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Return-to-Competition Progression After Exertional Heat Stroke in an Adolescent Runner: A Case Report

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A 14-year-old runner indigenous to the tropics collapsed during his first 10-km race in a hot and humid climate. Dizziness, stumbling, and loss of consciousness were symptoms of exertional heat stroke. Two days postcollapse, blood tests revealed elevated hepatic transaminases consistent with hepatic injury. We discuss the return-to-competition progression, which included a gradual increase in exercise duration, initially in a cool and then in a tropical climate, and 2 field-based (wet-bulb globe temperature $> 29.0°C$) running evaluations that simulated the environmental conditions and training intensity the athlete would encounter in his sport. The thermoregulatory results guided the training progression, his tolerance to running in the heat improved, blood values normalized, and he was cleared to compete in a tropical climate. This exploration case report presents a novel field-based protocol that replicates the physiological demands of training in the tropics to evaluate thermoregulatory responses during exercise-heat stress in young runners after exertional heat stroke to facilitate a safe return to competition.

Key Words: young athletes, hyperthermia, tropical climate, acclimatization

Key Points

- Laboratory-based heat tolerance tests used in adults may not be sufficient to determine readiness to return to competition after exertional heat stroke in young runners due to their low intensity and artificial environmental conditions.
- Using field-based running tests in the tropical outdoors, we documented improvements in thermoregulation and exercise tolerance in a young runner recovering from exertional heat stroke over a 15-week return-to-competition progression.

Young athletes have a high level of participation during the sports in hot summer months, may not recognize the symptoms of heat illness, may feel pressured to compete despite not feeling well, and usually do not drink enough during exercise.[1,2] Exertional heat stroke (EHS) is a life-threatening condition that results from extreme hyperthermia during strenuous exercise, especially in a hot and humid climate. It is characterized by symptoms of central nervous system (CNS) dysfunction such as confusion, irrational behavior, or loss of consciousness and a core body temperature (T_c) usually $\geq 40.0°C$.[3,4] Predisposing risk factors for EHS include a lack of heat acclimatization and dehydration.[5] In runners, dehydration leads to a higher rate of increase in T_c,[6] which can result in dangerous hyperthermia, especially in highly motivated athletes who are not well acclimatized to hot and humid conditions. Hyperthermia and dehydration impair cardiovascular function and hinder body heat transport and dissipation.[7] Early recognition of symptoms and prompt body cooling are crucial for minimizing damage to the liver, kidneys, brain, and skeletal muscle.[4]

In adult athletes, the decision to return to training or competition after EHS is based on clinical signs and blood analysis, laboratory-based heat tolerance testing, and their responses during the heat-acclimatization progression.[8–11] However, no evidence has shown that laboratory-based heat tolerance tests are adequate to determine readiness to return to training and high-level competition in adolescent athletes who experience EHS.[12] Opportunities to implement return-to-competition guidelines in young runners after EHS, based on the requirements of the event and the tropical conditions in which the athlete lives and trains, are rare. We report the case of a 14-year-old runner who collapsed with EHS during his first 10-km race in a hot and humid climate. We describe his thermoregulatory and perceptual responses during exercise in a hot and humid climate, which guided his return-to-competition progression. The young athlete and his parents gave permission to use his clinical and personal data to present this case.

CASE PRESENTATION

Patient

A 14-year-old, highly competitive male middle-distance runner (3:25 min/km in 3 km; maximal oxygen consumption [$\dot{V}O_2max$] consistently measured over 2 years at 72–78 $mL \cdot kg \cdot min^{-1}$) collapsed during his first 10-km road race in a

Table 1. Blood Variables at 2, 9, 16, and 33 Days Postcollapse

Variable	Normal Range	Days Postcollapse			
		2	9	16	33
Sodium, $mEq \cdot L^{-1}$	137–145	142	NE	141	141
Potassium, $mEq \cdot L^{-1}$	3.6–5.0	4.7	NE	4.6	4.6
Blood urea nitrogen, $mg \cdot dL^{-1}$	7–25	18.1	NE	21.7	18.9
Creatinine, $mg \cdot dL^{-1}$	0.7–1.3	0.94	NE	0.92	0.90
Hemoglobin, $g \cdot dL^{-1}$	14–18	15.8	NE	NE	NE
Hematocrit, %	42–52	46.4	NE	NE	NE
Aspartate aminotransferase, $\mu \cdot L^{-1}$	15–45	1923	100	55	59
Alanine transaminase, $\mu \cdot L^{-1}$	9–72	1996	424	170	64
Alkaline phosphatase, $\mu \cdot L^{-1}$	38–126	273	208	192	208
Creatine kinase, $\mu \cdot L^{-1}$	30–200	NE	NE	151	NE

