CASE REPORT
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A Case of Rhabdomyolysis Associated with Use of a Pneumatic Tourniquet during Arthroscopic Knee Surgery

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The common causes of rhabdomyolysis include trauma, hypoxia, drugs, toxins, infections and hyperthermia. Operative insults, including direct trauma and ischemia, have the potential to cause the development of rhabdomyolysis. Pneumatic tourniquets used during arthroscopic knee surgery to prevent blood loss have led to many complications such as nerve paralysis and vascular injuries. Rhabdomyolysis can also be caused by prolonged pneumatic tourniquet application without a midapplication release, and also from an increased application pressure, but the actual incidence of this is low. In order to prevent rhabdomyolysis, the clinicians must be aware of such risks and follow strict guidelines for the application time, the midapplication release and also the inflation pressure. Vigorous hydration and postoperative patient surveillance are helpful to prevent rhabdomyolysis. We have recently experienced a case of rhabdomyolysis after the arthroscopic knee surgery, and the rhabdomyolysis could have been associated with the use of a pneumatic tourniquet. **(Korean J Intern Med 2010;25:105-109)**

Keywords: Rhabdomyolysis; Tourniquets; Kidney failure, acute

INTRODUCTION

Bywaters and colleagues first reported on pigmented casts in the renal tubules during the time of World War II after performing autopsies on four cadavers with severe crush injuries that induced acute renal failure, and they named this condition as rhabdomyolysis [1]. Rhabdomyolysis is a syndrome that is characterized by the necrosis of myocytes and leakage of myocytic components into the bloodstream; this syndrome encompasses a spectrum of maladies from asymptomatic increases of muscle enzymes to death due to acute renal failure [2].

The causes of rhabdomyolysis can be divided into two groups: traumatic and nontraumatic. The nontraumatic causes include infection, toxin, CO poisoning and drug abuses. The traumatic causes include crush injuries, traffic accidents and surgery [2]. The surgical causes include orthopedic surgery-related trauma such as prone positioning due to direct compression of the rectus femoris muscles [3].

Pneumatic tourniquets have been commonly utilized during arthroscopic knee surgeries for creating a bloodless field and facilitating operative procedures. However, tourniquets could result in complications such as nerve paralysis and vascular injuries [4]. Although rhabdomyolysis could possibly result from the vigorous use of tourniquets, previous reports on tourniquet induced rhabdomyolysis are rare. There have been only three previous reports on the tourniquet-induced rhabdomyolysis [5-7]. Shenton et al. [5] first reported on a case of rhabdomyolysis after total knee replacement. The most recent published case was reported by Sheth et al. [6], in which a college football player who was taking creatinine supplements developed rhabdomyolysis following arthroscopic knee surgery, which was due to ischemia from intra-operative tourniquet application, and the creatinine supplements increased the

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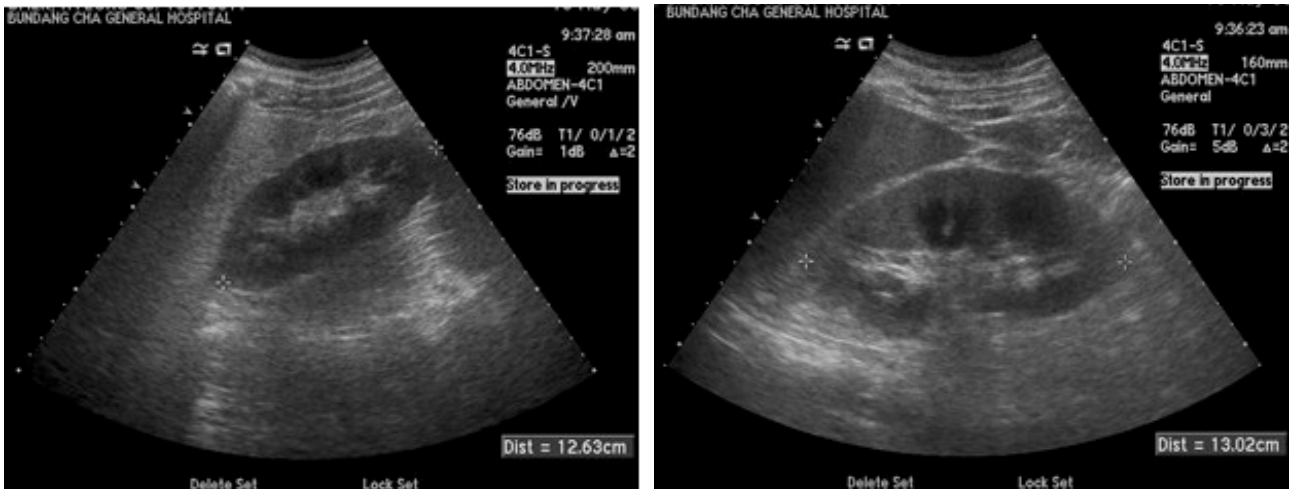

