

BIOCHEMICAL PROFILE CHANGES DURING EXERTIONAL HEAT STROKE

D.S. Moran, Y. Epstein and Y. Shapiro

Heller Institute of Medical Research, Sheba Medical Center, Tel-Hashomer 52621, Department of Physiology, Sackler Faculty of Medicine, Tel-Aviv University, Israel.



INTRODUCTION

Exertional heat stroke (EHS) is a state of extreme hyperthermia that occurs when excess heat generated mostly by muscular exercise exceeds the ability of the body to dissipate it at the same rate. EHS presents an ergonomic maladaptation between environmental condition and body activity. The severity of the illness depends on the degree of maladaptation and the resultant level of hyperthermia and its duration. It is a life-threatening medical emergency caused by the failure of the thermoregulation system and is diagnosed by measuring core temperature above 40°C. EHS is associated with leukocytosis. Hypokalemia is found in the early stages of EHS (1) and may later develop to hyperkalemia as a result of acidosis. Hematocrit (Hct) values are elevated, and sodium (Na⁺) levels might be increased due to dehydration. Hypocalcemia and hypomagnesemia may occur as a result of increased loss of calcium and magnesium in urine and/or sweat (2). Elevation of serum creatine phosphokinase (CPK) and serum glutamate oxaloacetate transaminase (sGOT) enzymes are considered in EHS. However, EHS is often misdiagnosed, especially because many of the first signs are nonspecific and associated with loss of consciousness (3-5). The purpose of this study was to examine and follow the biochemical profile changes, which occur in EHS patients, during the period of 96 h from the admittance to the emergency room.

METHODOLOGY

Biochemical profiles of 50 EHS patients at its acute phase on admission to the emergency room were analyzed at our institute. The follow-up of the patients and their biochemical profiles was for 48 to 96 h. All the patients were young males, highly motivated, healthy soldiers during the years 1988-1996. The physical characteristics of the patients are summarized in Table 1. Most of the heat strokes occurred during marches and runs while the soldiers were at different

Table 1. Physical characteristics of the 50 EHS patients

	age (yr)	weight	height (cm)	BSA (m ²)	BMI (kg·m ⁻²)
Mean ± SD	20 ± 3	72.11-10.1	176 ± 6	1.89 ± 0.15	22.9 ± 2.9
range	18-25	47-104	159-189	1.55-2.27	14.9-31.9

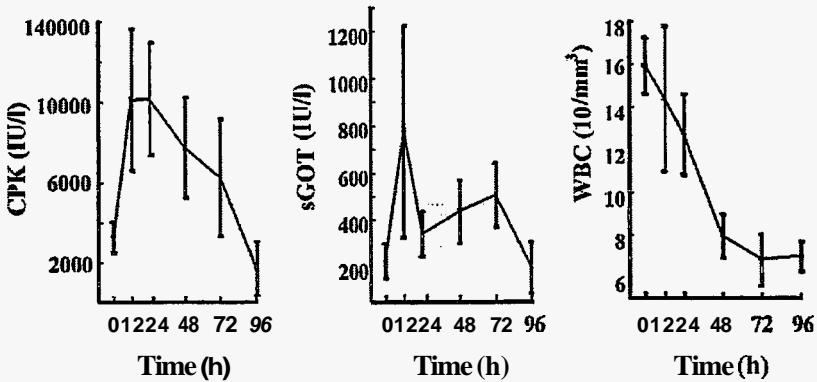


Figure 1. Changes in the profile of CPK, sGOT and WBC in 50 EHS patients.

basic training courses where exercise intensity was unmatched to the soldiers' capacity and fitness.

Analyzing the biochemical manifestations upon admission to the hospital revealed that in more than 50% of the patients no conclusive abnormalities could be detected. However, all the biochemical results were in a very wide range as depicted in Figures 1-2 and not necessarily with correlation between them. Mean values of hemoglobin (Hb), potassium (K⁺), Na⁺, Hct and glucose were within normal, but mean values of white blood cells (WBC) and enzymes (CPK and sGOT) were elevated as depicted in Figure 1.

DISCUSSION

In many EHS cases, Tre is not measured on site. In some cases, although Tre probably exceeds the critical temperature at the moment of collapse, heat illness might not be diagnosed. The discrepancies in Tre between the measurements may be significant and Shibolet et al. (6) reported a 3.3°C difference in Tre on site of collapse and at the emergency room. This may lead to a misdiagnosis of the real condition. In addition, many of the EHS patients arrive at the emergency room after being treated on site. The treatment, which usually includes fluid replacement, will result in a hematological picture within a normal range. Specific dynamics were noted for CPK and sGOT. The former peaked at 24 h of hospitalization and the latter peaked at 12 h and then at 72 h. The first sGOT peak is concomitant with rhabdomyolysis and correlates with elevated CPK. The second peak of sGOT reflects an acute hepatic dysfunction that develops in some cases during a later phase of exertional heat stroke. The results from this study, along with other works, confirm that peak values of CPK occur 24 to 48 h after EHS collapse (7). Although the dynamics in CPK levels are not pathognomonic, it is mostly helpful in differential diagnosis of heat stroke because in most other febrile states enzyme levels will be within a normal range. Moreover, since CPK levels correlate with the severity of the medical condition it is also a good indicator of the level of this ergonomic maladaptation.

