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Case Report

Acute bilateral foot drop in a chronic alcoholic patient

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ABSTRACT

Peroneal palsy is compression neuropathy of the peroneal nerve, which presents with foot drop. Patients with unilateral peroneal nerve palsy are frequently encountered in clinical practice. Although bilateral peroneal nerve palsy is rare, bilateral foot drop due to peroneal nerve palsy is much less common. The main complaint is often walking difficulty due to weakened ankle dorsiflexor muscles. Medical history and physical examination are always a part of the diagnosis, and the most useful method is electroneuromyography to evaluate the degree of the lesion. In this report, we present a 52-year-old male unconscious patient with chronic alcoholism admitted with acute bilateral foot drop and discuss clinical assessment, diagnosis, and treatment planning of this rare case of peroneal palsy after lying in the prone position in the light of literature data.

Keywords: Alcoholic neuropathy; electroneuromyography; peroneal nerve compression neuropathy.

Foot drop is a clue symptom with a wide range of diagnosis including cerebral, spinal, and peripheral causes. History taking and careful clinical examination are often useful in the neurotopographic classification. Peroneal palsy is a frequent cause of foot drop, and mostly due to the pressure on the fibular neck just below the knee.[1] Patients with unilateral peroneal nerve palsy are frequently encountered in clinical practice. However, peroneal nerve palsy is rarely bilateral.[2] Herein, we report a case of bilateral common peroneal nerve palsy who was left in the prolonged lying position in a drunk state.

CASE REPORT

A 52-year-old man with no relevant past history was admitted to our outpatient clinic with symptoms of bilateral leg weakness and walking difficulty. He had a history of chronic alcoholism and typically drank a glass of alcoholic drink per day for three decades. The patient who lived alone was found lying unconscious at home 20 days ago and he was urgently transferred to the emergency department by his neighbors. The patient who was estimated to have been lying unconscious for about two days was treated and followed for 10 days in the intensive care unit due to chronic liver disease, acute renal failure, and lobar pneumonia. After his treatment completed, he was discharged home; however, he complained about difficulty in ambulation and decreased strength of bilateral lower extremities with bilateral foot drop. The patient was, then, admitted to our clinic. Physical examination revealed that he had difficulty in walking and moving his ankles. On neurological examination, paresis of the extension and eversion of both feet and all toes were found, whereas inversion and plantar flexion were normal. Sensation was also diminished in the anterolateral side of the leg and the dorsal surface between the first and second toe bilaterally. The Achilles reflexes were absent in both lower extremities, and no pathological reflexes were noted (i.e., Babinski, ankle clonus). He had a steppage gait. The remaining physical examination findings were normal. A comprehensive laboratory testing including white blood cells and platelet counts, sedimentation rate, C-reactive protein, liver and kidney function tests, serum ionogram, muscular enzymes, folic acid and vitamin B12, thyroid

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functions, immunoglobulins, and autoantibodies were normal (Table 1). However, the albumin level (2.5 g/dL, reference range: 3.5-4.5 g/dL), 25 OH-vitD3 level (12 ng/mL, reference range: >30 ng/dL=normal, <20 ng/dL=deficient, 20-30 ng/dL insufficient), and prealbumin level (14.6 mg/dL, reference range: 20-40 mg/dL) were low. The patient was treated with conventional pharmacological treatment, including human albumin and vitamin supplementation and formal nutritional support. Alcoholic polyneuropathy or bilateral common peroneal nerve palsy was suspected as the preliminary diagnosis and electroneuromyography (ENMG) was performed which revealed normal conduction velocities of the bilateral sural, median, and ulnar nerves. The nerve conduction study (NCS) showed an absence of motor nerve action potentials in bilateral common peroneal nerves (Table 2). The findings of needle ENMG are shown in Table 3. The electrodiagnostic study demonstrated total axonal damage of bilateral common peroneal nerve. No electrophysiological finding was found to be compatible with alcoholic polyneuropathy. Magnetic resonance imaging (MRI) of the knee showed hyperintensities on T_2-weighted images such as bilateral soft tissue distances around the fibular heads, primarily signal changes thought to be compatible with edema (Figure 1a, b, 2a, b). Pressure-induced common peroneal nerve palsy was considered, and conservative treatment was planned initially. In the differential diagnosis, central or spinal lesions, radicular or sciatic lesions, plexopathy, polyneuropathy, and motor neuron disease were ruled out by clinical and electrophysiological tests. Although hereditary neuropathy was unable to be completely eliminated, genetic testing was unable to be performed. However, the medical history of the patient for previous pressure palsies was negative. The treatment protocol generated superficial heat for 20 min, followed by neuromuscular electrical stimulation and exercise program. There was no sign of improvement, and nerve conduction abnormality persisted after eight weeks. At four months of follow-up, foot drop improved by 30 to 40%.

