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Exertional heat stroke: the runner's nemesis

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Heat stroke in distance runners is increasing in frequency. A case is reported of a 41-year-old man who collapsed during a 10-km "fun run" held when the temperature was 31.6°C and the humidity 80%. Acute renal failure (serum creatinine level 1530 $\mu\text{mol/l}$ [17.3 mg/dl]), rhabdomyolysis, disseminated intravascular coagulation and hepatic damage complicated the clinical picture. Repeated peritoneal dialysis and one cycle of hemodialysis because of a very high serum level of uric acid (1.23 mmol/l [20.7 mg/dl]) were required. Although the illness was prolonged, recovery was almost complete, and 4 months after the man's collapse the serum creatinine level had fallen to 133 $\mu\text{mol/l}$ (1.5 mg/dl).

Chez les coureurs de fond la fréquence des coups de chaleur vont en augmentant. On signale le cas d'un homme de 41 ans qui s'effondra pendant une course "d'agrément" de 10 km tenue par une chaleur de 31.6°C et une humidité de 80%. Une insuffisance rénale aiguë (avec un taux de créatinine sérique de 1530 $\mu\text{mol/l}$ [17.3 mg/dl]), une lyse des fibres musculaires avec myoglobulinurie, une coagulation intravasculaire disséminée et des dommages hépatiques vinrent compliquer le tableau clinique. Des dialyses péritonéales répétées et une session d'hémodialyse rendue nécessaire par une uricémie très élevée (1.23 mmol/l [20.7 mg/dl]) furent requis. Bien que la maladie fût longue

la guérison fut presque complète, et 4 mois après le collapsus de cet homme le taux de créatinine sérique s'était abaissée à 133 $\mu\text{mol/l}$ (1.5 mg/dl).

Prolonged physical activity in the presence of high environmental temperature and humidity enhances the risk of heat stroke. This condition has been reported in military recruits, distance runners, cyclists and North American football players. With an estimated 25 million Americans and 1 million Canadians participating in organized road races annually, it is not surprising that exertional heat stroke is occurring with increasing frequency among distance runners.¹⁻⁴

Casualties are more common in novices who exceed their training efforts when racing and in well trained competitors who strive for improved performance by suddenly increasing their pace midway through a long-distance event. But the problem does not end here; under adverse environmental conditions even the most accomplished athlete is at risk.

The purpose of this report is to describe a case of heat stroke in an experienced runner who participated in a race conducted under extreme environmental conditions.

Case report

A 41-year-old man collapsed after 9 km of a 10-km "fun run" in Hamilton June 10, 1979. He had been physically active since adolescence and had been jog-

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ging consistently for the previous 9 years. Fourteen months before the Hamilton race he had begun a training program in which he ran 11 to 14 km per day during the week and slightly more on weekends. At no stage did he train in heat or excessive humidity. He was accustomed to competitive road running, having completed three standard marathons and a number of shorter races between June 1977 and May 1979. One month before the June 10 run he had completed a 42.2-km race in 3 hours and 17 minutes.

The Hamilton run began at 1.45 pm; at that time the temperature in the vicinity of the race was 31.6°C, the relative humidity 80% and the humidex 40.5°C. The patient drank 250 ml of water just before the race and 300 ml at the halfway mark.

He reported no premonitory symptoms. At the 9-km mark, however, he was seen to increase his speed. Shortly thereafter he veered off the track and collapsed. Bystanders immediately doused him with cold water and an ambulance was called. The interval between collapse and arrival at the Joseph Brant Memorial Hospital, Burlington was approximately 20 minutes.

At the time of admission to hospital the man was

comatose and unresponsive to painful stimuli. He was sweating profusely. The pulse rate was 180 beats/min and the rhythm regular, the blood pressure was 140/90 mm Hg and the respiratory rate was 36/min. He weighed 66 kg. An electrocardiogram (ECG) showed sinus tachycardia and a chest roentgenogram was normal. The rectal temperature was 40.3°C.

