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Case Report

Bilateral gluteal compartment syndrome complicated by rhabdomyolysis and acute kidney injury in a patient with alcohol intoxication

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ABSTRACT

Bilateral gluteal compartment syndrome is a rare clinical entity that can be complicated by rhabdomyolysis or acute kidney injury (AKI). We report the a case of a 30-year-old woman without any comorbid diseases who was diagnosed with bilateral gluteal compartment syndrome complicated by rhabdomyolysis and dialysis-requiring AKI, which was caused by prolonged immobilization under the influence of alcohol. Although the patient's renal function recovered fully after 5 sessions of hemodialysis, sciatic neuropathy caused by gluteal compartment syndrome led to permanent foot drop.

Introduction

Bilateral gluteal compartment syndrome is a rare, but devastating clinical syndrome that results from elevated pressure in confined gluteal space that may complicate rhabdomyolysis and acute kidney injury (AKI). The most common known etiologies leading to gluteal compartment syndrome are crush injury, prolonged immobilization due to substance abuse or surgery [1]. Increased intracompartmental pressure results in ischemic damage to muscles and nerves. Clinical manifestations are characterized by pain, tenderness of involved muscle area and neurological deficit [2]. Surgical decompression is known to be effective within 6 to 12 hours of onset. The presence of underlying diabetes mellitus or hypertension is known to be a predisposing factor in the development of compartment syndrome by further compromising vascular perfusion. We are reporting a case of bilateral gluteal compartment syndrome in a young and healthy woman who was left in prolonged sitting position in drunken status, and complicated by severe rhabdomyolysis and dialysis requiring AKI.

Case report

A 30-year-old woman visited emergency room with symptoms of both lower extremity weakness and myalgia. She had been intoxicated by alcohol and was sleeping in a sitting position when found at the sidewalk by passerby on the day before admission. Approximate duration of immobilization due to sleeping was 16 hours. Since she woke up, she complained of myalgia and paralysis in both lower extremities. Her body mass index was 19.3 kg/m^2, and she had no comorbid diseases as well as no familial medical history. The initial vital signs were body temperature 36.6 °C; pulse rate 78 beats/minute; respiratory rate 20 breaths/min; and blood pressure 130/90 mmHg. Both thighs were swollen and tender. On neuro-

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logical examination, both knee and ankle joints showed decreased motor function (Grade 4/4, 0∼1/0∼1 respectively) with intact sensory function in above damaged lesions. Deep tendon reflexes were absent in both lower extremities (areflexia) and no pathologic reflexes were noted (Babinski, ankle clonus). Laboratory examinations showed white blood cell count 17.2×10^9/L, hemoglobin 135 g/L, platelet count 178×10^{12}/L. Serum creatine phosphokinase 101,960 IU/L, lactic dehydrogenase 5,794 IU/L, myoglobin $>$ 2,000 μg/L, aspartate transaminase 1130 IU/L, and alanine transaminase 517 IU/L. Urinalysis showed dark-colored urine with a pH of 8.0, negative glucose, specific gravity of 1.010 kg/l, 2+protein, 3+occult blood but no red blood cells in microscopy. No myoglobinuria was detected. Although initial blood urea nitrogen (BUN) and creatinine were 330 mg/L and 17 mg/L respectively, her urine output remained $<$ 100 ml/day necessitating initiation of renal replacement therapy, despite fluid resuscitation and furosemide challenge.

An initial chest X-ray showed no pathologic finding. Three-phase bone scan showed increased uptake in gluteal muscles

