

Review

Rhabdomyolysis: Diagnosis and Treatment in Bariatric Surgery

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Background: Rhabdomyolysis (RML) and subsequent acute renal failure can be serious problems following bariatric operations. Early diagnosis and treatment are important to avoid the complications of RML.

Methods: This review was achieved by searching the key words: Rhabdomyolysis, diagnosis, treatment and bariatric surgery. We included prospective, retrospective, case reports and review articles.

Results: RML diagnosis can be done by: signs and symptoms, physical evaluation, laboratory findings and imaging examinations. Muscle weakness, myalgia, decubitus ulcer, proteinuria and myoglobinuria are the more mentioned findings. Elevation of CPK levels is the most sensitive diagnostic evidence of RML. Treatment is geared toward preserving renal function by avoiding dehydration, hypovolemia, tubular obstruction, aciduria, and free radical release. Early recognition allows the administration of fluids, bicarbonate, and mannitol.

Conclusion: Prophylactic measures and early diagnosis and treatment of rhabdomyolysis in bariatric surgery are imperative to prevent the potential fatal complications of this condition.

Key words: Rhabdomyolysis, bariatric surgery, diagnosis, treatment, morbid obesity, acute renal failure, CPK

Abbreviations:

RML = Rhabdomyolysis
ARF = Acute renal failure
CPK = Creatine phosphokinase
AST= Aspartate aminotransferase
ALT= Alanine aminotransferase
LDH= Lactate dehydrogenase
EMG = Electromyographic
MRI = Magnetic resonance imaging
CT = Computed tomography
US= Ultrasound
GFR= Glomerular filtration rate
CVVH = Continuous Venovenous Hemofiltration
BIC/MAN = Bicarbonate and Mannitol
CPK MM = Creatine phosphokinase MM-isoenzyme (muscle type)
CPK MB = Creatine phosphokinase MB-isoenzyme (cardiac type)

Introduction

Rhabdomyolysis (RML) can be defined as a disorder that consists of striated muscle disintegration resulting in the release of muscle toxic cell constituents into the extracellular fluid and systemic circulation.¹⁻⁷ Damaged skeletal muscle fibers break down and lose integrity of the sarcolemmal membrane, releasing their contents and challenging the kidney's filtering system. RML and subsequent acute renal failure (ARF) can be serious complications resulting from operative position compression of bariatric opera-

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tions. The incidence varies from 6 to 75%.^{2,8} Postoperative RML occurs due to the prolonged muscle compression in many non-physiological surgical positions, but mainly in procedures longer than 4⁹ to 5^{8,10} hours. In bariatric surgery, the excessive weight, the presence of diabetes, an ASA physical status >II,⁹ and prolonged surgical time lead to RML.¹⁰ Full recovery can be expected with early diagnosis and treatment of the many complications that can develop in patients with this syndrome.¹¹

Materials and Methods

PubMed, Medline, Bireme, Scielo and Lilacs libraries besides textbooks, specialized journals and the internet were searched between December 2005 and January 2006. The work includes prospective, retrospective, case reports and review articles in Portuguese and English languages. A total of 52 articles were obtained and appraised.

Published Results

Diagnosis of Rhabdomyolysis in Bariatric Surgery

Clinical Findings and Physical Evaluation

The initial expression of RML can be sudden, and an early diagnosis requires a high degree of suspicion.¹⁰ The syndrome has local and systemic features. Local signs and symptoms are non-specific and may include muscle pain, tenderness, swelling, bruising and weakness. Systemic features include tea-colored urine, fever, malaise, nausea, emesis, confusion, agitation, delirium and anuria¹¹ (Table 1). During the physical examination, decubitus ulcer and eruptions can be present in pressure zones, mainly at the hips, limbs and buttocks.^{12,13}

Usually, the first systemic clinical sign is the appearance of urine with altered color that can range from pink, to brown, and black.^{7,10,11} Myoglobinuria is suspected with the presence of altered urine color⁷ and requires differential diagnosis among several entities.^{13,14}

