POSTOPERATIVE RHABDOMYOLYSIS AFTER BARIATRIC SURGERY REPORT OF TWO CASES

María Victoria Zea Medina, M.D.*, Oliva Hernández Lazcano M.D.*

INTRODUCTION

The term rhabdomyolysis refers to massive muscle necrosis following disintegration of striated muscle. Clinically, a less severe form exists, and is characterized by an increase in serum creatine kinase (CK) and absence of red cell casts in urine, that on occasions develops into a complicated and serius form of acute renal failure.[1, 2] We report two cases of adult surgical patients who developed severe postoperative rhabdomyolysis after bariatric surgery.

Key words: Necrosis, muscle striated, laparoscopic.

CASE REPORT - CASE 1

A 31 years-old male, 248-kg in weight, with a body mass index (BMI) of 76.5 kg/m^2 was scheduled for laparoscopic gastroyeyunoanasthomosis. He had been healthy and had no history of muscle or renal disease. He was not taking any medications.

Surgical and anesthesic history included general anesthesia for a surgical reconstruction of a ruptured tendon in his right arm, that followed an uncomplicated course.

Upon admission the patient complained of dyspnea on exertion, and progressive fatigue that had worsened in the previous year, after noticing an increase in his body weight up to 240 kg. Estimated cardiovascular and functional capacity was greater then 5 METs. Respiratory functional capacity and arterial blood gas analysis were within normal parameters. Evaluation of the muscle and excleletic systems was normal. Distal pulse palpation in lower extremities was difficult because of edema. Distal cyanosis was apparent, as were superficial venous dilations in both legs.

Laboratory data before surgery were normal including a basal serum CK of 140 IU/L.

The patient was premedicated with ranitidine the night before the procedure. Once in the operating room, the patient was placed under standard monitoring, along with a radial artery catheter and central venous pressure (CVP). The intubation was carried satisfactorily with the patient awake and, after that, general anesthesia was induced with Propofol, Fentanyl and Vecuronium. No masseter muscle rigidity was noted during direct laringoscopy. Maintenance of anesthesia was conducted administering Sevofluranne, Fentanyl and Vecuronium during the 8-h period of surgical procedure. Patient was positioned supine with lower extremities extended and at a 45 degree angle. The patient's position was changed to Trendelemburg or Fowller as required by the surgical team.

Hemodynamic parameters remained within a 20% range of preoperative values. Respiratory values were modified immediately after pneumoperitoneum started, however, the Pet CO_2 was maintained between 28 and 35 mmHg throughout the procedure. Blood loss was estimated in 200 ml. The urine output was maintained at a minimum of 100 ml/h throughout the procedure. Serial arterial blood gas analysis indicated normocapnia and pH 7.34, Sat O_2 96% ($F1O_2$ of 0.5-0.6) bicarbonate 21 mmol/l and EB-4.8 mmol/l. Potassium levels remained normal. There were no incidents during the surgical procedure. However, shortly after the patient arrived to the recovery unit, he complained of severe back pain.

On postoperative day one, he continued to complain of severe back pain despite adequate analgesia. This pain impeded him to sleep. Phisical exam was relevant for tenderness and pain in the lumbar and sacral region. His urine had become dark, without reduction in volume. Urianalysis was negative for proteins and blood cells, and positive for epithelial cells (2 per field) mucine ++, and scant granulose and hyaline casts. Other exams revealed increased levels of serum alanineaminotranspherase (ALT 179 IU/L), asparte-aminotranspherase (AST 682 IU/L) and serum CK 13,179 IU/L. On postoperative day 2 CK decreased to 3387 IU/L. Serum electrolytes, blood urea nitrogen, creatinine and alkaline phosphatase were within normal values. Studies for serum and urine levels of myoglobin were not available at our hospital and computed tomographic scan and muscle biopsy were not performed.

The patient was discharged 3 days after his surgical procedure and was seen as an outpatient a week later. He complained of moderate low-back pain and urine had turned to its normal color, without any decrease in urine output.

CASE 2

A 23 years-old, obese female (BMI 50 kg/m^2) was scheduled for a laparoscopic gastroyeyunoanasthomosis. Family history was remarkable for diabetes mellitus. The patient had not used tabacco or alcohol. She had been healthy and had no history of muscle or renal disease. She was not taking any medications.

Upon admission, physical examination revealed exertional dyspnea. Estimated cardiovascular and functional capacity was

Recibido: marzo 23, 2001 – Aceptado: Enero 30, 2002. *Assistant Professor, Department of Anesthesiology. María Victoria Zea Medina. Vasco de Quiroga 15, Colonia Sección XVI, Tlalpan México City, CP 14000. Tel. 5573-1200 Ext 5020 E-mail address: mariavictoriazea@hotmail.com Instituto de Ciencias Médicas y Nutrición Salvador Zubrán.

greater than 4 METs. Respiratory functional capacity and arterial blood gas analysis were normal. Preoperative laboratory data including serum CK (160 IU/L) were within normal parameters.

In the operating room standard monitoring devices were placed, along with a radial artery and CVP catheters. General anesthesia was induced with Propofol, Fentanyl and Vecuronium. No masseter muscle rigidity was noted during direct laringoscopy. Maintenance of anesthesia was conducted with Sevoflurane, Fentanyl and Vecuronium during the 5-h surgical procedure. Body position for the operation was supine with extended lower extremities.

