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# Bilateral Sciatic Neuropathy due to Drug Overdose with Coma

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Limb compression in an unattended comatous patient often results in peripheral nerve injuries; however, bilateral sciatic neuropathy in these patients has rarely been described in literature. A 37-year-old woman with bilateral numbness and paralysis of the lower limb after drug overdose with coma was reported. After onset of unconsciousness, she was in a seated position with trunk and hips flexed and knees nearly fully extended for approximately ten hours. In the department of emergency, she was found to have bilateral thighs and buttocks swollen as well as myoglobulinuria. Electrodiagnostic studies supported a diagnosis of complete denervation bilaterally in the distribution of the sciatic nerve. After 19 months of intense rehabilitation, some evidence of reinnervation was noticed in follow-up electrodiagnostic testing. There were significant improvements in motor function with some remaining neurological deficits. According to our findings, severe and disabling sequelae may result from such peripheral nerve injuries, however, rehabilitation for these patients is helpful. (Tw J Phys Med Rehabil 2006; 34(1): 33 - 39)

**Key words:** sciatic neuropathy, coma, rehabilitation

## INTRODUCTION

Peripheral nerve injuries are common sequelae of limb compression during coma.<sup>[1,2]</sup> More than one peripheral nerve may be affected, and the site is usually proximal and unilateral. Often there is an accompanying compartment syndrome that may cause neuromuscular injury. To our knowledge, only six cases of bilateral sciatic nerve injuries have ever been reported; none are in the literature of physical medicine and rehabilitation.<sup>[3-8]</sup>

Here we reported a rare case of bilateral sciatic nerve injury from gluteal compartment syndrome caused by limb compression and stretch injury during drug-induced coma and discussed the rehabilitation for the patient.

## CASE REPORT

A 37-year-old divorced woman with a long history of depression and insomnia was prescribed with antidepressants and benzodiazepine drugs regularly through a psychiatric outpatient department. She attempted suicide

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by taking an overdose of sertraline hydrochloride, midazolam and flupentixol. After taking the drugs, she fell into a deep coma in a seated position on the side of a bed with wooden rims such that her knees were nearly fully extended and her trunk was bent forward with both palms touching the floor (Fig. 1). On awakening nearly 10 hours after taking the drugs, she found herself being unable to walk or move her legs and fell to the floor in an attempt to go to the toilet.

After being awoken for several hours, she was transported to the department of emergency via ambulance, in a state of lethargy and nervousness with body temperature of 37.0°C, pulse rate of 90 beats per minute and respiratory rate of 20 breaths per minute. General physical examination showed marked bilateral swelling of buttocks, posterior and medial thighs. Neurological examination showed a clear but agitated mental status with flaccid legs. Dysuria was also present. Lab data showed elevated creatine phosphokinase (CPK) of 10000U/L, blood urea nitrogen (BUN) 57 mg/dL, creatinine 3.9 mg/dL, potassium 6.4 meq/L and myoglobinuria. Under the impression of acute rhabdomyolysis with acute renal failure, she was treated conservatively, including aggressive hydration and diuretic and bicarbonate therapies.

As the patient's mental status improved over the next day, a more detailed examination of both lower extremities was performed. Manual muscle testing (MMT) showed grade 4 in bilateral hip abduction, grade 1 in bilateral knee flexion, and grade 0 in bilateral ankle dorsiflexion, plantar flexion, inversion, and eversion. Knee extension and hip flexion, extension, rotation, and adduction were normal (Table 1). Ankle jerk and hamstring jerk were absent bilaterally. Hypoesthesia and hypalgesia were present over the lateral, dorsal and ventral sides of her calves and feet.

By day 3, the patient was in a stable condition with lab data improving: CPK 7482 U/L, BUN 15 mg/dL, creatinine 2.1 mg/dL, and potassium 3.8 meq/L. However, severe sharp, shooting bilateral pain over the calves and soles of the foot persisted. Although no improvements in muscle power were seen, she was able to transfer from bed to wheelchair independently. She was still unable to ambulate independently.