Table 1. Clinical features of rhabdomyolysis

Local features	Systemic features
Muscle pain	Tea-colored urine
Tenderness	Fever
Swelling	Nausea
Bruising	Malaise
Weakness	Emesis
	Confusion
	Agitation
	Delirium
	Anuria

From: Sauret JM, Marinides G, Wang GK. Rhabdomyolysis. *Am Fam Physic* 2002; 65: 907-12.¹¹

Laboratory Findings

Although history and physical examination can provide clues, the actual diagnosis of RML is confirmed by laboratory studies.^{5,10,15} Once RML is suspected, the diagnosis can be confirmed by identifying high levels of creatine phosphokinase (CPK). Serum CPK 5 times the normal value is considered as a biochemical diagnosis of RML.² The elevation in CPK levels is the most sensitive diagnostic evidence of muscle injury^{8,11,14} and is present in 100% of RML cases.¹⁰ When the RML syndrome is present, extreme quantities of CKMM are released into the blood system and peak concentrations of 100,000 IU/ml or more are not unusual. No other condition will cause such extreme CPK elevations.¹⁰ Small amounts of CKMB may also be present.¹⁶ Serum CPK peak values occur 4 to 7 days after injury and remain elevated for up to 12 days.¹³ In some cases, the CPK isoenzymes MM and MB are measured to distinguish a cardiac from a skeletal source.⁷ An electrocardiogram must also be done to differentiate RML from myocardial infarction.²

Urinary myoglobin provokes a typical reddish-brown color. Myoglobin can be detected in urine when these values exceed 1500 to 3000 ng/m.¹⁶ Kim et al¹⁵ found in a prospective study that a urine myoglobin concentration >300 ng/ml was associated with an increased risk of RML and ARF. Urinalysis in patients with RML will also reveal the presence of protein, brown casts in tubules and uric acid crystals and may reflect electrolyte wasting consistent with renal failure.¹⁰

In a retrospective analysis, Grover et al¹⁸ studied the lack of clinical utility of urine myoglobin detection by microconcentrator ultrafiltration in the diagnosis of RML. They concluded that this method has a poor and clinically inadequate sensitivity in detection and diagnosis of RML.

When RML is present, there is generally an increase in blood urea nitrogen and creatinine due to prerenal causes of ARF from dehydration and myoglobinuria.¹⁹ Both ARF and increased release of creatine from skeletal muscle cause the serum concentration of urea nitrogen and creatinine to increase in RML.

A classical pattern of changes in serum electrolytes occurs in RML. At the outset, serum levels of potassium and phosphate increase as these components are released from the cells, then levels decrease as they are excreted in the urine. Serum concentration of calcium initially decreases as calcium moves into the damaged muscle cells, then gradually increases during the recovery phase due to the release of calcium from injured muscle and elevated 1,25-dihydroxyvitamin D levels.¹⁶ Severe hyperuricemia may develop because of the release of purines from damaged muscle cells.^{16,19} High anion acidosis can also occur with RML.¹⁹ Clotting studies are useful for detecting any indication of disseminated intravascular coagulation.¹⁰

Serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), aldolase, troponin I and lactate dehydrogenase (LDH) enzymes can increase due to muscular injury.^{13,19} Serum carbonic anhydrase III has also been suggested as a marker for the diagnosis of RML.¹³

Arterial blood gas analysis is helpful for detecting underlying hypoxia and metabolic acidosis and monitoring sodium bicarbonate therapy.¹⁰ Muckart et al²⁰ concluded in a prospective study that venous bicarbonate (VBC) concentration has an important role as a predictive factor that allows identification of patients at risk of developing myoglobin-induced acute renal failure. A VBC <17 mmol/L was significantly predictive of ARF development.²⁰

In a retrospective study, Al-Shehlee et al²¹ investigated the electromyographic (EMG) features of acute RML. They concluded that EMG is an important diagnostic tool in the work-up of patients presenting acute or sub-acute severe muscle weakness and significantly elevated CPK when the differential diagnosis includes RML and inflammatory myopathies.

