Acute Renal Failure (ARF) due to Rhabdomyolysis (RM) in Narcotic Drug Users (NDU)

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Introduction

Lysis of skeletal muscles resulting in extravasation of the intracellular toxic metabolites into the circulatory system and the accompanying manifestations, constitute the clinical syndrome of rhabdomyolysis (RM) (1). RM may occur in numerous clinical conditions that are separated into physical (trauma, occlusion of vessels, strainful muscle exercise, electrical current, hyperthermia) and non-physical (metabolite myopathies, drugs and toxins, infections, electrolyte abnormalities, endocrine disorders) (2). In peacetime, the main causes of RM are drugs and toxins. Among them, narcotic drugs (heroin, cocaine, etc) are common causative agents (3, 4).

After the occurrence of the causative factor, there is an influx of calcium in the myocyte that results in persistent muscle contraction, depletion of ATP and cell death. A large number of free radicals are produced and the migration of activated neutrophils triggers an inflammatory, self-sustaining myolytic reaction. Necrosis of the muscles results in the accumulation of large amounts of fluids in the affected area. Release of organic acids may cause acidosis. In the early stages, the accumulation of calcium in the affected area causes hypocalcemia and in later stages, the release of large amounts of calcium from the recovering tissue causes hypercalcemia. Massive myolysis causes hyperkalemia and hyperuricemia. The massive entrapment of fluids in the affected area results in serious hypovolaemia and occurrence of the so-called compartment syndrome that can lead to dangerous ischaemia of the local tissues.

Acute Renal Failure (ARF) is a serious and, sometimes, fatal complication of RM. It occurs in 8-20 % of RM incidents (5). The basic pathophysiologic mechanisms that can lead to ARF are: I) Renal vasoconstriction due to accumulation of large amounts of fluids in the "third space" and the diminution of circulating blood volume; II) Dehydration and renal vasoconstriction enhance water reabsorption and cause an increase of myoglobin concentration in the tubules resulting in myoglobin cast formation while hyperuricemia and massive urinary excretion of uric acid contributes to the formation of uric acid casts. Low urine pH due to the underlying acidosis favors the formation of myoglobin and uric acid casts. III) Iron and myoglobin can cause lipid peroxydation, formation of free radicals and renal injury (2,6-8).

In the present study we evaluated the severity of RM and ARF in narcotic drug users (NDU).

Materials and methods

Eleven patients (male 9, female 2, mean age 28.7±5.0 years) with RM associated with ARF were studied. They were classified in two groups according to the kind of narcotic drug they used; A: 6 users of heroin and B: 5 users of drug other than heroin. We estimated the severity of RM and ARF using objective clinicolaborative data. The severity of RM was evaluated by estimation, on admission, of serum CPK, SGOT, LDH, Phosphorus (P) and Calcium (Ca) and by the presence of paraplegia (PPL) and the severity of ARF by estimation of serum Creatinine (CR) and the presence of oligoanuria (OA), the days of hospitalization (DH), the total courses of hemodialysis (THD) and the total of pts who took blood transfusions (BT). The statistical analysis was performed with the use of uncorrelated t-test and p values less than 0,05 were considered to be statistically significant.

Results

The severity of RM on admission is shown in Table I. The CPK (U/L) mean values on admission were 135453±79144 for heroin users (Group A) and 39000±13638 for users of other narcotic drugs (Group B) ($p=0.045$). Mean SGOT (U/L) for Group A was much higher than that for Group B (1868±1190 vs. 390±152, $p=0.042$). Mean LDH (U/L) values (25542±20873 vs. 1660±1425, $p=0.035$) denounced a higher grade of cell destruction in heroin users. Mean phosphorus (P) levels (mg/dL) were substantially higher in the group of heroin users (9.32±1.35 vs. 5.58±2.05, $p=0.005$). Hypocalcemia (mg/dL) was statistically more severe in Group A (6.60±0.94 vs. 7.80±0.52, $p=0.032$). Finally it was noticed that 4 patients of group A were admitted with paraplegia and no patient from group B presented with any kinetic loss ($p<0.05$).

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Table I. Severity of RM

	CPK (U/L)	SGOT (U/L)	LDH (U/L)	P (mg/dL)	Ca (mg/dL)	PPL (n)
Group A	135453±79144	1868±1190	25542±20873	9.32±1.35	6.60±0.94	4
Group B	39000±13638	390±152	1660±1425	5.58±2.05	7.80±0.52	0
p	0.045	0.042	0.035	0.005	0.032	<0.05

Table II. Severity of ARF.

	CR (mg/dl)	OA (n)	DH	THD	BT (n)
Group A	6.28±0.75	5	25.0±11.52	9.0±5.51	5
Group B	3.28±3.04	1	9.4±7.92	1.81±4.02	1
p	0.042	<0.05	0.031	0.038	<0.05

We also estimated the severity of ARF, on admission, of the two groups of patients (Table II). The Renal Failure was more severe in Group A than in Group B [CR (mg/dL) 6.28±0.75 vs. 3.28±3.04, p=0.042]. Oligoanuria (n) was much more common for the pts of Group A (5 vs. 1, p=0.042). A more extended period of hospitalization was necessary for Group A patients (25.0±11.52 days vs. 9.4±7.92 days, p=0.031). Patients of Group A underwent a bigger number of hemodialysis courses than patients of Group B (9.0±5.51 vs. 1.81±4.02, p=0.038). The total of patients who took blood transfusions was higher in Group A than in Group B (5 vs. 1, p<0.05).

Conclusions

The data demonstrated that RM and the resulting ARF are more severe in heroin users than in users of other narcotic drugs possibly due to a direct myotoxic effect of heroin apart from the factor of compression that can cause RM in any type of narcotic drug use (2,9,10).

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