The serum potassium and chloride levels were elevated to 5.3 and 107 mmol/l respectively, and the blood glucose level was elevated to 10.3 mmol/l (185 mg/dl). The serum sodium, blood urea nitrogen (BUN), glutamic oxaloacetic transaminase (SGOT), creatine phosphokinase (CPK) and lactate dehydrogenase (LDH) levels were all within the normal range. Arterial blood gas analysis showed metabolic acidosis with a bicarbonate level of 16.3 mmol/l and respiratory alkalosis probably resulting from hyperventilation; the partial pressure of oxygen (corrected for temperature) was 82 mm Hg. The platelet count and the prothrombin and partial thromboplastin times were normal. The initial and subsequent laboratory findings are presented in Table I.

Intravenous administration of Ringer's lactate was started. Following transfer to the intensive care unit

Table I—Laboratory data in a patient with heat stroke

Variable	Admission	24-48 hours	60-72 hours	1 week	4 weeks	6 weeks	8 weeks	10 weeks	14 weeks	Normal values
Levels of serum constituents										
Sodium, mmol/l	144	120	129	128	132	143	143	142	145	135-145
Potassium, mmol/l	5.3	5.9	5.6	5.8	3.7	5.4	4.9	4.4	4.2	3.5-5.0
Chloride, mmol/l	107	93	93	87	93	106	106	105	106	95-105
Carbon dioxide, mmol/l	—	12	14	18	—	—	—	28	25	21-29
Blood urea nitrogen, mmol/l (mg/dl)	5.0 (14)	34.6 (97)	37.8 (106)	55.3 (155)	35.7 (100)	12.1 (34)	12.1 (34)	11.4 (32)	9.6 (27)	3.6-7.1 (10-20)
Creatinine, μ mol/l (mg/dl)	—	1061 (12)	1229 (13.9)	1529 (17.3)	601 (6.8)	194 (2.2)	168 (1.9)	168 (1.9)	141 (1.6)	44-133 (0.5-1.5)
Uric acid, mmol/l (mg/dl)	—	—	1.23 (20.7)	0.54 (9.0)	0.36 (6.1)	—	0.37 (6.2)	—	0.40 (6.8)	0.12-0.42 (2.0-7.0)
Glucose, mmol/l (mg/dl)	10.3 (185)	—	5.5 (99)	5.8 (105)	6.4 (116)	—	5.9 (107)	—	5.6 (100)	3.9-6.1 (70-110)
Bilirubin, μ mol/l (mg/dl)	—	—	—	—	—	—	—	—	—	—
Total	—	116 (6.8)	154 (9.0)	123 (7.2)	8.6 (0.5)	—	5.1 (0.3)	—	8.6 (0.5)	0-17.1 (0-1.0)
Direct	—	75 (4.4)	—	—	—	—	—	—	—	0-3.4 (0-0.2)
Alkaline phosphatase, IU/l	—	—	122	222	141	—	131	—	60	30-120
Glutamic oxaloacetic transaminase, IU/l	30	2 850	3 500	420	187	—	33	—	35	< 35
Creatine phosphokinase, IU/l	154	112 300	123 900	—	—	—	—	—	—	25-145
Lactate dehydrogenase, IU/l	207	4 380	6 080	—	—	—	—	—	—	110-250
Arterial blood gas values (corrected for temperature)										
pH	7.35	—	—	—	—	—	—	—	—	7.35-7.45
Oxygen tension, mm Hg	82	—	—	—	—	—	—	—	—	80-105
Carbon dioxide tension, mm Hg	31	—	—	—	—	—	—	—	—	35-45
Bicarbonate level, mmol/l	16.3	—	—	—	—	—	—	—	—	24-30
Coagulation values										
Platelet count, $\times 10^9/l$	257	85	50	166	—	261	—	—	—	150-400
Prothrombin time, s (control time, s)	14.5 (12)	25 (13.5)	21 (11)	10 (11)	—	—	—	—	—	70%-100% of control
Partial thromboplastin time, s (control time, s)	32 (28)	43 (29)	45 (30)	32 (31)	—	—	—	—	—	24-36
Fibrinogen degradation products, μ g/ml	—	—	192	96	—	—	—	—	—	20
Protamine sulfate test	—	—	Pos.	Neg.	—	—	—	—	—	Neg.
Urine values										
Protein	—	4+	3+	3+	0	0	—	—	—	0
No. of erythrocytes per high power field	—	0-1	6-8	5-10	0	0	—	—	—	0
Myoglobin	—	Pos.	Pos.	Pos.	Neg.	Neg.	—	—	—	Neg.

the patient was placed on a cooling blanket and given oxygen by mask. He remained unconscious for 6 hours and required intravenous administration of diazepam to control early signs of convulsion and decerebrate posturing of one arm.