Image Examinations

Radiographic evaluation can also be valuable for diagnosing RML when clinical findings and physical examination are not elucidating (Table 2). Magnetic Resonance Imaging (MRI) and Computed Tomography (CT) are helpful in the diagnosis of RML.²²

MRI accurately identifies muscular edema in the affected muscle groups. In Lamminen's et al²³ prospective study, MRI had a higher sensitivity in the detection of abnormal muscles than CT or ultrasound (US) (100%, 62% and 42% respectively). CT evaluation can reveal muscle necrosis and calcification that occur early in the course of RML.²⁴ CT for the diagnosis of RML must be non-contrast enhanced to avoid acute renal failure.²⁵

¹¹¹In-labeled antimyosin monoclonal antibody and Technetium-99m pyrophosphate (99mTc-PYP) scintigraphy have also been used to make the diagnosis of RML and evaluate muscle injury.¹³

Ultrasound has also been known to have some value in identifying injured musculature in RML by revealing hyperechoic areas within the muscles examined.²⁶ Plain muscle X-ray does not have value in RML.²⁷ A muscle biopsy in the affected site can be done if any doubt remains.¹³

Rhabdomyolysis Treatment in Bariatric Surgery

The treatment of RML is geared toward preserving renal function, which is done by preventing factors that can lead to ARF, which are dehydration, hypovolemia, tubular obstruction, aciduria, and free radical release.²⁸ Early recognition allows the administration of fluid, bicarbonate, and mannitol.^{5,8,10,29} These measures help to prevent volume depletion, tubular obstruction, aciduria, and free radical release which is the mechanism for renal failure in rhabdomyolysis.^{5,8}

Table 2. Image examinations to detect RML and the findings

Magnetic Resonance Imaging - Muscular edema
Computed tomography - Muscle necrosis and calcification
Ultrasound - Hyperechoic areas
Technetium-99m pyrophosphate scintigraphy - Accumulation of the radioactivity in the damaged skeletal muscle

Hypovolemia may result from sequestration of water by muscles and must be prevented by the early and aggressive administration of intravenous fluids.^{1,11,30} Expanding the intravascular volume maximizes renal excretion by flushing out the tubular debris and limiting the time that nephrotoxins are in contact with renal tissues.^{10,30} Treatment of RML requires aggressive administration of fluids to ensure urine output >1.5 ml/kg/h³² or 150-300 ml/h until myoglobinuria has ceased.^{5,10,11,33} Maintaining a urine output this high may require intravenous infusion of fluids between 500 and 1000 ml/h,³³ and all patients should have a urinary catheter placed in order to adequately monitor fluid output.^{1,34} Sinert et al³⁵ showed in a retrospective chart analysis that forced diuresis within the first 6 hours of admission prevented episodes of ARF.

Diuretics are also used, mainly mannitol and loop diuretics. The addition of mannitol to the fluid regimen serves several purposes: mannitol increases renal blood flow and glomerular filtration rate; mannitol is an osmotic agent that attracts fluids from the interstitial compartment, thus counterbalancing hypovolemia and reducing muscular swelling and nerve compression. Mannitol is an osmotic diuretic that increases urinary flow and prevents obstructive myoglobin casts, and mannitol scavenges free radicals.¹ Many authors assert that loop diuretics (furosemide, bumetanide, and torsemide) must be used if fluids and mannitol are insufficient to maintain a brisk urine output.^{5,33} They increase tubular flow and calcium losses and decrease the risk of precipitation of myoglobin,¹ although they may acidify the urine.^{1,36} However, there is no evidence that diuretics improve the final outcome.¹¹

The use of sodium bicarbonate helps to correct the acidosis induced by the release of protons from damaged muscles to prevent precipitation of myoglobin in the tubules and reduce the risk of hyperkalemia.¹ Bicarbonate and acetazolamide are used for producing more alkaline urine when blood pH is >7.45 .³² Some investigators assert that the urine must be alkalized to pH 6.0,³⁷ 6.5,³⁸ 7.0,⁵ or even 7.5³⁹ to prevent the dissociation of myoglobin into its nephrotoxic components. On the other hand, there are also some concerns about the use of sodium bicarbonate because it may worsen hypocalcemia or precipitate calcium phosphate deposition in various tissues.¹¹

