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Case Report

Rhabdomyolysis in an obese patient after total knee arthroplasty

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We report the case of a morbidly obese patient who developed rhabdomyolysis with acute renal failure, hepatic dysfunction, and an increase of cardiac troponin-1 after total knee arthroplasty. Postoperative rhabdomyolysis has a wide range of triggers and differential diagnoses that should be considered by the anaesthesiologist and surgeons. We would like to emphasize that morbidly obese patients have an increased risk of developing postoperative rhabdomyolysis potentially leading to life-threatening disease. Intensified postoperative observation seem justified in these patients.

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Obese patients are generally believed to be at a higher risk of developing perioperative complications.[1][2] A number of smaller case series link massive obesity and long duration of the operation to the risk of developing postoperative rhabdomyolysis.[3–6] Rhabdomyolysis is an acute disease of the skeletal muscle leading to leakage of myocytes with the release of muscle cell contents such as myoglobin, electrolytes, creatinin and creatine kinase (CK). Depending on the degree of muscle cell damage, rhabdomyolysis can lead to severe metabolic derangement and acute renal failure through myoglobin and urate crystallization and direct cell-toxic effects of urinary myoglobin.[6] In non-traumatic patients after elective surgery rhabdomyolysis usually occurs as a result of compression because of unsuitable positioning or tourniquet use, but there are a variety of additional eliciting factors that should be considered in patients with signs of rhabdomyolysis.[7] We report the case of a morbidly obese patient who developed rhabdomyolysis with acute renal failure and liver dysfunction after total knee arthroplasty.

Case report

A 62-yr-old woman underwent left total knee arthroplasty. The morbidly obese patient (BMI 42.5) had type-2 diabetes mellitus which was being treated by diet. Renal and liver function tests taken before surgery were normal [creatinine 80 μmol litre^{-1}, blood urea nitrogen 3.45 mmol litre^{-1}, aspartate aminotransferase (AST) 18 u litre^{-1}, alanine aminotransferase 32 u litre^{-1}, lactate dehydrogenase (LDH) 128 u litre^{-1}, albumin 7.4 g litre^{-1}, prothrombin activity 120% of normal]. Before operation, the patient occasionally took diclofenac 50 mg when she had pain. After uncomplicated catheter placement for an inguinal femoral nerve block for postoperative pain management, general anaesthesia was induced by administration of i.v. thiopentone (4 mg kg^{-1}), sufentanil (0.2 μg kg^{-1}) and rocuronium bromide (0.35 mg kg^{-1}). Anaesthesia was maintained with sevoflurane and sufentanil. The procedure lasted 70 min, with blood circulation arrested by tourniquet for 50 min. The inflation pressure used in the tourniquet was 350 mm Hg. During the operation and in the recovery room, 1500 ml cristalloids and 500 ml hydroxy-ethyl starch (130/0.4) were administered. Pulsoxymetric oxygen saturation and heart rate were measured continuously. Blood pressure was measured at 5 min intervals. No untoward events such as hypoxaemia or significant hypotension occurred during the operation and in the early postoperative phase. We used a continuous infusion of ropivacaine (0.2%) 6 mg h^{-1} for the inguinal femoral nerve block. No motor blockade occurred. The catheter was removed on the fourth postoperative day. In the postoperative period, serum CK levels rose and reached a peak of 7152 u litre^{-1} on the third postoperative day, while the MB

isoenzyme of CK remained low. Similarly, on the second postoperative day acute renal failure developed with oliguria (<400 ml/24 h) and elevated serum creatinine and blood urea nitrogen (194 μmol litre^{-1} and 7.2 mmol litre^{-1}, respectively). The postoperative course was additionally complicated by hepatic dysfunction with peak levels on the second postoperative day of AST 8150 u litre^{-1}, ALT 4113 u litre^{-1}, LDH 8014 u litre^{-1}, prothrombin activity 61% of normal, and albumin 4.7 g litre^{-1} (lowest value: third postoperative day). Two days after surgery, the patient complained of retrosternal pain without radiation. The ECG and CK to CK-MB ratio were normal. However, cardiac-specific troponin-1 (cTn-1) was elevated (2.7 ng dl^{-1}). The patient could be discharged from the ICU on the seventh postoperative day. CK levels decreased to normal levels on the ninth postoperative day. Renal function had fully recovered, but mild elevation of liver enzymes persisted until the patient's transfer to a rehabilitation facility on the 15th postoperative day (AST 55 u litre^{-1}, ALT 67 u litre^{-1}).