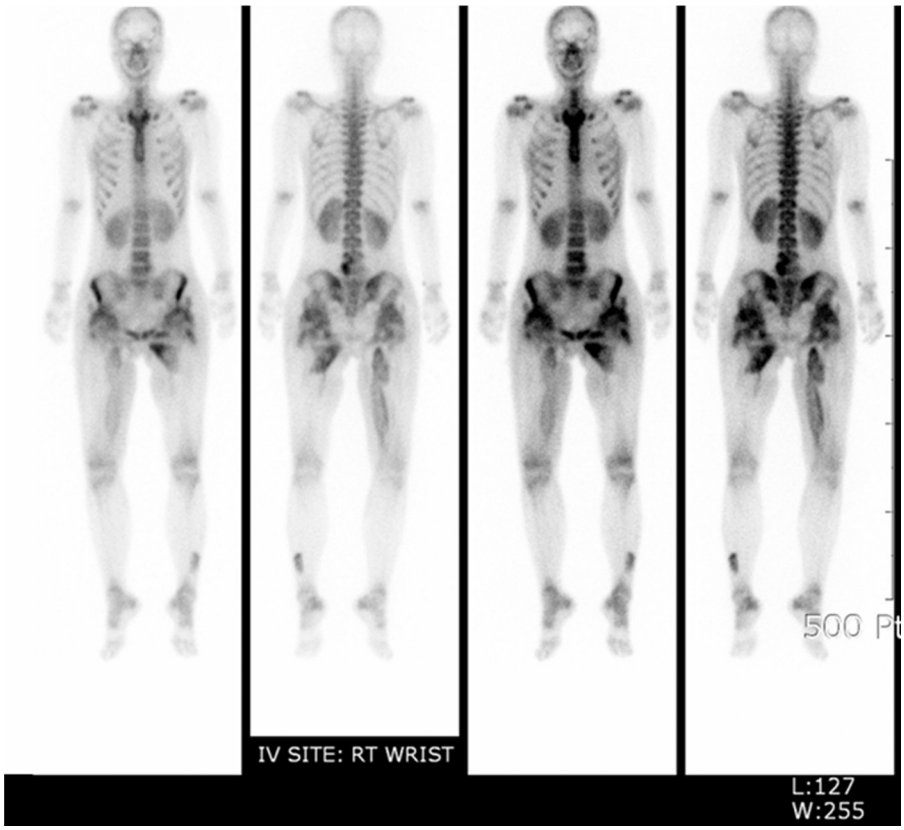

Figure 1. Bone scan showing diffusely increased uptakes in bilateral gluteal muscles, left *piriformis* and right *sartorius* muscle. Tracer retention is noted in both kidneys with diffuse uptake in the soft tissue, indicating rhabdomyolysis-induced AKI. Intense oval-shaped uptake in the left distal tibia is also noted, suggesting post-traumatic change.

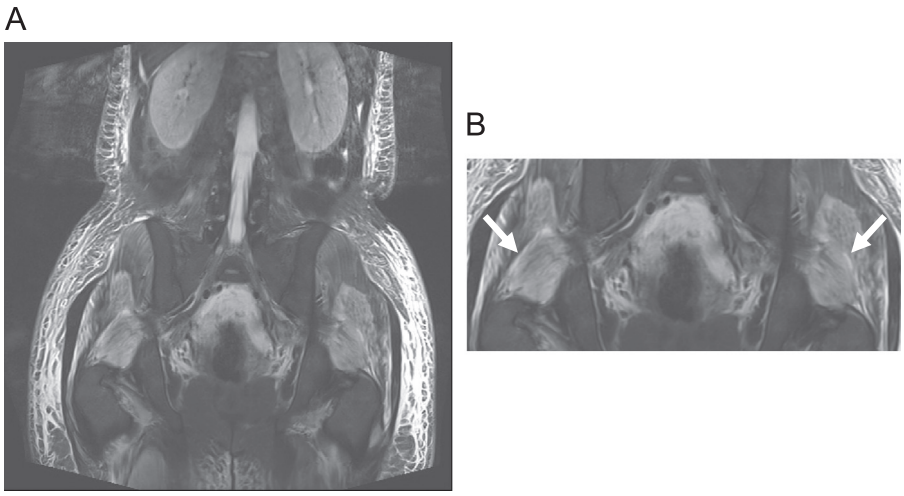

Figure 2. Coronal T2 weighted image of pelvic MRI. High signal intensity of bilateral *piriformis* muscle and *gluteus minor* muscle is noted (white arrows).

