Gluteal Compartment Syndrome Due to Prolonged Immobilization after Alcohol Intoxication: A Case Report

Shinichi IIZUKA[*1], Naoyuki MIURA[*1], Tomokazu FUKUSHIMA[*1], Tomoko SEKI[*1], Katuhiko SUGIMOTO[*1] and Sadaki INOKUCHI[*2]

[*1]*Department of Emergency and Critical Care Medicine, Odawara Municipal Hospital*
[*2]*Department of Emergency and Critical Care Medicine, Tokai University School of Medicine*

(Received March 1, 2011; Accepted April 12, 2011)

Gluteal compartment syndrome is a relatively rare condition that mostly result from atraumatic causes such as prolonged immobilization due to drug abuse or alcoholic intoxication and incorrect positioning during surgical procedures rather than traumatic causes. Early diagnosis is difficult and sometimes delayed or overlooked because of poor physical signs resulting from altered mental status and inappropriate diagnosis by clinicians. It has been reported that more than half of the cases of gluteal compartment syndrome are associated with crush syndrome and sciatic nerve palsy. Early diagnosis and immediate fasciotomy are necessary to improve the functional prognosis.
Here, we report the case of a patient with gluteal compartment syndrome caused by prolonged immobilization after acute alcoholic intoxication. After disease onset, the patient developed complications of crush syndrome and sciatic nerve palsy, but immediate fasciotomy improved his condition.

Key words: gluteal compartment syndrome, crush syndrome, prolonged immobilization, alcoholic intoxication, fasciotomy

INTRODUCTION

Compartment syndrome refers to the loss of organ function and perfusion in a limited space, which leads to an increase in internal pressure. When this condition occurs within the osteofascial compartments, it result in muscle ischemia, and a delayed or missed diagnosis lead to crush syndrome due to muscle necrosis [1]. Hargens stated that a fasciotomy should be performed if a pressure exceeding 30 mmHg persists in the osteofascial compartment for 6–8 h [2]. In a case series of limb compartment syndrome, a delayed fasciotomy (over 12 h after onset) was found to worsen the functional outcome [3]. In case of suspected compartment syndrome, early diagnosis and immediate fasciotomy are necessary to improve the functional outcome.

The compartment syndrome mostly occurs in the lower limbs and forearm after trauma, and it is rarely seen in gluteal lesions. Gluteal compartment syndrome occurs in approximately 0.9% of trauma patients [4] and mostly results from atraumatic causes such as prolonged immobilization due to drug abuse or alcoholic intoxication, in which case, the functional outcome is worse than that when the condition occurs from other causes. This is because the prolonged immobilization after onset leads to an altered mental state [5–8].

CASE REPORT

A 20-year-old man (BMI = 38.8) was found to be lied on his back for more than 6 h after heavy alcohol consumption, and was admitted to our hospital. On admission, he was comatose (Glasgow coma scale [GCS] score = 4 best eye response, E = 1 best verbal response, V = 1, best motor response, M = 2) and was easily intubated, and his vital signs were as follows: respiratory rate, 24 breaths/min; pulse rate, 120 beats/min; blood pressure, 100/55 mmHg; oxygen saturation (SpO_2), 95% under 6 L O_2/min; body temperature, 32°C (rectal temperature). The patient was treated on site for hypothermia by using warming blanket and transfusion, and 2 h later, on admission, his body temperature had improved to 36°C. Initial physical examination showed a gluteal lesion that showed reddening but no swelling and the urine color was dusky-red. Laboratory tests showed that he had metabolic acidosis and myoglobinuria (urinary myoglobin, 150000 ng/mL). Other blood test results were as follows: creatinine posphokinase (CPK), 14140 IU/L (normal level, 35–200 IU/L); lactate dehydrogenase (LDH), 450 IU/L (normal level, 120–220 IU/L); blood alcohol level, 394 mg/dL. His renal function and serum electrolyte levels were normal. He was diagnosed with acute alcohol intoxication, acute rhabdomyolysis, and accidental hypothermia and was subsequently hospitalized in the intensive care unit (ICU).