A nerve conduction study (NCS) done on day 18

failed to identify bilateral sural and superficial peroneal sensory nerve action potentials (SNAP) and bilateral peroneal and tibial compound muscle action potentials (CMAP) (Table 2). Needle electromyography (EMG) showed absence of motor unit action potentials (MUAP) during voluntary contraction bilaterally in muscles innervated by the sciatic nerve. Denervation potentials and increased polyphasic potentials were found bilaterally in the tensor fascia lata, right L5 and S1 paraspinal muscles, left gluteus medius muscle, and right external anal sphincter (Table 2).

Bilateral magnetic resonance imaging (MRI) of buttocks and thighs showed enlarged bilateral posterior compartment muscle groups with abnormal signal increase interpreted as edema or hemorrhage. The subcutaneous layers also showed edema in the medial aspect of both thighs. Some areas of dark signal within the abnormal muscles were interpreted as hemosiderin deposition, especially in the bilateral posterior muscle groups. The sciatic nerves were embedded within the lesions. MRI was compatible with rhabdomyolysis mainly involving the posterior compartment of both thighs, with nerve compression (Fig. 2 and 3).

The patient received rehabilitation therapy including passive exercise for range of motion, galvanic muscle stimulation bilaterally over the hamstring muscles and tibialis anterior muscles, progressive resistive exercises, and ambulation training with bilateral ankle-foot orthoses and a large-base quadricane.

At the initial follow-up at around 5 months post-injury, the patient showed some improvement in knee flexor strength with grade 2- bilaterally (Table 1). A later nerve conduction study (day 222) showed the appearance of left tibial CMAP with reduced amplitude and prolonged distal latency (Table 2).

Follow-up at nearly 19 months post-injury showed impressive improvements in muscle power with grade 5 in the right knee flexor, 4 in the left knee flexor, 4 bilaterally in ankle dorsiflexion, and 3+ bilaterally in plantar flexion (Table 1). Nerve conduction study showed findings similar to those obtained on day 222, and needle electromyography showed partial reinnervation bilaterally in the hamstring medial heads, biceps femoris short head, gastrocnemius, peroneus longus, peroneus brevis, tibialis anterior, and extensor hallucis longus (Table 2).

Table 1. Manual muscle testing after days of injury

Days after injury	2	18	147	222	378	468	564
Hip extension	5/5	5/5	5/5	5/5	5/5	5/5	5/5
Hip abduction	4/4	4/4	5/5	5/5	5/5	5/5	5/5
Knee extension	4/4	5/5	5/5	5/5	5/5	5/5	5/5
Knee flexion	1/1	1/1	2-/2-	3/2+	2/2+	4/4	5/4
Ankle dorsiflexion	0/0	0/0	0/0	0/0	1/1	3-/3-	4/4
Ankle eversion	0/0	0/0	0/0	0/0	0/0	2-/2-	3+/3+
Ankle plantarflexion	0/0	0/0	0/0	0/0	0/0	2/2	3+/3+
Ankle inversion	0/0	0/0	0/0	0/0	0/0	2/2	4/4
Toe extension	0/0	0/0	0/0	0/0	0/0	1/1	3-/3-
Toe flexion	0/0	0/0	0/0	0/0	0/0	0/0	3-/3-

Dater expression: right side / left side

5=normal; 4=good; 3=fair; 2=poor; 1=trace; 0=zero.

Table 2. Electrodiagnostic studies

Days after injury	EMG findings	NCS findings
18	EMG: Complete denervation of bilateral sciatic nerves with partial denervation of bilateral inferior gluteal nerves, superior gluteal nerves, right L5 and S1 roots and right S2-4 roots.	NCS: absence of CMAPs in bilateral peroneal nerves, tibial nerve, absence of SNAPs in bilateral sural nerves, H-reflex and F-waves.
147	EMG: Denervation of bilateral sciatic nerves with partial regeneration in bilateral hamstring medial head.	NCS: absence of all CMAPs and SNAPs in the nerves of both lower extremities.
222	EMG: Denervation of bilateral sciatic nerves with partial regeneration in bilateral biceps femoris short head and bilateral hamstring medial head.	NCS: Absence of CMAPs and SNAPs in the nerves of both lower extremities except left tibial nerve, which showed marked reduction in CMAP amplitude. (0.05mV)
378	EMG: Partial regeneration as above plus bilateral tibialis anterior, bilateral gastrocnemius and bilateral peroneus longus.	NCS: same as above.
468	EMG: Partial regeneration as above plus bilateral extensor hallucis longus, bilateral peroneus brevis.	NCS: same as above.
564	EMG: Partial regeneration of affected nerves (similar to day 468).	NCS: same as above.

