

CASE REPORT

Thigh compartment syndrome complicated by sciatic nerve palsy, rhabdomyolysis, and acute renal failure

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Introduction

A compartment syndrome is a well-known complication that could develop secondary to shin or forearm injuries; however, its manifestation in the thigh region remains unusual [1]. Thigh Compartment syndrome (TCS) is a severe condition that arises due to elevated pressures within the three compartments of the thigh fascia [2]. There are several causes of TCS such as blunt traumatic injuries (with or without bone fracture), frank bleeding into the myofascial spaces, and vascular injuries accompanying ischemic reperfusion [3–5]. Moreover, earlier studies have suggested a relationship between the development of TCS and massive deep vein thrombosis [6], prolonged use of tourniquet [7], closed intramedullary nailing of femur fracture, use of anti-shock trousers, and exercise-related contusions [8]. It has been observed that age, polytrauma, femoral fracture, and time to fasciotomy are primary determinants of the functional outcomes in patients who have TCS [9]. Though, the underlying mechanisms of TCS are well described, the occurrence and outcomes of patients with TCS complicated by rhabdomyolysis and sciatic nerve palsy are not well known due to the rarity of such combination. Herein, we reported an unusual case of thigh compartment syndrome

Key Clinical Message

We reported a rare case of thigh compartment syndrome (TCS) complicated by sciatic nerve palsy, rhabdomyolysis, and acute renal failure in an alcoholic patient. Intensive care measures and immediate posteromedial decompressive fasciotomy were performed. These timely interventions resulted in improvement of the nerve injury and restoration of the kidney function.

Keywords

Alcohol abuse, rhabdomyolysis, sciatic nerve palsy, thigh compartment syndrome.

complicated by sciatic nerve palsy, rhabdomyolysis, and acute renal failure in a patient with alcohol abuse.

Case Report

A 29-year-old Thai male patient presented to the emergency department with fever, agitation, and confusion after an alcoholic binge. Due to language barrier, the exact details and consequences of the incident were not easily extractable from the patient. The physical examination revealed a swelling over medial and posteromedial aspect of the left thigh. This swelling was tense, tender, and painful. The distal pulse was intact and a weakness of foot dorsiflexion was observed. On admission, the laboratory investigations revealed high blood level of alcohol (3 mmol/L), white blood cell count ($29.1 \times 10^3/\mu\text{L}$), C-reactive protein (39 mg/L), serum lactate (5.73 mmol/L), myoglobin (59885 ng/mL), urea nitrogen (16.6 mmol/L), creatinine (430 $\mu\text{mol/L}$), and creatine kinase (4100 U/L). The patient was found to have an acute renal failure and ketoacidosis.

The patient was admitted to the medical Intensive care unit (MICU) and immediately started on hemodialysis therapy in addition to other ICU supportive measures.

Initially, the differential diagnosis included necrotizing fasciitis and selective compartment syndrome resulted from rhabdomyolysis. The Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score was 7 based on C-reactive protein (39 mg/L), total white blood cell count ($29.1 \times 10^3/\mu\text{L}$), hemoglobin (15 g/dL), serum sodium (122 mmol/L), creatinine (430 $\mu\text{mol/L}$), and glucose (16.4 mmol/L). It has been suggested that LRINEC score ≥ 6 as an indicator of significant necrotizing fasciitis [10]. In agreement with our hospital policy, the general surgeon dealt with the necrotizing fasciitis with early and appropriate antibiotic therapy and surgical debridement to prevent delayed complications of necrotizing fasciitis.

Lower limb magnetic resonance imaging (MRI) study with multisequence and contrast was performed using a 1.5 Tesla MRI scanner (Siemens, Germany) and the sequence of MRI was STIR image. MRI showed significant muscles edema involving the gluteus muscles bilaterally but more to the left side, and the muscles edema extended distally and became more extensive and more aggressive at the left upper adductors muscles group. It also involved the hamstring muscles with significant edema involving the muscular fascia, subcutaneous edema, and swelling of the entire left thigh with subtle heterogeneity, mainly involving the adductor muscle group, most probably denoting early degeneration. The quadriceps muscles showed mild degree of edema. The common femoral, popliteal, and posterior tibial arteries were patent bilaterally with no evidence of occlusion of the main veins of the legs. The underlying bones of both lower limbs were grossly unremarkable and there was no evidence of gross collection. Therefore, pathology mainly involved the left thigh, which was most probably represented the compartment syndrome with significant fasciitis and subcutaneous edema with early degeneration involving the upper adductor muscles (Fig. 1).

