Heat stroke may appear as a result of exposure to high environmental temperature or strenuous exercise. It represents a medical emergency characterized by an elevated core body temperature and multi-organ failure. We have described a case of a 41-year-old female after sun exposure, who was admitted to the hospital with the temperature of 42°C. Because of high plasma bilirubin level the Molecular Adsorbent Recirculating System (MARS) was started. Three sessions of MARS, which lasted for eight hours each, were conducted on 15th, 18th, and 23rd day of hospitalization. The procedure was well tolerated by the patient and resulted in a sustained decline of plasma bilirubin from 33.5 to 14.7 mg/dl. The female was discharged from the hospital in good general condition. The two months follow up showed that the patient felt very well, and the plasma bilirubin was reduced to 2.2 mg/dl.

Introduction

Heat stroke (HS) may appear as a result of exposure to high environmental temperature or strenuous exercise. It represents a medical emergency characterized by an elevated core body temperature and multi-organ failure (MOF) [2,3].

We have described a case of a 41-year-old female, who was admitted to the hospital because of HS caused by prolonged sun exposure.

Case report

In July 2006, a 41-year-old female with epilepsy in anamnness was admitted to the hospital because of coma, and hyperpyrexia. The patient was found unconscious on the beach where she was taking a sun bath. The female was completely obtunded with a towel which had prevented skin burns.

The approximate sun exposure time was about 7 hours. At the time of admission to the hospital the patient was in deep coma (Glasgow Coma Scale 3), blood pressure was 100/40 mmHg, MAP 60 mmHg, pulse 110/min, respiratory rate 32 breaths/min, and temperature 42 degrees of Celsius (107.6 degrees of Fahrenheit).

Four hours of intensive external cooling and intravenous fluids decreased the body core temperature to 36.8°C although there was no improvement in the patient’s neurological condition.

Chest X-ray showed signs of acquired respiratory distress syndrome (ARDS), with pH 7.24; HCO₃ 11.4 mEq/l; pCO₂ 17.2 mmHg; pO₂ 45.6 mmHg, and NZ - 15 mEq/l; lactate 7.8 mmol/L. Because of progressive respiratory insufficiency the patient was intubated and mechanical ventilation was applied for seven days. There was also a sign of disseminated intravascular coagulation (DIC) with decrease in platelets count, and increase in INR from the beginning of hospital admission. We did not notice any significant hemodynamic instability of the patient and resulted in a sustained decline of plasma bilirubin from 33.5 to 14.7 mg/dl. Because of high plasma bilirubin level the Molecular Adsorbent Recirculating System (MARS) was started. Three sessions of MARS, which lasted for eight hours each, were conducted on 15th, 18th, and 23rd day of hospitalization. The procedure was well tolerated by the patient and resulted in a sustained decline of plasma bilirubin from 33.5 to 14.7 mg/dl. The female was discharged from the hospital in good general condition. The two months follow up showed that the patient felt very well, and the plasma bilirubin was reduced to 2.2 mg/dl.
Discussion

Heat stroke is a potentially fatal disorder that is caused by an extreme elevation of body temperature [2,3,6]. The pathophysiology of HS is not fully understood, however, endotoxins and cytokines appear to play a major role in the central nervous system and in the peripheral tissues.

According to Francesconi et al., in both classical and exertional heat strokes there is an important role of potassium and potassium depletion [4]. Protracted K+ deficiency, extreme hyperthermia, dehydration, and excessive exertion may reduce membrane potentials and conductance, futile cycling of the Na-K pump with concomitant energy depletion and highly increase metabolic heat production, reduced arteriolar vasodilation, altered neurotransmitter release, or cell swelling, each of which could contribute to the pathophysiology of heat injury [4]. Other risk factors for HS include age, period of exposure to high temperature, and obesity [7].

Slightly elevated liver enzymes, lacking clinical significance, seem to be quite frequent in the heat stroke, whereas severe, and clinically relevant hepatocellular injury has been observed in only a minority of cases [3].

In our patient there was a severe multi-organ failure with deep coma, ARDS, DIC, rhabdomyolysis and developed acute renal failure. The ALF in our case could be connected both with direct thermal injury of the hepatocytes but also hypovolemic shock with liver cell hypoxia. The first mechanism can be connected with high body temperature of our patient whereas the second is probable because of little delay of increase in transaminases level.

We noticed a good response to intensive supportive therapy, however, after an initial improvement there was a constant increase of plasma bilirubin accompanied by deterioration of patient’s neurological state.

According to the medical literature there is no agreement for the most effective treatment of ALF caused by heat stroke, and there are unanimous criteria pointing to the right stage in which to conduct the liver transplantation (LTx) [2,5].

Some authors present patients who fulfilled the London and Clichy criteria for LTx but recovered spontaneously without the procedure. In contrast, the outcome of some of the emergency liver transplantation was dismal [1,3,5,8]. Other authors propose continuous veno-venous hemofiltration or intermittent haemodialysis but the efficacy of these procedures is not encouraging [1,6].

The authors agree with Biais et al. that in some cases the introduction of Molecular Adsorbent Recirculating System may improve the patients state, and prevent the necessity of LTx [2].

References


Table I

Biochemical results of the patient.
Wyniki badań biochemicznych pacjentki.

<table>
<thead>
<tr>
<th>Biochemical tests results</th>
<th>Days of hospitalization</th>
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<tr>
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<tr>
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