



CASE REPORT

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An unusual double crush syndrome: femoral and sciatic neuropathy secondary to intra-abdominal firearm

Hilal Telli¹, Serkan Telli²

¹Ministry of Health Erzurum Regional Training and Research Hospital, Physical Medicine and Rehabilitation Clinic, Erzurum, Turkey

²Ministry of Health Erzurum Regional Training and Research Hospital, Anesthesiology and Reanimation Clinic, Erzurum, Turkey

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Abstract

Sciatic and femoral nerve neuropathies are commonly encountered causes of lower extremity dysfunctions. The sciatic nerve originates from the L4-S3 spinal nerves of the lumbosacral plexus, and the femoral nerve from the L2-L4 roots of the lumbar plexus. Traumatic, compressive, ischemic, neoplastic or idiopathic factors can lead to neuropathies of these nerves. We report a case of a 17-year-old male patient undergoing right hemicolectomy surgery following an abdominal firearm injury and with weakness around the right hip, knee and ankle in the postoperative period and paresthesia in the right knee, lateral part of the leg and dorsum of the foot.

Keywords: Femoral nerve, lumbosacral plexus, neuropathy, sciatic nerve

Introduction

The sciatic nerve originates from the L4-S3 spinal nerves and exits the pelvis by emerging from the large sciatic foramen beneath the piriformis muscle. It consists of lateral (peroneal fibers) and medial (tibial fibers) trunks. The sciatic nerve has no sensory branch. The lateral trunk remains taut without elasticity when opens into the sciatic notch, and has less flexibility in the face of tension since it is larger, contains less connective tissue, and has fewer fascicles than the medial trunk [1,2]. Since the sciatic nerve runs around the hip joint, trauma is the most common cause of proximal sciatic neuropathies. Compression of the sciatic nerve may occur due to hip operations, fracture, needle biopsy, sitting on hard surfaces, scar tissue and hematoma after injection, endometriosis inside the pelvis and long-term immobilization in bed [1,3]. The most common symptom in patients with sciatic neuropathy is weakness. In severe nerve lesion, paralysis develops in the hamstring muscles and all the muscles below the knee. Lack of sensation and paresthesia are also commonly encountered, and most patients experience dysesthetic pain inside the nerve's area of innervation. Loss of sensation occurs below the knee, and the Achilles reflex is often not elicited or else is significantly hypoactive [1,2]. Nerve conduction studies and needle EMG may be useful in the diagnosis and evaluation of traumatic sciatic nerve lesions. Nerve conduction study can help differentiate a lesion in the proximal part affecting one or both trunks of

the sciatic nerve from a lesion occurring in the distal part after the nerve has divided into the peroneal and tibial nerves [1,2].

The femoral nerve passes through the pelvis retroperitoneally by combining fibers from the L2, L3 and L4 roots and enters the lower extremity by passing beneath the inguinal ligament. It innervates the iliacus, sartorius, pectineus and quadriceps muscles. Sensory branches leaving the femoral nerve innervate the anterior internal part of the pelvis, while the saphenous nerve innervates the inner part of the leg [1,2]. The femoral nerve may be subjected to compression in association with pelvic or retroperitoneal hematoma, tumor or trauma. Neuropathy is characterized by a decrease in quadriceps muscle strength causing instability in the knee, by a decrease sensory symptoms in the anterior thigh and the inner surface of the calf. Localized pain may sometimes be present in the inguinal region or iliac fossa [1,2]. Patellar reflex may not be present at examination. Weakness in the iliopsoas muscle indicates that the superior lumbar plexus or L2 and L3 roots are affected. Weakness in the hip adductors similarly suggests lumbar plexopathy or L2 and L3 radiculopathy [1,2]. Femoral and saphenous nerve conduction studies permit differentiation of femoral neuropathy from lumbar radiculopathy. Weakness in the quadriceps muscle and myopathy leading to atrophy, radiculopathy and myelopathy must be distinguished at differential diagnosis. Careful clinical examination and electrophysiological investigation will assist with confirmation of the diagnosis [1,2].

We report a case of a 17-year-old male patient who had undergone right hemicolectomy surgery following an

*Corresponding Author: Serkan Telli, Ministry of Health Erzurum Regional Training and Research Hospital, Anesthesiology and Reanimation Clinic, Erzurum, Turkey
E-mail: md.serkantelli@gmail.com

abdominal firearm injury and who developed simultaneous femoral and sciatic nerve neuropathy in the postoperative period.

