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Acute Compartment Syndrome Due to Ruptured Baker Cyst After Nonsurgical Management of an Anterior Cruciate Ligament Tear

A Case Report

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Acute compartment syndrome is a clinical entity seen most often after extremity trauma. However, acute compartment syndrome can also follow atraumatic bleeding into a closed compartment.[2,19,28,41] This case report describes an occurrence of acute compartment syndrome in the setting of bleeding from a ruptured Baker cyst. Five previous cases of acute compartment syndrome in the setting of a ruptured[12,23,31,38] or dissecting[16] Baker cyst have been reported. This article highlights a sixth case and emphasizes the importance of potential contributing factors such as antithrombotic medications and mechanical factors such as leg curls and venous compression devices.

CASE REPORT

A 49-year-old man was initially evaluated with a 1-month history of a ruptured left ACL. He was treated with activity modification and gradual range of motion restoration. He began working aggressively on hamstring strengthening exercises using prone leg curls with a roller that contacted on the proximal posterior calf. Three weeks later, he came to the clinic with increased left leg pain and swelling. Examination at that time revealed pain with passive toe dorsiflexion (Homans sign). A duplex ultrasound revealed no evidence of deep venous thrombosis (DVT). He was diagnosed with superficial thrombophlebitis. That day, the patient underwent an MRI study, which confirmed the ACL tear in addition to a tear of the posterior horn of the medial meniscus as well as a ruptured Baker cyst. He was treated with rest, elevation, warm compresses, and scheduled doses of ibuprofen. The patient did well for the next 5 days. At that time, he had an acute increase in pain with increased swelling. A repeat ultrasound was negative for DVT. Compartment pressures were measured and ranged between 15 and 20 mm Hg. A diagnosis of possibly evolving acute compartment syndrome was made, and a venous compression device was placed on the foot (Plexipulse, NuTech, San Antonio, Tex) to assist with venous return and decrease swelling. The patient was discharged home with this device. He came to the emergency department 6 hours later with markedly increased swelling and tense compartments.

The patient's medical history was significant for Gilbert syndrome, benign prostatic hypertrophy, and depression. He had no history of coagulation or bleeding problems. Routine medications included fluoxetine (Prozac) 10 mg orally once a day and ibuprofen.

Physical examination revealed a healthy-appearing man in moderate pain. On musculoskeletal examination of the left knee, there were posterior fullness, tense swelling of all lower leg compartments, and increased pain with passive flexion and extension of the left ankle. Dorsalis pedis and posterior tibial artery pulses were palpable. On neurologic examination of the affected extremity, there was slightly decreased sensation to light touch in the deep peroneal nerve distribution. Compartment pressures were as follows: superficial posterior, 78 mm Hg; deep posterior, 69 mm Hg; lateral, 79 mm Hg; and anterior, 78 mm Hg—confirming a diagnosis of acute compartment syndrome.

Radiographs of the left lower extremity were notable for soft tissue swelling. Complete blood count showed a mildly

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elevated white blood cell count, with a normal differential, normal hematocrit, and normal platelets. The patient's bleeding time was elevated to 17.5 minutes, the prothrombin time was 10.8 minutes, and the partial thromboplastin time was 26.3 minutes. The patient was taken emergently to the operating room and underwent 4-compartment fasciotomy. At the time of surgery, the muscle appeared viable and had no evidence of necrosis. After the fascial releases, intramuscular pressures were below 20 mm Hg in all compartments. The wound was carefully inspected, and no active bleeding was noted from either the skin edges or the deeper tissues. The skin edges were reapproximated using vessel loops and staples. The patient was admitted and started on intravenous antibiotics. His pain was markedly improved postoperatively. His numbness also gradually improved during the next several days.

For the first 12 hours postoperatively, the wound remained relatively bloodless. During the next 12 hours, he had a significant increase in bleeding from the wound. A hematology consultation was obtained. During the first 4 days of the admission, the patient had elevation of his bleeding time to greater than 20 minutes and a drop in his hematocrit from 45% to 18.4%. With the recommendation of the hematology service, the patient was treated with transfusions of packed red blood cells and platelets. In addition, his fluoxetine was discontinued. He was started on tranexamic acid, a potent thrombolysis inhibitor.[17] The patient's hematologic status improved with these interventions.

On postoperative day 5, the patient underwent an MR angiogram to rule out an underlying arteriovenous malformation or aneurysm as a possible cause for the initial compartment syndrome. The MR angiogram showed a ruptured Baker cyst and an adjacent hematoma. No arteriovenous malformation or aneurysms were noted.

He was taken back to the operating room on postoperative day 5 and underwent irrigation and debridement of his wounds. A large hematoma was revealed in the lateral compartment wound. This hematoma was debrided and sent for bacteriology culture, which had negative results. A minimal amount of superficial muscle appeared nonviable and was debrided. The underlying muscle appeared normal and was contractile. All wounds were closed.

After complete healing of the skin incisions, the patient returned to a progressive rehabilitation program. He elected not to undergo an ACL reconstruction. He eventually returned to all of his activities, including expert snow skiing and long-distance cycling.

