

Gluteal Compartment Syndrome Complicated with Bilateral Sciatic Neuropathy

Chih-Yang Liu¹, Tzong-Shi Wang², Hsu-Chao Chang³, Shinn-Kuang Lin^{1,4}



Figure 1. (A) Coronal STIR MR image of lumbar spine shows diffuse high signal intensity of paraspinal and gluteal muscles, predominately on the left side (small arrows). (B) Coronal T1-weighted MR image after intravenous gadolinium administration shows myonecrosis of bilateral adductor muscles (arrows). (C) Transaxial fat-suppressed T1-weighted MR image after intravenous gadolinium administration shows myonecrosis of bilateral adductor and hamstring muscles. Enhancement of bilateral enlarged sciatic nerves is also seen (arrowheads).

A 27 year-old man was admitted for bilateral legs weakness and rhabdomyolysis after a prolonged sitting posture for 10 hours due to an overuse of drugs for schizophrenia. Neurological examination showed feet drop, absent ankle tendon reflexes and hypesthesia to pin-prick stimulation below knees. The straight-leg-raising test was normal. A lumbar spine MRI study did not show abnormal spinal lesion but found diffuse hyperintense change of lower back muscles with predominance on the left side. Serum creatine phosphokinase (CPK) level was 31478 IU/L (normal 39-308 IU/L). The nerve conduction velocity study showed absent responses of bilateral peroneal, tibial and sural nerves and electromyographic study showed active denervation at sampling muscles below the Hamstrings, while sparing the tensor fascia lata. A follow-up pelvic MRI study disclosed necrosis of bilateral gluteal muscles with marginal contrast enhancement (Figure). Bilateral gluteal compartment syndrome (GCS) with

From the ¹Stroke Center and Departments of Neurology, ²Psychiatry, and ³Diagnostic Radiology, Taipei Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation, Taiwan, ⁴School of Medicine, Tzu Chi University, Hualien, Taiwan.
Received March 15, 2014. Revised April 8, 2014.
Accepted August 4, 2014.

Correspondence to: Shinn-Kuang Lin, MD. Department of Neurology, Taipei Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation, Taiwan, No. 289, Jian Guo Road, 231, Xindian district, New Taipei City, Taiwan.
E-mail: jy0428@totalbb.net.tw, sk2022@tzuchi.com.tw

rhabdomyolysis (RM) and entrapment neuropathy of bilateral sciatic nerves were diagnosed. The patient remained feet drop with normal erythrocyte sedimentation rate and CPK level three months later.

Bilateral GCS is an uncommon condition. The clinical features of GCS range from sciatic nerve palsy to massive RM, acute renal failure, multiple organ failure, and death. The most common cause is gluteal muscle compression after prolonged immobilization, either due to substance abuse or trauma. Sciatic nerves can be compressed at femoral neck, posterior thigh⁽¹⁾, piriformis and gluteal muscle region⁽²⁾ from various etiologies⁽³⁾. Prolonged sitting position may damage the sciatic nerve by compressing it against the sciatic notch. The compression may have been worsened by hip flexion, since this tightens the nerve against the sciatic notch. In this case, prolonged sitting position resulted in both entrapment neuropathy of bilateral sciatic nerves and compartment of bilateral gluteal muscles. The GCS might further aggravate the severity of the sciatic neuropathy. MRI of pelvis provides a good imaging study for both GCS and sciatic entrapment syndrome. Care should be needed owing to the potential life-threatening process of multiple organ failure and death.

Acta Neurol Taiwan 2014;23:122-123

ACKNOWLEDGEMENT

The study was supported by a grant from the Taipei Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation (TCRD-TPE-103-RT-16).

REFERENCES

1. Tyrrell PJ, Feher MD, Rossor MN. Sciatic nerve damage due to toilet seat entrapment: another Saturday night palsy. *J Neurol Neurosurg Psychiatry* 1989;52: 1113-1115.
2. Rudolph T, Lokebo JE, Andreasen L. Bilateral gluteal compartment syndrome and severe rhabdomyolysis after lumbar spine surgery. *Eur Spine J* 2011;20 Suppl 2:S180-S182.
3. Plewnia C, Wallace C, Zochodne D. Traumatic sciatic neuropathy: a novel cause, local experience, and a review of the literature. *J Trauma* 1999;47:986-991.