Case

A 17-year-old male patient with no previous known disease suffered a abdominal firearm gunshot injury one month before presentation. He was brought to hospital in an unconscious state secondary to intestinal perforation and underwent partial right hemicolectomy. Difficulty in walking, weakness in the right lower extremity, diffuse pain from the lower back to the posterior surface of the right leg, paresthesia and fatigue accompanying pain has developed in the postoperative period. The patient consulted with these symptoms to the Erzurum Regional Training and Research Hospital Physical Medicine and Rehabilitation clinic. The pain was continuous, but was unassociated with movement and did not worsen with strain. All joint extensions were full and painless in both upper and lower extremities at locomotor examination. He moved with steppage gait pattern without support, but had difficulty in walking up and down steps. Lumbar joint range of motion was full and painless. Scar tissue was present in an entry hole 2 cm to the right of the L4 vertebra in the dorsal region and in an exit hole in the lateral aspect 7 cm to the right of the umbilicus. The straight leg raise and Laseque test were negative. Disesthesia was present in the anterolateral surface of the left lower extremity and the dorsum of the foot at sensory examination. Muscle tone in the upper and lower extremities was normal. When muscle strength examination was performed based on the Medical Research Council scale, right hip flexion was 4/5, knee extension 3/5, ankle dorsiflexion 0/5, ankle plantar flexion 2+/5, and hallux dorsiflexion 0/5. With the exception of these muscles, all motor examinations in the upper and lower extremities were within normal limits. Right-side Patellar reflex was normal, right-side Achilles reflex was hypoactive, and there was no pathological reflex positivity. The patient's superficial, pain and joint position sensation was normal. There are not diameter variation was determined at extremity measurements and are not vasomotor symptoms, such as swelling, increased temperature or color change were observed around the affected right foot and ankle. Complete blood count, erythrocyte sedimentation rate and C-reactive protein blood biochemistry were within normal limits. Sacralization and a fracture line were present in the right sacrum and the L5-S1 fusion region at lumbosacral x-ray. Zone 1 fracture in the right sacrum and the L5-S1 fusion region was evaluated at abdominal computerized tomography (CT). At abdominal magnetic resonance imaging (MRI) performed post-injury determined were interpreted as indicating psoas hematoma that occasional hemorrhagic densities, relatively greater thickening in the right iliopsoas muscle compared to the left. Sacral MRI revealed cortical irregularity, edematous bone marrow and an existing fracture line in the right sacrum, and also significant thickening in the right sciatic nerve from the point of

emergence from the sacrum to the proximal thigh compared to the left, and edema (sciatic nerve injury).

The examination and imaging findings were evaluated as femoral nerve neuropathy secondary to psoas hematoma and sciatic nerve neuropathy secondary to sacrum zone 1 fracture. There are any findings were encountered at lumbar MRI, and electroneuromyography (ENMG) was performed. Right peroneal motor amplitude was not recorded from the distal aspect, while very low amplitude was recorded from the proximal aspect. Tibial and femoral motor and saphenous and sural sensory amplitudes were lower than left-side measurements. Needle electromyography (EMG) revealed intense acute denervation potential and pronounced thinning in muscles with right peroneal innervation, chronic neurogenic motor unit potentials (MUP) and a mild thinning pattern in muscles with tibial innervation, but any pathological findings were in the paravertebral and other muscles. These findings were reported as compatible with severe right sciatic peroneal branch neuropathy, mild partial tibial branch neuropathy and mild partial femoral nerve neuropathy.

The patient was enrolled in the rehabilitation program in our clinic. Pregabalin 75 mg in 2 oral doses daily was given as medical treatment for the lower extremity pain and disesthesia. Foot care instruction was given in order to prevent pressure injuries. Reflex AFO / low foot brace were prescribed to assist walking, and walking training was provided with orthosis. An 8-week physiotherapy program was initiated consisting of electrostimulation (20 min) of the lower extremity muscles (particularly the bilateral dorsiflexor, plantar flexor, evtor and toe dorsiflexor muscle groups), proprioceptive, strengthening, tensioning and walking exercises after hotpack (20 min), right hip and knee proprioceptive, strengthening, tensioning and walking exercises and ambulation training.

At muscle strength assessment following the rehabilitation process, hip flexion and knee extension were 5/5, ankle dorsiflexion 4/5, ankle plantar flexion 4+/5, and hallux dorsiflexion 2/5 and the patient's ambulation had improved significantly. The patient stated that he was able to walk up and down steps on the stairs more easily, that he could walk on rough surfaces without tripping, and that the disesthesia (neuropathic pain) in the anterolateral surface of the leg and the dorsum of the foot had also decreased following pregabalin therapy. The patient was informed of the results achieved during treatment and of potential conditions that might arise. We also obtained a written consent form from the patient for these results to be published as a case report. The patient was discharged with a home program and clinic check-ups.

Discussion

This case is significant due to development of footdrop and walking difficulty following sciatic and femoral nerve injury (peripheral cause) concurrently with psoas

hematoma secondary to intra-abdominal firearm injury and sacrum fracture.