The rhabdomyolysis was treated with adequate hydration and alkaline dieresis, and a satisfactory urine flow was established. Three hours after hospitalization, the patient recovered consciousness, and was extubated successfully. At 10 h after hospitalization, he complained of a tense erythematous indurated left buttock

Shinichi IIZUKA, Department of Emergency and Critical Care of Medicine Odawara Municipal Hospital, 49 Kuno, Odawara-City, Kanagawa 250-8558, Japan Tel: +81-465-34-3175 E-mail: eriizuka@hotmail.com

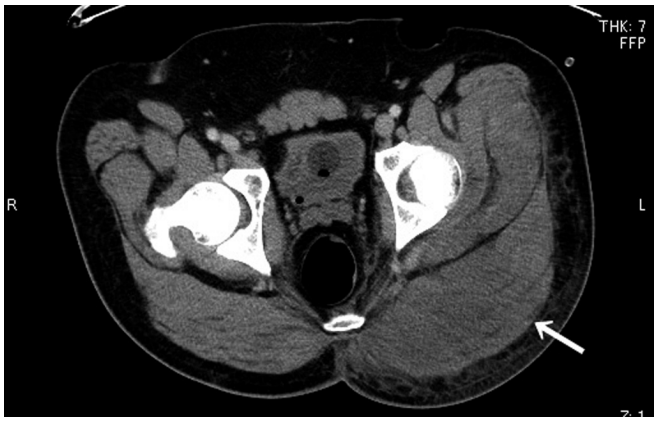

Fig. 1 CT scan of the pelvis showing massive edema of the gluteus maximus and gluteus medius/minimus on the left side (arrow).

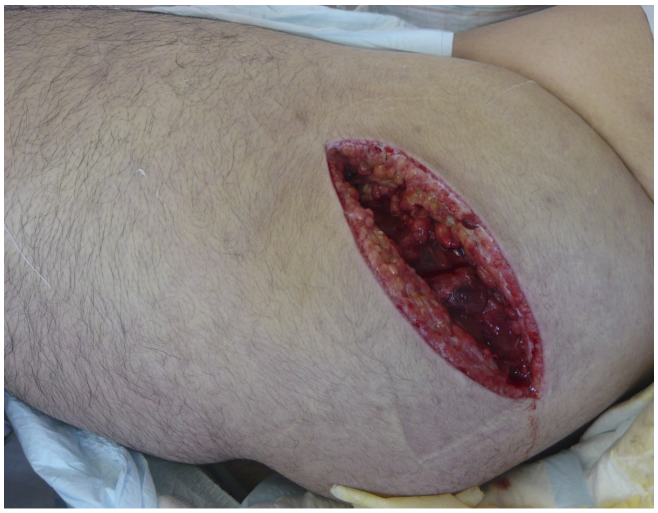

Fig. 2 Fasciotomy of the left gluteal compartment (posterior approach) showing muscle edema of the gluteal compartment.

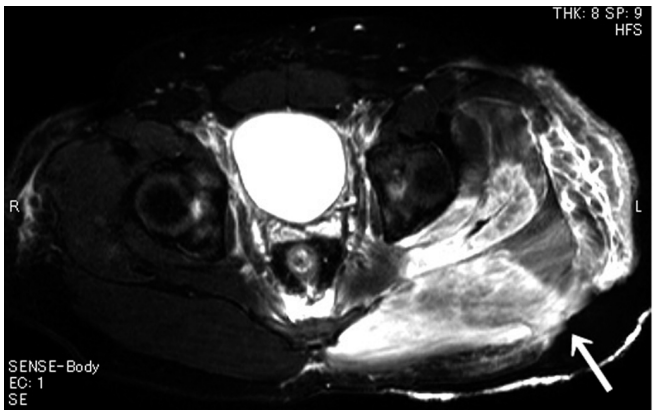

Fig. 3 MR image of the pelvis on the 5th d after the operation showing persistent muscle edema of the gluteus maximus and gluteus medius/minimus and no intramuscular abscess (arrow).

with severe tenderness, paresthesia of the left big and second toes, and decreased sensation on the left lateral foot to pinprick and light touch. On examination, we found that his serum CPK levels were elevated to 71000 IU/L. We started him on conservative management however, 18 h later, physical examination showed decreased motor power of MRC grade 2/5 in the left tibialis and extensor hallucis longus muscle. We suspected a clinical diagnosis of left gluteal compartment syndrome with complications of rabdomyolysis and left sciatic nerve palsy. The intra compartmental pressure in the left gluteal lesion had increased to 37 mmHg (right gluteal lesion, 0 mmHg). In addition, computed tomography revealed diffuse swelling of the left glutus maximus and medius (Fig. 1). Hence, left gluteal compartment syndrome was definitively diagnosed, and fasciotomy of the left gluteal lesion was immediately performed. A posterior approach was chosen for the operation on the left buttock (Fig. 2). On decompression of the entire gluteal compartment, we found dusky colored and bulging muscles, however, after a while, the muscle color improved and necrotic muscles were not present. We kept the wound open after the operation and covered it with saline soaked-gauze.

