CASE REPORT

PEROPERATIVE DIAGNOSIS OF RHABDOMYOLYSIS

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ABSTRACT

Crush syndrome or traumatic rhabdomyolysis constitutes the systemic changes seen after crush injury. The pressure causes necrosis of muscle, and during revascularisation diffusion of calcium, sodium and water into the muscle cells is seen, together with loss of potassium, phosphate, lactic acid, myoglobin and creatinine kinase. Untreated these changes can lead to: hyperkalemia, acidosis, acute renal failure and hypovolemic shock. This case report describes a patient developing rhabdomyolysis following fracture of femur. It was diagnosed and treated early during the course of anesthesia.

Keywords: Crush injury, Rhabdomyolysis

RABDOMİYOLİZİN PEROPERATİF TANISI

ÖZET

'Crush' sendromu veya travmatik rabdomiyoliz 'crush' hasarı sonrası görülen sistemik değişiklikleri içerir. Basınç ve travmanın neden olduğu kas nekrozu ve revaskülarizasyon sırasında kalsiyum, sodyum ve su hücre içine girerken, potasyum, fosfat, laktik asit, miyoglobin ve kreatin kinaz hücre içinden hücre dışına çıkar. Tedavi edilmediği takdirde bu olaylar hiperkalemi, asidoz, akut böbrek yetmezliği ve hipovolemik şoka neden olur.
Bu olgu sunumu femur fraktürü sonrası rabdomiyoliz gelişen bir hastayı anlatmaktadır. Peroperatif erken dönemde teşhis edilmiş ve tedavi başlanmıştır.

Anahtar Kelimeler: Ezik yaralanmaları, Rabdomiyaliz

INTRODUCTION

Rhabdomyolysis is a potentially life-threatening syndrome resulting from the breakdown of skeletal muscle fibers with leakage of muscle contents into the circulation. The most common causes of traumatic origin are chrush injury, overexertion, seizures, and electric shock. Nontraumatic causes are myopathies, alcohol abuse, certain drugs and toxic substances[1]. Here we report a patient who developed rhabdomyolysis following fracture of the femur after a car accident.

CASE REPORT

A 66-yr-old man (weight 90 kg, height 180 cm) was scheduled for popliteal exploration and external-internal fixation of the fractured femur. His past medical history was unremarkable. The patient was struck by a car and was admitted to a nearby hospital where he had undergone explorative laparotomy. The lacerations in the jejunum and sigmoid colon were repaired and then the patient was transferred to our hospital. Any information regarding fluid replacement and the vital signs during the previous surgery could not be obtained. Chest radiography revealed fracture of the eleventh rib and contusion of the left upper lobe. Pulses from the left popliteal, dorsalis pedis, and tibialis posterior arteries were not detected. Computed tomography showed linear fracture at the base of the anterior fossa without any sign of epidural / subdural or intracerebral hematoma. All preanesthetic laboratory results and blood gas analysis were within normal limits except lactate dehydrogenase (LDH) (981; normal range 50-150U/L) (Table I). Anaesthesia was induced with thiopental, 5 mg/kg IV and fentanyl, 3µg/kg IV. Endotracheal intubation was facilitated with vecuronium

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Marmara Medical Journal 2005;18(1);35-38

bromide, 0.1 mg/kg IV. Anaesthesia was maintained with 1 MAC sevoflurane and 66 % nitrous oxide in oxygen. Monitoring included direct arterial blood pressure, central venous pressure (CVP), end-tidal carbondioxide, esophageal temperature, and urinary output. At the third hour of anesthesia laboratory values from arterial blood were as follows: hemoglobin 8.8 g/dl, normal platelet count, pHa,7.22; $Paco_2$, 45.7; Pao_2, 136.3; HCO3, 18.6; base excess, -8.9. Despite hydration with colloids and cristalloids the patient remained hypotensive throughout the surgery (90/60 mmHg), CVP was 3 mmHg and the heart rate was between 90-95/min. Urine output at the time was 200 ml (0.5 ml/kg/h). Due to ongoing hypotension and metabolic acidosis blood electrolytes were checked and were as follows: Na, 143 mEq/L; K, 6.6 mEq/L; blood-urea nitrogen (BUN), 32 mg/dl; creatinine, 1.42 mg/dl. There was no sign of hyperkalemia on the electrocardiography.