Abbreviation: NE, not evaluated.

hot (approximately 32°C–35°C) and humid (approximately 60%–70% relative humidity) climate during the summer month of September. His father saw him stumbling in the eighth kilometer, and soon after, the athlete collapsed and became unconscious. While waiting for help at the roadside, the father poured 2 bottles (about 500 mL each) of water (not cold) over the runner's head; he was still on the pavement in wet running gear, exposed to the sun, and unresponsive. After about 10 minutes, help arrived, and the runner was picked up by his father and taken in a police car, still unconscious, to a local community-based emergency room (ER) close to the competition site.

According to ER records, upon arrival about 15 minutes postcollapse, he was still unconscious, wet, and tachycardic (138 beats/min) and had low blood pressure (100/50 mm Hg). He was immediately given an intravenous (IV) infusion of normal (0.9%) saline (500-mL bag) and supplemental oxygen via a nasal cannula at a rate of 5 L/min. Body temperature was not measured, nor was blood analysis ordered. Body-cooling methods were not used by the ER medical staff other than room-temperature IV fluids. At approximately 25 minutes postcollapse, he regained consciousness but was disoriented and irrational. He did not remember collapsing or being transported to the ER. After 1 hour, he voided for the first time since the collapse. At 2.5 hours, he was given another IV infusion of normal (0.9%) saline (500 mL). After 4 hours, he was alert and felt better, his heart rate (HR) was 88 beats/min and blood pressure 120/70 mm Hg, and he was discharged home. Upon arrival home, he showered with cold water.

Two days postcollapse, the young runner was evaluated by his primary physician, who ordered blood tests that revealed elevated hepatic transaminases (alanine transaminase = 1923, aspartate aminotransferase = 1996) consistent with hepatic injury. The physician advised him not to exercise until further evaluation by a sports medicine team. One week postcollapse, he presented to our center with his parents. A primary care sports medicine physician performed a detailed assessment of the athlete, in the presence of his parents, which included a complete history and physical examination as well as a review of blood tests.

Tests and Results

History, Physical Examination, and Blood Tests. The runner was a healthy, lean, young male with normal vital signs (height = 172.4 cm, weight = 57.7 kg, heart rate = 68 beats/min, blood pressure = 116/68). The head, ears, eyes, nose, throat, and abdomen and pulmonary, cardiovascular, and neurologic systems were within normal limits. He did not have a personal or family history of heat stroke or exercise-associated collapse, had no cardiac or other health conditions, and was not taking medications or nutritional supplements. He did not report having symptoms of infection or other illness on the day of or days before the race. He trained mostly in the coolest hours of the day and seldom in hot conditions similar to those on race day.

Before the incident, his longest race distance was 5 km (finishing time = 15:23; first place overall in a fun run). He was ranked at the top of his age group at the national level in 1500 m, 1 mile, and 3000 m. His average pace during the 10-km race up to the eighth kilometer was 3:26 min/km ($17.4\ km \cdot h^{-1}$), similar to his best time for 3 km. He felt dizzy after the sixth kilometer but did not slow down because he was following the adult elite runners who were leading, and his goal was to win the race. He collapsed soon after the 8-km mark. According to data recorded and saved in his sports watch, his time up to the eighth kilometer was 27:27. He did not ingest fluids during the race and did not remember having collapsed or his initial treatment at the ER. We ordered repeat blood tests, which revealed that hepatic transaminases were still considerably elevated 9 and 16 days postcollapse (Table 1).