EMG= electromyography; NCS= nerve conduction study.

CMAP= compound muscle action potential; SNAP= sensory nerve action potential.

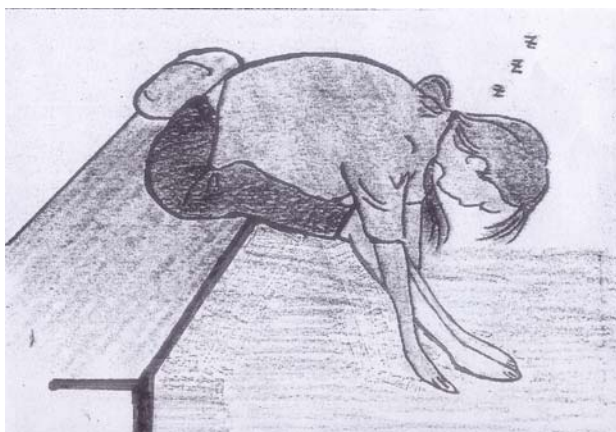


Figure 1. Position of the patient during state of coma.

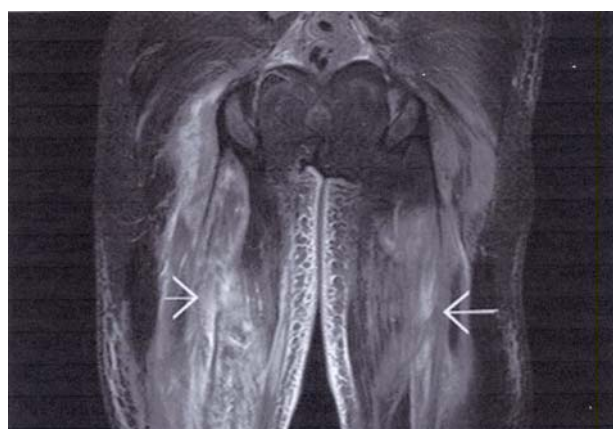


Figure 2. Coronal magnetic resonance image with a T2 spin echo, showing increased signals in the gluteal muscles, thigh muscles and subcutaneous tissues. Arrows point to the sciatic nerves embedded within the muscle groups.

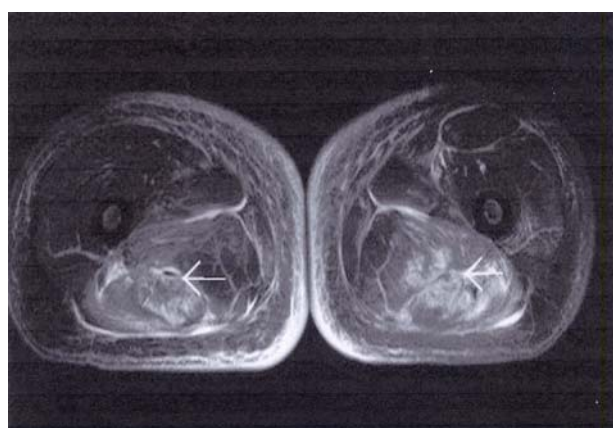


Figure 3. Axial magnetic resonance image with a T2 spin echo showing increase in the signals of the muscles in the posterior thigh compartment. Arrows point to the sciatic nerves embedded within the muscles with increased signals.

After almost 19 months of rehabilitation, she was able to walk without an assistive device but neurological deficits including hypesthesia and muscle weakness of both feet and legs remained.

