Exertional Collapse of a Male Collegiate Rower

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Exertional heat exhaustion is defined as an inability to continue exercise as a result of cardiovascular insufficiency and energy depletion, which may result in physical collapse.[1] In active populations, this is the most common heat-related condition.[2] A heat-related condition may be difficult to distinguish from hypoglycemia, lactic acidosis, or cardiogenic shock, particularly when exposure to extreme heat is not a factor. Presence of trained medical personnel and the availability of proper evaluation tools are essential in such a situation. The purpose of this report is to review a situation in which conflicting signs and symptoms resulted in a failure to recognize and properly manage a heat-related condition that developed during an indoor rowing event.

Key Points

- Exertion-related conditions should not be categorized solely on the basis of environment or sport.
- Both intrinsic and extrinsic factors can negatively influence thermoregulatory mechanisms during exertional activity.
- A potentially life-threatening condition should be managed by trained medical personnel in accordance with an established protocol that is specific to the condition.

Case Report

An 18-year-old male crew athlete (body mass 74.8 kg, height 180.3 cm) presented with disorientation, confusion, and overall weakness immediately after completing a strenuous 2000-meter indoor race on a rowing ergometer (Figure 1). The athlete attempted to stand, but was unable to do so, and fell next to the ergometer. Initial assessment of his status revealed a rapid and weak pulse and cold, clammy, and pale skin. Assistance from nearby teammates was needed to move the athlete from the ergometer to an appropriate area for further assessment. The athlete reported nausea, dizziness, headache, muscle burning in his hips and quadriceps, and stomach pains. The athlete vomited clear fluids for the next five minutes, which was followed by 10 minutes of dry heaving. There were two fainting episodes that both resulted in approximately five seconds of loss of consciousness. Excessive sweating continued for 10 minutes following completion of the race.

When asked about urine color, he indicated that it was light-colored when he last urinated about 30 minutes prior to the race. He mentioned having consumed 20 ounces of water and 10 ounces of a sports drink within two hours of the race time, but he had not eaten (due to competition anxiety). He denied a history of any glucose metabolism disorder, use of any medications, or ever having experienced a similar episode. The athlete was well-conditioned, having trained for two months prior to the event and having completed ergometer testing at a comparable level of intensity and duration. The information regarding the athlete's status and history was derived from one of his teammates who was certified as an American Heart Association first responder. This individual intervened within 10 minutes after completion of the race and obtained information about the athlete's status from other members of

the rowing team who did not possess any training for management of a potentially life-threatening condition.

Differential Diagnosis

Possible conditions that could have been responsible for the symptoms presented in this case include cardiomyopathy, cardiogenic shock, lactic acidosis, and exertional heat exhaustion (EHE).

Treatment

The athlete was given approximately 20 ounces of water and a sports drink, but he immediately vomited upon its ingestion. Towels doused in cold water were then placed on the athlete's neck and forehead. After dry heaving had stopped, within 10 minutes following completion of the race, the athlete assumed a supine position on the ground with the cold towels on his neck and forehead (Figure 2). The athlete was instructed to continue ingesting fluids, and his condition improved.

Figure 1 Indoor race on a rowing ergometer.

Figure 2 Athlete assumed a supine position on the ground with the cold towels on his neck and forehead.

At approximately 30 minutes after completion of the race, the athlete was able to walk independently. Skin appearance and heart rate returned to normal, and he was able to eat, but his headache lingered for the remainder of the day.

Unique Aspects and Considerations

Athletes participating in outdoor sports on hot summer days are clearly at risk for development of exertional heat illness.[1] The reported exertional collapse did not occur in a hot environment. The cluster of symptoms presented made it difficult to identify its cause. A variety of conditions can present similar symptoms to those associated with extreme exertion in a climate-controlled environment.

EHE is associated with pallor, weakness, hyperventilation, and fainting.[1] Thermoregulation can be challenged by both intrinsic and extrinsic factors. Indoor training of elite oarsmen has been demonstrated to potentially result in elevation of blood lactate concentration and body temperature.[3] Poor cardiorespiratory fitness and dehydration would present thermoregulatory challenges, but the athlete was well trained and was apparently adequately hydrated. The presentation and patient history in this case defy most of these factors, given that the event was held indoors and occurred in January and the athlete was wearing shorts and a mesh jersey. Cardiogenic shock could be responsible for the collapse, confusion, excessive sweating, and rapid heart rate that were recognized by his teammates upon completion of the race. Whether or not the athlete would have been able to continue the race if it had been longer than 2000 meters is unknown, but he would have undoubtedly adjusted his exertion level downwardly if this had been the case. In any case, his collapse immediately upon completion of the event dictates that hypertrophic cardiomyopathy should be ruled out. Poor pre-event nutrition may have resulted in a hypoglycemic state that contributed to the episode. However, gradual improvement in mental and physical status during the 30-minute period following the collapse suggests that hypoglycemia was not a primary factor.

Lactic acidosis is another possible contributing factor that should be considered (Figure 3). Research has demonstrated that inadequate buffering results in decreased rowing performance.[4] Oxygen delivery to working muscles decreases with lowered pH, which results in a hyperventilatory response. This problem