Rhabdomyolysis and Myonecrosis in a Patient in the Lateral Decubitus Position

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Rhabdomyolysis, from muscle necrosis, is known to occur in certain surgical positions, most notably the dorsal lithotomy and knee chest. This complication is often attributed to compromise of venous and arterial blood flow. Several cases of rhabdomyolysis and gluteal myonecrosis have been reported to occur in patients in the lateral decubitus position undergoing hip surgery. These complications have been attributed to the posterior clamp pressing directly on the buttocks instead of the sacrum area and to the anterior pubic clamp being placed against the anterior part of the dependent thigh, causing vascular compromise at the groin.1,2 We report a case of rhabdomyolysis occurring solely from the direct and prolonged pressure of the operating room bed against the gluteal and flank muscles in a patient in the lateral decubitus position.

Case Report

A 29-yr-old, 110-kg man with a long history of recurrent left nephrolithiasis underwent partial left nephrectomy for a large left upper pole calyceal diverticulum with “milk of calcium stones” that had caused recurrent renal colic. The patient had undergone multiple procedures in the past, including open and percutaneous surgery and extracorporeal shock wave lithotripsy, in an attempt to correct this problem at other institutions.

The patient underwent an uneventful induction of anesthesia with midazolam, sodium thiopental, pancuronium, and metocurine. After induction, the patient was placed in a right flexed lateral decubitus position on a padded table with the kidney bolster raised. All pressure points were padded. Anesthesia was maintained with a combination of nitrous oxide, isoflurane, and intravenous fentanyl. The patient had no episodes of hypotension and had a urine output >0.75 ml kg⁻¹ h⁻¹. The patient remained in a lateral position for 7 h during the surgical procedure. The long duration was due to extensive scarring surrounding the kidney. The patient did not complain of flank or hip pain in the recovery room. Biochemical testing was performed on discharge of the patient from the recovery room because of periods of renal ischemia during the partial nephrectomy. The results were remarkable for an increased serum glutamic oxaloacetic transaminase (SGOT) of 95 u/l (normal <35 u/l) and lactic dehydrogenase (LDH) 365 u/l (normal 90–250 u/l). The SGOT and LDH had been noted to be normal on preoperative screening. The patient was given a patient-controlled analgesia morphine pump for postoperative pain control. Two hours after completion of surgery, the patient was noted to have new onset of tea-colored urine.

On postoperative day 1, the patient began to complain of severe right flank and hip pain and numbness. Repeat test values of SGOT and LDH were further increased at 876 u/l and 711 u/l, respectively. Subsequently, creatinine kinase (normal 50–180 u/l) and aldolase (normal 1–10 u/l) were found to be markedly increased at 28,522 u/l and 660 u/l, respectively, and myoglobin was present in the urine. Serum potassium, calcium, and phosphorous remained within normal limits.

A vascular surgeon was consulted to evaluate the patient for possible gluteal muscle compartment syndrome. The right hip was noted to be indurated. The patient had normal pulses and capillary refill in both lower extremities. Duplex ultrasound of the right leg was negative for deep venous thrombosis or hematoma. Compartment pressures were not measured. The patient was treated with intravenous hydration, sodium bicarbonate, and mannitol, keeping urine output >100 ml·h⁻¹ with urine pH >7.0. By postoperative day 5, the urine myoglobin was negative. The patient had no evidence of renal damage from the rhabdomyolysis.

Three weeks after surgery, the patient was evaluated by a neurologist for persistent numbness and pain over his right flank, iliac crest, and upper lateral portion of his thigh. The neurologic examination was remarkable for weakness in abduction of the right hip and decreased sensation to pinprick and cold over the right lower flank and lateral iliac crest. Electromyogram results were consistent with a
right superior gluteal mononeuropathy and evidence of injury to the right L3–S1 paraspinal muscles from pressure necrosis during surgery.

The patient continued to undergo physical therapy. On follow-up examination 4 months after surgery, the patient’s pain had resolved, but he continued to complain of right flank numbness. However, his examination showed marked improvement in abduction of his right hip strength.

Discussion

Few cases of rhabdomyolysis occurring in the gluteal muscles in patients undergoing surgery while in the lateral decubitus position have been reported. Dahlberg and Howard reported rhabdomyolysis and renal failure occurring from necrosis of the dependent gluteal muscles in a patient in the lateral decubitus position for 4.5 h after pyelolithotomy and partial nephrectomy.\(^3\) Rommel et al. described a gluteal compartment syndrome occurring in a patient undergoing pyelolithotomy and dismembered pyeloplasty while in the flexed lateral decubitus position for 8 h.\(^4\)

Direct, prolonged pressure on the dependent gluteal and flank muscles was the etiology of rhabdomyolysis and superior gluteal nerve damage in our case. Direct external pressure decreases compartment size, resulting in rising tissue pressure.\(^5\) Soft padding is estimated to decrease dependent leg compartment pressures only 16% compared to hard surfaces.\(^6\) Thus, adequate padding will not necessarily prevent pressure necrosis. Whitesides et al. estimated that muscle ischemia develops when tissue pressures are within 10–30 mmHg of the diastolic pressure.\(^7\) Thus, hypotensive anesthesia increases the risk of myonecrosis from direct pressure.

