

CASE REPORTS

likely.⁸ Despite the severity of rhabdomyolysis in the present case, no cardiac dysrhythmia leading to cardiac arrest and no acute renal failure occurred. This result is particularly uncommon because less severe cases of rhabdomyolysis related to MH have been reported to lead to cardiac arrest or to require renal extracorporeal epuration.^{1,2}

The diagnosis of MHS was suspected in the early postoperative period, and the administration of dantrolene was discussed in the recovery room. Dantrolene decreases the intensity of the contractures of skeletal muscles and reduces hypermetabolism by inhibiting the release of calcium from intracellular stores. The efficacy of dantrolene to lower CPK blood levels has been suggested in children with masseter muscle spasm, but it remains uncertain in adults.¹⁰ Therefore, dantrolene was available but not given immediately to our patient, in the absence of life-threatening cardiac episode or uncontrolled hypermetabolism. In the present case, the prompt diagnosis of myoglobinuria, the early fluid challenge, and the forced diuresis avoided dialysis and cardiac complications.

In summary, this case emphasizes that MHS may be revealed by an almost isolated and extremely severe rhabdomyolysis. Under such circumstances, dantrolene has to be available, and appropriate fluid loading with forced diuresis may be helpful to prevent life-threatening organ failures. In a second step, a patient with perioperative rhabdomyo-

lysis or isolated myoglobinuria needs to be investigated for MHS after full recovery.

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Massive Postoperative Rhabdomyolysis after Uneventful Surgery: A Case Report of Subclinical Malignant Hyperthermia

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MALIGNANT hyperthermia (MH) is generally thought of in its fulminant form, manifesting as severe hypercapnia,

uncontrollable hyperthermia, and accompanied by hyperkalemia with cardiac dysrhythmias. We report a case of an adult surgical patient who developed severe postoperative rhabdomyolysis without other symptoms and who was subsequently found by muscle biopsy to be MH susceptible.

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Case Report

A 50-yr-old, 100-kg male was scheduled for a radical prostatectomy for prostatic adenocarcinoma. Medical history included depression, chronic left upper extremity weakness for 23 yr, and peptic ulcer disease. Surgical and anesthetic history included general anesthesia for cholecystectomy, hemorrhoidectomy, and three procedures on

CASE REPORTS

his left lower extremity (for vein stripping). Halothane and succinylcholine were used in at least one of the anesthetics, without recorded complications. Family history was unremarkable. Medications included nortriptyline, naproxen, cimetidine, valproic acid, and isosuxiprine. The patient had not used tobacco or alcohol since 1991 and had no allergies to medications. Preoperative physical examination was unremarkable except for some dystrophic changes and lessened motor strength (3/5) in the left upper extremity. Laboratory data 6 days before surgery were normal except for an incomplete right bundle branch block on his electrocardiogram.

The patient received no premedication for sedation or analgesia until his arrival to the preoperative holding area where he received 2 mg of intravenous midazolam during placement of an epidural catheter. Standard monitors were placed, along with a radial artery catheter. The patient was placed in the supine position, and general anesthesia was induced with midazolam, fentanyl, sodium thiopental, d-tubocurarine, and succinylcholine. No masseter muscle rigidity was noted during direct laryngoscopy. Maintenance of anesthesia was conducted with isoflurane, nitrous oxide, and fentanyl during the 8-h period. Positioning for the surgical procedure was supine with a 30° flexed back and an elevated kidney rest. Hemodynamic values remained within 30% of the preoperative values. Serial arterial blood gas analysis indicated consistent normocapnia and a pH between 7.33 and 7.37. The capnograph display indicated a $P_{ET}CO_2$ between 28 and 35 mmHg. The arterial oxygen tension ranged from 110 to 148 mmHg while the patient received an FiO_2 of 0.35–0.44. A serum potassium measured 45 min after induction was 4.0 mEq/l. The potassium value remained stable throughout the case.

The blood loss was estimated to be 3000 ml. Fluids administered included 6% hetastarch, autologous blood, 0.9% saline, and lactated Ringer's solution. At the end of the surgical procedure, neuromuscular blockade was reversed, the endotracheal tube was removed, and the patient was taken to the postanesthesia care unit (PACU). Vital signs remained within preoperative range during this time. The patient was awake and expressed no specific complaints. Results of a blood gas analysis were normal. He was discharged from the PACU to the intensive care unit, and an epidural infusion of morphine and bupivacaine was initiated for analgesia. Postoperative vital signs remained within normal limits until approximately 13 h postoperatively when he became febrile to an oral temperature of 37.6°C. The oral temperature remained close to 37.8°C throughout the postoperative course.