## DISCUSSION

The causes of unilateral sciatic mononeuropathy are numerous. In a study of 100 patients by Yuen et al.,<sup>[9]</sup> the most common cause of sciatic neuropathy was total hip arthroplasty. Other possible mechanisms for sciatic nerve injury may be caused by overstretch injury, direct nerve trauma, hip dislocation or fracture, acute compression, and various other reasons. Bilateral sciatic neuropathy is a rare condition. It has been described as a consequence of traumatic hip dislocation,<sup>[3]</sup> hip surgery,<sup>[4]</sup> after an 8-hour operation in a sitting position,<sup>[5]</sup> after alcohol intoxication,<sup>[6]</sup> associated with weight loss,<sup>[7]</sup> as well as toilet seat entrapment.<sup>[8]</sup>

In our patient with acute bilateral lower extremity paralysis, weakness involved bilateral ankle dorsiflexors, plantar flexors, invertors, evertors, and knee flexors, with paresthesias and dysesthetic pain in lateral calves and soles of both feet. Nerve conduction study on day 18 showed absence of bilateral peroneal and tibial CMAP, bilateral sural and superficial peroneal nerve SNAP, and also bilateral H-reflex and F-wave. Needle electromyography at the same time showed positive sharp waves and fibrillations in all sciatic-innervated muscles bilaterally, as well as the right L5 and S1 paraspinal muscles and left gluteus medius muscle. Absence of voluntary MUAP in bilateral hamstring, gastrocnemius, tibialis anterior, and peroneus longus muscles were detected; however, the recruitment and interference pattern of bilateral paraspinal and gluteal muscles were within normal limits. All femoral nerve-innervated muscles were spared. Magnetic resonance imaging showed bilateral gluteal compartment syndrome with bilateral signal increase in the gluteal muscles and posterior thigh compartments. All of these findings suggested that the main problem in this case was ischemic compression of the bilateral sciatic nerves due to compartment syndrome in both buttocks and posterior thighs. There was also a possible injury to the lumbosacral roots and superior and inferior gluteal nerves due to the awkward position or ischemic compression. However,

the injury was mild and EMG examination revealed good recruitment of paraspinal and gluteal muscles.

The most possible cause of sciatic nerve injury in this patient was an external compression of the buttocks on the edge of the bed to both sciatic nerves, as well as a posture-induced overstretch injury to the lumbosacral roots. Our patient is a slender woman with body weight of 48 kilograms and body height of 160 centimeters (body mass index = 18.75) and this may be why she is more prone to a compression injury. In a study of 103 patients, Katirji et al. reported weight loss as the most common cause of common peroneal mononeuropathy.<sup>[10]</sup> Animal experiments of acute compression lead to paranodal and segmental demyelination.<sup>[11]</sup> However, severe compression injuries also affect axonal transport. In an experiment with rabbit sciatic nerve which was elongated and compressed, Ikeda et al.<sup>[12]</sup> also showed that even mild compression can cause severe damage to the elongated nerve. Therefore, an overstretched nerve is vulnerable to compression injury. On the other hand, a precompressed nerve may also easily develop stretch injury. Probably, this is one of the important reasons why our patient suffered from a severe nerve injury. In human volunteers, Gelverman et al. and Szabo et al. increased external pressure within the carpal tunnel for a maximum of 4 hours and demonstrated that all nerve function were lost when the pressure was up to 50 mmHg.<sup>[13]</sup> Electrophysiological studies and the clinical course of this patient indicated a possible axonotmesis injury bilaterally to the sciatic nerves.

Could the presence of acute rhabdomyolysis without neuropathy have produced electrodiagnostic findings similar to those found in this patient? Al-Shekhlee et al.<sup>[14]</sup> performed electrodiagnostic study on 15 patients with acute rhabdomyolysis within 2 weeks of symptom onset. He reported that all patients showed normal nerve conduction study results. Furthermore, needle electromyography showed either normal or only slightly increased on insertional activity or subtle myopathic MUAP in proximal muscles and normal recruitment. Therefore, it is unlikely that the abnormal electrodiagnostic findings in this case resulted solely from acute rhabdomyolysis.

Drugs taken by our patient included sertraline, midazolam, and flupentixol. None of these drugs taken by this patient has been reported to cause severe neurotoxic

effects in healthy individuals.<sup>[15]</sup> Moreover, drug-induced rhabdomyolysis complicated by compartment syndrome has been reported in association with alcohol, heroin, barbiturate, and benzodiazepine abuse.<sup>[16]</sup> This suggests that the nature of the drug is not a critical factor for development of rhabdomyolysis or compartment syndrome.

