

WATER INTOXICATION

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Definition and Causes:

Water intoxication is an acute condition that may be potentially life threatening due to hyponatremia. Hyponatremia is defined as a sodium level <135mmol/L, severe hyponatremia is defined as a sodium level <115mmol/L. This condition may be seen in individuals who consume large amounts of water in a short period of time without solute replacement causing a drastic decrease in sodium levels. This consumption may be exercise induced, related to psychological disease, iatrogenic, or intentional causes (ie abuse). This discussion will focus on exercise induced hyponatremia.

Hyponatremia related to water intoxication may be influenced by improper functioning of renal diluting mechanisms, hormonal control, or large amount of sodium loss combined with large amount of water intake. An excess of water in the body causes weaker concentrations of sodium. In an attempt for the body to maintain hemostasis the cells within the body absorb more water to equalize concentrations. As the cells absorb more water the swelling of the cells becomes greater, this swelling is the greatest concern in the brain as cerebral edema ensues. Severe cases of hyponatremia have a 50% mortality rate due to its effects on the nervous system.

Several factors that may increase risk in the development of water intoxication are listed below.

- -Exercise duration >4 hours or slow running/exercise pace.
- -Female gender (may be explained by lower body weight)
- -Low body weight
- -Excessive drinking (>1.5L/hr) during an event
- -Pre-exercise overhydration
- -Abundant availability of drinking fluids at the event
- -Nonsteriodal anti-inflammatory drugs
- -Extreme hot or cold environment

History and Studies:

Studies investigating water intoxication predominantly come from marathon runners and military recruits. There have also been several documented cases of backcountry hikers developing water intoxication. Studies were conducted related to fatalities in military recruits between 1989-1996. During this period of time recruits were encouraged to consume large amounts of water (>1.5L) for every hour exposed to temperatures >30C. The result of these studies concluded that severe hyponatremia due to water intoxication was the cause of death in these recruits (Rosner and Kirven 2007).

The prevalence of water intoxication in marathon runners and endurance athletes was fairly uncommon until a change in the practices of water consumption was advised. Athletes began to be encouraged to overcompensate for water loss during activity. After this period 10 deaths were noted as related to water intoxication in a 10 year time frame (Lingampalli 2013) A study done on Boston Marathon runners in 2002 showed correlation between fluid intake and incidence of hyponatremia. This study looked at pre and post race weights and water consumption, as well as sodium levels. It was found 13% had of study participants had hyponatremia, and of that only 0.6% were critical levels(Rosner and Kirven 2007). It was noted in this study that weight gain was the most prevelant predictor of hyponatremia.

Signs and Symptoms:

Typically symptoms are not seen until sodium levels drop below 120mmol/L, this is considered a critical level. Some individuals may appear asymptomatic. Nausea is the most common first sign in water intoxication. Other early signs of water intoxication may include altered mental status, confusion, inappropriate behaviors, delusions, or poor coordination.. As sodium levels drop further and the condition progresses signs and symptoms of increasing intracranial pressure begin to appear due to increasing cerebral edema. Patients may begin to have vital sign changes to include bradycardia, widened pulse pressures and irregular breathing patterns (Cushings Triad). Seizure, coma and even death may occur due critically low sodium levels. Non-cardiogenic pulmonary edema may also be seen in some cases.

Treatment:

Treatment of water intoxication is based on severity of hyponatremia and signs and symptoms. During the early stages of hyponatremia IV fluids with electrolytes or increased consumption of salty foods is advised. More severe cases of intoxication requires hospitalization in order to manage symptoms and correct imbalances. Management of cerebral and pulmonary edema, control of blood pressure and correcting electrolyte imbalances are the primary focus in treatment. Correction of sodium levels must be done carefully, as an undercorrection or overcorrection may also be harmful and potentially deadly.

It is very important to realize that sodium levels may be initially underestimated on lab values due to water that may still be retained within the GI tract. The source that blood is aquired for testing sodium levels is also important. Venous levels may be higher than arterial levels. The difference may be as much as 4mmol/L due to the rapid absorption of water (Rosner and Kirvin 2007). Treatment should be based on arterial values, not venous values.

Cases of severe hyponatremia, NA <115mmol/L, require rapid correction of sodium levels. It is recommended that initial serum sodium levels should be targeted to increase by 4-6mEq/L. If symptoms continue after initial treatment further elevations may be nessesary (Tzamaloukas, Malhotra, Rosen, Raj, Murata & Shapiro 2013). Patients that have hyponatremia with encephalopathy in the setting of water intoxication should receive a 100ml bolus of 3% NaCl infusion over 10 minutes. If no clinical improvement this may be repeated 2 additional times (Sterns, Nigwekar & Hix 2009). These patients should be admitted to an ICU for continued treatment and close monitoring. Monitoring of lab values and clinical status are imperative.

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