Within 5 hours of admission the patient's rectal temperature fell to 38°C. An ECG then showed a nodal rhythm and a heart rate of 75 beats/min. Two hours later both the cardiac rhythm and the temperature were normal. He was conscious and had no neurologic deficit after the sixth hour in hospital.

During the first 9 hours in hospital the patient voided 570 ml of urine. Losses through emesis and diarrhea, which began 3 hours after admission, amounted to 2725 ml. Over the ensuing 24-hour period his urinary output was only 200 ml and the gastrointestinal loss was a further 700 ml. He subsequently became anuric. The emesis and diarrhea remitted spontaneously. No infecting organisms were ever isolated from blood or stool cultures.

Within 24 to 48 hours after admission the patient's serum sodium and chloride levels fell below normal. The serum potassium, BUN, creatinine and bilirubin levels rose above normal and the SGOT, CPK and LDH levels became grossly elevated. The platelet count fell to $85 \times 10^9/l$ and the prothrombin and partial thromboplastin times became prolonged. His urine was strongly positive for protein and myoglobin, and an occasional erythrocyte was detected.

A diagnosis of heat-induced acute renal failure, rhabdomyolysis, disseminated intravascular coagulation and hepatic necrosis was made and the patient was transferred to St. Joseph's Hospital, Hamilton for further management.

There he was given peritoneal dialysis on alternate days. An elevated serum uric acid level, 1.23 mmol/l (20.7 mg/dl), was recorded 60 hours after admission; the level remained elevated in spite of the administration of allopurinol, 300 mg/d, but following a single cycle of hemodialysis it fell to 0.54 mmol/l (9.0 mg/dl).

The patient's renal function was slow to recover. The first indication of a diuretic phase occurred 3 weeks after admission; peritoneal dialysis was then discontinued. A week later the BUN level was 35.7 mmol/l (100 mg/dl) and the serum creatinine level 601 $\mu\text{mol/l}$ (6.8 mg/dl). By 10 weeks the levels had fallen to 11.4 mmol/l (32 mg/dl) and 168 $\mu\text{mol/l}$ (1.9 mg/dl) respectively. At 14 weeks the serum creatinine level was almost within the normal range, at 141 $\mu\text{mol/l}$ (1.6 mg/dl), and at 22 weeks it was normal, at 133 $\mu\text{mol/l}$ (1.5 mg/dl).

By 4 weeks after admission all the other laboratory values had stabilized within their normal ranges and the urine was clear.

The patient recovered uneventfully, and following discharge from hospital he continued to make good progress. He subsequently returned to normal activity and resumed running.

Discussion

Some of the protean manifestations of exertional

heat stroke were well illustrated by our patient. The body temperature is invariably raised above 38°C during a marathon run, and if the runner is dehydrated the rectal temperature may rise above 40°C. The dividing line between such elevated temperatures and overt heat stroke is tenuous and may depend on individual differences in thermoregulation and the predisposition to heat stroke.

The steady-state body heat content (H) is a function of gain from metabolism (M), net gain or loss from radiation (R) and from conduction and convection (C), and losses from evaporation (E) and from work (W). These factors are summarized by the heat-balance equation:

$$H = M \pm R \pm C - E \pm W$$

High gains from metabolic heat production and solar radiation are crucial factors when long-distance running is considered.⁵

In cool weather, heat is easily lost from the skin by radiation, conduction and convection. However, with increasing environmental temperature, heat dissipation through conduction and convection decreases, and at ambient temperatures above about 34°C the only mechanism available for heat loss is evaporation of sweat (C. Gisolfi: personal communication, 1980). High temperature and humidity and a high level of solar radiation can overwhelm this mechanism, and metabolic heat production will have to be controlled if heat stroke is to be avoided. This implies immediate cessation of activity or the institution of an effective method of cooling.