(Fig. 1). Pelvic magnetic resonance imaging also revealed increased signal intensities in both *piriformis* muscles and *gluteus minor* muscles, which were thought to result from muscular swelling that cause sciatic nerve compression (Fig. 2). Nerve conduction study demonstrated Wallerian degeneration in sensorimotor fibers of the bilateral sciatic nerves with conduction block in the right sciatic nerve, and myopathy in the bilateral hip girdle muscles. Although consultation to orthopedics was taken, a rehabilitation program instead of operative fasciotomy was initiated because delayed surgical decompression was less likely to be helpful for recovery of sciatic neuropathy. Hemodialysis was stopped after 5 dialysis sessions and renal function remained stable with a serum creatinine concentration of 9.0 mg/L. She was transferred to the rehabilitary medicine department for long-term rehabilitation and discharged with a condition of independent ambulatory state. However, during admission and since, during a follow-up period of 19 months, bilateral foot drop became a permanent sequela that required ankle-foot orthosis to make ambulation possible.

Discussion

Although gluteal compartment syndrome in our case is thought to have resulted mainly from prolonged immobilization under the influence of alcohol and increased pressure in confined space subsequently caused ischemic damage to gluteal muscles and sciatic nerves, alcohol-induced direct toxicity to muscles is also thought to contribute to the occurrence of rhabdomyolysis [3,4]. Rhabdomyolysis in compartment syndrome occurs as elevated compartment pressure causes damage to muscles. This results in release of osmotically active agents into the interstitial space of fascial compartments, ultimately leading to tissue necrosis [5]. Following the diagnosis of impending or true compartment syndrome, immediate measurement of compartment pressure is known to be necessary to ensure that the deleterious sequelae of compartment syndrome do not occur. Measured compartment pressure above 30 mmHg is an absolute indicator for fasciotomy. However, it is known that the initial presence of neurological deficit or prolonged duration of clinical evidence of compartment syndrome with a suspected duration of 8 hours or longer, usually exclude the viability of involved nerves or muscles either due to multiple injuries or severe tissue ischemia. In such cases, fasciotomy is not usually beneficial. Instead, the involved limb should be aggressively splinted to maintain functional positions as the muscles are prone to undergo fibrosis and contracture [2]. In our case, the patient was admitted after an interval of approximately 16 hours and initially presented bilateral foot drop as a consequence of sciatic neuropathy, that lead to the conclusion that measuring compartment pressure was not needed because of suspected duration of over 8 hours of evidence of compartment syndrome and presence of a neurological deficit, so a noninvasive, rehabilitary program was chosen rather than surgical decompression.

Gluteal compartment syndrome complicated by severe rhabdomyolysis and nerve injury is known to be associated with the diverse comorbid diseases such as diabetes mellitus or hypertension [6,7]. The presence of vasculopathy in diabetes or hypertension is expected to further compromise vascular perfusion in compartment syndrome [8]. However, our patient was a healthy young woman without any comorbid diseases, and prolonged immobilization in a sitting position and alcohol intoxication are thought to be factors leading to bilateral gluteal compartment syndrome. Although increase in pressure in the gluteal space due to prolonged sitting is thought to be an initiating event leading to sciatic nerve injury and rhabdomyolysis, there is also a possibility that concomitant occurrence of rhabomyolysis in gluteal muscles secondary to alcohol also contributes to further increase in pressure in the gluteal space, aggravating injury [9]. In addition, alcohol-induced volume depletion in our patient is thought to play an important role in the development of myoglobinuric AKI, requiring renal replacement therapy [10–12].

We report a case of alcohol-induced bilateral gluteal compartment syndrome complicated by sciatic neuropathy, rhabdomyolysis, and AKI leading to permanent gait disturbance. In an alcohol-intoxicated patient presenting with muscle tenderness following a period of immobilization, it is important to consider compartment syndrome immediately, as it requires early fasciotomy to reduce the risk of permanent damage to nerves.

Conflict of interest

There is no conflict of interest.

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