Discussion

CK is a sensitive marker for rhabdomyolysis-related myocyte damage when elevated 5-fold and heart or brain ischaemia is ruled out. Renal toxicity is mainly mediated by myoglobin. Determination of plasma myoglobin might be more specific as a prognostic marker of renal function, but little is known about the kinetics of myoglobin during rhabdomyolysis, and most clinical laboratories do not offer routine measurement of myoglobin. Postoperative rhabdomyolysis has a wide range of triggers and differential diagnoses that should be considered by the anaesthesiologist and surgeons. A number of these risk factors are well defined and will be discussed using the example of the case reported here. In serious cases of rhabdomyolysis, severe electrolyte disorders and acute renal failure may occur, leading to life-threatening disease.[8]

Mognol and colleagues[3] reported that obesity is an independent risk factor for rhabdomyolysis. The authors found an incidence of 22.7% for rhabdomyolysis in obese patients undergoing laparoscopic gastric banding or gastric bypass. These authors identified the duration of the operation as another important risk factor for rhabdomyolysis in obese patients. Elevated liver enzymes as concomitant phenomenon of rhabdomyolysis are a sign of the reversible hepatic dysfunction that occurs in 25% of cases as reported by Akmal and Massry.[9] Our patient had a marked elevation of liver enzymes with mild impairment of liver synthesis function (decrease of albumin concentration and prothrombin activity, but no signs of hepatic encephalopathia or impairment of blood glucose concentrations). We could not identify the cause for hepatic dysfunction with CT-imaging, ultrasound examinations, viral serology testing and blood tests. We attributed the liver dysfunction to the severe rhabdomyolysis. However, the molecular mechanism of liver function impairment after rhabdomyolysis is not well understood and may be multifactorial.

The incidence of acute renal failure in patients with rhabdomyolysis ranges from 17% to 33%. In a study of 2083 posttraumatic patients with rhabdomyolysis, Brown and colleagues[10] found a CK level >5000 u litre^{-1} to be associated with renal failure in one out of five patients. Severe courses of acute renal failure that require dialysis occur in up to 28%.[11] Our patient developed acute renal failure without the need for renal replacement therapy. Urine output was restored by loop diuretics and i.v. crystalloids. Additionally, the patient received acetazolamide for urine alkalization. It is not clear whether renal replacement therapy contributes to the elimination of myglobin.[8]

It is common to see elevated cTnI during rhabdomyolysis; in most cases it is not a sign of myocardial damage. Benoist and colleagues reported 18 cases of rhabdomyolysis, 33% of which had elevated serum cTnI levels.[12] Furthermore, Punukollu and colleagues[13] found that 21% of patients with rhabdomyolsis had cTnI levels >0.6 ng ml^{-1} without evidence of acute coronary syndrome in the absence of ECG signs of ischaemic heart disease. In our patient, no signs of acute coronary syndrome were present despite elevated serum cTn-1 levels. However, echocardiography was not performed. Nevertheless, we believe that myocardial ischaemia was not present in our patient. The precise mechanisms accounting for the elevation of cTnI levels in patients without acute coronary syndrome are not known, but could be the result of cardiotoxicity attributable to increased levels of free radicals, circulating cytokines, acidaemia, hypoperfusion or increased heart wall stress.[13]

Besides direct surgical trauma, improper positioning of the patient during the operative procedure can cause muscle damage. Several cases of rhabdomyolysis have been reported in patients who underwent procedures while in a prone or lithotomy position. Some authors report cases of rhabdomyolysis caused by the tourniquet itself. Defects in the pressure gauges can cause abnormally high tourniquet pressures.[14 15] Therefore, avoiding tourniquet use in total knee replacement has been demonstrated to be effective.[16] In our case, both the duration of the transient circulatory blockade by tourniquet (50 min) and the total operation time (70 min) were in a time range that should not be associated with an increase in the prevalence of rhabdomyolysis.[17 18] No signs of compartmental syndrome such as local swelling and erythema were found, but swelling might have been masked by obesity.

We do know whether the rhabdomyolysis was generalized or restricted to the leg on which surgery was undertaken.

Drugs and toxins are responsible for up to 80% of rhabdomyolysis cases in adults.[19 20] Besides excessive alcohol consumption and drug abuse, hydroxymethylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors and propofol are of importance because of their widespread use. In recent years a number of reports have described the development of

rhabdomyolysis under long-term sedation with propofol in children as well as in adults leading to the term propofol infusion syndrome (PRIS).[19 21] Our patient, however, received neither propofol nor HMG-CoA reductase inhibitors perioperatively. No potential nephrotoxins such as NSAIDs or gentamicin were administered in the postoperative period.

Frequent clinical findings in patients who develop malignant hyperthermia include the development of rhabdomyolysis, muscle rigidity and myoglobinuria. Our patient had received a number of general anaesthesias in the course of previous operations, including inhalational anaesthetics and depolarizing neuromuscular blocking agents, without developing signs of malignant hyperthemia. This makes a predisposition for malignant hyperthermia unlikely. However, the diagnosis of malignant hyperthermia should be considered in obese patients as depolarizing neuromuscular blocking agents are frequently used for rapid sequence induction to prevent aspiration of gastric contents.

In conclusion, we present a case of rhabdomyolysis in a morbidly obese patient associated with acute renal failure, hepatic dysfunction, and an increase of cardiac troponin-1 after total knee arthroplasty. Given the fact that rhabdomyolysis occurs frequently in obese patients, intensified postoperative observation seems justified in these patients.

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