-maksimum) semptom süresi 20,5 (2-140) haftaydı. Peroneal sinirin etkilenen tarafı %43,3 sağ, %43,3 sol ve %13,3 bilateral idi. Peroneal sinir yaralanmalarının en sık nedeni kompresyona bağlıydı (%40). On iki olgudan beşinin olası kompresyon nedenleri iyatrojenikti. Hastaların %10'unda kilo kaybı saptandı ve bir hastada (%3,3) tekrarlayan ganglion kisti öyküsü vardı. Lezyonların yaklaşık %23'ü traksiyon yaralanmasına bağlıydı ve olguların %23'ü idiyopatikti. Elektrofizyolojik incelemelere göre, 16 olgu ağırlıklı olarak aksonal yaralanma olmaksızın demiyelinizan idi. Kalan 14 olguda (%46,6) aksonal yaralanma saptandı ve aksonal yaralanmalı olguların

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Abstract

of cases were idiopathic. According to electrophysiologic investigations, 16 cases were predominantly demyelinating without axonal injury. The axonal injury was detected in the remaining 14 cases (%46.6) and half of the cases with axonal injury were accompanied by demyelinating injury. Six cases had mild, 3 cases had severe, and 5 cases had a total axonal injury.

Conclusion: Compression is the most commonly seen etiological factor in peroneal neuropathy. Electrophysiologic investigations play a significant role in the differential diagnosis, prognosis, management plan, and follow-up of recovery. Further detailed studies are needed to clarify the relationship between electrophysiologic findings and prognosis to form an algorithm for the treatment and follow-up.

Keywords: Electrodiagnosis, entrapment neuropathy, peroneal nerve

Öz

yarısına demiyelinizan yaralanma eşlik etti. Altı olguda hafif, 3 olguda ağır ve 5 olguda total aksonal yaralanma vardı.

Sonuç: Kompresyon, peroneal nöropatide en sık görülen etiyolojik faktördür. Elektrofizyolojik araştırmalar ayırıcı tanıda, prognoz, tedavi planı ve iyileşmenin takibinde anahtar rol oynar. Tedavi ve takip için bir algoritma oluşturmak için elektrofizyolojik bulgular ile prognoz arasındaki ilişkiyi netleştiren daha detaylı çalışmalara ihtiyaç vardır.

Anahtar kelimeler: Elektrodiagnoz, peroneal sinir, tuzak nöropati

Introduction

The peroneal nerve is derived from the L4, L5, and S1 nerve roots, which travel through the lumbosacral plexus and eventually join the sciatic nerve. Within the sciatic nerve, the fibers are destined to become the peroneal nerve run separately from the fibers forming the tibial nerve. Before separation from the sciatic nerve at proximal to popliteal fossa, peroneal nerve fibers innervate the short head of biceps femoris and distally form the common peroneal nerve, also known as common fibular nerve. The common peroneal nerve supplies the sensation of the upper lateral leg. It passes around the fibular head where it becomes superficial and bifurcates into the superficial and deep peroneal nerves. This naming is according to the relationship between branches and bone. The superficial and deep peroneal nerves innervate the dorsiflexors of the ankle, toes, and the evertors of the ankle, respectively (1,2).

Peroneal neuropathy is the most common entrapment neuropathy in the lower limb. It accounts for 15% of all peripheral entrapment neuropathies and takes third place after median and ulnar neuropathies. The common site of injury is around the fibular head, where the nerve is superficial and eligible for compression and trauma. However, more distal lesions at the level of the calf, ankle, and foot affecting the deep and superficial peroneal nerve as well as proximal lesions occur less frequently. Patients often present with foot drop and sensory disturbance over the dorsum of the foot and lateral calf (1,2).