Heat stroke invariably occurs when the rectal temperature exceeds the critical thermal maximum of about 42°C.⁵ It should be suspected if a runner shows any of the following premonitory signs: irritability and aggression, emotional instability, hysterical weeping, apathy, disorientation or unsteadiness of gait.⁶

The maximum rectal temperature recorded in our patient seems lower than one might expect in heat stroke. However, it was measured in an air-conditioned hospital 20 to 30 minutes after the collapse, and the rectal and central nervous system temperatures may have been considerably higher at the time of collapse. Also atypical was the absence of premonitory symptoms except for some disorientation shortly before the collapse.

Gastrointestinal problems

Gastrointestinal symptoms are common in heat injury, especially during the first 24 hours. Shibolet and colleagues⁷ studied 36 patients in the acute phase of heat stroke. Diarrhea was evident in 13 and vomiting in 19. The extent of gastrointestinal fluid loss obviously varies, but such losses were particularly severe in our patient, and this factor may have accounted, at least in part, for the severity of this case.

Renal involvement

A spectrum of renal abnormalities from acute renal failure to chronic interstitial nephritis has been described in association with heat stroke.⁸⁻¹⁰ Acute ne-

phropathy occurs in 10% to 35% of diagnosed cases of heat stroke.¹¹

Hyperkalemia is often a problem during the early part of the hospital stay. Multiple system involvement, especially muscle damage, contributes to the hypercatabolic state that is thought to be the main contributor to an elevated BUN level.¹²

Myoglobinuria is the direct link between rhabdomyolysis and renal insufficiency. Schiff, MacSearraigh and Kallmeyer,¹³ studying runners in the 88-km Comrades Marathon in South Africa, found that 25 of 44 competitors had myoglobinuria following the race but none had it before the race. Since myoglobin is normally found only in muscle cells, its presence in urine is regarded as a sensitive indicator of muscle breakdown and is significantly and positively correlated with serum enzyme and uric acid levels.¹³ The serum uric acid level rises significantly following exertion, and high levels are known to precipitate renal failure. Recent work by Sutton and coworkers¹⁴ has indicated that skeletal muscle is the main source of uric acid during vigorous exercise.

The myoglobinuria and excessively high serum CPK and uric acid levels in our patient thus indicated substantial muscle damage and provided some explanation for the severity of his renal decompensation. Furthermore, failure to regain full renal function within the first few days following heat stroke is reputed to signal a poor prognosis for renal recovery.¹⁰ Over a 14-week period, however, our patient's serum creatinine level gradually fell toward normal and was normal at 22 weeks — a more favourable outcome than had been expected.

Coagulopathy

Impaired coagulation is often associated with severe heat injury,¹⁵⁻¹⁷ and its presentation in this situation is little different from that in other circumstances. The manifestations include thrombocytopenia, prolongation of bleeding and clotting times, lowered plasma fibrinogen and prothrombin levels and increased fibrinolytic enzyme activity. Endothelial cell damage following an elevation in body temperature is the most likely triggering mechanism of disseminated intravascular coagulation in heat stroke.¹⁸ A review of Table I will confirm the short duration of this complication in the present case.

Hepatic damage

As with renal impairment in heat stroke, hepatic damage varies in severity. Physical exertion alone causes elevated serum levels of hepatic enzymes, but there is a distinct difference between enzyme patterns seen after uncomplicated exercise and those seen in heat stroke.^{19,20} Hyperbilirubinemia has been reported in heat stroke but is less consistent than elevated serum enzyme levels.²¹ Although it is sometimes difficult to establish the source of the enzymes SGOT and LDH, indicators such as isoenzyme LDH-5, carbamyl transferase and citrate dehydrogenase can provide data specific to hepatic damage following heat injury.²¹

Ultrastructural abnormalities include degeneration

or desquamation of sinusoidal lining cells, ballooning or flattening of the microvilli and changes in the mitochondria.²²

In our patient the outstanding features included gross elevations in the serum levels of SGOT and LDH as well as transient hyperbilirubinemia.

