# Bilateral Femoral Neuropathy with "Hanging Leg" Syndrome: Report of a Case

Ping-Chang Yang[1] and Shu-Feng Chang[2]

**Abstract-** The most frequent cause of femoral nerve injury is trauma and unfortunately often iatrogenic owing to direct or indirect compression during operative procedures. Another common compressive cause is hematoma caused by coagulopathy in the iliopsoas. We report a case of femoral neuropathy without any predisposing factor. A detail history and a clinical electrodiagnostic study are most important in establishing the nature of injury, anatomic localization, and prognosis. Review of the literature suggests that the injury is related to nerve stretching or ischemia with prolonged maintenance of a "hanging leg" position.

**Key Words:** Femoral neuropathy, Non-compression lesion, Heroin, "Hanging leg" syndrome

*Acta Neurol Taiwan 2003;12:136-138*

## INTRODUCTION

The traumatic[1] or non-traumatic compressive lesions are the major causes of the femoral neuropathy. The femoral nerve may be damaged by a penetrating trauma, bony fracture, or surgical procedures involving abdominal, pelvic or hip areas[2-4]. Other compressive lesions are benign or malignant mass, abscess, hematoma in iliacus or psoas muscles. There are other uncommon injuries[5-9]. We report a case of bilateral femoral neuropathies that were caused by hyperextension of the hip during deep sleep.

## CASE REPORT

A 33-year-old man had been a heroin addict for 10 years. On June 28, 2002, immediately after an intravenous bolus of 10 ml of heroin at his left elbow, he felt asleep in supine position with both legs hanging on the edge of the bed for about 6 hours. On awaking, he was unable to stand up and also had burning pain in both anterior thighs.

He was admitted to a local hospital 3 days later. Physical examination revealed weakness of quadriceps, and pain and numbness at both anterior thighs. Both knee jerks were decreased. There was no obvious swelling or tenderness at thigh or inguinal area. Radiography of lumbar spines and knees were normal. Serum muscle enzyme was elevated, including creatine kinase (5570 IU/L), lactate dehydrogenase (854 IU/L), aspartate aminotransferase (94.3 IU/L), and alanine aminotransferase (43.8 IU/L). Other laboratory data

From the [1]Department of Neurology, Pao Chien Hospital, Ping Dong, Taiwan; and [2]Department of Neurology, Ming Chung Hospital, Ping Dong, Taiwan.
Received January 29, 2003. Revised March 12, 2003.
Accepted May 23, 2003.

Reprint requests and correspondence to: Ping-Chang Yang, MD. Department of Neurology, Pao Chien Hospital, No. 123, Zhong Shan Road, Ping Dong, Taiwan.
E-mail: nicegone@yahoo.com.tw

including complete blood count were within normal limits. Nerve conduction velocity (NCV) study of the lower limbs (including bilateral tibial, peroneal, and sural nerves) and F-waves of tibial and peroneal nerves were normal. However, electromyography (EMG) and NCV of the femoral nerves were not done. The patient was referred for physiotherapy under the impression of myositis. His recovery was gradual. He was able to walk independently, though incompletely, about 2 months later.

Five months after the injury, he visited our orthopedic clinic to correct knee in stability. He complained of persistent numbness of left anteromedial thigh numbness and weakness of bilateral proximal thigh muscles. Neurological examination revealed marked bilateral quadriceps wasting, left side predominant decreased voluntary muscle power (about 3/5 ~ 4/5), and absence of bilateral knee reflex. Pinprick, touch, and temperature was decreased over left distal anteromedial thigh. He could ambulate independently but with an unsteady gait, especially in climbing stairs. He was unable to stand up from squatting position. NCV study revealed normal conductions of bilateral tibial, peroneal, and sural nerves, decreased amplitude of the compound muscle action potentials of right femoral nerve, no pick up of left femoral nerve and bilateral saphenous nerves (Table). Needle EMG study revealed rare positive waves, no fibrillation at rest, markedly decreased insertional activity, polyphasic motor units of abnormal long duration of bilateral vastus medialis and vastus lateralis. EMG study of bilateral fourth lumbar paraspinal muscle, iliopsoas, adductor magus, anterior tibialis, and gastrocnemius muscle was normal. Pelvic CT scan and lumbar myelogram revealed no compressive lesion or hematoma. No contributory causes were noted in laboratory studies, including complete cell count, serum myoglobulin, Venereal Disease Research Disease Laboratory, human immunodeficiency virus, and prothrombin time.

## DISCUSSION

The femoral nerve arises from the posterior divisions of the second, third, and fourth lumbar spinal roots. It penetrates the psoas major muscle to situate in a groove between the psoas and iliacus muscles. Here it is covered by the fascia iliacus. Passing over the superior pubic ramus and under the inguinal ligament, it enters the femoral triangle, where it divides into a number of terminal branches which supplies the pectineus, sartorius, vastus intermedius, vastus lateralis, vastus medialis and rectus femoris. The intermediate and medial cutaneous branches innervate the anteromedial thigh, and the saphenous nerve branch is responsible for cutaneous sensation along the medial aspect of the calf and foot. The femoral nerve receives its blood supply in the pelvis from the small iliolumbar artery and the deep circumflex