Figure 1. Abdominal ultrasonographic findings. Both kidneys were enlarged (the right kidney 12.6 cm and the left kidney 13.0 cm in length), and their echogenecity increased.

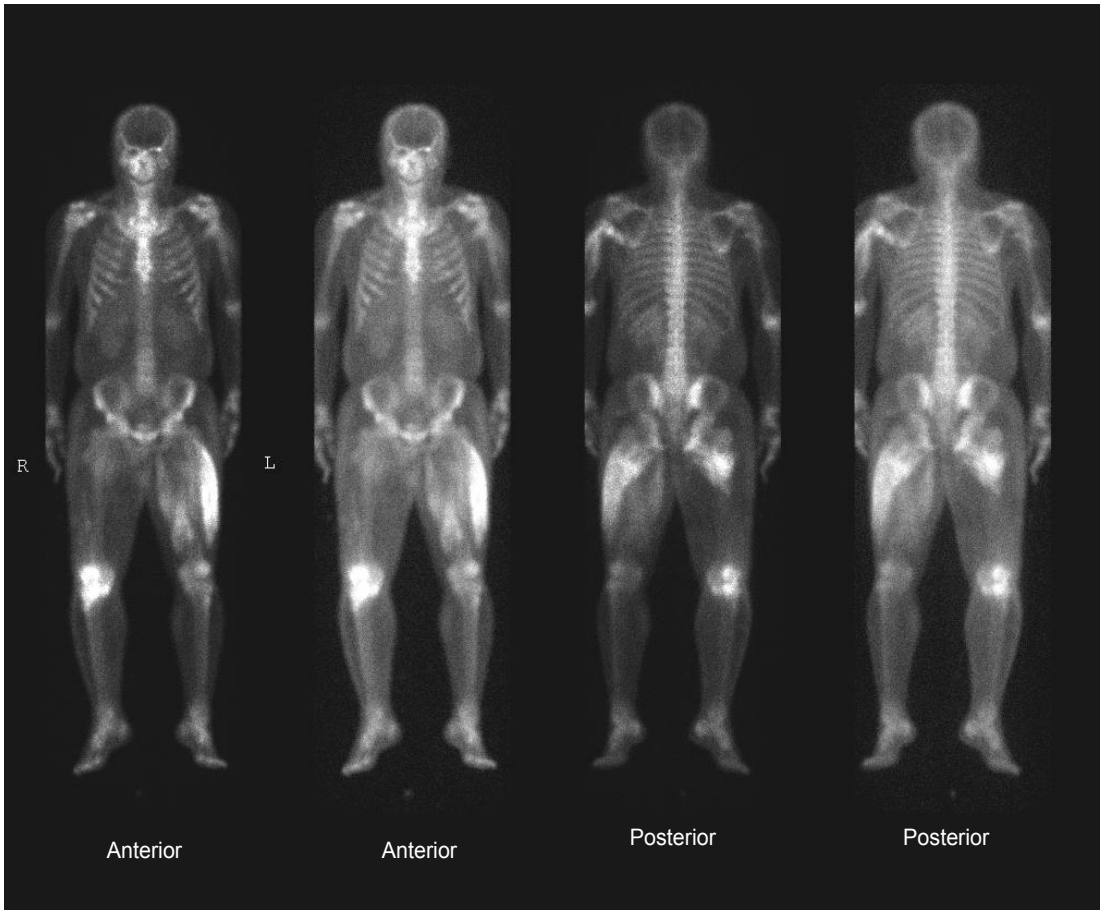

Figure 2. Technetium-99m HDP whole body bone scan. Focal soft tissue uptakes are noted at the right proximal femur area and left thigh area, which is consistent with rhabdomyolysis. HDP, hydroxymethylene diphosphonate.

risk of rhabdomyolysis. We have recently experienced a case of rhabdomyolysis after arthroscopic knee surgery, and this could have been associated with the use of a pneumatic tourniquet. We report here this case and we include a literature review.

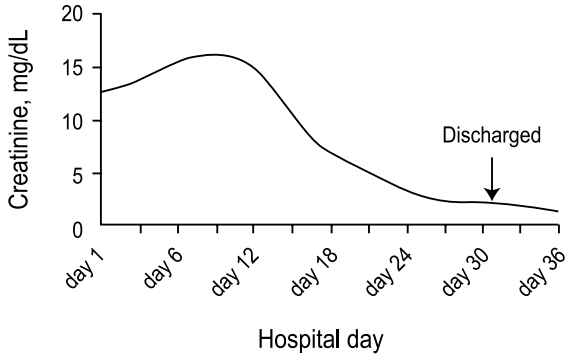

Figure 3. The changes of the serum creatinine level during the hospitalization.

CASE REPORT

A 38 year-old male patient was transferred to our hospital from a local orthopedic clinic due to oliguria, and this occurred 5 days after anterior cruciate ligament reconstruction with a bone-patellar tendon-bone autograft. The total urine output at the time of transfer on post operative day 5 was 300 mL/day. The patient was a current smoker with history of 18 pack-years and he was also was social drinker. He was an office worker. The patient's weight was 83 kg and his height was 168 cm, and the body mass index was 29.4 kg/m^2. Other than being diagnosed with a fatty liver, his medical history was unremarkable; he had never been previously admitted to a hospital and never underwent any kind of surgery until this time. The patient completely tore his right anterior cruciate ligament while playing soccer one day prior to the surgery. At the time of injury, the patient visited the local orthopedic surgery clinic and decided to undergo arthroscopic repair. The arthroscopic surgery procedure included general anesthesia with the patient in the supine position, and utilization of a pneumatic tourniquet to prevent bleeding. There were no significant events such as shock, prolonged surgery and excessive bleeding during the surgery. After the surgery, the patient was in a generally good condition with stable vital signs and no evidence of bleeding, and he was without any other possible signs of postoperative complication. The patient received ceftriaxone and Non-steroidal anti-inflammatory drugs for 4 days before the transfer, but he did not receive any aminoglycoside. Except for the pain at the operative site, the patient did not have any other complaints.