Sciatic neuropathy can develop due to focal lesion at the hip or pelvic level and also femoral neuropathy can develop due to focal lesion in the retroperitoneal region or at the pelvic level. Lesions may include demyelinating, axonal or mixed axonal-demyelinating injury and partial or complete nerve severance [4, 5]. Traumatic, compressive, ischemic, neoplastic or idiopathic causes may be involved in the etiology of sciatic and femoral neuropathy. Traumatic injuries may include femur fracture, hip dislocation or fracture, laceration or posterior compartment syndrome, or, as in this case, may be associated with firearm injury [5,6]. One of the causes of femoral nerve compression is retroperitoneal hemorrhage in patients using heparin or oral anticoagulants [7,8]. Retroperitoneal hemorrhage can lead to femoral neuropathy femoral in two locations along the course of the femoral nerve. One location is in the iliopsoas gutter due to the rigid surrounding fascia and the other location is at the inguinal ligament [9]. Sciatic and femoral neuropathy can appear with motor and sensory deficits [4]. Sensory loss may be observed in the posterior thigh, lateral leg and the entire foot in complete sciatic nerve lesion and in the anteromedial thigh in femoral nerve lesion.

Sciatic and femoral neuropathy occurred in our patient in association with a firearm injury at the level of the right lumbar region 4 months previously. ENMG examination was compatible with severe partial neuropathy of the right sciatic peroneal branch, mild partial neuropathy of the tibial branch and mild partial neuropathy of the femoral nerve. According to the generally accepted view, the peroneal part of the sciatic nerve is more severely affected by injury than the tibial part. The common peroneal branch in our patient was also more severely damaged than the tibial branch. Drop foot, weakness in right hip flexion and knee extension, hypoesthesia in the sural nerve dermatome and disesthesia in the posterolateral part of the lower extremity compatible with the level of injury were present at clinical examination. There was not Achilles reflex. Sciatic and femoral nerve damage in our patient developed in association with secondary to sacrum fracture and psoas hematoma related to intra-abdominal firearm injury. Retroperitoneal hematoma can be diagnosed using imaging methods such as USG and CT [10]. Hematoma was initially diagnosed in our patient with USG and CT, and USG was used during follow-up.

One retrospective study scanning 303 patients presenting with footdrop reported central causes in 31% of cases and peripheral causes in 68%. Peripheral causes include common peroneal nerve lesions, L5 radiculopathies, polyradiculopathies and, rarely, pure sciatic lesions [11]. Kim et al. observed that 22% of sciatic nerve injuries at the hip and thigh level treated surgically between 1968 and 1999 were associated with firearm injuries, 18% with intragluteal analgesic, antiemetic, antibiotic or local anesthetic injection, 17% with hip or femur fracture and dislocation and 5% with hip arthroplasty [12]. In

addition to the low level of hip arthroplasty, increased injury due to firearm injury and injection may account for these factors being frequently seen in earlier decades.

A retrospective screening study of 190 patients with sciatic nerve injury at the gluteal level between 1962 and 1997 by Huang et al. determined etiological causes in the form of injection in 164, knife injuries in 14, pelvic fracture and hip dislocation in 11 and contusion in one. Positive results were achieved at a level of 78.2% following neurolysis, epineural neuroorrhaphy, nerve graft and nerve exploration performed for therapeutic purposes [13]. Yuen and Onley investigated the clinical characteristics of 73 patients for whom electrodiagnostic examination was requested due to sciatic nerve neuropathy. They described absence of paralysis of the dorsiflexor/plantar flexor muscles of the foot as significant evidence for early or better clinical improvement [14].

The treatment of femoral neuropathy developing in association with hematoma in the iliacus muscle is controversial. Treatment varies depending on the rate of hematoma formation, the size of the hematoma and the level of neurological injury. Conservative treatment is recommended in hemodynamically stable cases, with mild or moderate neurological deficit, with no active hemorrhage and with hematoma developing in association with bleeding disorder and anticoagulant therapy, while transarterial embolization or surgical decompression are recommended in other cases [10]. Whether or not surgery is performed, it is important to initiate a rehabilitation program in the early period in cases of femoral neuropathy. Methods such as an appropriate exercise program for the quadriceps and hip flexors, walking aids (sticks, knee locking pads), EMG biofeedback and electrical stimulation can be used as a rehabilitation program. Improvement may be expected within 1 year in 70% of cases [7,16,17].