Allopurinol may be useful because it reduces the

production of uric acid and also acts as a free-radical scavenger. Another purine analog pentoxifylline has been considered in the management of RML because of its capacity to enhance capillary flow and decrease neutrophil adhesion and cytokine release.¹

Electrolyte disorders should be prevented or promptly treated.¹⁶ Control of hyperkalemia is an important therapeutic goal. Calcium salts and calcium kayexalate (sodium polystyrene sulfonate and exchange resin) should be used with caution because they enhance the risk of intramuscular calcium deposition.¹ Hypocalcemia usually does not require correction, particularly because this would increase the risk of intramuscular calcium deposition.¹

Dialysis is necessary if the kidneys no longer respond to the above-mentioned supportive measures and severe renal dysfunction has set in.^{2,32} Dialysis is indicated not only in patients with overt hyperkalemia but also in patients whose serum potassium rises rapidly and those with acidosis.¹

Compartment syndrome may be an early or late complication that results mainly from direct muscle injury.^{10,11,16,40} This complication occurs primarily in muscles whose expansion is limited by tight fascia. Peripheral pulses may still be palpable, and in these cases, nerve deficits (mainly sensory) are the more important finding. Compartment syndrome may develop or worsen during fluid resuscitation due to the development of edema of limbs and/or muscles. Decompressive fasciotomy, muscular debridement and escharotomies should also be considered in patients with evidence of neurovascular compression and decubitus ulcer if the compartment pressure is >30 mmHg.^{10,11,16,40,41}

Discussion

The early diagnosis of RML is especially important so that adequate treatment can be initiated and complications avoided. Features of RML can easily be unrecognized in critically ill patients. Physical examination in obese patients is difficult and may be non-contributory due to the amount of fat tissue in areas submitted to greater pressure. Furthermore, the diagnosis could be delayed in obese patients due to postoperative analgesia, late extubation and pre-existing symptoms, which morbidly obese patients

often have in addition to myoskeletal discomfort in the lumbar region and gluteal muscles.^{32,42} Laboratory and imaging tests must be done to confirm RML.

Elevation of CPK levels is present in 100% of RML cases. CPK measurement must be done not only in the postoperative phase but also in the preoperative period to compare enzyme levels. The CPK level to diagnose RML varies from each author. Some of them use CPK >1000 UI/l.² Others prefer CPK levels >5000 UI/l¹³ and even >10000 UI/l.⁷ The first one (>1000 UI/l) is safer for making prompt diagnosis and avoiding RML complications. Faintuch et al⁴³ stratified the patients according to muscle pain and peak serum CPK value in minor RML (inconspicuous pain and CPK <8000 UI/L) and extensive RML (severe shoulder back or buttock pain, swelling and weakness and CPK >8000 UI/L).

Since degradation of CPKMM is slow and the enzyme is not removed by the kidneys or dialysis, the plasma concentration of CPK remains elevated for much longer and in a more consistent fashion than that of myoglobin. Consequently, CPK is more reliable than myoglobin in assessing the presence and intensity of damage to the muscles.¹ Serum levels of myoglobin also increase markedly in RML, but this increase is not a reliable indicator of RML because myoglobin is rapidly cleared from the plasma.¹⁰

In their reviews, Wiltshire et al¹³ and Lane et al⁴⁴ stated that the laboratory values >300 ng/ml and >250 ng/ml were compatible with myoglobinuria, respectively.

Li et al⁴⁵ established a poor correlation between CPK peak and cardiac troponin I peak levels in a retrospective study. They found a prevalence of 17% of false positive troponin I levels in Emergency Room patients with RML.

When a CT scan is utilized, the iodinated radio-contrast should not be used due to renal toxicity, and it is limited to patients weighing <140 kg in our institution. MRI is an excellent method for diagnosis of RML, but is it not regularly utilized due to its costs and the patient's size.

Aggressive treatment must be done after diagnosis of RML to avoid complications. The first step is to preserve the affected zones and avoid RML in new areas of pressure, encouraging early ambulation and the use of special mattresses or pneumatic beds.⁴⁶ Early and adequate treatment of RML is imperative. However, no randomized trials concern-

ing treatment or prevention have been published. The only effective treatment is the intravascular volume expansion with saline solution.