The clinical examination revealed tense and firm posteromedial compartment, which indicated the need for surgical decompression. The medial and posteromedial compartments were released through a medial approach as this was the tensest site of the thigh which was found under pressure, but looked otherwise healthy and popped-up from the wound extension (Fig. 2). So, the release of the sciatic nerve compression was achieved and the wound was left open (covered by sterile dressing only) for secondary look. The patient stayed in the MICU for 1 month until his renal function improved and weaned off the hemodialysis. The fasciotomy wound was repaired by partial skin graft, general body exercise was routinely performed, and the leg was supported by ankle-foot orthosis. The patient was discharged from the hospital with a regular outpatient clinic follow-up. The wound

was completely healed and the foot drop got improved with a dorsiflexion grade 3 on the second follow-up visit.

Discussion

This is an unusual case report, which demonstrates that sciatic nerve palsy could happen in patient with TCS associated with alcohol-induced rhabdomyolysis.

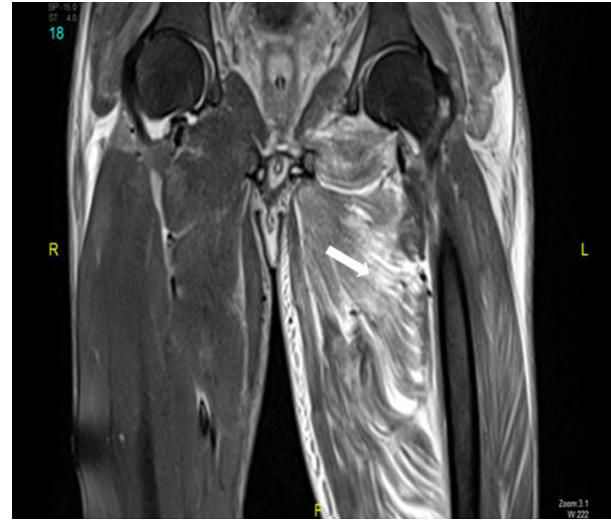


Figure 1. Magnetic resonance imaging (MRI) study was performed with multisequence and contrast study of the lower limb on a 1.5 Tesla MRI scanner (Siemens, Germany) and the sequence of MRI was STIR image. It showed pathology involving mainly the left thigh, which is most probably represented the compartment syndrome with significant fasciitis and subcutaneous edema with early degeneration involving the upper adductor muscles. High signal intensity of left thigh muscles secondary to edema with compartment syndrome (white arrow).

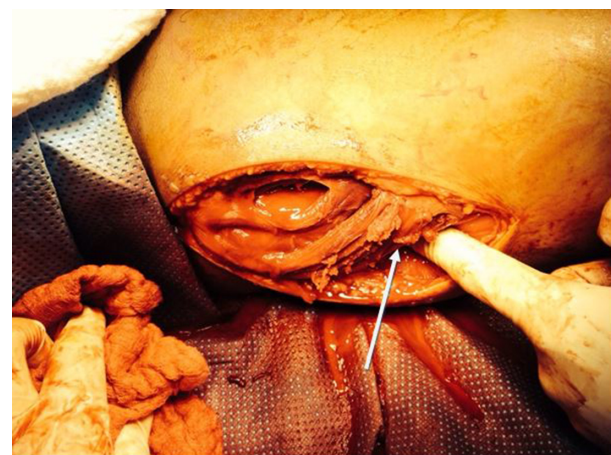


Figure 2. posteromedial release of the adductor group; the white arrow point to sciatic nerve deep in the wound.

