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Case reports

Plasma exchange in rhabdomyolysis

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Abstract. The effect of plasma exchange (PE) was evaluated in 4 patients with rhabdomyolysis. A single 2 l PE produced a transient fall of creatinine phosphokinase and did not prevent renal failure. Theoretically PE would need to be performed very frequently to remove toxins in appropriate amounts. Since renal failure in myoglobinuira has a relative good prognosis, we do not recommend intensive PE as therapy.

Key words: Rhabdomyolysis – Myoglobinuria – Acute renal failure – Plasma exchange

Rhabdomyolysis may be associated with myoglobinuria and acute renal failure (ARF). Causes of muscle cell injury include trauma, muscle ischemia, toxin exposure, infections and myositis. The number of recognized diseases associated with rhabdomyolysis is increasing rapidly [1]. It seems probable that any process interfering with energy utilization of muscle tissue may predispose to muscle cell injury or necrosis.

Several factors are important in the pathogenesis of pigment nephropathy. They include: (a) tubular obstruction following precipitation of high molecular weight heme proteins and (b) direct nephrotoxicity of heme pigments, in particular in the presence of systemic acidosis and/or decreased renal blood flow [1].

Volume expansion, alkalinization and forced diuresis are procedures usually applied to protect the kidney in pigment nephropathy. Plasma exchange (PE) has been recommended as an additional form of therapy in rhabdomyolysis and myoglobinuria [2]. It has been suggested that PE may postpone or even prevent ARF by removing myoglobin from the plasma. The literature concerning PE in rhabdomyolysis is scarce and no controlled studies exist. In this paper we report the effects of PE in four patients. The results show that a single 2 l PE has no beneficial effects in the treatment of patients with rhabdomyolysis.

Case reports

Patient 1. A 38-year-old woman was admitted with a compartment syndrome of the left leg complicated by rhabdomyolysis. Fasciotomy was performed on the day of admission and on the same day she developed ARF due to myoglobinuria. The exchange of 2 l plasma resulted in a transient decrease of creatinine phosphokinase (CPK) (Fig. 1, Table 1). Renal function returned to normal within 20 days and during this period hemodialysis was needed twice.

Patient 2. A 15-year-old boy with crushed legs following a road traffic accident was transferred to our hospital for osteosynthesis. Myoglobinemia and myoglobinuria were detected on admission. Renal function remained normal during his stay in hospital of 6 weeks. A 2 l PE was performed once and produced a transient fall of CPK (Table 1).

Patient 3. A 64-year-old man was transferred to our hospital because of severe Legionella pneumonia. Mechanical ventilation was begun and he developed ARF due to massive myoglobinuria.

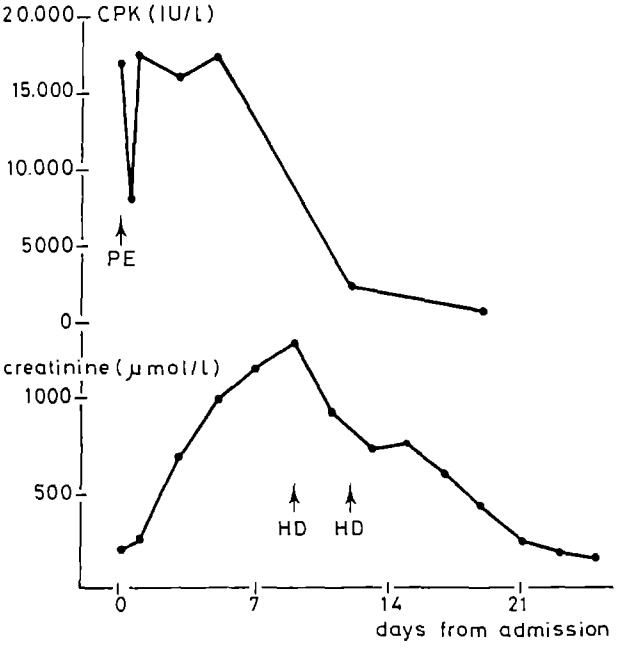

Fig. 1. Course of serum creatinine and CPK in patient no. 1. *HD*, hemodialysis; *PE* plasma exchange