Myoglobin may have direct toxic effects on the tubular epithelium and this toxicity is increased when nephronal flow-rates are low and urine is concentrated. In addition, urine pH may play a role with increased toxicity when the urine is acidic. Thus, in the early phases of RML, renal injury can be prevented or diminished by maintaining adequate volume-repletion or even hypervolemic state with a urine output of at least 100 ml/h.

The use of mannitol remains controversial, and is mostly supported by experimental animal studies and retrospective clinical studies.^{11,47} The risk of using mannitol is the occurrence of volume overload if renal failure develops, in spite of the above-mentioned treatment. Bicarbonate administration to elevate urine alkalinity and help solubility of myoglobin casts is often recommended, although the benefits from this treatment have not been conclusively demonstrated.¹⁶ The only drawback of bicarbonate administration is the decrease of serum ionized calcium¹ and its deposition in soft tissues. If bicarbonate is used, calcium levels should be carefully monitored because hypocalcemia may be aggravated by this therapy.¹⁶ Homsy et al⁴⁸ showed in a retrospective study that progression to establish ARF following RML could be totally avoided with prophylactic treatment in which volume repletion was achieved, using saline alone, and the use of bicarbonate and mannitol was unnecessary. In another retrospective study, Brown et al⁴⁹ state that bicarbonate and mannitol therapies do not prevent ARF and the need of dialysis or mortality in patients with CPK <30,000 U/L.⁴⁹

Treatment of compartment syndrome by means of fasciotomy is controversial. Some physicians advise immediate decompression of the muscles by surgical intervention, thereby decreasing the pressure in the injury region. However, this creates a potential source of infection.⁵⁰

Some authors have suggested that immediate hemodialysis may be useful for patients with RML and serum CPK >10,000 U/L before the development of customary biochemical and clinical indications of ARF.^{12,51-53}

The last measure is the maintenance of therapy and the monitoring of clinical and laboratory data. The CPK level should be determined every 6 to 12 hours, and all patients with RML require continuous elec-

trocardiographic monitoring for signs of hyperkalemia or cardiac irritability. This also includes sequential monitoring of urine output to guide further fluid resuscitation. Patients should be monitored closely to avoid the development of oliguric renal failure and to prevent fluid overload that can lead to the development of pulmonary edema and congestive heart failure. Patients with RML may benefit from invasive arterial and pulmonary artery pressure monitoring, which enables cognizance of the circulatory volume status. Other interventions include limiting the use of nephrotoxic antibiotics (e.g. aminoglycosides and amphotericin), non-steroidal anti-inflammatory drugs and iodinated radiocontrast to minimize further kidney damage¹⁰ (Figure 1).

Conclusion

Early diagnosis and treatment of rhabdomyolysis in bariatric surgery is imperative to prevent the potential fatal complications of this condition. The bariatric surgeon must be attentive and must know all clinical, biochemical and image examinations that indicate postoperative RML and should also establish early treatment for this syndrome. The treatment still has many controversies that will only be solved with prospective, controlled and multi-centered trials.