During the first postoperative day, the patient felt well, had return of bowel sounds, and had his nasogastric tube withdrawn. He experienced some left lower extremity numbness, and because of plans to ambulate, the bupivacaine was withheld from his epidural infusion. During the second postoperative day, the patient began to complain of lumbar pain. Physical examination of the lumbar region revealed paravertebral muscle tenderness bilaterally. Also during the second postoperative day, his abdomen became slightly distended, and oral intake was withheld because of the possibility of an ileus. Blood was drawn to analyze multiple enzymes levels to determine if an abnormal intraabdominal process was occurring. The serum glutamic oxaloacetic transaminase (SGOT) and lactic dehydrogenase (LDH) levels were elevated (505 U/l and 1143 U/l, respectively).

On postoperative day three, the patient continued to complain of lumbar pain, and despite adequate analgesia of his wound pain, he requested withdrawal of his epidural catheter. He was then placed on intravenous patient-controlled analgesia (PCA) with morphine. His lumbar pain increased in severity after withdrawal of epidural

analgesia. Serum gamma-glutamyl transferase (GGT), serum glutamic pyruvic transaminase (SGPT), and creatine kinase (CK) levels were ordered to delineate any abnormal gastrointestinal processes and rule out a muscle source of the increased SGOT and LDH. The SGPT was 245 mU/l, the GGT was normal, and the CK level was >285,000 U/l. A diagnosis of rhabdomyolysis was made. At this time, the serum creatinine was 1.3 mg/dl. Further examination revealed no sign of compartment syndrome in any extremity. A nephrologist was then consulted, who suggested institution of intravenous normal saline, mannitol, and sodium bicarbonate to prevent acute renal failure from myoglobinuria (urine myoglobin = 226 mg/l). He was transferred to the intensive care unit.

A computed tomographic scan was performed to determine whether an abnormal intraabdominal or pelvic process was present. There were no abdominal or pelvic abnormalities noted, except for expected postoperative changes from his prostatectomy. The scan revealed edema surrounding the latissimus dorsi muscles and overlying the external oblique abdominal muscles bilaterally. There was also slight decreased attenuation of the paraspinal muscles.

Over the balance of his hospitalization, the patient's serum muscle enzyme levels gradually dropped to CK, 7,486; LDH, 880; and SGOT, 234 (table 1) by the time of discharge. His serum creatinine level remained stable. Although he continued to have lumbar pain, he experienced no other problems and was discharged 10 days after his surgical procedure. Laboratory analysis obtained during a postoperative clinic visit 1 week after discharge continued to indicate elevation of muscle enzymes (SGOT, 59; LDH, 411) and mild hyperkalemia (K^+ , 5.3 mEq/l).

Approximately 4 months after his surgical procedure, the patient was referred to our Malignant Hyperthermia Center for evaluation. Because strong signs of MH were lacking, the value of a muscle biopsy was questioned, but because of the severity of his rhabdomyolysis, we decided to obtain a muscle biopsy to test for MH. Vastus lateralis muscle was obtained during general anesthesia, performed with a non-triggering anesthetic technique. Three muscle fascicles were tested with caffeine, and contractures were initiated in each at 0.5 mm concentration, exhibiting an abnormal response. Two of these fascicles had isometric contracture tensions of 0.33 g and 0.75 g when exposed to 2.0 mm caffeine, exceeding the MH diagnostic threshold of 0.2 g.¹ Three other fascicles were tested for halothane-induced contracture response, and in two of these, tensions of 0.5 g and 0.7 g exceeded the diagnostic threshold range of 0.2–0.7 g. Other fascicles tested with caffeine plus halothane also produced abnormal responses. These results indicated MH susceptibility for this patient as judged by diagnostic testing standards set forth by the North American MH Protocol. The patient was counseled regarding the implications of the results for himself and his family. To our knowledge, he has not undergone any further surgical procedures requiring anesthesia.

Discussion

The clinical presentation of MH can be highly variable and may present anytime perioperatively.² Other cases of anesthetic-associated myoglobinuria reported as episodes of MH demonstrated myoglobinuria as the only presenting sign.^{3–8} As in our case, MH has been reported to occur in subjects who have received several anesthetic

CASE REPORTS

Table 1. Sequence of Laboratory Values over Time

	SGOT (5–35 U/L)	LDH (90–250 U/L)	Creatine Kinase (U/L)	Creatinine (mg/dl)	Potassium (mEq/L)
OP–6 days	50	148	NA	1.1	3.9
OP + 12 h	505	1,143	NA	1.3	5.0
OP + 36 h	947	1,733	NA	1.3	4.9
OP + 72 h	980	1,898	>285,000	1.0	4.7
OP + 96 h	866	1,828	57,583	0.9	3.8
OP + 120 h	597	1,462	27,995	0.8	3.4
OP + 144 h	415	1,363	18,501	0.8	3.6
OP + 168 h	304	1,055	10,146	0.8	4.2
OP + 192 h	234	880	7,486	0.9	4.6

OP = day of operation; SGOT = serum glutamic-oxaloacetic transaminase; LDH = lactic dehydrogenase; NA = not applicable.