Two days after the operation, the motor power of both the left tibialis and extensor hallucis longus muscle improved to MRC grade 5/5, and the paresthesia was relieved. On the 4th after the operation, the patient could walk using crutches; however, the very next day, his body temperature increased to 39°C. Magnetic resonance image (MRI) was conducted to check for abscess formation in the left gluteal muscle. The MR image showed edematous changes in the muscles but no abcesses (Fig. 3). Gram-negative rods were detected

in the wound and blood cultures, and the patient was therefore diagnosed with wound sepsis and treated with 13.5 g/d of tazobactum/piperacilin (PIPC/TAZ). A day later, we detected pseudomonas aeruginosa consequently, we continue a treatment with 13.5 g/d of PIPC/TAZ. On the 12th d after fasciotomy, we closed the wound. The patient was discharged on the 14th d. During follow-up a mounth later, we observed that the wound was healing and the sciatic nerve palsy was completely cured. The patient could become ambulatory without using crutches.

DISCUSSION

Three separate non-distensible compartments in gluteal lesions: (1) The gluteus maximus, which is the main extensor and external rotator of the leg, is covered on its superficial and deep side by a fibrous fascia. (2) The gluteus medius runs deep and superolateral to the gluteus maximus, and is separated from the gluteus maximus by a deep gluteal fascia, and superficially confined to the gluteus minimus by aponeurosis. (3) Finally, the smallest compartment consists solely of the tensor fascia lata, which is the lateral aspect of the gluteus medius and minimus compartments. The 3 compartments are covered with non-distensible gluteal fascia and aponeurosis, which confine them to a limited space [5, 9]. Damage to the muscles in these compartments because of ischemia caused by prolonged compression results in edema with traumatic contusion and posttraumatic hematoma formation, an increase in the internal pressure and subsequently, developed of compartment and crush syndromes.

Gluteal compartment syndromes mainly result from atraumatic causes such as prolonged immobilization due to drug abuse or alcoholic intoxication and incorrect surgical position, and the cause are very rarely traumatic, such as blunt trauma. In case of drug abuse, gluteal compartment syndrome often occurs due to prolonged immobilization in the prone, lateral or sitting positions [7], This syndrome also complicates other compartment syndromes such as those in the thighs [8] and lower limbs [5]. Gluteal compartment syndrome comprises 22% of all compartment syndromes caused by prolonged immobilization drug abuse [10]. Gluteal compartment syndromes caused by incorrect surgical positions often results in complication after surgery such as hip and knee arthroplasty [11, 12] or urological surgery [13, 14], with the patient in the prone or lateral decubitus or dorsal lithotomy positions [11–14]. The risk factors for such complications include obesity, prolonged operative time, and epidural anesthesia [11, 12]. Posttraumatic gluteal compartment syndrome develops because of edema with traumatic contusions [7, 9, 15], crush injuries [15], and hematoma formation due to blunt superior or inferior gluteal artery injuries [15, 16] in all compartments of the gluteal lesion.

For early diagnosis of gluteal compartment syndrome, this condition should be initially considered on the basis of the pathogenic mechanisms and clinical symptoms observed. Severe pain, tenseness, and swelling in or around the affected buttock are the most frequent symptoms of gluteal compartment syndrome. Aditionally, tenderness, hardness, skin abnormality, and pain on the passive motion in or around the affected buttocks are other findings. In the later stage of the condition, the patient may demonstrate sciatic nerve dysfunctions, such as deficits in the leg, thigh, and foot strength or pain in the posterior thighs and legs and lateral legs and feet. On testing with a dipstick, the urine may be tea-colored or positive for blood without the presence of any red cells, representing myoglobinuria. In addition, a palpable peripheral pulse is also observed in the affected limb.

In many previous instance, gluteal compartment syndrome has been misdiagnosed as venous thrombosis in the affected limb because of similar clinical symptoms of these conditions [6]. Measurment of the compartmental pressure, laboratory evaluations, and imaging tests are useful in the differential diagnosis of gluteal compartment syndrome.