Table 1. Effects of plasma exchange on myoglobin and CPK levels in four patients with rhabdomyolysis, including two with systemic acidosis

Case	Day	CPK IU/l	Myoglobin IU/l	Blood pH	Bicarbonate mmol/l
1	1 pre PE	17500	ND	7.31	16
	post PE	7850			
	2	18000			
2	1 pre PE	15700	5–10000	7.38	22
	post PE	4650	500–1000		
	2	8700	1–5000		
3	1 pre PE		25000	7.31	18
	2		25000		
4	1 pre PE	55000	100000	7.37	24
	post PE		10–100000		
	2	49000	$>10^6$		

ND = not determined

Hemodialysis was performed 7 times and PE was carried out once using a 2 l exchange. Renal function recovered completely with normal laboratory values within 25 days.

Patient 4. A 19-year-old boy was admitted to hospital after a road traffic accident. Osteosynthesis was needed to set several fractures. Myoglobinuira was diagnosed. A single 2 l PE was performed. Over the following days the serum myoglobin rose to values exceeding 1 000 000. Nevertheless, renal function remained normal.

Discussion

Acute renal failure due to myoglobinuria is a well known complication of rhabdomyolysis. The potential nephrotoxicity of myoglobin and its derivatives has been studied extensively. It is generally accepted that heme pigments are relatively non-nephrotoxic in themselves, but are highly nephrotoxic when renal ischemia and or systemic acidosis coexist [1]. It is recommended that urinary alkalinization is induced and volume expansion with forced diuresis is performed to prevent ARF. Beneficial effects from this regime have been reported by Ron et al. [3]. Several authors have stressed the good prognosis of ARF in rhabdomyolysis provided appropiate medical and surgical management is instituted promptly [3, 4].

It has also been recommended that PE is carried out to remove myoglobin from the plasma in order to prevent renal failure. Myoglobin is a low molecular weight protein (17 800 dalton) and is distributed over the intra- and extra-vascular compartments. The volume of distribution of myoglobin is about 28.5 l in a normal person [1]. If a myoglobin concentration of Y mg/ml is found, then the total amount of myoglobin would by $Y \cdot 1000 \cdot 28.5$ mg. One PE of 2 l will remove $Y \cdot 1000 \cdot 2$ mg, which represents only 8% of the total amount of myoglobin. If each procedure removes 8% of the amount of myoglobin present at the beginning of each procedure, then at least 9–10 PE procedures are needed to obtain a substantial 50% reduction, assuming that the release of myoglobin by the damaged muscles stops. Successful treatment of ARF due to myoglobinuria in two patients was reported by Kuroda et al. [2]. A PE of 3 l was performed three times in each patient. Despite this both patients developed ARF with complete recovery of renal function over the following two to three weeks.

In our patients a PE of 2 l was performed once. As expected, CPK and myoglobin values were lowered, but rapidly returned to pretreatment values after PE, probably as a result of redistribution between the extra- and intravascular compartments [5]. Such a transient fall of myoglobin has been observed previously by Freysz et al., who performed PE in a patient with rhabdomyolysis [6]. Despite PE, 2 of our patients (no. 1 and 3) developed ARF necessitating hemodialysis. Renal function recovered after 10–20 days. Patients 2 & 4 maintained normal renal function throughout, even in the presence of extremely high levels of myoglobin in one case (patient 4). The difference may be explained by a systemic acidosis, present in patients 1 & 3, which is known to provoke the nephrotoxicity of myoglobin. A beneficial effect of PE was not seen in our patients. Theoretically, to obtain a substantial decrease of myoglobin levels, PE should be performed very frequently. As ARF due to myoglobinuria has a good prognosis, an intensive PE program, with daily PE for more than a week, seems to be an excessively aggressive therapy. Such an approach may induce other problems such as increased risk of infection and bleeding due to hypogammaglobulinemia and the removal of coagulation factors. For these reasons we do not recommend PE as an additional therapy in patients with rhabdomyolysis.

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