ELEVATION OF CREATININE PHOSPHOKINASE IN HEAT SYNDROME

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SYNOPSIS

Enzymes (CPK, LDH, SGOT) were measured in ten patients diagnosed to have heat disorders viz, heat cramps, heat exhaustion or heat stroke. Creatinine phosphokinase was found to be elevated in 7 out of 10 patients. CPK-MB isoenzyme was elevated in 2 out of 4 cases measured. The significance of these results is discussed.

INTRODUCTION

Heat syndrome can be classified as heat cramps, heat exhaustion and heat stroke. The more severe forms are known to cause myocardial injury (1). which can be diagnosed clinically, electrocardiographically and by raised enzymes such as creatinine phosphokinase (CPK), lactate dehydrogenase (LDH) and glutamic oxaloacetic transaminase (SGOT) (2).

These enzymes have also been found to be raised after excercise especially marathon running (3,4,5,6). As heat injuries are normally precipitated by strenous or unaccustomed exercise in hot and humid environment, the raised enzyme levels may be due to the exercise rather than the heat injury per se. In this study, we report the levels of CPK, LDH and SGOT in ten patients with heat syndrome.

MATERIAL AND METHOD

Ten patients who were admitted for heat syndrome to the Department of Medicine, Toa Payoh Hospital between 1st April to 30th June 1983 were included in the study. All the patients had history of being engaged in strenuous physical activity such as running, marching, manual work under hot, humid environmental condition prior to their admission. Rectal temperature was recorded on arrival. Blood specimens were taken for CPK, LDH, SGOT and electrocardiograms were performed on all the patients on admission. As CPK-MB isoenzyme estimation was not routinely available, it was performed in four cases after the initial CPK levels were known to be elevated. The patients were categorised as heat cramps, heat exhaustion or heat stroke according to currently established criteria[7].

RESULTS

All the patients were males with the age ranging from 18 years to 55 years (Table 1). The core temperature on admission ranged from 37 °C to 41 °C. CPK was elevated in 7 of the patients. Patient 10 had the highest CPK level recorded in Singapore. The isoenzyme CPK-MB was performed in 4 patients and was present in 2 patients. LDH was elevated in 5 patients while SGOT was elevated in 4 patients. All the patients had normal ECG's.

TABLE 1: SERUM ENZYME LEVELS IN HEAT SYNDROME

No.	Patient	Sex	Age (Years)	Diagnosis	Rectal Temp.	CPK (u/l)	CPK-MB	LDH (u/l)	SGOT (u/l)
1	W.K.F.	M	25	Heat Cramps	37.5 °C	167	–	254	24
2	N.B.A.	M	19	Heat Cramps	37.5 °C	50,560	–	2,144	953
3	T.C.Y.	M	55	Heat Exhaustion	37 °C	1,081	–	380	27
4	N.H.S.	M	18	Heat Exhaustion	37.5 °C	3,176	Neg	528	55
5	L.K.S.	M	19	Heat Exhaustion	38 °C	206	–	359	29
6	K.T.H.	M	19	Heat Exhaustion	38 °C	172	–	381	26
7	W.Y.F.	M	19	Heat Exhaustion	40.8 °C	786	20	491	38
8	W.Y.C.	M	18	Heat Exhaustion	41 °C	242	23	525	23
9	R.M.	M	18	Heat Stroke	40.8 °C	3,534	–	380	27
10	O.M.H.	M	18	Heat Stroke with DIVC	40 °C	187,980	Neg	12,228	> 999

Normal Range: CPK 40-210 u/l
LDH 180-380 u/l
SGOT 15-33 u/l

DISCUSSION

Creatinine phosphokinase is an enzyme in skeletal muscle, heart and brain that catalyses a reaction that provides high-energy phosphate early in the anaerobic phase of organ function. It has three iso-enzymes: CPK-MB, CPK-BB, CPK-MM and is found elevated in many conditions that include hypothyroidism, myocardial infarction, polymyositis, myopathies, and heat stroke. Kew[8] in 1971, concluded that SGOT and LDH were specific in the diagnosis and prognostication of heat stroke, while CPK, though elevated in most cases of heat stroke was also found to be marginally elevated in healthy miners where it was attributed to strenuous physical exertion and minor muscle trauma. Thus an elevated CPK level in a patient with heat injury could be due to the heat injury or the preceding physical exertion. In our present series of ten patients, CPK was found raised in both cases of heat stroke, 1 case of heat cramps and 4 cases of heat exhaustion. The elevated CPK levels in heat injury could be due to the strenuous physical activity. Support for this hypothesis could be found in several studies done on marathon runners (3,4,5,) and less strenuous exercise such as swimming, weight lifting and short distance running (6).

It has been generally assumed that the increase in total CPK after exercise was entirely of the MM faction derived from skeletal muscles (3,5). Other studies, however, have shown that CPK-MB (4,9,10,11) and even CPK-BB (10) could be raised although CPK-MM faction showed the largest rise. In our present series where the CPK-MB isoenzyme was measured in 4 cases, it was raised in 2 cases.

Human myocardium contains up to 20% of total creatinine phosphokinase activity in the MB faction, in contrast to trace amount, at most, in normal skeletal muscle. Although elevated serum CPK-MB has the highest predictive accuracy of any biochemical indicator for myocardial necrosis and is widely used as the standard for diagnosis of such injury[12], it is by no means specific as it has been found to be elevated in skeletal muscle disorders such as dermatomyositis[13] muscular dystrophy[14] and alcoholic rhabdomyolysis[15]. Even after exclusion of the above conditions, an elevated CPK-MB isoenzyme, in people with heat injury or post-exercise, could not be taken as definite evidence of myocardial damage. Siegel (9) reported normal myocardial scintigrams in twelve marathon runners with raised CPK-MB and concluded that the CPK-MB could have come from non-cardiac source.

In conclusion, elevated CPK and CPK-MB, can be found in heat injured patients and may be due to the heat injury or physical activity, and should not be assumed to indicate myocardial damage in the absence of other evidence.

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