Table 1. Laboratory test results

Parameters	Results	Reference values
Hemoglobin (g/dL)	12.1	13.2-17.3
Platelet (10^3/μL)	327	150-450
White blood cell (10^3/μL)	8.2	3.57-11.01
Erythrocyte sedimentation rate (mm/h)	22	0-20
C-reactive protein (mg/L)	1.2	0-9
Creatinine (mg/dL)	0.56	0.84-1.25
Blood urea nitrogen (mg/dL)	16	17-43
Sodium (mEq/L)	138	136-146
Potassium (mEq/L)	4.18	3.5-5.5
Chloride (mEq/L)	103	101-109
Calcium (mg/dL)	9.04	8.8-10.6
Phosphorus (mg/dL)	4	2.5-4.5
Aspartate transaminase (U/L)	18	3-50
Alanine transaminase (U/L)	12	3-50
Alkaline phosphatase (U/L)	125	40-150
Creatine phosphokinase (U/L)	59	10-171
Total protein (g/dL)	5.91	6.4-8.3
Albumin (g/dL)	2.5	3.5-5.0
Prealbumin (mg/dL)	14.6	20-40
25(OH)Vit-D3 (ng/mL)	12	>30
Parathormone (pg/mL)	17.9	12-88
Vitamin-B12 (pg/mL)	224	126-505
Thyroid-stimulating hormone (uIU/mL)	1.4	0.27-4.2
Folic acid (ng/mL)	3.42	2.5-24

Table 2. Nerve conduction study

Nerve stimulation	NCV (m/s)	DML (ms)	AMP (2-4uV)
Motor			
Right peroneal nerve (EDB)			
1. Ankle	No response		
2. Fibulae head	No response		
3. Knee	No response		
Right peroneal nerve (EDB) 4th month			
1. Ankle		8.35	0.3
2. Fibulae head	41.8	14.20	0.2
3. Knee	42.1	16.35	0.2
Left peroneal nerve (EDB)			
1. Ankle	No response		
2. Fibulae head	No response		
3. Knee	No response		
Left peroneal nerve (EDB) 4th month			
1. Ankle		6.95	0.5
2. Fibulae head	28.3	17.60	0.4
3. Knee	30.0	21.60	0.3
Right peroneal nerve (tibialis anterior)			
1. Fibulae head	No response		
2. Knee	No response		
Left peroneal nerve (tibialis anterior)			
1. Fibulae head	No response		
2. Knee	No response		
Sensory			
Sural nerve			
Right sural nerve	36.0	3.86	7.50
Left sural nerve	36.9	3.60	12.20

NCV: Nerve conduction velocity; DML: Distal motor latency; AMP: Amplitude; EDB: Extensor digitorum brevis.

