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LETTERS TO THE EDITOR

Acute liver failure is frequent during heat stroke

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Abstract

Acute liver failure (ALF) is relatively frequent during heat stroke (HS). This risk must be emphasized, because its incidence is higher than is usually thought. In a recent study by Weigand *et al*, two cases were reported in which liver failure was the leading symptom. We have confirmed their conclusion in a study of 25 cases of HS with ALF, compared with 25 other cases without ALF. Moreover, we observed that hypophosphatemia on admission could predict occurrence of ALF during HS. As for clinical and other biological parameters, phosphatemia should be monitored for at least 3 d in all cases of HS, even when it is thought to be mild.

Key words: Heat stroke; Hypophosphatemia; Liver failure

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LETTER TO THE EDITOR

We were particularly interested in the excellent study by Weigand *et al*[1], who reported two cases of acute liver failure (ALF) after heat stroke (HS), one during a half marathon [exertional heat stroke (EHS)], the other classical, due to excessive environmental heat, and complicated by multi-organ failure. In addition to severe rhabdomyolysis, each patient was characterized by marked elevation of liver enzymes. Liver injury and ALF are known complications of HS[2]: serum alanine aminotransferase (ALAT) level usually rises within 30 min after HS and peaks within 3-4 d. The risk of ALF during HS must be emphasized, because its incidence appears to be higher than is indicated in this study. Irreversible ALF is rare and may require liver transplantation[3].

We have already reported some of our clinical data[4,5], from a cohort of 50 male subjects (which now consists of 110 cases); all investigated after EHS were confirmed by clinical and biological data, study of muscle metabolism by magnetic resonance spectrophotometry, muscle biopsy for pharmacodynamic tests, and pathology. Twenty-five of these consecutive patients (aged 25 ± 4 years) with EHS and ALF were compared with 25 other EHS patients, who were age-matched but without ALF (ALAT 3563 ± 1313 *vs* 590 ± 742 IU/L; factor V 30% ± 12% *vs* 66% ± 20%; $P < 0.001$ for each). ALF was defined as ALAT > 10 times the upper limit, and coagulopathy (factor V lower than 50%).

Age, body mass index, physical fitness, background, climatic conditions, drug and alcohol intake, clinical manifestations and laboratory findings were analyzed with reference to their prognostic significance in ALF. A logistic regression model was used. In 22 of 25 patients, ALAT level returned to normal within 10 d, but three patients died of ALF. Univariate analysis found that poor fitness ($P = 0.02$), hygrometry > 86% ($P = 0.03$), creatininemia > 160 μmol/L ($P < 0.001$) and hypophosphatemia < 0.5 mmol/L ($P < 0.001$) were significant predictors of ALF. In multivariate analysis, on admission, hypophosphatemia < 0.5 mmol/L was the only independent predictive factor of ALF (RR 3.8, 95% CI 1.1-6.2).

In conclusion, ALF is not uncommon in EHS of which we assert, as Weigand *et al*[1], that this one is an underestimated cause.

This ALF is strongly associated with early hypophosphatemia, of which a value < 0.5 mmol/L is predictive. However, there is no evidence that hypophosphatemia by itself causes important liver dysfunction[6]. Massive liver cell necrosis results from thermal shock, circulatory disruption, endotoxinemia (heat sepsis), high blood concentration of cytokines and acute-phase proteins.

Therefore, in EHS, measurement of phosphatemia, ALAT and factor V should be systematic on admission, and 3-4 d later. Phosphorus supply, usual in an intensive care unit, has not been evaluated in this situation.

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