Rectal temperature was not measured, even though it is best practice to determine the extent of hyperthermia when EHS is suspected; the runner demonstrated altered mental status that progressed to unconsciousness, which is a clinical indicator of significant hyperthermia during exercise. Our diagnosis was collapse due to EHS based on the symptoms of altered mental status (dizziness and stumbling) witnessed by spectators and his father before the collapse, loss of consciousness for approximately 25 minutes, and elevated liver enzymes for more than 2 weeks, combined with hot and humid environmental conditions. Based on the athlete's history, physical examination, and laboratory tests, we ruled out hyponatremia, rhabdomyolysis, cardiac anomalies, and exertional sickling.

Intervention. In the weeks after the EHS, the athlete followed our recommendations, which were based on the guidelines of the American College of Sports Medicine (ACSM)[4] and are summarized in Table 2. Starting at week 6, after thermoregulatory and perceptual responses were evaluated with a training simulation test in a hot and humid climate,

Table 2. Return-to-Sport Progression After Exertional Heat Stroke in an Adolescent Runner

Weeks Post–EHS	Activity	Duration or Intensity
2–3	Swim and run at reduced pace in a cool environment (after the 16-d blood analysis result).	20–60 min <70% of maximal HR
4–5	Gradually increase the duration, intensity, and heat exposure. Avoid ≥2% dehydration during exercise by replacing fluids following recommendations based on the sweat rate measured before the EHS.	≤60 min Approximately 70% of maximal HR
5	Training simulation test outdoors to evaluate the thermoregulatory and perceptual responses in a hot and humid environment.	Athlete completed 40 min of the 60-min test at 75% of the racing velocity of previous 3000 m in a hot and humid climate (85% of maximal HR).
6–7	Training sessions outdoors at moderate intensity with the goal of acclimatizing to hot and humid conditions. Supervised by coach in communication with the medical team.	Followed the training plan, supervised by coach
8–9	Start competing in short races (≤5 km) in mild climate.	Race pace
10–14	Compete in longer races (5–8 km) in mild climate.	Race pace
15	Repeat training simulation test outdoors in hot and humid environment.	75%–85% of racing velocity (82%–98% of maximal HR). Athlete completed the 60-min test.
15+	Cleared for competition in tropical climate.	Race pace

Abbreviations: EHS, exertional heat stroke; HR, heart rate.

the coach supervised the training sessions and communicated with the medical team. A description and the results of the training simulation tests at 5 weeks and 15 weeks post-EHS follow.

Training Simulation Test at 5 Weeks Post-EHS. At 5 weeks after the EHS, the runner underwent an exercise test at moderate intensity in a hot and humid environment so that we could assess the T_c response and fluid loss and intake patterns as suggested by Roberts et al[9] for a safe return to training-pace intensity in a tropical climate. Air temperature, humidity, wind, and solar radiation (wet-bulb globe temperature = 31.9°C) were typical of running conditions in a tropical climate. Six hours before the test, the athlete swallowed an ingestible sensor (Cortemp; HQ Inc) to measure gastrointestinal temperature as an indicator of T_c. The runner's coach provided information about the training intensity pace. Velocity was 12.8 km·h^{-1} (75% of his racing velocity for 3000 m) for 1 hour or until T_c reached 39.3°C. His HR (model M400 monitor; Polar) was recorded every minute.

The young athlete was able to run for 40 minutes before his T_c reached 39.3°C. His average T_c rate of rise was 3.1°C·h^{-1} (Figure 1). Average HR was 165 beats/min (85% of maximum; Figure 2), sweat rate was 1.8 L·h^{-1} (>75th percentile for age),[13] water intake was 200 mL (16% of fluid loss), and body fluid deficit was 2%. Ratings of perceived exertion and feeling hot or overheated are shown in Figure 3A and B. Based on these results, he would probably have shown a T_c <38.5°C at the low intensity of the typical heat tolerance test (5 km/h, 2% grade),[14] which represents about 20% of his $\dot{V}O_2$max, and passed the test. This supports the use of field-based running tests to evaluate thermoregulatory responses at higher exercise intensities in well-trained young runners after EHS. Our advice was to start training outdoors at low-to-moderate intensity with the goal of acclimatizing to hot and humid conditions. Following the ACSM guidelines[4] and after laboratory results showed the liver enzymes were close to normal values, we allowed the runner to compete in a cooler environment after 2 more weeks. At 8 weeks post-EHS (November), he competed in a 4-km race. After gradual and progressive increases in training distance and heat exposure, the absence of symptoms of heat intolerance, as well as normal blood tests, the health care team considered him ready for longer races. At 14 weeks, he competed in an 8-km race. Both races were in towns located in the central mountain region of the island with a milder

