

Dilutional hyponatraemia in athletes

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Hyponatraemia should always be included in the differential diagnosis when an endurance athlete collapses at or just over the finish line.

Sports physicians and GPs with an interest in sports medicine often find themselves working in the medical tent adjacent to the finishing line of an endurance event. Blisters and overuse problems are common and generally of little concern, but an athlete who collapses on the finish line or soon afterwards may well induce a significant sympathetic nervous system response in an attending doctor.

The vast majority of athletes who collapse have exercise associated collapse, which has been defined as inability to stand or walk unaided as the result of light-headedness, faintness, dizziness or syncope.¹ It is a benign condition caused by postural hypotension due to cessation of muscle pumping action in the legs. Rapid recovery occurs when the athlete is placed supine with his or her legs elevated.

More significant medical causes of collapse include hyperthermia, hypothermia, hypoglycaemia, cardiac arrest and convulsions. These are more likely causes of collapse that occurs during an event.

Less well known is the relatively recently described condition of hyponatraemia, which is defined as a serum sodium level of less than 135 mmol/L. Hyponatraemia was initially thought to occur only during or after ultra-endurance events, but it is now not infrequently seen at the conclusion of a standard marathon. The incidence in marathon runners requiring treatment in race medical facilities has been reported to be as high as 9%.²

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Pathophysiology

Risk factors for hyponatraemia include sporting activity in hot weather, female gender and a slow finishing time, such as completion of a standard marathon in over five hours. It has been suggested that dilutional hyponatraemia occurs in athletes who:³

- drink to excess during exercise
- retain excess fluid because of inadequate suppression of antidiuretic hormone secretion
- osmotically inactivate circulating sodium or fail to mobilise osmotically inactive sodium from internal stores.

Presentation

Some athletes with mild hyponatraemia are asymptomatic. Those with more significant reductions in serum sodium may present with nausea, vomiting, light-headedness, lethargy, oedema (particularly of the hands and fingers) and cramps – features that are not particularly helpful in making a specific diagnosis.⁴ More severe hyponatraemia is associated with changes to mental status, convulsions, pulmonary oedema and death.

Management

The initial assessment of a collapsed endurance athlete should include mental status, blood pressure, pulse rate, hydration status, rectal temperature, serum glucose and serum sodium.⁵ If the athlete's pre-race weight is available, comparison with post-race weight is useful because there is a significant inverse relationship between percentage weight loss during endurance events and serum sodium concentration. The more invasive investigations (blood tests and rectal temperature measurement) may not be required in those who have minimal symptoms and who respond rapidly to leg elevation. Most collapsed athletes have

normal blood glucose, electrolytes and body temperature.¹ However, it has been suggested that an endurance athlete who exhibits an altered state of consciousness and a normal rectal temperature should be considered hyponatraemic until proven otherwise.⁵

The majority of cases of hyponatraemia occur during prolonged activity in hot and humid conditions, and it is therefore tempting to make a diagnosis of dehydration or heat stress and to treat with intravenous fluids. As hyponatraemia is primarily a problem of fluid overload, administration of intravenous fluids – even normal saline – can exacerbate the problem so it is important to attempt to make a firm diagnosis beforehand. Hyponatraemia can only be definitively diagnosed by determination of the serum sodium level. After exercise associated collapse, a firm diagnosis of dehydration should be made before intravenous fluids are administered – they are indicated in an endurance athlete who:

- shows clinical signs of dehydration (e.g. dry mucus membranes, inability to spit, decreased skin turgor, sunken eyes)
- has dehydration that causes signs of cardiovascular instability or other medical problems, or
- cannot be treated appropriately with oral rehydration.

Once a diagnosis of hyponatraemia has been made, even a mildly symptomatic athlete should be transported to hospital for monitoring until spontaneous diuresis occurs and symptoms resolve. An athlete with more severe symptoms requires admission to an intensive care unit. Potential management tools in this setting include hypertonic saline, loop diuretics and mannitol – agents that should not be administered in the medical tent.

Prevention

Prevention of hyponatraemia involves appropriate replacement of fluid losses during exercise. Historical concern about the consequences of dehydration may have led to excessive recommendations for fluid replacement. Thirst may be the best clinical

guide and a hydration rate of 500 mL/hour or less is generally reasonable, particularly for those exercising at lower intensities; higher volumes may be required in more elite competitors or in more extreme conditions. Programmed drinking should be avoided. Although the ingestion of sodium-containing sports drinks is preferable to ingestion of water, overuse of these drinks (in an attempt to replace sweat related sodium losses) should be discouraged. The use of salt tablets to prevent hyponatraemia has not been shown to be effective.

Summary

In endurance events, collapse at or just over the finish line is usually due to postural hypotension. A diagnosis of dehydration should be made on firm clinical grounds and hyponatraemia should always be included in the differential diagnosis. **MT**

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