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Bilateral Gluteal Compartment Syndrome Following Right Total Knee Revision: A Case Report

Kristie D. Osteen, MD, Shireen H. Haque, MD[1]

Department of Anesthesiology, Ochsner Clinic Foundation, New Orleans, LA

ABSTRACT

Background: Gluteal compartment syndrome is a rare occurrence traditionally found in settings of extended immobilization. Thrombolytics and medications with myositis as a potential side effect have also been implicated in a few isolated cases of spontaneous compartment syndrome. Early signs are pain on passive stretching and pain out of proportion to physical examination findings. Failure to recognize and definitively treat compartment syndrome within the first 24 to 36 hours can lead to permanent limb loss and morbidity from a host of systemic complications such as hyperkalemia, renal failure, and sepsis.

Case Report: We report a case of bilateral gluteal compartment syndrome in a 52-year-old patient following a right total knee revision. On postoperative day 2, physical examination after the patient became agitated and in severe distress from bilateral buttock pain showed that the right and left gluteal regions were tense, hard, and erythematous. Creatinine phosphokinase and liver function tests were significantly elevated. Following emergency fasciotomy, physicians thoroughly reviewed the operative course, medication history, and imaging studies. We withdrew simvastatin, a medication associated with spontaneous compartment syndrome, from our patient's daily medications. By day of discharge, both creatinine phosphokinase and liver function problems were decreasing, and the gluteal pain had significantly resolved. The etiology of bilateral gluteal compartment syndrome in our patient could have been a combination of intraoperative length and positioning with simvastatin-induced myositis. Obesity presented an additional risk factor.

Conclusion: This case highlights the importance of identifying patients at increased risk of compartment syndrome in the preoperative assessment and following them with more intensive intraoperative and postoperative monitoring.

Address correspondence to
Kristie D. Osteen, MD
Department of Anesthesiology
Ochsner Clinic Foundation
1514 Jefferson Hwy.
New Orleans, LA 70121
Tel: (504) 842-3755
Fax: (504) 842-2036
Email: kosteen@ochsner.org

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[1]Dr Haque is now with Southeast Anesthesiology Consultants, Charlotte, NC.

INTRODUCTION

Compartment syndrome can be described as increased pressure within a closed osteofascial compartment that leads to neurovascular compromise. Factors associated with compartment syndrome include prolonged immobility, trauma, burns, and substance abuse.[1-10] In orthopedic procedures, deliberate hypotension, positioning, excessive traction, obesity, and prolonged operative time increase the risk of developing compartment syndrome.[2,7] In areas with an adequate vascular supply, such as the gluteal region, compartment syndrome is rare. To date, the literature includes only 44 cases of gluteal compartment syndrome.[10] Our case describes bilateral gluteal compartment syndrome following a right total knee revision.

CASE REPORT

An obese 52-year-old male was admitted for a right total knee revision secondary to increased pain and immobility. The original knee replacement surgery was without complications. Preoperative laboratory values were within normal limits. The patient had well-controlled hypertension and hyperlipidemia that had been treated over the prior 3 years with a combined regimen of valsartan, hydrochlorothiazide, and simvastatin.

Surgical anesthesia involved a combined spinal epidural placed using sterile technique. Using a loss-of-resistance technique, we introduced a 17-gauge, 3.5-inch Tuohy needle midline to the epidural space with the patient in the sitting position. A 25 g Whitacre needle was then directed to the subarachnoid space, and 15 mg of 0.75% bupivacaine was injected. A single attempt was made for the spinal anesthesia,

Table. Laboratory Values Before and After Fasciotomy

Date	CPK (U/L)	LFT		Cultures
		AST	ALT	Blood, Gluteal Tissue, Right Knee
10/21 prefasciotomy	30,000			No growth
10/22 postfasciotomy	12,462			
10/23		236	110	No growth
10/24	6,031	239	131	
10/26	2,539	150	113	No growth

ALT, alanine aminotransferase; AST, aspartase transaminase; CPK, creatinine phosphokinase; LFT, liver function test.

and the procedure was completed without complications. The epidural space was flushed with saline and a 20-gauge epidural catheter was guided through the Tuohy needle. After a negative test dose through the epidural catheter, the surgery began when the patient achieved sensory block to T12. The tourniquet time during the case was 109 minutes, and the total operative time (200 minutes) was prolonged secondary to scar tissue.