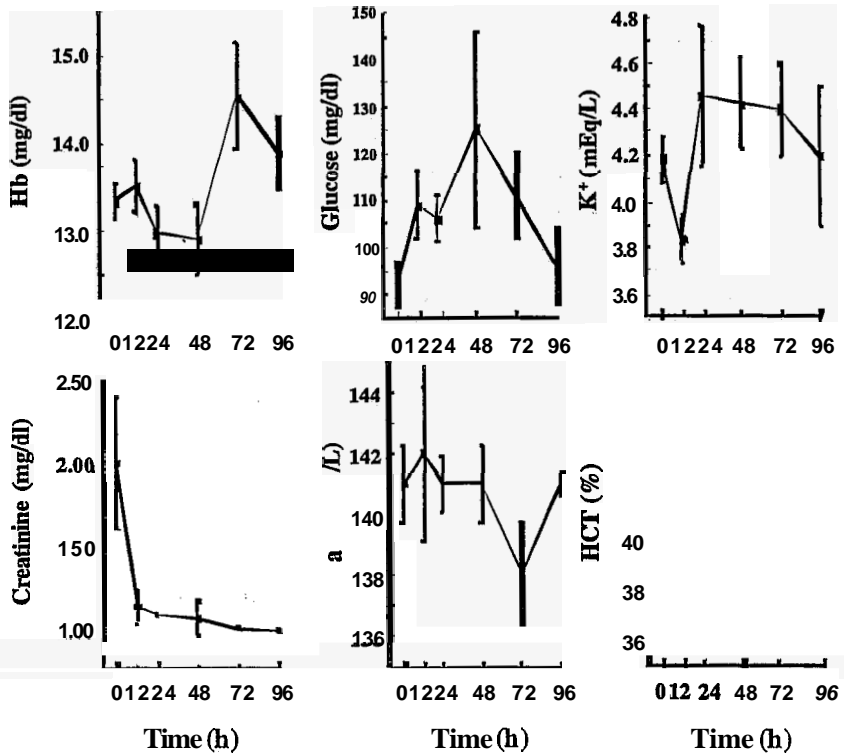


Figure 2. Changes in the profile of Hb, K⁺, Hct, creatinine, Na⁺ and glucose in 50 EHS patients.

SUMMARY

The biochemical picture upon admission to the emergency room can be misleading in the diagnosis of EHS. This is attributed mainly to a large range in the hematological and enzymatic picture during the first hours of the event. It is suggested that suspected heat stroke patients be hospitalized for 48 h, during which the dynamics of the various parameters will be clarified, and together with the nature of the event and other background factors, will substantiate the diagnosis of EHS.

REFERENCES

1. Hart, G. R., Anderson, R. J., Crumpler, C. P., Shulkin, A., Reed, and G., Knochel, J. P. 1982, Epidemic classical heat stroke: clinical characteristics and course of 28 patients, *Medicine*, 61, 189-197.
2. Shapiro, Y. and CristaIN. 1987, Hyperthermia and heat stroke: effect on acid-base balance, blood electrolytes and hepato-renal function, in J.R.S. Hales and D.A.B. Richards (eds.), *Heat Stress: Physical Exertion and Environment*, (Amsterdam: Elsevier Science), 289-296.

3. Shapiro, Y. and Seidman, D.S. **1990**, Field and clinical observations of exertional heat stroke patients, *Medicine and Science in Sports and Exercise*, **22**, 6-14.
4. Savdie, E., Prevedoros, H., Irish, A., Vickers, C., Concannon, A., Darveniza, P., and Sutton, J. R. **1991**, Heat stroke following rugby league football, *Medical Journal of Australia*, **155**, 636-639.
5. Shibolet, S., Lancaster M.C. and Danon, Y. **1976**, Heat stroke: a review, *Aviation, Space, and Environmental Medicine*, **47**, 280-301.
6. Shibolet, S., Coll, R., Gilat, T. and Sohar, E. **1967**, Heatstroke: its clinical picture and the mechanism in 36 cases, *Quarterly Journal of Medicine*, **36**, 525-548.
7. Epstein, Y., Sohar, E. and Shapiro, Y. **1995**, Exertional heat stroke: a preventable condition, *Israeli Journal of Medical Science*, **31**, 454-462.