Our patient stated that he had been admitted to the emergency surgery department due to intra-abdominal hemorrhage and intestinal perforation but that he had not undergone surgery for repair of the sciatic nerve. He was admitted to a physiotherapy and rehabilitation program for the resolution of loss of muscle strength and walking difficulty. Joint-protection therapies were recommended. An orthosis was prescribed when instability was determined at gait evaluation even though the muscle strength of the ankles was good, and the patients received training in this. Our patient's knee flexor and extensor muscle strengths were 3/5 before rehabilitation, but were fully restored after rehabilitation. Footdrop was present on the right, for which reason he experienced frequent tripping and difficulty going up and down steps. On a flat surface he was independently mobile with a steppage gait. The patient's gait pattern and inclination, elevation and difficulty in walking up steps were overcome with a reflex AFO/short walking device. Lumbosacral plexus lesion, radiculopathy or mononeuropathy may be missed at differential diagnosis in patients presenting with similar clinical findings. Electrodiagnostic study and imaging must be performed

for differentiation [5]. Our patient was investigated in terms of lumbar pathology, but this was not considered at differential diagnosis. Our patient benefitted partially from all treatments and was discharged in an independent ambulatory condition. We think that the desired target was reached since our patient was started on neurological rehabilitation in the first month after injury.

Conclusion

Traumatic injuries of the sciatic and femoral nerves are multifactorial in origin. In the event that sciatic and/or femoral neuropathy is determined enrolling the patient early on a rehabilitation program is important in terms of preventing permanent nerve damage and potential future disabilities.

We report a case of a male patient who developed femoral nerve and sciatic nerve injury associated with sacrum fracture and psoas hematoma due to firearm injury and consequent footdrop. However, disability was prevented since rehabilitation was initiated in the early period.

Conflict of Interest and Financial Support Declaration

The authors declare no conflict of interest in this study, and that no financial support was received.

References

1. Drake RL, Vogl W, Mitchell AWM; illustrations by Tibbitts RM and Richardson P; photographs by Horn A. Paul Gray's anatomy for students. Philadelphia: Elsevier/Churchill Livingstone. 2005.
2. Akarırmak Ü: Tuzaknöropatileri. In: Beyazova M, Gökçe-Kutsal Y, eds, Fiziksel Tıp ve Rehabilitasyon. Ankara: Güneş Kitabevi, 2000;2071-2089.
3. Akı S, Alev L, Boyacıyan A, Karan A: Siyatik sinir nöropatisine sebep olan etyolojik faktörlerin değerlendirilmesi. Türkiye Fiziksel Tıp ve Rehabilitasyon Dergisi. 1998;1(1):65-67.
4. Sunderland S. A classification of peripheral nerve injuries producing loss of function. Brain. 1951;74:491-516.
5. Yuen EC, So YT. Entrapment and other focal neuropathies: sciatic neuropathy. Neurol Clin. 1999;17(3):617-31.
6. Plewna C, Wallace C, Zochodne D. Traumatic sciatic neuropathy: a novel cause, local experience and review of the literature. J Trauma. 1999;47(5):986-99.
7. Kocatürk H, Yurtman V, Karaca L. Warfarin kullanımına bağlı gelişen subakut retroperitoneal kanamaya bağlı sol femoral sinir nöropatisi. Genel Tıp Derg. 2013;23(2):56-8. 11
8. Zago G, Appel-da-Silva MC, Danzmann LC. Iliopsoas muscle hematoma during treatment with warfarin. Arq Bras Cardiol. 2010;94(1):1-3.
9. Krause ML, Post JA. 73-year-old woman with anterior thigh pain. Mayo Clin Proc. 2011;86(4):21-4.
10. Murray IR, Perks FJ, Beggs I, Moran M. Femoral nerve palsy secondary to traumatic iliacus haematoma- a young athlete's injury. BMJ Case Rep. 2010;2010.
11. Van Langenhove M, Pollefliet A, Vanderstraeten G. A retrospective electrodiagnostic evaluation of footdrop in 303 patients. Electromyogr Clin Neurophysiol. 1989;29(3):145-52. 20.
12. Kim DH, Murovic JA, Tiel R, Kline DG. Management and outcomes in 353 surgically treated sciatic nerve lesions. J Neurosurg. 2004;101:8-17.
13. Huang Y, Yan Q, Lei W. Gluteal sciatic nerve injury and its treatment. ZhongguoXiu Fu Chong Jian Wai Ke Za Zhi. 2000;14(2):83-6.
14. Yuen EC, Onley RK, So YT. Sciatic neuropathy: clinical and prognostic features in 73 patients. Neurology. 1994;44(9):1669-74.
15. Wang JQ, Yi MJ, Zhu Y, Wang MY, Zhang BS, Liu DQ, Wei J. Surgical treatment of shotgun injuries of the sciatic nerve. Zhonghua Wai Ke Za Zhi. 2004;42(2):81-3.
16. Kong WK, Cho KT, Lee HJ, Choi JS. Femoral neuropathy due to iliacus muscle hematoma in a patient on warfarin therapy. J Korean Neurosurg Soc. 2012;51(1):51-3.
17. Çeliker R, Alt Ekstremitelerde Tuzak Nöropatiler, Türk Fiz Tıp Rehab Derg. 2009;55(1):30- 15.