On postoperative day 2, the serum creatinine level increased to 1.8 mg/dL, and when it rose to 10.9 mg/dL on postoperative day 4, the patient was then transferred to our hospital. At the time of admission to our hospital, the patient's blood pressure was 150/100 mmHg, the pulse rate was 85/min and the body temperature 36.7˚C. A blood sample was taken upon arrival. The serum creatinine level was 12.7 mg/dL and the cystatin C was 5.41 mg/L with the glomerular filtration rate being 18 mL/min/1.73 m^2. The serum creatinine kinase level was 42,820 U/L, the aspartate aminotransferase was 755 IU/L and the serum lactate dehydrogenase level was 1,521 U/L; the serum calcium, phosphorus and uric acid levels were 8.5, 5.8 and 13.5 mg/dL, respectively. Urinalysis included a pH of 5.0, a SG of 1.025, proteinuria of 3 + and microscopic hematuria of 3 to 5 red blood cells. The urine myoglobin tested negative, but the serum myoglobin level was greater than 3,000 ng/mL. Abdominal ultrasonography showed bilaterally enlarged kidneys with increased echogenecity (Fig. 1); a severe fatty liver, gall bladder edema and pelvic ascites were also revealed. The Tc-99m hydroxymethylene diphosphonate whole body bone scan revealed focal soft tissue uptakes at the right proximal femur area and left thigh area, which was consistent with rhabdomyolysis (Fig. 2). Thus, the patient was diagnosed with rhabdomyolysis induced acute renal failure. The patient underwent nine sessions of hemodialysis, and on the thirty-second hospital day his serum creatinine level reached 1.7 mg/dL and the urine output was 2,000 mL/day. He was then discharged from the hospital and scheduled for outpatient follow-ups (Fig. 3).

DISCUSSION

Rhabdomyolysis is a syndrome that's characterized by the necrosis of myocytes and intravasation of cellular materials. The manifestations of rhabdomyolysis range from asymptomatic elevation of the muscle enzymes to life threatening electrolyte imbalance and acute renal failure. Injuries to skeletal muscle lead to the secretion of cellular materials into the blood vessels and this damages the membrane bound Na-K ATPase, which then alters the Na-Ca exchange, and so this activates neutral protease and eventually the destruction of myocytes [2].

The pathogenesis of acute renal failure in the rhabdomyolysis includes the tubular obstruction and cytotoxicity caused by the myoglobin and uric acid, the renal ischemia that's due to fluid shifting to the injured muscles, inhibited production of endothelium-derived relaxing factor by the

myoglobin, which results in vasoconstriction, and the production of toxic free radicals by the metabolites of myoglobin [2].

The clinical features of rhabdomyolysis are nonspecific. The local and systemic features include muscle pain, tenderness and swelling, malaise and tea-colored urine. The typical clinical signs include increased creatinine phosphokinase, pigmenturia and myoglobinuria. However, myoglobinuria had been overlooked in many previous case reports since any myoglobin was usually excreted before the patients could reach the hospital, leading to the result of elevated creatinine kinase without myoglobinuria.

The complications of rhabdomyolysis can be classified as early and late. The early complications include hyperkalemia that could result in cardiac arrhythmia and cardiac arrest, hypocalcemia potentiated by the phosphate released from the lysed muscle cells, and hepatic inflammation caused by the protease released from injured muscle. Late complications such as acute renal failure and disseminated intravascular coagulation are apparent at 12 to 24 hours after the initial injury. Acute renal failure is the most serious complication due to its high morbidity and mortality, and the prevalence of acute renal failure in patients with rhabdomyolysis can reach 15% [2]. Another serious complication is compartment syndrome, and this is caused by direct muscle injury or vigorous muscle activity. A delay of more than six hours in diagnosing compartment syndrome may lead to irreversible muscle damage or death [8]. Recognizing the severe complications and instituting adequate treatment are of the utmost priority for managing rhabdomyolysis.

Pneumatic tourniquets have been utilized in orthopedic surgery to reduce blood loss during surgical procedures and to facilitate the operative procedure. Likewise, arthroscopic surgery has also utilized pneumatic tourniquets for achieving a bloodless operative field. However, the use of pneumatic tourniquets has resulted in many complications such as vascular injury, nerve paralysis, postoperative swelling and delay in regaining muscle power. These complications are thought to be the consequences of the tourniquet's direct pressure on the nerve and vessels. Cardiac and respiratory complications such as acute pulmonary edema and cardiac arrest can happen following the release of tourniquets [4].