Currently, there are few case series in the literature, which highlight the clinical presentation and outcome of acute compartment syndrome of the thigh [11, 12]. Generally, a compartment syndrome in a limb is characterized by enhanced tissue pressure within the closed fascial spaces. Moreover, delayed or missed diagnosis could result in severe limb ischemia and damage to the surrounding nerves and muscles. Therefore, prompt diagnosis and surgical decompression is crucial to avoid the potential irreversible muscular ischemia and nerve palsy [12]. Furthermore, the outcomes of acute TCS depend upon the underlying etiology and time to surgical intervention. Blunt trauma and vascular injuries are the most frequently associated mechanisms of TCS [11–13]. Earlier case reports identified other predisposing factors such as positional ischemia, crush injury, thigh contusion, aggressive resuscitation in the trauma setting, aneurysm, joint replacement, and fracture [2]. Kuklo *et al.* [14] reported bilateral gluteal, thigh, and leg compartment syndrome with severe rhabdomyolysis following physical exertion. The present case developed acute TCS, massive rhabdomyolysis, and acute renal failure.

A high index of suspicion is needed to diagnose acute TCS, as it is a rare complication of thigh injury [11]. The diagnosis of compartment syndrome is usually based on clinical examination along with the measurement of intracompartmental pressure [15]. Early diagnosis is important to prevent permanent damage to muscles and nerves. Pain and tenderness of the affected area and neurological deficit of the sciatic nerve are important clinical manifestations of TCS [15]. Consistent with manifestations of other compartment syndromes, TCS is also presented with extreme pain at rest and pain on stretching muscles [16]. Kanlic *et al.* [2] identified tissue edema with palpable tenderness of thigh to be the most persistent physical finding that raises the suspicion of TCS. Holbein *et al.* [17] reported a case of polytrauma who developed TCS with sciatic nerve palsy after intramedullary nailing of femur fracture. Cho *et al.* [18] described bilateral compartment syndrome of the gluteal region with rhabdomyolysis and acute kidney injury secondary to alcohol intoxication. These authors suggested that prolonged sitting under the influence of alcohol could be the provocative of sciatic nerve palsy and rhabdomyolysis. Moreover, alcohol-induced rhabdomyolysis might be responsible for development of acute renal failure secondary to myoglobinuria [19]. Also, it has been speculated that the development of rhabdomyolysis following alcohol binge might contribute in the elevated pressure within the thigh spaces and subsequently substantiate further injury [18].

Since the TCS is atraumatic in our case, it could be explained in part by the prolonged immobilization,

particularly toward the left side. The elevated pressure in the left thigh fascia might eventually lead to damage of thigh muscles and sciatic nerve as well. It has been reported that the development of rhabdomyolysis in compartment syndrome might result from progressive increase in the compartmental pressure causing osmotic changes which lead to tissue necrosis [20]. Moreover, rhabdomyolysis could be explained also in part by alcohol-induced direct toxicity to the muscles.

An earlier case report demonstrated that delayed surgical intervention, mainly fasciotomy, is associated with poor outcomes [16]. Therefore, surgical decompression should be considered immediately after confirming the diagnosis and preferably within the first 12 h [18]. Notably, the surgical approach for thigh fasciotomy varies according to the muscle groups involved [21]. An earlier study suggested that all muscle compartments should be treated by fasciotomy in order to minimize the risk of subsequent ischemic changes following TCS [22]. Riede and colleagues [23] presented a case of acute TCS sustained blunt trauma that was successfully managed conservatively. Therefore, selective nonoperative management could be possible in patients without fracture or vascular damage. To date, there is lack of reports to evaluate the selection criteria and outcome in patients who are managed conservatively versus those who are treated surgically. So, there is a need to look carefully for TCS patients who could be selectively treated nonoperatively [16].

In conclusion, we described a rare case of acute thigh compartment syndrome complicated by sciatic nerve injury, rhabdomyolysis, and acute renal failure in a patient with binge drinking. We suggested that early diagnosis and timely decompressive fasciotomy will minimize the risk of irreversible sciatic nerve damage.

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Conflict of Interest

None declared.

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