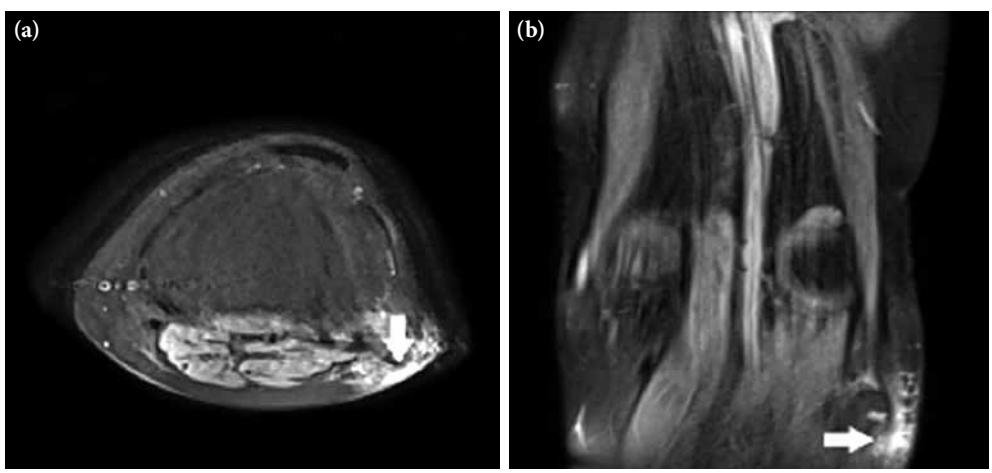

Figure 1. (a) Magnetic resonance imaging of left leg showing hyperintensity in fibular head. **(b)** Axial T_2-weighted magnetic resonance image of the leg below the knee. Coronal T_2-weighted magnetic resonance image of the leg below the knee. Hyperintensities on T_2-weighted images (white arrows) compatible with edema, compressing the common peroneal nerve and resulting in a peroneal neuropathy.

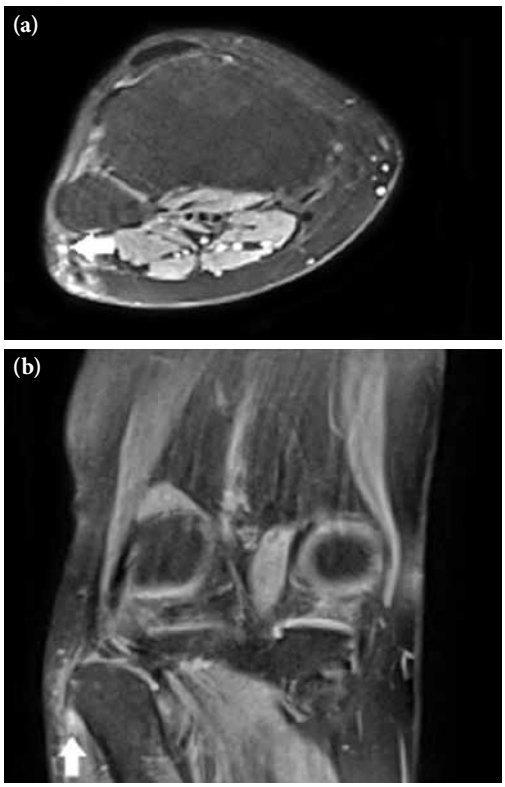

Figure 2. (a) Magnetic resonance imaging of right leg showing hyperintensity in fibular head. **(b)** Axial T_2-weighted magnetic resonance image of the leg below the knee. Coronal T_2-weighted magnetic resonance image of the leg below the knee. Hyperintensities on T_2-weighted images (white arrows) compatible with edema, compressing the common peroneal nerve and resulting in a peroneal neuropathy.

Clinically, moderate peroneal nerve dysfunction was confirmed. The results of repeated NCS and ENMG studies are shown in Tables 2 and 3. The patient used ankle-foot orthosis for ambulation alone.

DISCUSSION

Peroneal nerve palsy is the most common entrapment neuropathy of the lower extremity.[3] Patients with unilateral peroneal nerve palsy are frequently encountered in clinical practice. Although bilateral peroneal nerve palsy is rare, bilateral foot drop due to peroneal nerve palsy is much less common, and prolonged squatting, holding and pressing the lateral aspect of the flexed knees in a supine position, following skeletal traction for bilateral femoral fractures, complication of total knee arthroplasty, use of the pneumatic compression devices, and nerve engagement of the giant cell arteritis have

Table 3. Needle electroneuromyography findings

Muscle	Spontaneous					Motor unity action potential			Recruitment
	Initial activity	Fibrillation	Positive sharp wave	Fasciculation	High frequency	Amplitude	Duration	Polyphase	Pattern
Right tibialis anterior	Normal	2+	2+	None	None	Normal	Normal	Normal	No activity
Right tibialis posterior	Normal	None	None	None	None	Normal	Normal	Normal	Normal
Right biceps femoris (short head)	Normal	None	None	None	None	Normal	Normal	Normal	Normal
Left tibialis anterior	Normal	2+	3+	None	None	Normal	Normal	Normal	No activity
Left tibialis posterior	Normal	None	None	None	None	Normal	Normal	Normal	Normal
Right tibialis anterior 4th month	Normal	None	1+	None	None	1-	1+	2+	Reduced
Left tibialis anterior 4th month	Normal	None	1+	None	None	1-	1+	2+	Reduced