Many etiological factors may contribute to peroneal neuropathy such as external compression (casts, immobilization), direct trauma (fracture), traction injuries (forcible ankle inversion), mass lesions (ganglion, tumor), entrapment (fibular tunnel), vascular conditions, diabetes mellitus, weight loss, leprosy and idiopathy. However, the most encountered mechanism is compression and it can develop by prolonged squatting, habitual leg crossing, immobilization, or even ankle-foot orthoses (1-4).

For the diagnosis of neuropathy, a detailed history of symptoms, distribution, duration and course of the neuropathy should be obtained. The presentation of sciatic neuropathy, lumbosacral plexopathy, or L5 radiculopathy may be similar to peroneal neuropathy. In addition to a good clinical examination and laboratory investigations, electrodiagnostic tests (EDT) play a key role in the differential diagnosis. EDT identifies the site of the lesion, reveals the underlying pathology, establishes the prognosis by determining the severity of the injury, and helps to monitor recovery (1,3). In this study, we aimed to evaluate the etiologic factors and electrodiagnostic findings in peroneal neuropathy.

Materials and Methods

Subjects

A total of 5.570 electrodiagnostic studies were screened retrospectively. The number of cases diagnosed with the entrapment neuropathy was 1.252. A total of 30 patients with clinical features compatible with peroneal neuropathy (19 males, 11 females, median age 30 years, range 21 to 66), who presented to the electromyography (EMG) Laboratory of İstanbul Medipol University Hospital between January 2016 and December 2019, were retrospectively analyzed.

Patients meeting the following criteria were included in the study: (1) weakness present in at least one of the muscles innervated by the peroneal nerve (ankle dorsiflexion, toe extension, foot eversion) with or without sensory loss

involving anterolateral leg and dorsum of foot, (2) needle EMG findings consistent with peroneal neuropathy. The exclusion criteria were as follows: (1) polyneuropathy or a disease that might cause polyneuropathy such as diabetes mellitus, (2) neurological examination findings like weakness of muscles not innervated by the peroneal nerve (hip abduction, knee flexion, foot inversion, and dorsiflexion), positive straight leg raise test, fasciculation, hyperreflexia, spasticity, (3) EDT findings suggestive of the sciatic mononeuropathy, lumbosacral radiculopathy or plexopathy, motor neuron disease (4) a history of lumbosacral radiculopathy or plexopathy, and (5) neurodegenerative diseases.

Imaging modalities including magnetic resonance imaging and conventional radiograph were conducted in cases with suspected lumbar pathologies or anatomical anomalies.

Demographic data such as age, gender, comorbid diseases (coronary artery disease, malignity, etc) and clinical characteristics including the side of the peroneal nerve injury, duration of symptoms, and etiology of injury were noted. The study protocol was approved by the İstanbul Medipol University Ethics Committee (10840098-772.02-E.34268).

Electrodiagnostic Tests

All patients had a detailed neurologic examination. Standard motor and sensory nerve conduction studies and needle EMG were performed by the same author (F.A.) using a Dantec, Keypoint G3 EMG & Evoked Potential Response Unit Equipment. Needle EMG was performed for the leveling of the injury. The peroneal motor nerve conduction study was carried out recording the extensor digitorum brevis muscle, stimulating the ankle, below the fibular head, and lateral popliteal fossa. Also, recording the tibialis anterior muscle (stimulating below the fibular head and lateral popliteal fossa) was performed when there was no conduction block at the fibular neck. The superficial peroneal sensory nerve conduction study was performed recording the lateral ankle and stimulating the lateral calf at the level of 10-12 cm proximal to the ankle. Motor unit action potentials (MUAPs) and the presence of active denervation were analyzed using concentric needle electrodes. When recruitable MUAPs on needle examination in the affected muscles were absent, complete nerve injury was considered. When preserved voluntary control of MUAPs in the affected muscle was detected, incomplete nerve injury was considered.

Statistical Analyses

Data were analyzed using the IBM SPSS for Windows version 23.0 software (IBM Corp. Armonk, NY, USA). The Shapiro-Wilk test was used to evaluate the normality of data. Descriptive analyses were presented as median, minimum, and maximum for continuous variables. Categorical data were summarized as counts and percentages.