After the knee revision, pain was controlled with an epidural infusion of fentanyl 2 mcg/mL and ropivacaine 0.2% at a rate of 6 cc per hour with a patient-controlled bolus of 4 cc every 12 minutes. The visual analogue scale (VAS) pain score on arrival was 0/10. Nine hours after the procedure (postoperative day 1, 1:34 am), the patient reported bilateral lower extremity numbness. After examining the patient, the acute pain service decreased the epidural infusion to 5 cc per hour. On the morning of postoperative day 1 (8:15 am), nursing staff noted redness on the left buttock with minimal blanching. Over the course of the day, the patient again developed numbness, more prominent on the left leg. The epidural catheter was withdrawn 1 cm, and the infusion held for 30 minutes. The numbness resolved, and the epidural was restarted at 5 cc per hour. Three hours following the second episode of numbness, the patient reported 7/10 bilateral lower extremity pain; the nurse advised him to continue to push the button for additional medication. On postoperative day 2 during early morning rounds, the patient appeared comfortable, and the VAS score was 0/10. Mid-morning, the acute pain service was called because the patient's left leg was numb. At this time, the epidural infusion was stopped and the epidural removed per postoperative day 2 protocol.

Approximately 1 hour after epidural removal, the patient appeared agitated, in severe distress, and reported 10/10 pain in the right and left gluteal muscles. The acute pain service was notified. During physical examination, the patient could not tolerate any type of motion, and the gluteal region was tense, erythematous, and hard. A neurological examination could not be performed secondary to extreme pain. Complete blood count, creatinine phosphokinase (CPK), comprehensive metabolic panel, and coagulation studies were performed. Broad-spectrum antibiotic coverage was started with vancomycin and piperacillin/tazobactam, and the patient immediately underwent surgical exploration and fasciotomy of the gluteal area. During the surgical exploration, the gluteal muscle appeared intact with no signs of necrosis or purulent drainage.

Abnormal laboratory values included a significant elevation of CPK and a marginal rise in liver function studies. Of note, the CPK drawn prior to fasciotomy was 30,000 U/L (Table) and subsequently trended toward normal by the day of discharge. The greatest increase in liver function occurred on postoperative days 4 and 5.

After finding isolated case reports in the literature of compartment syndrome associated with the use of simvastatin, we held this medication beginning on postoperative day 4. Magnetic resonance imaging demonstrated subcutaneous edema in the gluteal region with extensive edema in the gluteal muscle bilaterally, the right gluteus medius, and the anterior compartment muscles of the left lower extremity (Figure). The patient had an uneventful course following the fasciotomy and was discharged 5 days later with no neurological deficits or residual pain in the gluteal region.

DISCUSSION

The gluteal area is surrounded by layers of fascia that create 3 independent compartments. The fascia lata runs cephalad from the thigh to form the tensor compartment anteriorly and then divides posteriorly to enclose the gluteus maximus muscle. Between these 2 closed spaces, a third compartment formed from the overlying fascia contains the gluteus medius and minimus.[6,11] Bony structures—including the ilium, sacrum, and femoral head—reinforce the boundaries of these spaces and limit their ability to adjust to changes in compartment volume or pressure.[6-9,11]

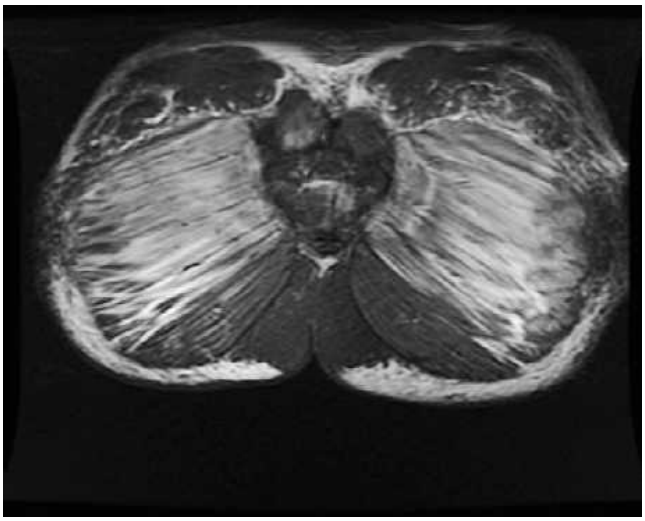

Figure. Magnetic resonance imaging showing extensive edema within the gluteal muscles, specifically the gluteus maximus, the right gluteus medius, and the anterior compartment of the left lower extremity.