Because of suspected rhabdomyolysis, forced diuresis was induced with mannitol 20% (40 g) and, 20 mg furosemide intravenously. Hyperkalemia was treated with 10% dextrose with insulin. At the end of surgery estimated blood loss was 3300 ml and urine output 600 ml (1 ml/kg/h). 3000 ml of whole blood, 3000 ml isotonic saline and 1000 ml gelatin were administered during the course of the operation. After an uneventful surgery and anesthesia, patient was transferred to the intensive care unit (ICU) where his lungs were mechanically ventilated. Upon admission to the ICU,blood-gas analysis revealed severe metabolic acidosis (pHa, 7.05; Paco2, 47.2; Pao_2, 169; HCO_3, 12.8; base excess, - 17.5).Intravenous bolus and infusion of sodium bicarbonate were administered (total dose of 250 mEq). Oliguria developed and renal function continued to deteriorate (Table I). Dopamine, furosemide, and mannitol infusions were administered to maintain urine output. On the fourth postoperative day, venovenous hemodialysis was performed. He died of circulatory insuffiency on the sixth postoperative day.

DISCUSSION

Extensive skeletal muscle injury, whether caused by mechanical crush or by extreme physical exertion, is incompatible with life, unless treated early and vigorously. The immediate cause of morbidity is leakiness of the sarcolemmal membrane to cardiotoxic or nephrotoxic cations and metabolites (potassium, phosphate, lactic acid, creatine kinase, myoglobin and urate) of the sarcoplasma, and rapid massive uptake by the muscles of extracellular fluid, sodium, and calcium, leading to profound hypovolemic and

Table I: Laboratory Results

Laboratory tests	Normal range	Preop	Perop	PD-0	PD-1	PD-2	PD- 3	PD- 4	PD- 6
Na (mEq/L)	135-147	141	143	144	148	144	139	143	142
K (mEq/L)	3.5-5.0	4.1	6.6	5.0	4.52	4.15	3.9	5.1	5.4
BUN (mg/mL)	8-18	33	32	33	48	67	84	101	120
Cr (mg/mL)	0.6-15	1.36	1.42	1.55	1.87	3.31	4.74	5.81	6.32
LDH (U/L)	50-150	981		851					
AST (U/L)	0-35	140		195	3675	5770		866	89
ALT (U/L)	0-35	62		122	1696	1503		50	15
Aldolase (U/L)	3.1-7.1			3.1					
Ca (mg/dL)	8.5-10.5			6.3				5.4	6.3
CPK	40-174 U			4929		3400		2715	
Urine Output	_	_	600	1200	650	400	250	50	_
Urine Myoglobin	0-200 ng/ml			31000			25000		
Urine pH				6.0	5.5		5.0		
Urine Density				1030	1033		1015		
Urine Bilirubine	Neg			Neg	Neg		Neg		

PD-0: postoperative day 0; Na: sodium; K: potassium; BUN: blood-urea nitrogen; Cr: creatinine; LDH: lactate dehydrogenase; AST: aspartate transaminase; ALT: alanine transminase; Ca: calcium; CPK: creatinine phosphokinase, Neg: negative