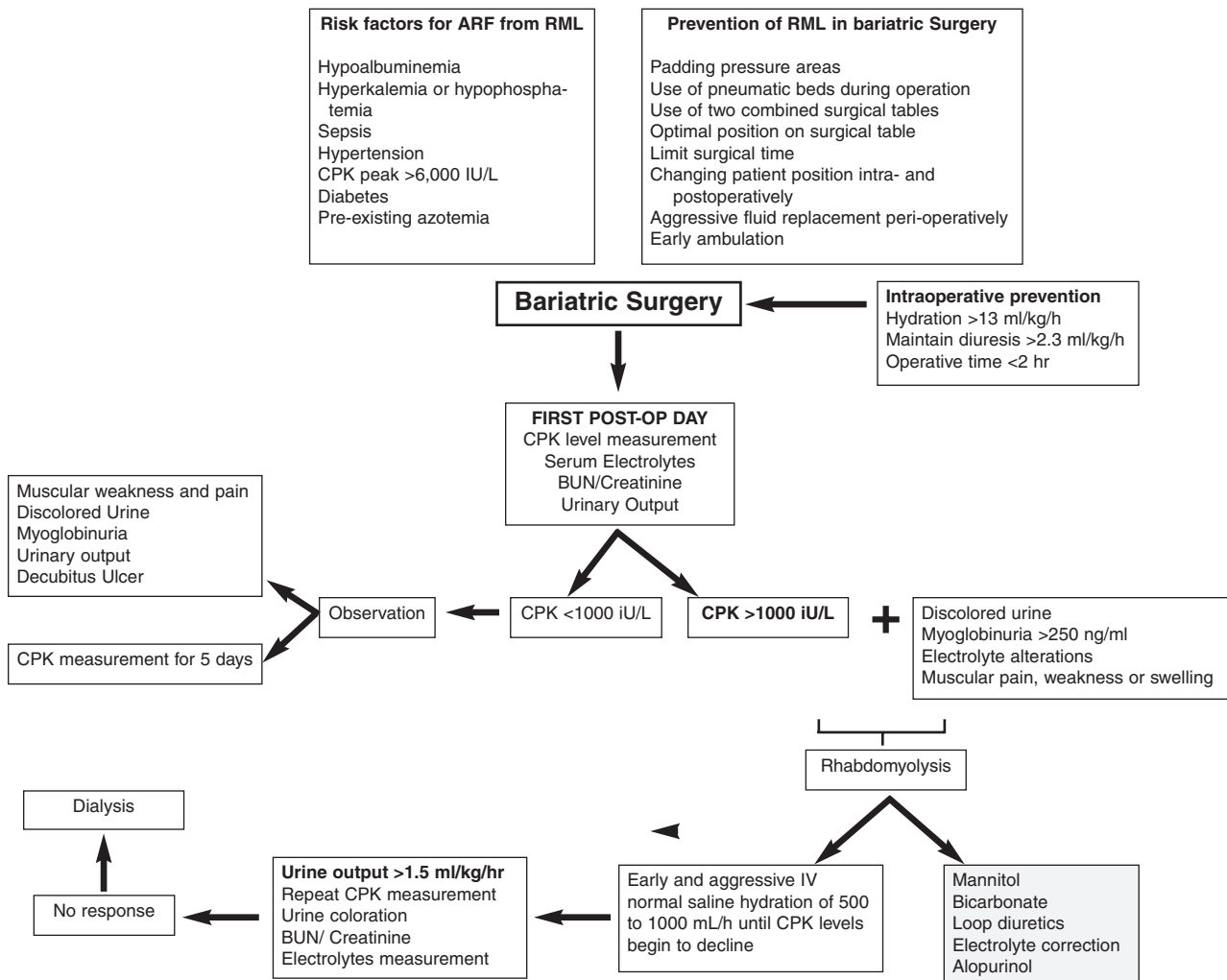


Figure 1. Algorithm for diagnosis and treatment of rhabdomyolysis in bariatric surgery.