The cases of tourniquet-induced rhabdomyolysis have usually been associated with total knee replacements and arthroscopic knee repair [5-7]. There also was a case of rhabdomyolysis after applying a tourniquet on a hypothyroidism patient during anterior cruciate ligament repair, and the rhabdomyolysis was probably caused by the interaction of hypothyroidism and precipitating factors [9]. The most recent case in the literature was reported by Sheth et al. [6], in which a college football player who was taking creatinine supplements developed rhabdomyolysis following arthroscopic knee surgery. Sheth et al. [6] argued that the rhabdomyolysis was due to ischemia from the intraoperative tourniquet application, and the creatinine supplements much increased the risk. The mechanism of muscle injury in a previous case of tourniquet induced-rhabdomyolysis was suggested to be ischemia that resulted from vascular occlusion, since the skeletal muscles are more prone to the ischemic injuries. In this case, the possibility that the hypotension was the cause of ischemia could be ruled out because the patient had been hemodynamically stable throughout the surgery.

Many factors such as general health, age and the presence of peripheral vascular disease all have an influence on the safe tourniquet time. However, the optimal tourniquet time and pressure have currently not been determined. The minimal pressure necessary to maintain a bloodless operative field is currently used. The safest tourniquet application time is less than one hour. There are reports of minimal tissue damage with 180 minutes of tourniquet application with a 5 minute midapplication release time, implying that such an application time/procedure could be safe [10]. The application time was more than 4 hours for the reported cases of tourniquet-induced rhabdomyolysis; the tourniquets were applied for 4.5 hours continuously in one case and for 4 hours, twenty-one minutes of total inflation time with a midapplication release time in another case [5]. When the application pressure is too high, such as 520 millimeters of mercury in one case, the rhabdomyolysis could result even though the application time was only forty-five minutes [5]. Thus, in order to prevent rhabdomyolysis from tourniquet use, the clinicians must abide to the optimal time and pressure for applying a tourniquet and vigorous hydration is also necessary. Recognizing other risk factors that may precipitate rhabdomyolysis is also quite important. Performing keen postoperative surveillance to detect problems in the at-risk patients is very important to prevent the often severe complications of rhabdomyolysis.

Conflict of interest

No potential conflict of interest relevant to this article was reported.

REFERENCES

1. Bywaters EG, Beall D. Crush injuries with impairment of renal function: 1941. J Am Soc Nephrol 1998;9:322-332.
2. Kim HY, Choi SO, Shin SJ, et al. Analysis of 250 cases of rhabdomyolysis. Korean J Nephrol 1994;13:810-817.
3. Prabhu M, Samra S. An unusual cause of rhabdomyolysis following surgery in the prone position. J Neurosurg Anesthesiol 2000;12: 359-363.
4. Murphy CG, Winter DC, Bouchier-Hayes DJ. Tourniquet injuries : pathogenesis and modalities for attenuation. Acta Orthop Belg 2005; 71:635-645.
5. Shenton DW, Spitzer SA, Mulrennan BM. Tourniquet-induced rhabdomyolysis: a case report. J Bone Joint Surg Am 1990;72: 1405-1406.
6. Sheth NP, Sennett B, Berns JS. Rhabdomyolysis and acute renal failure following arthroscopic knee surgery in a college football player taking creatine supplements. Clin Nephrol 2006;65:134-137.
7. Palmer SH, Graham G. Tourniquet-induced rhabdomyolysis after total knee replacement. Ann R Coll Surg Engl 1994;76:416-417.
8. Schwartz JT Jr, Brumback RJ, Lakatos R, Poka A, Bathon GH, Burgess AR. Acute compartment syndrome of the thigh: a spectrum of injury. J Bone Joint Surg Am 1989;71:392-400.
9. Vold PL, Weiss PJ. Rhabdomyolysis from tourniquet trauma in a patient with hypothyroidism. West J Med 1995;162:270-271.
10. Sapega AA, Heppenstall RB, Chance B, Park YS, Sokolow D. Optimizing tourniquet application and release times in extremity surgery: a biochemical and ultrastructural study. J Bone Joint Surg Am 1985;67:303-314.