Management of sciatic neuropathy depends on the clinical setting and the suspected cause. In our patient, surgical decompression of the gluteal compartment could have been helpful. Rollins et al.<sup>[17]</sup> showed that at pressures of 120 mmHg, complete conduction block may occur after only 30 to 60 minutes, but fasciotomy within 4 hours may be followed by return of near normal conduction. Unfortunately, our patient arrived at the emergency department nearly 15 hours after onset of coma, which was too late for fasciotomy to be performed for prevention of disabling neuromuscular sequelae. Therefore, only conservative treatment was given, consisting of emergent medical treatment and, later, a rehabilitative program. Galvanic muscle stimulation was used to slow muscle atrophy and facilitate reeducation of the impaired muscle.<sup>[18]</sup> Passive range of motion exercise and stretching exercise should also be done to avoid contractures. Once the patient has improvement in muscle strength, isometric and isotonic strengthening exercises should be performed with gradual increase in frequency and intensity to build up strength and endurance. Ambulation training with crutches or canes and ankle-foot-orthosis should be started as soon as possible.

As with all other mononeuropathies, prognosis of sciatic neuropathy depends on its etiology and severity. In a study of 52 patients who had acute sciatic neuropathy and removal of the inciting factor (caused by hip replacement, hip dislocation or fractures, gunshot wound, acute external compression, nerve infarction, femoral fracture, or gluteal contusion), improvement in MMT to grade 2 or by at least 2 grades occurred in 30% of patients by 1 year, 50% by 2 years, and 75% by 3 years. Predictive factors of good prognosis included the presence of a recordable extensor digitorum brevis CMAP and absence of ankle paralysis.<sup>[19]</sup> In another report on a series of 52 patients who had sciatic neuropathy after total hip arthroplasty, the main factor predicting better neurological outcome was the presence of partial motor function on

initial examination.<sup>[20]</sup> In our case, the bilateral sciatic nerves most likely suffered axonotmesis injury, but not neurotmesis (or complete transection) injury, given subsequent significant reinnervation of the initially paralyzed muscles. In axonotmesis injuries, the basal laminal tubes of the nerve fiber remain intact, forming conduits for regenerating axon and Schwann cells.<sup>[21]</sup> Therefore, motor recovery was possible, with axons regenerating in proximal muscles (bilateral medial hamstrings and biceps femoris short head) first. Although our patient was not particularly tall in body height, the course of the patient's recovery was prolonged because the sciatic nerve is a long nerve. At 19 months post-injury, needle electromyography showed nerve regeneration reaching distally innervated muscles with good functional recovery.

## CONCLUSION

Bilateral sciatic neuropathy can be caused by prolonged ischemic compression to an elongated nerve as a consequence of prolonged sitting on the edge of a bed with trunk flexion and knee extension. Significant recovery is possible if the nerve is not transected and an appropriate rehabilitation program is provided.

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## 藥物過量昏迷後之雙側坐骨神經病變

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意識不清所造成的周邊神經損傷並不罕見，但因昏迷而導致雙側坐骨神經病變則十分罕見。

本文報告一位因藥物過量而導致昏迷，並引起雙側坐骨神經病變之案例。個案是一位三十七歲女性，因憂鬱症和長期失眠經常服用安眠藥及抗憂鬱藥劑。某日患者服用過量藥劑後神智變差，且以雙膝伸直的姿勢昏睡了將近十幾個小時。她醒來後發現自己無法移動雙腳而跌倒在地上。送醫後發現個案有雙側臀部及大腿腫脹及肌蛋白尿(myoglobulinuria)。電學檢查發現雙側坐骨神經有完全去神經現象(complete denervation)。經過十九個月的復健治療後，電學檢查追蹤可見部份神經再生(reinnervation)。雖然個案在神經學方面有後遺症但是功能上有明顯的進步。如此可見，周邊神經損傷可能造成嚴重的功能喪失，但是復健治療有助於此類病患康復。(台灣復健醫誌 2006；34(1)：33 - 39)

**關鍵詞：**坐骨神經病變(sciatic neuropathy)，昏迷(coma)，復健(rehabilitation)