Normal levels of gluteal compartment pressure are 13 mmHg in the gluteus maximus and 14 mmHg in the gluteus medius muscles. Previously, gluteal compartment syndrome has often been diagnosed on the basis of a compartment pressure exceeding 30 mmHg [5, 7–9]. However, sometimes, it may be difficult to diagnose this condition by examining only the focal symptoms in an equivocal case in which the patient has impaired consciousness. In such cases, measurements of the internal pressure in the affected gluteal compartment is helpful for early diagnosis [5]. On the other hand, when the clinical symptoms are consistent with compartment syndrome but the pressure is less than 30 mmHg, further examination is recommended depending on the patient's condition [8].

The MR image and computed tomography (CT) scan delineate the extent of muscle edema, necrosis, and hematoma formation in the affected gluteal compartments [13, 16, 17]. Some authors advocate the use of these image techniques as they are useful examination tools in the diagnosis of gluteal compartment syndrome [5, 7]. Futher in our case as well, the preoperative pelvic CT scan and MR image clearly showed the muscle edema and damaged area, respectively. The findings of the CT scan, in particular, played an important role in the diagnosis of left gluteal compartment syndrome.

Crush syndromes refers to the condition that results when rhabdomyolysis and multiple organ dysfunctions are accompanied by compartment syndrome or crush injuries [1]. Owing to the large muscle mass, gluteal compartment syndromes are usually accompanied by the crush syndrome [5–9, 12–14, 17]. In cases of atraumatic gluteal compartment syndromes specifically, measurement of CPK and electrolyte levels, renal function examination, and electrocardiography should be conducted to rule out crush syndrome [10]. Early and vigorous fluid resuscitation followed by mannitol-alkaline diuresis prevents acute renal failure in crush victims [18]. In a few cases of gluteal compartment syndrome, temporary hemodialysis was needed for acute renal failure following rhabdomyolysis [5–8]. Via a case series of bariatric surgery, Boatanjian reported that 3 patients died from acute renal failure following rhabdomyolysis due to gluteal muscle myonecrosis [19].

Neurologic findings that appeared to be caused by sciatic nerve palsy have been reported in more

than half of the patients with gluteal compartment syndrome. In cases where sciatic nerve palsy developed before fasciotomy, most patients suffered from persistent gait deficit or impaired sensation for a few months or a few years [6–9, 11, 14, 15]. This persistent sciatic nerve palsy commonly occurs when the diagnosis of gluteal compartment syndrome is delayed. The sciatic nerve does not lie within the fascial envelope of any muscle except in instances in which it perforates the piriformis. Since the nerve lies between the pelvis-external rotator complex and the deep surface of the gluteus maximus, the nerve and its supply may be vulnerable to compression by swelling in these muscles [9]. In a few other case reports, the patients recovered without residual disability because fasciotomy was performed in the early phase of sciatic nerve palsy following gluteal compartment syndromes [7, 12], as in our case.

The sciatic nerve is a large nerve fiber derived from the L4–S3 nerve roots. It arises from the lumbosacral plexus and is composed of 2 distinct trunks: the lateral (peroneal division) and the medial (tibial division). These divisions lie next to each other in the mid-thigh to the distal-thigh region and form the common peroneal and tibial nerves [20]. A complete sciatic nerve lesion can cause sciatic distribution pain and paresthesia; difficulty with knee flexion; and a flail foot with loss of dorsiflexion, plantarflexion, invertors, and evertors. It can also cause sensory loss in the posterior thigh, lower lateral leg, and the entire foot [21]. However, some studies have shown that the peroneal division has greater susceptibility to sciatic nerve palsy than the tibial division; therefore, sciatic nerve palsy can lead to symptoms, such as foot drop, that mimic those of peroneal nerve palsy [22]. When physical examination and clinical history are not conclusive, electrodiagnostic evaluation may help in locating the nerve injury [20].

Typically, gluteal compartment syndromes is treated with immediate fasciotomy, but via the case series of atraumatic gluteal compartment syndrome with crush syndrome and sciatic nerve palsy, Lachiewicz reported that 2 of 6 patients showed improvements with conservative management, and even in the 2-year follow-up, they had no residual disability from sciatic nerve palsy. However, in other case reports of conservative management, residual disability from sciatic nerve palsy or muscle atrophy was found to persist for long [6, 12, 14]. Conservative management for gluteal compartment syndrome with crush syndrome and sciatic nerve palsy is highly controversial. The therapeutic policy for gluteal compartment syndromes is essentially fasciotomy in the acute phase after disease onset.