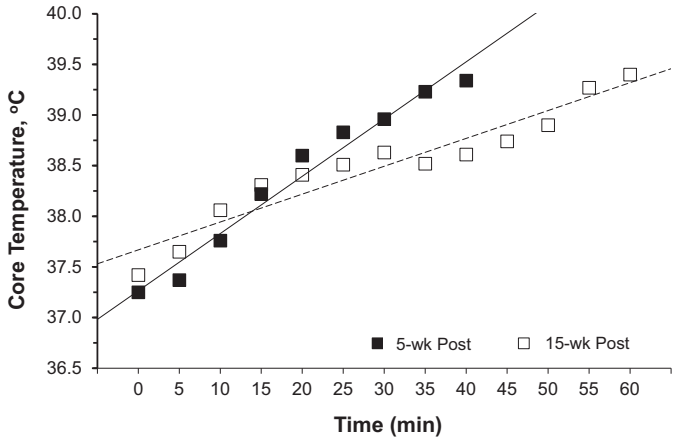

Figure 1. Core temperature during a training simulation test outdoors at 5 and 15 weeks post–exertional heat stroke. The solid line represents the trend in values at 5 weeks, and the dashed line represents the trend in values at 15 weeks.

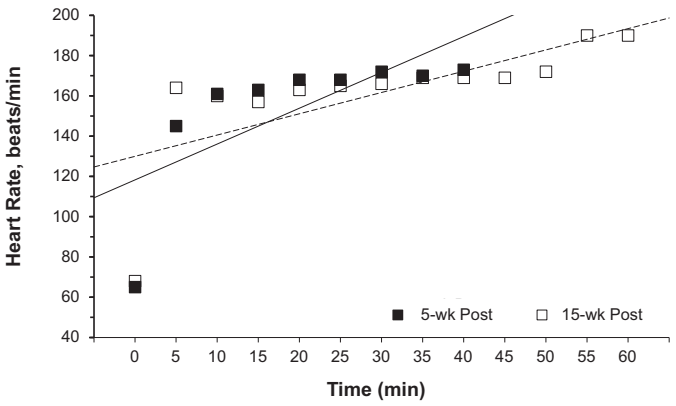

Figure 2. Heart rate during a training simulation test outdoors at 5 and 15 weeks post–exertional heat stroke. The solid line represents the trend in values at 5 weeks, and the dashed line represents the trend in values at 15 weeks.

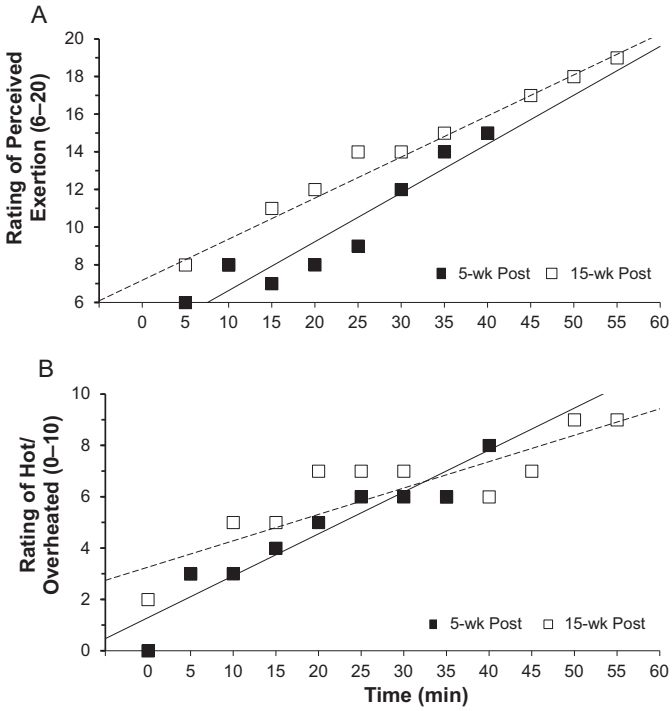

Figure 3. A, Rating of perceived exertion and B, feeling hot or overheated during a training simulation test outdoors at 5 and 15 weeks post–exertional heat stroke. The solid line represents the trend in values at 5 weeks, and the dashed line represents the trend in values at 15 weeks.

climate (approximately 27°C and 70%–75% relative humidity) than his location on the day of the EHS.