Neurologic abnormalities

Coma is a feature of moderate to severe heat stroke.²³ Depression of the central nervous system and extreme hyperirritability often occur simultaneously in the form of delirium, hallucinations, status epilepticus with decerebrate rigidity, oculogyric crises and opisthotonus. Cerebellar syndromes with ataxia and a chronic thalamic syndrome have also been reported following heat stroke.^{11,15}

Failure to regain consciousness within 2 hours of collapse seems to carry a poor prognosis.¹⁵ Although our patient's coma lasted much more than 2 hours clinical examination following recovery revealed no neurologic deficit.

Cardiovascular changes

Sinus tachycardia invariably accompanies heat stroke. Heart rates greater than 160 beats/min occur in severe cases and may be accompanied by hypotension.¹⁵ ST-segment depression and T-wave changes are often seen on the ECG, and conduction abnormalities, especially incomplete bundle branch block, have been reported.^{11,24,25}

Myocardial damage is common and is best reflected by elevated serum levels of cardiac-specific enzymes. Subendocardial hemorrhages have been described, and fragmentation or rupture of cardiac muscle fibres can further complicate the condition.²³

A transient nodal rhythm was recorded in our patient, but recovery from this was uneventful, and cardiac decompensation did not occur.

Management

Early recognition is essential to effective management of heat stroke. The cornerstones of management include prompt cooling, appropriate rehydration and correction of circulatory collapse.

Early immersion in ice water, though frequently recommended,^{11,23} is not without hazard, for it causes a sudden drop in temperature, and the resultant vasoconstriction and shivering impede further heat loss. It is more appropriate to transfer the victim to a shady area, fan the body surface and place wet towels packed with crushed ice on the neck, trunk, axillae and groin. Studies by Richards and colleagues²⁶ have demonstrated that such simple measures, together with administration of intravenous fluids, can reduce the core temperature from 43°C to 38°C in under 35 minutes.

In mass participation runs, provision for rapid cooling at the race site is vitally important, as the outcome of heat stroke is best when cooling is begun immediately.²⁶ After initial cooling, the victim of heat stroke should be transferred to hospital, where his or her status should be monitored for 24 to 36 hours

so that complications can be identified and treated. When the rectal temperature has fallen to about 38.5°C with active cooling, passive cooling (such as placing the person on a cooling blanket) can be used to further reduce the temperature to normal.

The administration of antipyretics, phenothiazines and anesthetics has been the subject of much debate, but there is little to support the routine use of these agents in heat stroke.

Hypotension, a manifestation of marked peripheral vasodilatation during hyperpyrexia, will usually respond to prompt cooling and intravenous administration of fluids.

The fluids initially administered should include glucose-electrolyte solutions such as dextrose-saline. Rapid intravenous infusion of fluid at room temperature is advised during the acute phase, but precautions to prevent fluid overload are mandatory. In severe cases a central venous pressure catheter is useful in monitoring fluid balance.

Mannitol, 12.5 g, may be administered intravenously in the presence of impending renal shutdown. However, if cooling is rapid and the circulating fluid volume is restored quickly with intravenous administration of fluids, the use of mannitol may be unnecessary.

Serum levels of electrolytes, BUN, creatinine, SGOT, CPK and LDH should be determined without delay so that organ damage can be rapidly assessed and appropriate treatment initiated.

Even under ideal circumstances of diagnosis and management the prognosis in moderate to severe heat stroke remains guarded. Atypical features, as described in our patient, can easily complicate the course and outcome of this potentially fatal condition.

Full recovery from heat stroke is no reason for complacency, as heat intolerance in former heat stroke patients has been reported,²⁷ and recurrences in susceptible individuals are therefore possible.

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Shown on the cover of this issue of the Journal is a runner in distress at the finish area of the Montreal International Marathon, Aug. 24, 1979 (photo courtesy of Entre 2 Design, Montreal and the marathon's organizing committee).