References

1. Singh D, Chander V, Chopra K. Rhabdomyolysis. *Methods Find Exp Clin Pharmacol* 2005; 27: 39-48.
2. Mognol P, Vignes S, Chosidow D et al. Rhabdomyolysis after laparoscopic bariatric surgery. *Obes Surg* 2004; 14: 91-4.
3. Criner JA, Appelt M, Coker C et al. Rhabdomyolysis: the hidden killer. *Medsurg Nurs* 2002; 11: 138-43.
4. Sulowicz W, Walatek B, Sydor A et al. Acute renal failure in patients with rhabdomyolysis. *Med Sci Monit* 2002; 8: 24-7.
5. Vanholder R, Sever MS, Erek E et al. Rhabdomyolysis. *J Am Soc Neph* 2000; 11: 1553-61.
6. Zager RA. Rhabdomyolysis and myohemoglobinuric acute renal failure. *Kidney Int* 1996; 49: 314-26.
7. Torres-Villalobos G, Kimura E, Mosqueda JL et al. Pressure-induced rhabdomyolysis after bariatric surgery. *Obes Surg* 2003; 13: 297-301.
8. Bostanjian D, Anthonie GJ, Hamoui N et al. Rhabdomyolysis of gluteal muscles leading to renal failure: a potentially fatal complication of surgery in the morbidly obese. *Obes Surg* 2003; 13: 302-5.
9. Lagandre S, Arnalsteen L, Vallet B et al. Predictive factors for rhabdomyolysis after bariatric surgery. *Obes Surg* 2006; 16: 1365-70.
10. Criddle LM. Rhabdomyolysis. Pathophysiology, recognition, and management. *Crit Care Nurs* 2003 23: 14-22, 24-6.
11. Sauret JM, Marinides G, Wang GK. Rhabdomyolysis. *Am Fam Physic* 2002; 65: 907-12.
12. Collier B, Goreja MA, Duke BE. Postoperative rhabdomyolysis with bariatric surgery. *Obes Surg* 2003; 13: 941-3.
13. Wiltshire JP, Custer T. Lumbar muscle rhabdomyolysis as a cause of acute renal failure after Roux-en-Y gastric bypass. *Obes Surg* 2003; 13: 306-13.
14. Masuda A, Hirota K, Satone T et al. Pink urine during propofol anesthesia. *Anesth Analg* 1996; 83: 666-7.
15. Khuara RN, Baudendistel TE, Morgan EF et al. Postoperative rhabdomyolysis following laparoscopic gastric bypass in the morbidly obese. *Arch Surg* 2004; 139: 73-6.
16. Polderman, K.H. Acute renal failure and rhabdomyolysis. *Int J Artif Organs* 2004; 27: 1030-3.
17. Kim KK. Exogenous causes of myoglobinuria – review of 26 cases. *J Korean Med Sci* 1996; 11: 342-6.
18. Grover DS, Atta MG, Eustace JA et al. Lack of clinical utility of urine myoglobin detection by microconcentrator ultrafiltration in the diagnosis of rhabdomyolysis. *Nephrol Dial Transplant* 2004; 19: 2634-8.
19. Harriston S. A review of rhabdomyolysis. *Dimens Crit Care Nurs* 2004; 23: 155-61.
20. Muckart DJ, Moodley M, Naidu AG et al. Prediction of acute renal failure following soft-tissue injury using the venous bicarbonate concentration. *J Trauma* 1992; 33: 813-7.
21. Al-Shekhlee A, Hachwi R, Jaber MM et al. The electromyographic features of acute rhabdomyolysis. *J Clin Neuromusc Dis* 2005; 6: 114-8.
22. De Freitas Carvalho DA, Valezi AC, de Brito EM et al. Rhabdomyolysis after bariatric surgery. *Obes Surg* 2006; 16: 740-4.
23. Lamminen AE, Hekali PE, Tiula E et al. Acute rhabdomyolysis: evaluation with magnetic resonance imaging compared with computed tomography and ultrasonography. *Br J Radiol* 1989; 62: 326-30.
24. Messing ML, Feinzimer ET, Brosnan JJ et al. CT of rhabdomyolysis associated with malignant hyperthermia and seizures. *Clin Imaging* 1993; 17: 258-9.
25. Russ PD, Dillingham M. Demonstration of CT hyperdensity in patients with acute renal failure associated with rhabdomyolysis. *J Comput Assist Tomogr* 1991; 15: 458-63.
26. Steeds RP, Alexander PJ, Muthusamy R et al. Sonography in the diagnosis of rhabdomyolysis. *J Clin Ultrasound* 1999; 27: 531-3.
27. Nakahara K, Tanaka H, Masutani K et al. The value of computed tomography and magnetic resonance imaging to diagnose rhabdomyolysis in acute renal failure. *Nephrol Dial Transplant* 1999; 14: 1564-7.
28. Sharon GC. Rhabdomyolysis. *Orthop Nurs* 2005; 24: 443-7.
29. Pasnik K, Krupa J, Stanowski E et al. Successful treatment of gastric fistula following rhabdomyolysis after vertical banded gastroplasty. *Obes Surg* 2005; 15: 428-30.
30. Better OS, Stein JH. Early management of shock and prophylaxis of acute renal failure in traumatic rhabdomyolysis. *N Engl J Med* 1990; 322: 825-9.
31. Haskins N. Rhabdomyolysis and acute renal failure in intensive care. *Nurs Crit Care* 1998; 3: 283-8.
32. Filis D, Daskalakis M, Askoxylakis I et al. Rhabdomyolysis following laparoscopic gastric bypass. *Obes Surg* 2005; 15: 1496-500.
33. Abassi A, Hoffman A, Better O. Acute renal failure complicating muscle crush injury. *Semin Nephrol* 1998; 18: 558-65.
34. Moghtader J, Brady WJ, Bonadio W. Exertional rhabdomyolysis in an adolescent athlete. *Pediatr Emerg Care*. 1997; 13: 382-5.
35. Sinert R, Kohl L, Rainone T et al. Exercise-induced rhabdomyolysis. *Ann Emerg Med* 1994; 23: 1301-6.
36. Slater MS, Mullins RJ. Rhabdomyolysis and myoglobinuric renal failure in trauma and surgical patients: a