Comparative Outcomes at 15 Weeks Post–EHS. Fifteen weeks after the EHS, the athlete underwent a second exercise test outdoors (wet-bulb globe temperature = 29.5°C), starting at a similar intensity as the first test, so that we could assess his progress for clearance to compete in a hot and humid climate. He showed a plateau in T_c after 30 minutes (Figure 1). At 50 minutes, the T_c was 38.9°C, and we increased the pace to 14.8 km·h^{-1} (85% of racing velocity) for the final 10 minutes to evaluate his tolerance to a higher metabolic heat production combined with a high level of heat stress. The T_c did not reach 39.3°C until 60 minutes. Heart rate was 160 to 190 beats/min (82%–98% of maximum; Figure 2).

The results of this follow-up field test indicated that the runner was better able to tolerate the heat stress conditions. In addition to the T_c plateau, the average T_c rate of rise decreased (1.8°C·h^{-1} versus 3.1°C·h^{-1} at 5 weeks). Total exercise time increased 33% from the previous test before reaching the endpoint T_c. Sweat rate rose 22% L·h^{-1} to 2.2 L·h^{-1}, suggesting improved heat acclimatization.[15] Fluid intake (950 mL) was higher than during the first test and replaced 46% of fluid loss. However, the body fluid deficit (2%) did not decrease. Ratings of perceived exertion and feeling hot or overheated were slightly augmented, which may have indicated that the athlete's perception of heat stress improved (Figure 3A and B). Based on the increased tolerance to exercise-heat stress, the runner was cleared for competition in cross-country events in a hot and humid climate. At 20 weeks post–EHS, he won the 8-km National Cross-Country Age Group Championship (3:26 min/km pace).

DISCUSSION

This exploration case report presents the evaluation and progression for return to competition in an adolescent runner who collapsed during a 10-km road race on a hot and humid summer day. The runner arrived at the ER unconscious, tachycardic, and hypotensive. The ER physician did not relate the symptoms to EHS, and aggressive cooling strategies were not implemented. Rapid transport to the ER, exposure to a cool environment (air conditioning), and rapid IV fluid infusion may have contributed to reducing the body temperature and thereby prevented major organ damage or death. Cold or room-temperature IV infusions achieve greater cooling rates than other cooling methods commonly used in clinical settings, such as ice bags placed on major arteries, fanning and wetting with water, and passive cooling.[16] The ACSM guidelines[4] as well as physiological data obtained during running tests outdoors guided the return-to-competition progression. The novel aspect of our test protocol is that we replicated the mode, heat stress, and intensity of running training and competition outdoors. Our recommendations are based on thermoregulatory responses observed in the training field rather than in artificial environmental conditions, such as those in a climatic chamber.

Elite male runners often exhibit core temperatures >40°C during races,[17] but whether adolescent runners reach these levels during competition and what effect hyperthermia may have on organ function are unknown. Exertional heat stroke is the principal cause of preventable death in youth sports.[18] The diagnosis of EHS relies primarily on the triad of hyperthermia, CNS dysfunction, and exercise in a hot climate.[19] Unfortunately, T_c, a key component in the recognition of EHS, can seldom be measured immediately in an athlete who collapses in a recreational road race. Furthermore, ER physicians may not always adhere to best-practice guidelines for proper recognition of EHS, such as measuring rectal temperature. In these cases, signs of CNS dysfunction related to hyperthermia during exercise in the heat and ruling out other diagnoses by medical history, physical examination, and laboratory results can establish the diagnosis of EHS.