Hemodynamic parameters remained stable with mean blood pressure of 120/70 mmHg. Respiratory values were modified immediately after pneumoperitoneum, and mechanical ventilation was used to maintain the PET CO_2 between 27 and 30 mmHg. Estimated blood loss was 50 ml. Urine output was maintained at a minimum of 90 ml/h throughout the operation. Surgical procedure was uneventful and the patient promptly recovered from anesthesia and remained for an hour in the postanesthesia care unit.

On postoperative day one she complained of severe back pain. Her urine had changed to a dark, cola-like color with decreased output. Urianalysis was abnormal: pH 6.5, density 1.020, proteins ++++, ketones +, negative glucose, bilirrubins and nitrites, with 1-2 blood cells per field, 1-2 leukocytes per field. The rest of the laboratory work-up was relevant for increased levels of serum ALT (1152 IU/L), AST (350 IU/L) and CK 53,000 IU/L, Creatinine 2.2 mg/dl. Rhabdomyolysis was diagnosed and therapy was started. Urinary volumes increased to normal levels and this was followed by normalization of serum creatinine. Serum CK levels decreased to 15,000 IU/L in 5 days after initial diagnosis. There was no neeed for hemodyalisis. Low-back pain subsided progressively, and the patient left the hospital 6 days after surgery. On the 10th postoperative day, serum CK was 1091 IU/L. Serum potassium remained normal throughtout hospitalization. CT scan and muscle biopsy were not performed.

DISCUSSION

There are several causes of the rhabdomyolysis in the perioperative setting. The main factors involved are: a) inadequate patient body postures coupled with prolonged operating time which cause muscle compression and secondary ischemia; b) thrombosis, embolism or clamping of vessels during surgery which may result in servere muscle ischemia and consequent extensive cell necrosis, and c) rhabdomyolysis associated with malignant hyperthermia.

Bertrand et. al.[3] described 9 cases (frequency of 4% in their series) of lumbar muscle rhabdomyolysis after abdominal aortic surgery. This form of rhabdomyolysis occurred in younger patients who were more frequently obese. As in the current cases, the main clinical manifestation was low-back pain accompanied by a moderate elevation of serum CK, which did not surpass the 75,000 IU/L. Review of literature reveled that CK levels after intraoperative muscle pressure ischemia are usually lower Than 75, 000 IU/L.[4] In the both cases we described diagnosis was not confirmed by detection of myoglobin in urine, muscle biopsy or CT scan.

Malignant hyperthermia (MH) is a rare clinical syndrome characterized by hyperthermia, muscle rigidity, and metabolic acidosis most often seen in genetically susceptible individuals undergoing general anesthesia. Inhaled volatile anesthesics are the most frequent medications that trigger MH. These agents include holothane, enphlurane, sevoflurane and succinycholine.[5] The probability of MH in adult patients with anesthesia-induced rhabdomyolysis is only 0.05 to 0.0005%.[2] The presence of rhabdomyolysis secondary to MH is a remote possibility in the current cases. The only anesthesic factor involved in both cases was the use of sevoflurane, which has been previously related to the development of MH[2]. Fentanyl, propofol and vecuronium are considered low-risk agents for the development of MH[2]. This diagnostic possibility was suspected based on previous reports that describe a subclinical form of MH in which myoglobirubina and increased serum CK are the only findings.[4, 6, 7] Others causes of rhabdomyolysis were considered and ruled out.[1,2]

This constitutes the first report of rhabdomyolysis in obese patients undergoing laparoscopia bariatric surgery. We believe that the most probable mechanism by which rhabdomyolysis was caused was the combination of an extreme body posture during and extended time in subjects with obesity that caused severe ischemia and necrosis of the muscles in the lumbar region. Although diagnosis was not confirmed with imaging studies and biopsy, the appearance of severe low-back pain, muscle spasm, dark urine and greatly increased CK is highly suggestive of rhabdomyolysis. In the second case, rhabdomyolysis was complicated with acute renal failure that promptly responded to conventional therapeutic maneuvers.

REFERENCES

1. Vanholder R, Sever MS, Erek E, Lameire N: rhabdomyolysis. Disease of the Month. Journal of the American Society of Nephrology 2000; 11:1-16.
2. Bertorini TE. Myoglobinuria, malignant hyperthermia, neuroleptic malignant syndrome and serotonin syndrome. Neurologic Clinics 1997; 15: 649-671.
3. Bertrand M. Lumbar muscle rhabdomyolysis after abdominal aortic surgery. Anesth Analg 1997; 85: 11-5.

4. Harwood TN, Nelson TE. Massive postoperative rhabdomyolysis after uneventful surgery: A case report of subclinical malignant hyperthermia. Anesthesiology 1998; 88: 265-268.
5. Chan TC, Evans SD, Clark RF. Drug induced hyperthermia. Critical Care Clinics 1997; 13: 785-808.
6. Birmingham PK, Stevenson GW, Uejima T, Hall SC. Isolated postoperative myoglobinuria in a pediatric outpatient. A case report of malignant hyperthermia. Anesth Analg 1989; 69: 846-9.
7. Rubiano R, Chang JL, Carrol J, Sonbolian N, Larson CE. Acute rhabdomyolysis following halothane anesthesia without succinylcholine. Anesthesiology 1987; 67: 856-7.