Results

Our clinical rate for peroneal entrapment was 7.5 cases per year and the percentage of peroneal entrapment among entrapment neuropathies was 2.3%. Thirty patients with peroneal nerve injury were included in the study. 63.3% of the patients were male and the median (minimum-maximum) age of the patients was 30 (21-66) years. Four (13.3%) patients had a comorbid disease. The median (minimum-maximum) symptom duration was 20.5 (2-140) weeks. The side of the peroneal nerve injury was 43.3% right, 43.3% left, and 13.3% bilateral. The demographic and clinical characteristics of the patients were summarized in Table 1.

Etiological factors of peroneal nerve injury were summarized in Figure 1. The common causes of peroneal nerve injuries were due to compression (40%). Underlying predisposing factors were lying on with pressure on the fibular head (4 cases, 13.3%), pressure wrapping around the knee including (2 cases, 6.7%), prolonged squatting or excessive exercise (5 cases, 16.7%), and habitual leg crossing (1 case, 3.3%). Five out of 12 cases with compressive etiologies were iatrogenic. Casting (2 cases),

Table 1. Demographic and clinical characteristics of patients with peroneal nerve injury

	n	%	Median (min-max)
Age (year)	-	-	30 (21-66)
Gender	-	-	
Male	19	63.3	
Female	11	36.7	
Symptom duration (week)	-	-	20.5 (2-140)
Comorbidities			
Coronary artery disease	1	3.3	-
Malignancy (lymphoma, lung cancer, esophagus cancer)	3	9.9	-
Side of the peroneal nerve injury			
Left	13	43.3	-
Right	13	43.3	-
Bilateral	4	13.3	-

n: Number of individuals, min: Minimum, max: Maximum

prolonged bed rest in the intensive care unit (2 cases), and protracted positioning during surgery (1 case) were the iatrogenic reasons that we obtained. Weight loss after bariatric surgery was found in 10% of patients (3 cases) and one patient (3.3%) had a history of a recurrent ganglion cyst. Approximately 23% of lesions (7 cases) were due to the traction of the nerve. 23% of patients (7 cases) did not have any etiological factor and remained idiopathic.

Results of nerve conduction studies and EMG are shown in Table 2. According to EDT, 16 cases were predominantly demyelinating without axonal injury. The axonal injury was detected in the remaining 14 cases (%46.6) and half of the cases with axonal injury were accompanied by demyelinating injury. Six cases were mild, 3 cases were severe, and 5 cases were total axonal injury. Prolonged bed rest in the intensive care unit, sustained compression, and casting were found in the history of patients with severe axonal injury. It was found that surgical treatment was applied to 5 cases with total axonal injury, 1 case with a ganglion cyst, and 1 case without axonal injury. The rest 23 cases were managed by watchful waiting and conservative treatment composed of lifestyle modifications, pain management, exercise, and splints if necessary. Unfortunately, outcomes of treatments were unavailable because of the recall bias and the lack of objective assessment.

Table 2. Results of nerve conduction studies and electromyography

Measured parameters		n	%
CMAP	Absent	7	23.3
	Reduced	23	7.7
SNAP	Absent	5	16.7
	Reduced	11	36.7
	Normal	14	46.7
Denervation potentials	Absent	18	60
	Present	12	40
MUAPs	Absent	5	16.7
	Neurogenic	9	30

n: Number of nerves, CMAP: Compound muscle action potential, SNAP: Sensory nerve action potential, MUAPs: Motor unit action potentials

