CASE REPORTS


Rhabdomyolysis after Abdominal Surgery in the Hyperlordotic Position Enforced by Pneumatic Support

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ENFORCED patient positioning in surgery may lead to serious complications such as rhabdomyolysis, peripheral nerve palsy, or other tissue damage.1–5 We report a patient who developed rhabdomyolysis and renal insufficiency after 11 h in the hyperlordotic supine position.

Case Report

A 61-yr-old, 164-cm, 65-kg male was scheduled for a pancreato- duodenostomy for pancreatic carcinoma. He had been healthy and had no history of muscle disease, renal disease, or hypertension. He was taking no medications.

Upon admission, physical examination was unremarkable, but high levels of serum glutamic-oxaloacetic transaminase (150 U/l), serum glutamic-pyruvic transaminase (364 U/l), serum y-glutamyl transpeptidase (249 U/l), and alkaline phosphatase (485 U/l) were noted. Creatinine kinase level was 168 U/l.

The patient received 10 mg of diazepam for sedation. After general anesthesia was induced with thiopental, fentanyl, and vecuronium, maintenance was conducted with sevoflurane, nitrous oxide, and vecuronium. An epidural catheter was inserted at the level of Th12/L1. Bupivacaine 0.125% was intermittently administered throughout surgery.

After the induction of anesthesia, the inflatable bladder of a blood pressure cuff was placed under the patient's back at the level of the lower thoracolumbar spine. The cuff was inflated to obtain the desired hyperlordotic position for surgical access. Surgery was performed with the patient in the supine position with the cuff inflated continuously for 11 h. Throughout the procedure, systolic blood pressure was maintained at approximately 100–120 mmHg. The blood loss of 1,300 ml was replaced with albumin and crystalloid solutions. The procedure was complicated by an accidental right renal artery disruption, which was successfully reconstructed in 45 min. After this accident, to maintain blood pressure and urine output, in addition to hydration, dopamine 2–8 μg·kg⁻¹·min⁻¹ was started until the end of surgery. The

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ward the end of surgery and procedure. Epidural morphine was administered as a 3-mg bolus toward the end of surgery and 2.4 mg/day continuously for postoperative pain relief. The initial recovery from anesthesia was uneventful. However, shortly after the patient was taken to the postanesthesia care unit, he complained of severe back pain such that he could not stay in a single position.

On postoperative day 1, he continued to complain of severe back pain despite adequate analgesia of his wound pain with continuous epidural morphine. The laboratory data showed increased levels of serum glutamic-oxaloacetic transaminase (441 U/l), serum glutamic-pyruvic transaminase (425 U/l), lactate dehydrogenase (3,544 U/l), and creatinine kinase (13,409 U/l). Myocardial infarction was suspected, but electrocardiogram showed no abnormalities, and the isozyme of creatinine kinase was of muscle origin. His urine color had changed to brown. For his back pain, 5 ml of 0.5% bupivacaine was infused into the epidural catheter intermittently. On postoperative day 2, redness and small blisters were noticed on his lower back where the pneumatic device had been placed. He felt irritating pain in this area and tenderness in his bilateral paravertebral areas, which were swollen. The dermal alterations on his back spread wider during the following week. Serum myoglobin level was high (8,560 ng/ml) on postoperative day 2, and serum creatinine and blood urea nitrogen levels increased gradually up to 5.0 mg/dl and 68.5 mg/dl, respectively, in 7 days. Measurement of urine myoglobin level was not available in our hospital. Normal saline and loop diuretics were administered for 1 week to keep urine output more than 1.5 l/day for myoglobinemia. Fortunately, the patient recovered from renal insufficiency without hemodialysis. On postoperative day 5, an orthopedic surgeon was consulted for the patient’s back pain. Myofascial inflammation of the paravertebral muscles was suspected. Computed tomography scanning was performed on postoperative day 8. There was decreased attenuation of the bilateral paravertebral muscles, indicating muscle necrosis (fig. 1).

The patient’s back pain gradually ceased, and the laboratory data returned to normal range in 34 days. The skin alterations resulted in increased pigmentation with a rectangular configuration corresponding to the shape of the cuff. The patient was discharged without another complication.

Discussion

Perioperative rhabdomyolysis may be caused by malignant hyperthermia, preoperative intensive muscular exercise, or compression of musculature leading to muscle ischemia, but the former two are highly unlikely in this case because the patient did not develop any other signs of malignant hyperthermia and did not perform excessive physical exercise preoperatively. In addition, as reported by Harwood and Nelson, creatinine kinase levels after intraoperative pressure muscle ischemia were much lower (<75,000 U/l) than those observed in our patient.

Creatinine kinase of skeletal muscle origin and its elevated level (13,409 U/l), along with the computed tomography scan findings, strongly suggest paravertebral muscle cell destruction as the source of this patient’s severe pain and myoglobinemia. Therefore, it is reasonable to think that the paravertebral muscles were injured by some mechanism, and, as a result, a massive amount of myoglobin was released into the blood from the injured muscle cells. Subsequently, increased serum myoglobin levels may have destructed the renal tubules, causing renal dysfunction. The accidental injury to the right renal artery may have contributed to renal dysfunction. However, this is unlikely because the patient’s preoperative renal function was normal, and the renal artery was reconstructed in 45 min. Even if the right kidney had become nonfunctioning, the left kidney would have compensated for it.

In this case, the skin changes were in a dermal region that corresponded to the place under which the pneumatic support was placed. The patient was forced to hold a hyperlordotic position by this device. As a result, the weight of his body centered on the blood pressure cuff. The skin, subcutaneous tissue, and paravertebral muscles apparently received high pressure produced by the cuff and his own body weight, which disturbed the circulation to his back. Poor blood supply and low perfusion pressure for almost 11 h probably made the skin and paravertebral muscles ischemic. The paravertebral muscles must have become edematous and developed the compartment syndrome.

Many cases of surgical positioning-related rhabdomyolysis have been reported. Although this complication is most often reported for patients who undergo procedures while in a lithotomy position, it also has been reported for patients who undergo procedures while in a supine position. In the reports by Bertland et al. and Bukowski et al. long-lasting surgery and hyperlordosis

Fig. 1. Computed tomography scan of lumbar muscles showing hypodensities of bilateral paravertebral muscles. The arrows indicate lumbar muscle damage and necrosis.
posture possibly contributed to the genesis. Owen et al. reported that direct pressure to muscle could lead to compartment syndrome. The fascia, which lacks elasticity, limits the enlargement of the edematous muscle, resulting in a further increase in intramuscular pressure even after removal of the compression. Finally, the muscle may become necrotic because of deteriorated perfusion.

In conclusion, we believe that our patient developed paravertebral rhabdomyolysis and renal insufficiency because of compartment syndrome that resulted from a supine hyperlordotic position and long-lasting surgery. Although surgeons often require the hyperlordotic position for surgical access, it should be avoided if a prolonged surgery is anticipated. Positioning should be carefully considered and managed meticulously.

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