Evidence of Prolonged Myocardial Dysfunction in Heat Stroke*

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Heat stroke is a life-threatening syndrome of multi-organ dysfunction caused by elevated body temperature. It may result from failure of the thermoregulatory mechanisms or from inadequate heat dissipation and affects members of several risk groups. A number of authors reported cardiac involvement in heat stroke, but heart failure rarely has been documented. We report the cases of two heat stroke victims, one of whom developed pulmonary edema and the other, peripheral edema. In both, RVG demonstrated dilatation and diffuse hypokinesis of the right ventricle, which persisted for several weeks. We conclude that the heart may be involved in heat stroke and that heart failure is a potential complication of the syndrome that can have a prolonged subclinical course. Fluid replacement in heat stroke should be done under careful observation, which may be facilitated by noninvasive assessment of cardiac function.

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Heat stroke is a potentially fatal condition affecting mainly military recruits, athletes, laborers, alcoholics and the elderly, when exposed to high temperatures.1 Although clinical evidence of cardiac failure is rare in heat stroke, the heart may be involved in multi-organ damage, as has been demonstrated pathologically,2 enzymatically and electrocardiographically.3 We describe two cases of heat stroke complicated by heart failure, in which prolonged myocardial dysfunction, mainly right-sided, was demonstrated by RVG.

CASE REPORTS

CASE 1

An 18-year-old military recruit was admitted to our intensive care unit for suspected heat stroke. He had been in vigorous health and had participated in very strenuous training without difficulty. The patient's past medical history was totally unremarkable and he had been receiving no medications. On the morning of admission, the patient performed physical training in ambient temperature exceeding 40°C. During the morning hours he had two episodes of diarrhea; soon afterwards he complained of extreme fatigue and lightheadedness, and stopped exercising. Upon examination by a physician shortly thereafter, the rectal temperature was found to be 39.7°C. A presumptive diagnosis of heat exhaustion was made; treatment with intravenously administered fluids and cooling by means of ice-cold sponges and fanning was initiated; nevertheless, the rectal temperature increased and reached 40°C. He was transferred to our hospital, having received 6 L of Hartmann's solution in approximately four to five hours.

On admission, the patient was found to be prostrated, dyspneic and somnolent. The blood pressure was 110/70 mm Hg; the pulse, 128 beats per minute and regular; and the rectal temperature, 38.4°C. Positive physical findings included fine rales over both lung bases, rapid heart sounds without gallop, and a distended, mildly tender abdomen. The ECG revealed only sinus tachycardia. The chest x-ray film showed pulmonary edema and an abdominal film demonstrated marked colonic distention.

The urinalysis results were normal. The hemoglobin value was 13.9 g/dL and the white blood cell count, 8,400/cu mm. The platelet count was 96,000/cu mm. The sodium value was 139 mmol; the potassium level, 3.6 mmol; the calcium value, 2.14 mmol; the phosphorus level, 0.85 mmol; and the urea value, 4.3 mmol/L (10 mg/dL). The creatinine value was 95 µmol/L (1.04 mg/dL) and the CK, 766 µL. Liver enzymes, serum protein, LDH, glucose and uric acid values were normal. The prothrombin time was 16.3 s with a control value of 11 s, and the partial thromboplastin time was 54 s. Fibrinogen and fibrinogen degradation products were normal. A specimen of arterial blood, drawn while the patient was breathing oxygen through nasal prongs at a rate of 3 L/min, showed that the partial pressure of oxygen was 51 mm Hg, the partial pressure of carbon dioxide was 33 mm Hg, and the pH was 7.41. A central venous line was inserted and the central venous pressure was found to be 11 cm H₂O.

A diagnosis of heat stroke complicated by pulmonary edema was made and the patient received intravenous furosemide and a CPAP mask. External cooling was continued for two hours, after which the rectal temperature reached 37°C. With this treatment, the patient's condition improved over a few hours, although he remained exhausted and sleepy; the hypoxemia resolved upon institution of CPAP; the patient had a brisk diuresis and the pulmonary congestion subsided. The central venous pressure remained elevated (10 to 15 cm H₂O) for 24 hrs, at which time the line was removed. Ten hours after admission (at which time the pulmonary congestion was considerably improved) RVG demonstrated a LVEF of 72 percent with normal regional wall motion; the right ventricle was dilated (right ventricular to left ventricular diastolic volume ratio of 2.5) and the RVEF was only 32 percent (Fig 1).

The patient was discharged after an uneventful recovery. A repeat RVG 16 days after admission showed a LVEF of 61 percent with mild apical hypokinesis; the RVEF was 43 percent and the right ventricle was still somewhat dilated (the diastolic volume ratio was now 1.2). Twelve weeks later, another RVG demonstrated normal size and contraction of both ventricles; and LVEF rose normally with exercise. Serial ECGs showed no change.