Discussion

Entrapment neuropathies of the lower limb may arise from compression, trauma, or iatrogenic injury. Factors like the site of the injury, the severity of the lesion, etiology are important for the prognosis and deciding the treatment plan (1). With this regard, we aimed to identify the etiological factors causing peroneal neuropathy and electrophysiological findings in our study. There is no publication identifying the frequency of peroneal neuropathy, but the most common entrapment neuropathy of the lower extremity is peroneal neuropathy at the level of the fibular head because of its superficial course around the fibula and susceptibility to stretch injury than tibial nerve (1,3-5). Especially, ankle sprains with inversion and flexion cause traction injury with the mechanism of tearing of the vasa nervorum (6). The percentage of peroneal neuropathy due to traction was found 23.3% in our study. Traumatic injuries like fibular fractures and knee dislocation are also risk factors for peroneal neuropathy (7-9). Noble et al. (10) found the rate of peroneal neuropathy as 1-2% in patients with tibia or fibula fractures.

The most common and probable cause of peroneal neuropathy is external compression at the fibular head which may originate from habitual leg crossing, prolonged squatting, inappropriate positioning in bedridden patients, occupational factors, casting, and braces (5). The percentage of compressive etiologies in our study was 40% and this is compatible with the literature.

Weight loss is another risk factor for peroneal neuropathy. Some case reports implicate the relationship between

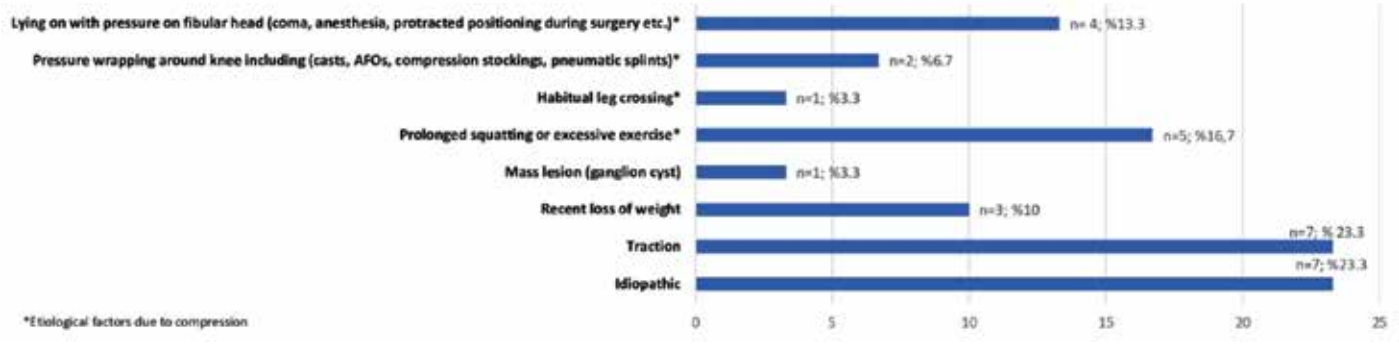

Figure 1. Etiological factors of peroneal nerve injury
n: Number of cases

weight loss and peroneal neuropathy (11-13). In one case series, the frequency of weight loss was reported as 20% in peroneal neuropathy (14). In our study, the frequency of weight loss was 10% and all patients who lost weight from 5 to 14 kg had a history of bariatric surgery. Only one patient had bilateral entrapment of the peroneal nerve.

Internal compression also presents with the same symptoms and is caused by mass lesions like neuroma, Baker cyst, tumors, and ganglion cysts (5). In one study, the percentage of mass lesions in peroneal neuropathy is found as 13%, and the mostly detected mass lesion is intraneural ganglion cyst (15). Additionally, the common peroneal nerve is the most common site of an intraneural ganglion cyst in the peripheral nervous system (16). Ganglion cysts are space-occupying lesions and cause internal compression resulting in progressive peroneal neuropathy (17,18). We had one case of a ganglion cyst (3.3%).