***Table.*** *Nerve conduction velocity (NCV) study of the patient*

| Nerve | Stimulation site | Recording site | Amplitude (mV) | Latency (m/s) | MNCV [a] (m/s) | SNCV [a] (m/s) |
|---|---|---|---|---|---|---|
| R Tibial | (ankle/knee) | AH[a] | 23.0/18.7 | 3.6/10.2 | 50 | |
| L Tibial | (ankle/knee) | AH | 20.1/17.7 | 3.4/10.0 | 50 | |
| R Peroneal | (ankle/knee) | EDB[a] | 9.3/ 8.9 | 3.1/10.0 | 57 | |
| L Peroneal | (ankle/knee) | EDB | 8.3/ 7.5 | 2.5/ 9.7 | 50 | |
| R femoral | inguen [b] | Quadriceps | 0.2 | 5.3 | | |
| L femoral | inguen | Quadriceps | no response | | | |
| R H-reflex | | | | 27.I | | |
| L H-reflex | | | | 26.7 | | |
| R Sural [c] | | | 21 | 2.0 | | 50 |
| L Sural | | | 22 | 2.0 | | 50 |
| R Saphenous | | no response on stimulation 14 cm above the ankle | | | | |
| L Saphenous | | no response on stimulation 14 cm above the ankle | | | | |

[a] Motor nerve conduction velocity (MNCV, meter/ second), sensory nerve conduction velocity (SNCV, meter/second), abductor hallucis muscle (AH), extensor digitorum brevis (EDB).

[b] The active surface recording is placed over the quadriceps 14 cm distal to the stimulation at the femoral nerve just below the inguinal liga ment and lateral to the femoral artery.

[c] The active surface stimulation site was placed 14 cm proximal to the recording site (lateral malleolus).

iliac artery. Outside of the pelvis the nerve receives blood from the lateral femoral circumflex artery[10].

Most cases of the femoral neuropathies following closed trauma are caused by rupture of iliopsoas muscle with formation of a hematoma, which may compress the femoral nerve. This kind of lesion usually follows a fall onto the back or side, and the location of the hematoma is usually proximal to the inguinal ligament[7-8]. The patient will complain of pain in the abdomen, groin, or lumbar area and a tender palpable mass or swelling is usually noted. Pain may be provoked on flexion and external rotation of the hip.

We report this case of femoral neuropathy without any predisposing factor. The history suggests that the femoral nerve was probably injured from a combination of acute stretch and ischemia after unattended sleep with "hanging leg" posture (Fig.). As the femoral nerve passes over the superior pubic ramus, the bone may act as a frucrum[5]. In this fixed position, with the hip forced into hyperextension, a stretch injury can occur. The obturator nerve is relative spared for a less restriction of motion and no bony prominence over its course.

Clinical electrodiagnostic study is useful in determining the distribution and prognosis for this patient. A complete or severe femoral nerve lesion indicate a severe injury with axonal loss. Consequently, severe deficit without dramatic rapid recovery is expected. Physicians should be aware that bilateral femoral neuropathies can result from a "hanging leg" position. The close femoral nerve injury probably results from a stretch or ischemia. If there is a hematoma or the lesion is progressive, exploration is indicated. When hematoma is not found or less likely clinically, such as our patient, a conservative treatment is indicated[8]. Depending on the severity of the injury, there can be partial or complete recovery which may take days, months, or years.

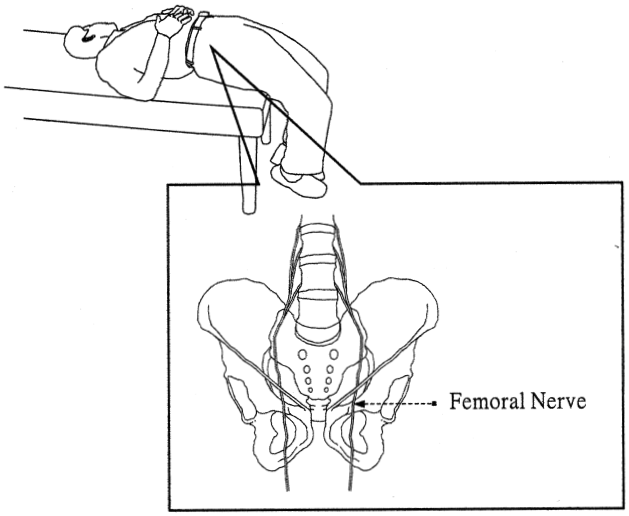


**Figure.** Bilateral "hanging legs," with the hip hyperextended and the buttock hanging against the edge of the mattress. Insert illustrates left femoral nerve as it passes over superior pubic ramus and under the inguinal ligament, a site of potential injury.

## REFERENCES


1. Kim DH, Kline DG. Surgical outcome for intra- and extrapelvic femoral nerve lesions. J Neurosurg 1995;83: 783-90.
2. Burnett AL, Brendler CB. Femoral neuropathy following major pelvic surgery: etiology and prevention. J Urol 1994;151:163-5.
3. Walsh C, Walsh A. Postoperative femoral neuropathy. Surg Gynecol Obstet 1992;174:255-63.
4. Kuntzer T, van Melle G, Regli F. Clinical and prognostic features in unilateral femoral neuropathies. Muscle Nerve 1997;20:205-11.
5. Rottenberg MF, DeLisa JA. Severe femoral neuropathy with "hanging leg" syndrome. Arch Phys Med Rehabil 1981;62:404-6.
6. Shields RWJ, Root KE, Wilbourn AJ. Compartment syndromes and compression neuropathies in coma. Neurology 1986;36:1370-4.
7. Green JP. Proximal avulsion of the iliacus with paralysis of the femoral nerve: report of a case. J bone Joint Surg Br 1972;54:154-6.
8. Brozin IH, Martfel J, Goldberg I. Traumatic closed femoral nerve neuropathy. J Trauma 1982;22:158-60.
9. Miller EH, Benedict FE. Stretch of the femoral nerve in a dancer: a case report. J Bone Joint Surg Am 1985;67:315-7.
10. Boontje AH, Haaxma R. Femoral neuropathy as a complication of aortic surgery. J Cardiovasc Surg (Torino) 1987; 28:286-9.