To investigate the possibility of diffuse pulmonary embolization (in the context of intravascular coagulation) we measured the lung's diffusion capacity, which was normal.

CASE 2

A previously healthy soldier was admitted to another facility after

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CHEST / 95 / 5 / MAY, 1989 1089
having collapsed during physical training on a hot day. His rectal temperature was 39.8°C and a diagnosis of heat exhaustion was made. He had received 2 to 3 L of intravenously administered fluids during the two hours preceding his admission. On the following day, prominent peripheral edema was noted and persisted for three days despite the discontinuation of intravenous fluid therapy. There was no evidence of jugular distention or pulmonary congestion. The ECG showed only sinus tachycardia. The RVC was performed a month after the acute event and demonstrated a RVEF of 43 percent; the right ventricular to left ventricular diastolic volume ratio was 2, signifying right ventricular dilatation. Left ventricular function was normal.

All scans were obtained by a portable Elscint gamma camera.

**DISCUSSION**

Heat stroke is an important medical problem among military recruits as well as among members of other risk groups. The frequency and severity of cardiac involvement in the condition have not been adequately established. While Shibolet and associates reported that the only electrocardiographic abnormality among 36 cases was sinus tachycardia, others found electrocardiographic changes, the most frequent of which were in the T wave and in the ST segment, in the majority of patients; conduction abnormalities have also been observed. Enzymatically, elevations of LDH isoenzyme 1 have been well characterized. Pathologically, subendocardial hemorrhages, mainly in the left ventricular septum, were described in cases of heat stroke; additional findings were dilatation of the right ventricle and myocyte necrosis.

Although myocardial damage has been documented in heat stroke, it is not a universal finding; Costrini et al found elevated levels of the MB fraction of CK in none of their 13 heat stroke patients.

The usual cardiovascular response to heat stroke is hyperdynamic, that is an increase in cardiac output and a decrease in peripheral resistance. Severe cases may present with a hypodynamic circulation and shock, but pulmonary edema is a rare finding. Whereas in a series of 26 cases no patient had this complication, others reported in detail the case of a young patient who had acute myocardial infarction and pulmonary edema in the setting of heat stroke; these investigators mentioned two other patients whose course had been complicated by pulmonary edema. Of 17 cases of fatal heat stroke described in an older report, eight were caused by pulmonary edema.

It may be argued that pulmonary and peripheral edema in our patients was iatrogenic. Although rapid administration of fluids may have been a contributing factor, we believe myocardial dysfunction had to be present as well; we believe it is unlikely for healthy young men to develop pulmonary or peripheral edema after receiving the amounts of fluids our patients did; furthermore, heat stroke often is accompanied by dehydration, and our first patient suffered diarrhea on the morning he became ill. Direct evidence for myocardial depression was offered by the RVC. Although the LVEF was normal, the RVEF was severely reduced, documenting myocardial dysfunction. We assume that some degree of left ventricular dysfunction had been present at the time of admission but ten hours later, when the first scan was obtained, the patient's pulmonary congestion was markedly improved as, probably, was left ventricular function. The patient's stress may have caused ventricular hypercontractility and so a falsely "normal" function was recorded ten hours after admission. The second RVC demonstrated hypokinesis of the left ventricular apex.
We interpret our findings as consistent with global myocardial dysfunction due to heat-related myocardial injury, and assume that in the first patient left ventricular function improved more rapidly than the right. The prolonged nature of myocardial depression in both our patients supports the hypothesis that myocardial damage, rather than mere fluid overload, had been the cause of the decompensation on admission. We cannot exclude the possibility that our first patient's pulmonary edema was caused by a combination of fluid overload and pulmonary capillary damage; however, even if this were so, the radionuclide studies documented severe, prolonged, myocardial depression.

Myocardial dysfunction has not, to our knowledge, been directly demonstrated, as it was in our patients; moreover, our cases illustrate the possibility of subclinical myocardial depression persisting for weeks after the acute event. The significance of this prolonged abnormality of cardiac contractility and its natural history are unknown at present. The more severe injury to the right ventricle we observed, combined with pathological data,7 may indicate that the right ventricle is more vulnerable than the left in heat stroke. Our data suggest that hemodynamically compromised victims of heat stroke should be evaluated (either by echocardiography or by RVG) for the presence of impaired myocardial contractility; if such impairment is demonstrated the patient should be followed on a long-term basis or until the abnormality resolves.

Our findings should prompt caution in the administration of intravenous fluids to victims of heat stroke; although dehydration often coexists, the fluid deficit should be carefully assessed on an individual basis and replacement should be guided by measurement of the heart's filling pressures or, at least, by careful clinical monitoring.

REFERENCES

10 Austin MG, Berry JW. Observations on 100 cases of heat stroke. JAMA 1956; 161:1525-29