DISCUSSION

Common diagnostic dilemmas involve the distinction between a ruptured Baker cyst and thrombophlebitis, as both syndromes present with extremity swelling, pain, and pain with passive toe dorsiflexion (Homans sign).[7,20] Misdiagnosis of a ruptured Baker cyst as thrombophlebitis may lead to nonindicated anticoagulation with progression to compartment syndrome.[7,12,19,20,31,41] Concurrent popliteal vein thrombosis can also occur along with a ruptured Baker cyst, increasing the diagnostic difficulty.[26] Five previous cases of a ruptured Baker cyst leading to compartment syndrome have been reported in the literature.[12,16,23,31,38]

Acute compartment syndrome is most commonly associated with extremity trauma. However, acute compartment syndromes induced by medication or concurrent disease-induced bleeding have been reported in the literature.[4,12,19,23,28,31,38,41] The medications most commonly implicated are aspirin, warfarin, and heparin.[2,4,19,41] Anouchi et al[2] reported misdiagnosis of rupture of the medial head of the gastrocnemius, "tennis leg," treated with heparin for suspected DVT with progression to compartment syndrome.

Our patient had been treated with the nonsteroidal anti-inflammatory drug (NSAID) ibuprofen. The NSAIDs are frequently used medications with approximately 60 million prescriptions written per year in the United States.[33] The mechanism of action is inhibition of prostaglandin formation from arachidonic acid by inhibition of the enzyme cyclooxygenase (Cox).[33] Two isoenzymes of Cox have been identified, Cox-1 and Cox-2. Cox-1 appears to be a "housekeeping" enzyme in multiple tissues such as the kidney, stomach, and blood.[33] Cox-2 appears to be the isoenzyme primarily responsible for the inflammatory response.[33] The NSAIDs inhibit platelet aggregation by inhibiting platelet production of thromboxane A2, which is required for platelet thrombus formation.[6,22] The NSAIDs such as nabumetone (Relafen) and etodolac (Lodine) have predominant activity at Cox-2. These medications appear to have improved clinical safety profiles with decreased gastric and hematologic complications.[10,22,25,36] In addition, in vitro studies have demonstrated that nabumetone has significantly less inhibition of platelet thromboxane A2 synthesis than do naproxen and indomethacin.[10,22] Newer Cox-2 specific inhibitors, such as celecoxib (Celebrex) and rofecoxib (Vioxx), have higher affinities for Cox-2 activity to provide a greater safety profile relative to older NSAIDs.[8,14,18,27] The patient in this case report was taking ibuprofen, an older NSAID with both anti-inflammatory and antiplatelet effects.[15,34,35]

In addition to ibuprofen, the patient was taking fluoxetine. Fluoxetine (Prozac) is an antidepressant medication that inhibits central nervous system neuronal uptake of serotonin.[21,29,30] It is metabolized through the liver to inactive metabolites, which undergo renal clearance. According to the *Physicians Desk Reference*,[32] increased bleeding time, anemia, ecchymosis, thrombocytopenia, thrombocythemia, petechiae, purpura, and other blood dyscrasias have been reported as rare side effects of fluoxetine. Increased bleeding has been reported in cases of coadministration with warfarin secondary to the inhibition of cytochrome P450.[9,11] However, in the literature, there are multiple reports of bleeding complications and bruising as a result of the use of fluoxetine without concurrent use of antithrombotics.[3,21,30,37,42] In addition, reversible bleeding time prolongation has been reported with fluoxetine use.[21] Despite these reports, no studies have proven fluoxetine causes bleeding dyscrasias. Several small prospective studies have failed to show significant hematologic changes from this drug.[1,5,24] These studies had small sample sizes (≤10 subjects)

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and short follow-up (4 weeks of treatment). Because the functional effect of fluoxetine on inhibition of platelet aggregation is rare, it has been hypothesized that clinical relevance occurs when patients have an underlying congenital or acquired platelet disorder.[21]

Our patient's hematologic system was most likely impaired as a result of treatment with ibuprofen in combination with fluoxetine use.

In addition to the pharmacologic and anatomical etiological factors, another possible contributor to the patient's acute compartment syndrome may have been the pneumatic compression pumps placed on his lower extremities. These compression devices decrease venous stasis and augment fibrinolytic activity.[13,40] There is only 1 case report in the literature of acute compartment syndrome resulting from a malfunctioning pneumatic compression boot.[39] The patient in this case report did not have a malfunctioning pneumatic compression device, but it is theoretically possible that the increased venous return toward the ruptured Baker cyst could have contributed to the increased pressure in the limb and, ultimately, to compartment syndrome.

This case highlights acute compartment syndrome in the setting of nonoperative treatment of an ACL injury. Specifically, the presence of a ruptured Baker cyst in combination with pharmacological and mechanical factors contributed to the development of compartment syndrome in this patient. A systematic approach to rule out DVT must be pursued. The common management of pain in the extremities with NSAIDs must be tempered by the risk of bleeding complications, particularly in patients with synergistic medications and potential underlying platelet abnormalities. If a compartment syndrome is suspected, standard management includes measurement of compartment pressures and emergent fasciotomies. Heightened clinical suspicion must be maintained to identify and prevent this devastating sequence of events.

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