been reported in the literature.[2-9] Iatrogenic injury is also common with acute foot drop, mainly resulting from surgery of the hip, knee, and ankle; positioning during anesthesia; prolonged bed rest; casting; bracing; compression wrapping; and the use of pneumatic compression devices.[3] However, as in our case, in certain instances, the patient and clinician may not be aware that external compression has occurred due to the fact that it may develop during sleep or under the influence of drugs or alcohol. External compression of peroneal or tibial nerves in the distal thigh, in the upper part of popliteal fossa, is uncommon due to the deep course of both nerves in this site.

Peroneal nerve palsies have been also reported following severe weight loss, as the thinner subcutaneous tissue over the fibula head carries a risk of injuring the peroneal nerve.[10,11] Simultaneous compression by the bed, chair, or contralateral knee may easily cause bilateral nerve palsies in patients with weight loss. Thus, the development of bilateral peroneal nerve palsies requires simultaneous compression to both legs.[4,8] The reported case in the present study seemed to have two risk factors for the development of peroneal nerve palsy. Compression due to prolonged immobilization in the lying position was the initial risk factor, while malnutrition seemed to be the other one. Although increased pressure in the fibular head due to prolonged lying is thought to be an initiating event leading to common peroneal nerve injury, there is also a possibility that alcohol-induced volume depletion and malnutrition also contribute to aggravated injury.

Diagnosis of peroneal neuropathy is based on the characteristics of motor and sensory abnormalities, patient history, and physical examination. Patients with a peroneal nerve injury often present with complete or partial foot drop in addition to a change in gait, such as a steppage gait. Reflexes are spared as well as the plantar flexion and inversion of the ankle. However, weakness of the ankle dorsiflexors and evertors is common, resulting in typical foot drop.[12]

Electrodiagnostic studies also help the clinician evaluate the motor and sensory axons of the peroneal nerve and its branches. They also are useful for localizing the site of injury, evaluating the severity of a lesion, and monitoring recovery after a nerve injury.[3] An electrophysiologic study should be performed baseline in all patients with new-onset foot drop, and it can be repeated every three months to monitor for improvement or deterioration.[3,13]

Furthermore, MRI has been increasingly utilized as an adjunct in the evaluation of mononeuropathies. Intrinsic and extrinsic lesions of the peripheral nerves and features of denervation of corresponding muscles can be identified using MRI. The nerve normally shows low signal intensity within high-intensity fat on T_1- and T_2-weighted sequences. Abnormal bulbous enlargement of nerve is seen at the site of compression with increased signal on T_1- and T_2-weighted sequences. The MRI scan of normal skeletal muscle shows an intermediate signal intensity on T_1-weighted sequence and lower signal intensity on T_2-weighted sequence. In acute muscle denervation, normal T_1- and T_2- signal pattern may be retained up to one month; however, increased short-tau inversion recovery (STIR) signal intensity may be reported within four days and gadolinium enhancement as early as 24 hours after denervation.[14]

Initial treatment of a peroneal neuropathy is typically conservative management including a variety of interventions such as stretching, range of motion exercises, and strength training. In the setting of substantial muscle weakness, electrical stimulation can be used to initiate muscle contractions. Depending on the severity of the lesion and extent of symptoms, an ankle-foot orthosis can be used for toe clearance during ambulation.[3,13] Surgical decompression should be considered for refractory cases and those with compressive masses, acute lacerations, or severe conduction changes. Tendon and nerve transfers can be used in the setting of failed decompression or for patients with a poor prognosis for nerve recovery.[3]

Our case with chronic alcoholism had bilateral common nerve compression syndrome caused by malnutrition and immobilization. As soon as we were informed that the patient was sleeping in the prone position, we considered that the formation of bilateral peroneal palsy was probably in the slept in a flexed spine kneeling position.

In conclusion, for patients with common peroneal nerve entrapment at the fibular head, watchful waiting until spontaneous recovery occurs has been advocated. In this case, spontaneous recovery was achieved four months after the injury. Prompt recognition of a peroneal neuropathy is important in ensuring appropriate treatment, preservation of maximum function, and resolution of the injury.

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