In this case report, we identified a lack of frequent exposure or training in hot and humid conditions and poor hydration habits as possible predisposing factors for EHS. The athlete lived and trained in the central mountain region of the island, where the mean average temperature is 3°C to 5°C cooler during the summer months than in the coastal region (race site).[20] He trained in the early morning and late afternoon hours, seldom trained in hot and humid conditions, and was not acclimatized to the level of heat stress present during the race. He was running at a very high exercise intensity (racing pace), which led to a sustained high rate of metabolic heat production.

Seven months before the incident, we measured the athlete's hydration habits while running continuously for 50 minutes at a high exercise intensity in a hot and humid outdoor climate. His fluid loss was 1.45 L, his water intake replaced only 4.4% of the fluid loss, and his body fluid deficit was 2.7%. Based on this information, we gave him specific guidelines to increase his fluid intake and avoid significant dehydration. During the training simulation test

at 5 weeks postincident, his fluid intake replaced 16% of the fluid loss and his body fluid deficit was 2%, which indicated that his hydration habits were still inadequate.

The athlete did not ingest fluids during the race, which most likely led to a mild fluid deficit due to his high sweat rate (approximately 1.4 L/h previously measured outdoors), especially if he started hypohydrated, as is often the case in young runners before training.[1] Dehydration increases cardiovascular and thermoregulatory strain and affects aerobic performance.[8] The combination of insufficient fluid intake, high metabolic and environmental heat loads, and high internal motivation to win while racing among elite adult runners led to the heat gain exceeding the heat loss and severe hyperthermia, as evidenced by collapse and CNS dysfunction. Exercise intensity was most likely the primary factor and dehydration a minor risk factor for the EHS in this young runner.

The heat tolerance protocol developed by the Israel Defense Forces,[14] commonly used to evaluate heat tolerance for return to sport after EHS in adults, consists of walking at moderate intensity in a climatic chamber and may not be appropriate in endurance athletes who exercise at higher intensities outdoors.[21] We had the unique opportunity to quantify thermoregulatory improvement and fluid intake patterns over time with 2 running tests that replicated training intensity in the tropical outdoors while the runner recovered from EHS. His responses indicated that he was not well adapted to exercising in the heat, had a possible detraining effect after the prescribed rest per medical advice, and had probably experienced EHS and was still recovering.

Similar to our report, Johnson et al[11] used a training simulation test to evaluate thermoregulatory responses and adaptations in an adult triathlete after EHS. The difference between studies is that they monitored the athlete during daily heat-acclimation exercise sessions in a climatic chamber (36°C, 50% relative humidity) for 2 weeks, whereas we recommended a longer period of progressive exercise and heat exposure in a tropical outdoor climate monitored by the coach, as well as competition in a milder climate, before allowing the runner to compete under greater climatic heat stress. In both cases, the runners showed heat adaptations such as decreased exercise HR and T_c and increased sweat rate at exercise intensities common to their sport and a safe return to competition in the heat. Similar to Roberts et al,[9] who evaluated an adult runner with a race-simulation test after EHS, we identified predisposing factors for the EHS. We advised the athlete and his coach to improve the duration of exercise-heat exposure and fluid intake habits during the progression to return to competition to avoid future heat-related health problems.

Based on the present case report, we suggest development of the following: (1) educational material for parents and spectators at road races about the symptoms of heat-related health problems in athletes and how to initiate body cooling if necessary; (2) return-to-sport protocols after EHS that are specific to different athletic events and can be implemented outdoors in the training field, without the need for a climatic chamber; and (3) guidelines for coaches to monitor the athlete (HR, fluid loss and intake, symptoms of heat stress) in the field of play as the athlete progresses toward competition in the heat.

CLINICAL BOTTOM LINE

This exploration case report demonstrated that a field-based test that replicates the mode, heat stress, and intensity of training and competition outdoors can be used to assess thermoregulatory, perceptual, and fluid loss and intake responses in adolescent runners after EHS. This information may guide coaches, athletic trainers, and physicians in assessing progress toward the safe return of adolescent runners to competition in a tropical climate. The adolescent runner in this case recovered completely, with no lasting health effects, and successfully returned to competition. At 20 weeks post–EHS, he won the National Cross-Country Championship in his age group and has continued competing successfully at the university, national, and international levels in a hot and humid climate with no heat-related symptoms.

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