The underlying pathophysiology of compartment syndrome includes an initial inciting event that generates an inflammatory response and leads to extravasation of fluid from capillary beds within the muscle. Increased interstitial pressure raises the compartment pressure and causes elevations in the venous pressure. As the venous pressure rises above the capillary perfusion pressure, the capillaries collapse, causing local ischemia, hypoxia, and tissue necrosis.[4-11] Vasoactive agents released in the presence of hypoxia continue the process of vasodilation and capillary leak, thus continuing the rise in compartment pressure. In the face of severe hypoxia, nerve damage begins within an hour and becomes irreversible at 8 hours.[2,11] Variable damage to muscle tissue occurs within 6 hours, with permanent necrosis at 8 hours.

Any factor that increases compartment volume or decreases compartment space has the potential to induce compartment syndrome. Common etiologies in the setting of orthopedic surgery are intraoperative positioning, obesity, excessive traction, prolonged external compression, postoperative bleeding, and inadvertent trauma to soft tissue. The lithotomy and lateral decubitus positions have the highest rates of compartment syndrome.[2,9] Nonoperative causes include crush injuries, insect bites, burns, military antishock trousers, and immobility from intoxication or altered mental status.[1,2,11,12] Patients on any type of anticoagulation regimen are at an increased risk of compartment syndrome after minor injuries. One case report describes a 60-year-old male on warfarin who developed compartment syndrome of the thigh after bumping into a desk.[2]

The most sensitive clinical indicator of compartment syndrome is pain with passive stretching or palpation of the compartment muscles. Pain out of proportion to physical examination, tense edema or erythema, and paresthesias are all associated with compartment syndrome.[3-5,11] The more classically taught pallor, pulselessness, and paralysis are late findings that occur with severe, irreversible ischemia.[9,10] The definitive diagnosis of compartment syndrome rests on physical examination. A patient with clear and unequivocal clinical signs should proceed directly to the operating room for fasciotomy. When the examination is less definitive, physicians should take serial compartment pressure measurements and frequently reexamine the patient. The recommended threshold for fasciotomy is an absolute compartment pressure $\geq$ 30 mmHg or a delta pressure between 20 mmHg and 30 mmHg. Delay in surgical treatment of compartment syndrome can lead to limb loss or other significant morbidity, including hyperkalemia, myoglobinuria, and acute renal failure from muscle necrosis. Mortality in this setting often arises from cardiac arrhythmias, sepsis, and shock.[2,3-7]

The concurrent use of medications that predispose patients to muscle breakdown increases the risk of postoperative compartment syndrome. Although the exact etiology of compartment syndrome in our patient remains unclear, simvastatin may have been a contributing factor. A thorough review of the literature reveals a few isolated case reports of spontaneous compartment syndrome with the use of simvastatin.[12-15] In one case, a patient confused simvastatin with a weight loss medication and took multiple excess doses that ultimately led to rhabdomyolysis with myoglobinuria, acute renal failure, and bilateral lower extremity compartment syndrome.[12] The patient had no other risk factors for compartment syndrome other than the high doses of simvastatin. In a second case, a patient developed left lower extremity compartment syndrome 3 weeks after a percutaneous transluminal coronary angioplasty with stent placement. The dose of simvastatin after stent placement had been increased from 20 mg to 40 mg per day.[15] At the time of fasciotomy, the absence of a hematoma and pus ruled out anticoagulation or infection as the etiology of compartment syndrome. These reported cases highlight the importance of reviewing the medication history of patients with unexplained spontaneous compartment syndrome.

CONCLUSION

The etiology of bilateral gluteal compartment syndrome in our patient could have been a combination of intraoperative length and positioning with simvastatin-induced myositis. Obesity presented an

additional risk factor.[7] Diagnosing and appropriately treating compartment syndrome in postoperative patients should be a team-based, multidisciplinary approach that includes ancillary staff involved in daily patient care. In a patient with epidural analgesia, a sudden increase in pain or a significant change in the pain score should be brought to the attention of the acute pain service.[16-18] Patients at increased risk of developing compartment syndrome should be identified preoperatively and followed for the course of their hospital stay. Risk factors include obesity, diabetes, lithotomy positioning, prolonged intraoperative time, and use of medications that predispose to muscle breakdown. With early identification and treatment, the long-term morbidity associated with compartment syndrome can be avoided. Special attention should be directed at operative position, the appropriate use of traction, and the need for cushioning areas at risk of compression. Increasing awareness of the signs of compartment syndrome with patient care technicians, nursing staff, and physical therapists could also facilitate early diagnosis and treatment.

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