Increased capillary permeability within the muscle bed occurs after 3 h of ischemia.\(^8\) Muscle necrosis and myoglobinuria may begin to occur after 4 h of muscle ischemia.\(^6,8\) However, myonecrosis, rhabdomyolysis, and compartment syndrome often do not occur until after surgery.\(^3\) The ischemic muscle bed undergoes reperfusion of a normal blood supply during the immediate postoperative period. This reperfusion leads to egress of fluid from the damaged, leaking capillary bed into the tissues, causing muscle edema and a further elevated compartment pressure, which promotes a vicious cycle of worsening muscle ischemia.\(^3\) This reperfusion phenomenon was seen with our patient 2 h postoperatively, when the patient’s urine was noted to change from yellow to tea color. The patient had no complaints of pain initially in the recovery room or that evening but began to complain of hip and flank pain on the dependent side the next morning.

In addition to the length of surgery, the only other risk factor the patient had for pressure necrosis was increased body mass. Our patient was a muscular 110-kg man. We believe the patient’s large muscle mass decreased the compliance of his gluteal muscle compartment and thus his ability to compensate for increased compartment pressure from direct pressure or muscle edema. Flexion of the table at the iliac crest and the use of the kidney bolster likely added to increased pressure over the dependent flank and hip areas. However, this position was needed for adequate access for the partial nephrectomy.

In conclusion, the operating team must recognize that patients placed in the lateral flexed position are at risk for these complications, especially when the duration of the procedure exceeds 4 h. Such measures as limiting the extent of kidney bolster and flexion to those critical times needed for optimal access to the kidney may decrease the severity of compression of the dependent muscle group. Also, the anesthesiologist should ensure that the operating room table is well padded and avoid prolonged hypotensive anesthetic technique. Nevertheless, these precautions will not necessarily prevent pressure myonecrosis as seen in our patient. Furthermore, we do not believe that monitoring the dependent limb pulse with pulse oximetry would necessarily alert the anesthesiologist to the onset of direct pressure muscle necrosis. Rhabdomyolysis and compartment syndromes often occur with normal distal pulses and capillary refill in the affected extremity.\(^5\) This patient’s rhabdomyolysis developed not from vascular compromise but from direct pressure of the affected muscle compartment. The perioperative physician needs to have a high index of suspicion for the onset of myonecrosis and rhabdomyolysis after any prolonged surgery in patients in the lateral decubitus position.

References

CASE REPORTS


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Rebound Hypertension and Acute Withdrawal Associated with Discontinuation of an Infusion of Epidural Clonidine


A small percentage of patients with cancer pain suffer from refractory pain despite aggressive therapy.1 Intra-spinal administration of opioids and local anesthetic agents may be helpful in such settings, but their use may be limited by side effects such as motor block and hemodynamic instability.2–5 Clonidine is a centrally acting α2-adrenergic agonist with established analgesic effects6,5 and has synergistic effects with spinal opioids6,7 and spinal local anesthetics.8,9 Epidural clonidine produces analgesia by a spinal mechanism in patients after surgery and in those with cancer pain,10 and it appears to be an effective treatment for severe cancer pain in patients for whom other treatments are ineffective.11

Although the risk of acute withdrawal and rebound hypertension is well recognized with sudden cessation of systemically administered clonidine,12 no such reports exist with regard to epidurally administered clonidine. We describe a case of acute withdrawal and rebound hypertension after abrupt cessation of epidural clonidine in a patient with intractable cancer pain.

Case Report

A 49-yr-old man was diagnosed with metastatic adenocarcinoma of the pancreas. The patient was normotensive and did not have a history of alcohol or other drug abuse. Despite chemotherapy with 5-fluorouracil and gemcitabine, increased tumor growth resulted in complaints of upper abdominal pain radiating through to the back. Pain management was further complicated by complaints of diffuse abdominal pain and intermittent constipation associated with longstanding irritable bowel syndrome. Treatment with escalating doses of sustained-release and immediate-release morphine failed to control his pain and exacerbated his constipation. A neurolytic retrocrural celiac plexus block with 40 ml of 100% anhydrous alcohol produced only minimal relief of the pain. A temporary thoracic epidural catheter was inserted at the T8–T9 interspace, and a combination of 0.125% bupivacaine and 40 μg/ml morphine administered at 12 ml/h resulted in adequate pain control. One week later, a subcutaneously tunnelled thoracic epidural catheter was inserted. The patient’s pain was successfully managed at home with this regimen for 6 weeks. Because of complaints of increasing diffuse abdominal pain and postural hypotension, the patient was readmitted for evaluation and pain control.

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