CONCLUSION

Early diagnosis of gluteal compartment syndrome is required to avoid the severe ensuing metabolic and physical deficits. When this pathological condition is suspected from clinical symptoms and pathogenic mechanisms of patients, further examination, including laboratory tests, image tests and measurement of compartment pressure, should be conducted, and fasciotomy should be performed as soon as possible.

REFERENCE

1) Mubarak S, Owen CA. Compartmental syndrome and its relation to the crush syndrome: a spectrum of disease. Clin Orthop 1975; 113: 81–89.
2) Hargens AR, Romine JS, Sipe JC, Evans KL, Mubarak SJ, Akeson WH. Peripheral nerve-conduction Block by High Muscle-Compartment Pressure. J Bine Joint Surg Am. 1979; 61-A No.2: 192–200.
3) Sheridan GW, Masten FA, Fasciotomy in the Treatment of the acute compartment syndrome. J Bone Joint Surg [Am] 1976; 58-A: 112–115.
4) Ritenour AE. Dorlac WC, Fang R, Woods T, Jenkins DH, Flaherty SF, *et al*. Complications after fasciotomy revision and delayed compartment release in combat patients. J Trauma. 2008; 64 (2): 153–162.
5) Bleicher RJ, Sherman HF, Latenser BA. Bilateral gluteal compartment syndrome. J Trauma. 1997; 42(1): 118–122.
6) Hynes JE, Jackson A. Atraumatic gluteal compartment syndrome. Postgrad Med J. 1994; 70: 210–212.
7) Yoshioka H. Gluteal compartment syndrome. Acta Orthop Scand. 1992; 63(3): 347–349.
8) Schmalzried TP, Neal WC, Eckardt JJ. Gluteal compartment and crush syndrome. Clin Orthop Relat Res. 1992; 277: 161–165.
9) Owen CA, Woody PR, Mubarak SJ, Hrgens AR. Gluteal compartment syndrome. Clin Orthop Relat Res. 1978; 132: 57–60.
10) Franc-Law JM, Rossignol M, Vernec A, Somogyi D, Shrier I. Poisoning-induced acute atraumatic compartment syndrome. Am J Emerg Med. 2000; 18(5): 616–620.
11) Kumar V, Saeed K, Panagopoulos A, Parker PJ. Gluteal compartment syndrome following joint arthroplasty under epidural anaesthesia: a report of 4 cases. J Orthop Surg. 2007; 15(1): 113–117.
12) Lachiewicz PF, Latimer HA. Rhabdomyolysis following total hip arthroplasty. J Bone Joint Surg. 1991; 73-B: 576–579.
13) Kneene R, Froelich JM, Milbrandt JC, Idusuyi OB. Bilateral gluteal compartment syndrome following robotic-assisted prostatectomy. Orthopedics. 2010; 33(11): 852.
14) Rommel FM, Kabler RL, Mowad JJ. The crush syndrome: a complication of urological surgery. J Urol. 1986; 135(4): 809–811.
15) Bosch U, Tscherne H. The pelvic compartment syndrome. Arch Orthop Trauma Surg. 1992; 111: 314–317.
16) Gee MJ, Tolat A, Sinha J. Acute gluteal and thigh compartment syndrome following pelvic fracture and superior gluteal artery bleeding. Eur J Trauma Emerg Surg. 2007; 33: 188–191.
17) Mustafa NM, Hyun A, Kumar JS, Yekkirala L. Gluteal compartment syndrome: a case report. Case J. 2009; 10(2): 190.
18) Gunal AI, Celiker H, Dogukan A, Ozalp G, Kirciman E, Simsekli H, *et al*. Early and vigorous fluid resuscitation prevents acute renal failure in the crush victims of catastrophic earthquakes. J Am Soc Nephrol. 2004; 15: 1862–1867.
19) Bostanjian D, Anthone GJ, Hamoui N, Crookes PF. Rhabdomyolysis of gluteal muscles leading to renal failure: a potentially fatal complication of surgery in the morbidly obese. Obes Surg. 2003; 13: 302–305.
20) Feinberg J, Sethi S. Sciatic neuropathy: Case report and discussion of the literature on postoperative sciatic neuropathy and sciatic nerve tumors. HSS J. 2006; 2(2): 181–187.
21) Baker GS, Parsons WR, Welch JS. Endometriosis within the sheath of the sciatic nerve. Report of two patients with progressive paralysis. J Neurology. 1966; 25(6): 652–655.
22) Yuen EC, So YT. Sciatic neuropathy. Neurol Clin. 1999; 17(3); 617–631.