- review. *J Am Coll Surg* 1998; 186: 693-716.
37. Curry SC, Chang D, Connor D. Drug- and toxin-induced rhabdomyolysis. *Ann Emerg Med* 1989; 18: 1068-84.
 38. Harper J. Rhabdomyolysis and myoglobinuric renal failure. *Crit Care Nurse* 1990; 10: 32-6.
 39. Kruse D. "Tyson squats" as a cause of rhabdomyolysis. *J Emerg Nurs* 1998; 24: 116-7.
 40. Stroh C, Hohmann U, Remmler K et al. Rhabdomyolysis after biliopancreatic diversion with duodenal switch. *Obes Surg* 2005; 15: 1347-51.
 41. Bocca G, Van Moorselaar JA, Feitz WFJ et al. Compartment syndrome, rhabdomyolysis and risk of acute renal failure as complications of the lithotomy position. *J Nephrol* 2002; 15: 183-5.
 42. Melissas J, Kontakis G, Volakakis E et al. The effect of surgical weight reduction on functional status in morbidly obese patients with low back pain. *Obes Surg* 2005; 15: 378-81.
 43. Faintuch J, de Cleve R, Pajecki D et al. Rhabdomyolysis after gastric bypass: severity and outcome patterns. *Obes Surg* 2006; 16: 1209-13.
 44. Lane R, Phillips M. Rhabdomyolysis. *BMJ* 2003; 327: 115-6.
 45. Fai Li S, Zapata J, Tillen E. The prevalence of false-positive cardiac troponin I in ED patients with rhabdomyolysis. *Am J Emerg Med* 2005; 23: 860-3.
 46. Ettinger JEMTM, Dos Santos PV, Azaro E et al. Prevention of rhabdomyolysis in bariatric surgery. *Obes Surg* 2005; 15: 874-9.
 47. Better OS, Rubinstein I, Winaver JM et al. Mannitol therapy revisited (1940-1997). *Kidney Int* 1997; 52: 886-94.
 48. Homsí E, Barreiro MF, Orlando JM et al. Prophylaxis of acute renal failure in patients with rhabdomyolysis. *Ren Fail* 1997; 19: 283-8.
 49. Brown CV, Rhee P, Chan L et al. Preventing renal failure in patients with rhabdomyolysis: do bicarbonate and mannitol make a difference? *J Trauma* 2004; 56: 1191-6.
 50. Swain R, Ross D. Lower extremity compartment syndrome. When to suspect acute or chronic pressure buildup. *Postgrad Med* 1999; 105: 159-62.
 51. Smoszna J, Pietrzak B, Wankowicz Z. Acute kidney failure in the course of rhabdomyolysis with hemodialysis in personal material from 1995-1999. *Pol Merkeriusz Lek* 2000; 9: 826-9.
 52. Acquarone N, Garibotto G, Pontremoli R et al. Hypermnatremia associated with severe rhabdomyolysis. *Nephron* 1989; 51: 441-2.
 53. Ettinger JEMT, Marcílio de Souza CA, Ázaro E et al. Clinical features of rhabdomyolysis after open and laparoscopic Roux-en-Y gastric bypass. *Obes Surg* (accepted for publication).

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