hypocalcemic shock. Rhabdomyolysis occurs frequently but is usually asymptomatic (laboratory abnormalities only). However, in more severe cases, severe electrolyte disorders and acute renal failure may (ARF) occur, leading to life-threatening conditions. Rhabdomyolysis develop in any situation where energy demands in muscle exceed the available energy supply 2. Multiple complications can occur and are classified as early or late. Early complications include severe hyperkalemia that causes cardiac arrhythmia and arrest. The most serious late complication is ARF, which occurs in approximately 15 percent of patients with the syndrome. Patients who survive the early stage of hyperkalemia and arterial hypotension are susceptible to myoglobinuric ARF which is due to the combination of renal vasoconstriction, nephrotoxicity, and tubular obstruction by myoglobin plugs and urate[3,4]. Myoglobinuric renal failure accounts for 2-5 % of all cases of ARF in the ICU. It should be considered a possible cause in all cases of ARF of uncertain etiology. If the serum content of sarcoplasmic enzymes is very high (creatine kinase values 10000 to 50000 units) myoglobinuria must be the cause[5].-Crush injuries resulting in traumatic rhabdomyolysis are an important cause of ARF. Ischemia reperfusion is the main mechanism of muscle injury. Intravascular volume depletion and renal hypoperfusion, combined myoglobinuria, result in renal dysfunction. In our case underlying cause of severe rhabdomyolysis was crush injury and ischemia by vascular obstruction which contributed to the development of ARF with concomitant volume depletion and hypotension[6]. In a recent study de Meijer et al evaluated the risk factors for the development of ARF and demonstrated that in patients with severe rhabdomyolysis the level of serum creatine kinase levels predicted the development of ARF[7]. Management includes immediate intravenous volume replacement followed by mannitol-alkaline diuresis. The alkali regimen ameliorates the acidosis associated with shock and the hyperkalemia, and protects against the nephrotoxicity of myoglobin and urate by alkalinization of the urine[8].

In the present case, anesthesia was commenced with a standart approach. The patient was diagnosed having an open femur fracture of which an external and internal fixation of the bone together with arterial anastomosis was planned. The time past after the accident to the second operation was twenty-four hours. At the third hour of anesthesia the result of blood gas analysis revealed metabolic acidosis together with increased level of preoperative LDH we correlated with nothing but rhabdomyolysis. Blood chemistry showed that he was hyperkalemic at that time (6.6 mg/dL) without any electrocardiographic changes. Test result for blood in urine was negative. Urine output was 200 ml at the third hour. After then treatment initiated immediately, aiming at a rapid correction of the extracellular volume with crystalloids, colloids and blood products. Hyperkalemia was treated accordingly. Mannitol and furosemide were employed to promote renal tubular flow, flush myoglobin plugs, and to enhance urinary elimination of nephrotoxic metabolites. In our case, despite early diagnosis and vigorous treatment, the patient died of circulatory insuffiency on the sixth postoperative day. Since physiologic amputation is a treatment option that halts myonecrosis, prevents myoglobinuria, and lessens the risk of associated acute renal failure [9,] we speculate that he could have benefited the procedure.

The reason we report this case is that anaesthesiologists who do not deal with trauma cases on daily bases would easily skip the diagnosis. Besides, patient's preoperative history and clinical picture did not direct us toward the possibility of rhabdomyolysis. It was the first case in our institute of rhabdomyolysis detected after a fracture of femur. Metabolic acidosis detected after a coincidental blood-gas analysis was the harbinger of rhabdomyolysis. We tried to rule out the possible causes which might have led to metabolic acidosis in this patient. Rightafter we checked the serum potassium level. Increased potassium level together with metabolic acidosis detected peroperatively and the preoperative increased LDH rendered us to the diagnosis of rhabdomyolysis. Furthermore this case demonstrates the importance of a careful and continous monitoring in traumatized patients.

We would like to emphasize that fracture of the long bones and pelvic fractures may lead to crush injuries (rhabdomyolysis) although skeletal muscle injury seems to be minimal clinically. Early recognition of rhabdomyolysis and prompt management of complications are crucial to a successful outcome. If the patient continues to deteriorate physiologic amputation should be considered.

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