Possible mechanisms for iatrogenic peripheral nerve injuries are direct nerve injury, mechanical factors (compression, stretch, ischemia), the toxicity of injected drugs, and double crush syndrome (19). Antoniadis et al. (20) reported that common peroneal nerve affected 13% among 340 iatrogenic nerve injuries treated surgically. According to our results, the iatrogenic peroneal nerve injury ratio was 16.6% and compression was the main mechanism detected. Systemic diseases like diabetes mellitus and inflammatory diseases are other etiological factors for peroneal neuropathy (1).

Peroneal neuropathy is classified as idiopathic when none of these predisposing factors are present. Aprile et al. (21) reported the rate of idiopathic peroneal neuropathy as 16% among 69 cases. According to our results, 23.3% of our cases were idiopathic.

Electrophysiologic investigations are accepted as the gold standard for the diagnosis of peroneal neuropathy (1,2). It is also important for the differential diagnosis. Symptoms of peroneal neuropathy may be drop foot or sensation deficits. And presentations of L5 radiculopathy, sciatic mononeuropathy, lumbosacral plexopathy, and polyneuropathy may be similar. Other diseases that should be considered in the differential diagnosis are cerebral and spinal cord lesions, motor neuron disease, and anterior compartment syndrome of the leg (2,22). Electrophysiologic investigations help to distinguish these various diseases by localizing the lesion precisely and determinate the severity of the condition. Thus, electrodiagnostic studies are “sine qua non” for peroneal neuropathy (1). Karakis et al. (23) showed that predominantly axonal pathologies were seen in the peroneal neuropathy in childhood and adolescence (72%) but this should be attributed to the percentage of traumatic etiology (56%) in their study. According to our results, pathophysiology was predominantly demyelinating in 53.4%, axonal in 23.3%, and mixed in 23.3%. The total axonal injury was seen at 16.6%. Two out of 5 cases with total axonal injury were associated with compression, but the duration of symptoms was longer than 6 months for both. The remaining three cases were iatrogenic due to prolonged bed rest in the intensive care unit and casting. It should be concluded from here that the duration of symptoms and the mechanism of injury are important risk factors for the development of total axonal injury.

Neuropathies of the lower limb cause morbidity and disrupt functionality. Peroneal neuropathy has a favorable prognosis generally. Poor prognostic predictors are the findings of denervation in EMG, age, and severe weakness at onset (24,25). Needle EMG helps prognostication by indicating the presence of denervation. In our study, denervation potentials in the needle EMG were found to be 40%.

Derr et al. (26) showed that peroneal neuropathy with non-traumatic compression origin had a good prognosis. In the case of external compression, the recommended treatment methods are watchful waiting, lifestyle modifications including the relief of the pressure, doing exercise, and usage of ankle-foot orthoses to prevent possible deformity. If there is peroneal neuropathy induced by internal compression, then surgical treatment should be chosen. Patients with progressive symptoms or who do not improve with conservative treatment should be referred for surgical treatment (24,27).

There are some limitations of this study that should be mentioned. The study was conducted retrospectively in a single center with a small population. EDT were done at varying times after the peroneal nerve injury and patients have not been followed up for their recovery process.

Conclusion

Peroneal neuropathy is the most common entrapment mononeuropathy of the lower extremity, and the nerve is affected especially around the fibular head. Compressive pathologies are the most frequently seen etiologies. Electrophysiologic investigations play a key role in the differential diagnosis, prognostication, management plan, and follow-up of recovery. Further detailed

studies are needed to clarify the relationship between electrophysiologic findings and prognosis to form an algorithm for the treatment and follow-up.

Ethics

Ethics Committee Approval: The study was approved by Ethics Committee of İstanbul Medipol University with decision no: 10840098-772.02-E.34268.

Informed Consent: Retrospective study.

Peer-review: Externally and internally peer-reviewed.

Authorship Contributions

Concept: S.T., F.A., N.H., Design: S.T., A.R., F.A., Data Collection or Processing: S.T., N.H., F.A., Analysis or Interpretation: A.R., N.H., Literature Search: A.R., F.A., Writing: S.T., N.H.

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