tics that produced no complications.^{9,10} Rhabdomyolysis has also been reported in patients undergoing surgical procedures in whom pressure necrosis of muscle occurs from positioning.^{11,12} A case of idiopathic rhabdomyolysis not related to surgery has also been reported in an adult male in whom no precipitating cause could be determined.¹³

The differential diagnoses for his rhabdomyolysis included: (1) MH, (2) compression of musculature leading to muscle ischemia, (3) dermatomyositis, (4) one of several enzyme deficiencies (glucose-6-phosphodiesterase, phosphofructokinase, carnitine palmityl transferase, muscle phosphorylase), and (5) familial periodic paralysis. The first two etiologies were deemed most likely because of the abrupt nature and timing of his rhabdomyolysis. Moreover, the other etiologies occur rarely. The likelihood of MH was considered less likely because of the patient's lack of perioperative tachycardia, hypercapnia, acidemia, and the low level of his postoperative hyperthermia. On the other hand, a review of the literature had revealed that reported levels of CK after intraoperative pressure muscle ischemia were much lower (<75,000 U/l) than observed in our patient.

Previous authors have described a syndrome of anesthesia-induced myoglobinuria as an abortive form of MH.⁵ Because of the lack of reports that analyze series of cases of perioperative rhabdomyolysis, no judgment can be made on the validity of this phenomenon as an expression of MH. The question of what exactly triggered rhabdomyolysis in this particular case remains unanswered. The patient had a muscle type that tested positive for MH and was given triggering agents. Also his weight, duration of surgery, and positioning could potentially lead to pressure ischemia.

Although the existence of false-positive MH diagnostic test results is unproven, one possible explanation for our finding is that the MH-like muscle responses represent a false-positive diagnosis. Another explanation for the findings in this patient is that some other underlying myopathic process has predisposed the muscle to the clinical and *in vitro* test responses. Neither histologic nor histochemical examination of the muscle tissue was performed to rule out specific or non-specific myopathy. However, false-positive MH contracture results in patients with specific muscle disease, *e.g.*, myotonia congenita, is an unproven entity. Hence, even finding a specific myopathy in this patient cannot rule out MH.¹⁴

In summary, we describe a patient, who later tested positive for MH, who developed severe postoperative rhabdomyolysis after a general anesthetic for radical prostatectomy. The patient demonstrated no signs or symptoms indicating MH intraoperatively. We believe that other cases of MH may occur that also go unrecognized. When patients demonstrate symptoms or laboratory evidence of significant muscle injury perioperatively, the possibility of MH should be considered.

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Transient Neurologic Toxicity after Spinal Anesthesia, or Is It Myofascial Pain? Two Case Reports

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THERE have been several publications of “transient neurologic toxicity” or “transient radicular irritation” attributed to intrathecal local anesthetics. Most described cases involved the use of hyperbaric lidocaine, 2–5%. Sumi *et al.*¹ recently reported a case in which tetracaine, 0.5%, was used. Reported cases present with low back or buttock pain radiating to the thighs or lower legs. The pain is usually moderate to severe, appearing 1–24 h postoperatively after recovery from an otherwise uneventful spinal anesthetic. In all reported cases, the symptoms disappeared within 1 week. We report two cases of similar symptoms and circumstances that have the appearance of simple musculoskeletal pain.

Case 1

A 30-yr-old man with chondromalacia of the right patella was scheduled for an arthroscopy and débridement. With the patient in the right lateral decubitus position, the lumbar area was prepped with an iodine-containing solution, draped, and the skin wiped dry. A skin wheal was raised using 2 ml of lidocaine, 1%, solution at the L4–L5 level using sterile technique, and a 25-gauge Quincke needle was used to obtain free-flowing, clear cerebrospinal fluid (CSF), on the first attempt. After adequate backflow was confirmed, 70 mg (1.4 ml) of 5% MPF-lidocaine in 7.5% dextrose (Hyperbaric Xylocaine®, Astra Pharmaceuticals, Westborough, MA) was injected over approximately 30 s. No paresthesias were elicited during needle insertion or injection of local anesthetic. Adequate anesthesia was obtained with a T10 sensory level. During surgery lasting 1 h, the patient was in the supine position. After an uneventful intraoperative and postanesthesia care unit (PACU) course, the patient was discharged home. On routine telephone call follow-up evaluation on postoperative day 1, the patient indicated that he was doing well, although later that day (approximately 24 h after the anesthetic), the patient called to complain of excruciating low back pain with paresthesias radiating to both legs. At that time, he was asked to come to our pain clinic for evaluation and treatment. The pain was confirmed to be in the lower back/buttocks area and radiating down both legs. The leg pain was noticed not to involve the feet. Results of Lasègue's test were negative. Because of the severity of symptoms, magnetic resonance imaging (MRI) was performed to rule out a structural lesion. Results of the MRI were negative. A diagnosis of myofascial low back pain with six active paraspinal muscle trigger points was made. This was confirmed by infiltrating a solution of 0.25% bupivacaine/0.5% etido-

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