any soft tissue injury that may require surgical repair, such as rupture of the conjoined tendon of the hamstring muscles.[15]

If there is displacement of the ischial apophysis of 2 cm or more, open reduction and internal fixation with two or more screws should be carried out early. Cannulated screws allow accurate placement using a guidewire. In chronic cases of a symptomatic fibrous non-union, excision of the fibrous tissue and internal fixation produces a good functional result. Excision of the avulsed fragment should be reserved for those cases in which satisfactory reduction cannot be achieved.[9]

If there is no or minimal displacement of the avulsed apophysis, then conservative management should be pursued. Non-apophyseal injuries involving the musculotendinous junction or the muscle belly itself should also be treated non-operatively.[1] Complete rupture of the conjoined hamstring tendon is rare but is best treated by surgical repair.[16]

Non-operative and post-operative management consists of a well planned rehabilitation programme. This should concentrate on a short period of rest with relaxation of the hamstring muscle group, followed by protected weight bearing, and then a progressive regimen of hamstring stretching and eccentric strengthening.[2]

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Iatrogenic acute hyponatraemia in a college athlete

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Abstract

Hyponatraemia is one of the most common electrolyte abnormalities, leading to significant morbidity and mortality. In the most basic sense, hyponatraemia can be due to sodium loss or fluid excess. The extracellular fluid status is used to clinically divide hyponatraemia into three categories to help to determine both the cause and treatment required. Hyponatraemic patients can be categorised on the basis of their fluid status as hypovolaemic, euvolaemic, or hypervolaemic. Another distinction to make in evaluating hyponatraemia is whether the onset was acute or chronic in nature. The case presented here is iatrogenic acute hypervolaemic hyponatraemia in a college athlete. The patient presented in respiratory distress with an altered mental status after the administration of hypotonic fluids for treatment of muscle cramps. Treatment included intubation, water restriction, and furosemide, to which he responded favourably. Hyponatraemia should be in the differential diagnosis for patients presenting after intravenous fluid administration.

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Keywords: athlete; hyponatraemia; iatrogenic; acute

Hyponatraemia is one of the most common electrolyte abnormalities that lead to significant morbidity and mortality. It is classified as either acute or chronic as well as hypervolaemic, euvolaemic, or hypovolaemic in order to provide appropriate treatment. We present a case of iatrogenic acute hypervolaemic hyponatraemia in a college athlete.

Case report

A 22 year old black male college football player experienced leg cramps after practice and reported to the team doctor. He was diagnosed as having muscle cramps secondary to dehydration. Therefore 5 litres 0.45% normal saline with 5% dextrose was administered intravenously along with 3 litres of liquids by mouth over a five hour period. He later experienced shortness of breath and mental status changes and was brought to the University of Kentucky emergency department. Physical examination showed that he was in respiratory distress with confusion. Vital signs were: blood pressure 146/78 mm Hg; respiratory rate 48 breaths/minute; temperature 37°C; pulse 89 beats/

minute. Examination of the head, eyes, ears, nose, and throat showed normal pupils, no nystagmus, positive gag reflex, and no evidence of trauma. Cardiovascular examination showed a normal rate without murmurs or rubs. Auscultation of the lungs disclosed rales and crackles in all lung fields. Breath sounds, however, were equal. The abdomen was soft and non-tender with normal bowel sounds. Extremities showed no rashes or oedema. Neurological examination showed the patient to be confused and agitated, and intermittently he would not follow commands. He could move all four extremities and had no focal deficits. Chest *x* ray detected pulmonary oedema with a normal cardiac silhouette. Arterial blood gas tests showed a pH of 7.37, P_{CO_2} 4.79 kPa, P_{O_2} 8.38 kPa, and O_2 saturation 91%. Electrolytes were as follows: sodium 121 mmol/l, potassium 3.8 mmol/l, chloride 91 mmol/l, bicarbonate 21 mmol/l; blood/urea nitrogen was 3.93 mmol/l, creatinine 106.1 μmol/l, glucose 6.38 mmol/l, and creatine kinase 40 000 U/l. Initial urine myoglobin was negative. A computed tomography scan of the head was normal.

The patient was diagnosed as having hyponatraemia, pulmonary oedema, and probable rhabdomyolysis. He was admitted to the intensive care unit where he responded to treatment, which included intubation, fluid restriction, and furosemide. Myocardial injury was ruled out by serial electrocardiograms and the results of cardiac enzyme assays. An echocardiogram was normal. He was transferred to the floor after two days in the intensive care unit and was discharged on day three without complications.

Discussion

Hyponatraemia is due to sodium loss or fluid excess.[1] Hyponatraemic patients can be categorised on the basis of their fluid status as hypovolaemic, euvolaemic, or hypervolaemic.[2] The case presented is classified as hypervolaemic hyponatraemia. The underlying causes of hypervolaemic hyponatraemia include congestive heart failure, liver cirrhosis, nephrotic syndrome, renal insufficiency, and iatrogenic water intoxication. Iatrogenic water intoxication is a common cause of inpatient hyponatraemia,[2 3] but is uncommon in the outpatient or prehospital setting.

Hyponatraemia has deleterious effects on the central nervous, cardiovascular, musculoskeletal, and renal systems. Owing to the effects on multiple organ systems, the signs and symptoms are multiple and non-specific. They include headache, nausea, vomiting, weakness, anorexia, and muscle cramps. More severe signs and symptoms such as rhabdomyolysis, disorientation, coma, seizures, diminished reflexes, pseudobulbar palsy, and focal neurological deficits are possible.[1 4] Acute hyponatraemia is associated with a higher mortality than chronic hyponatraemia.

Hyponatraemia may be acute (onset 24–72 hours) or chronic.[1 4–6] The severity and duration of onset both affect the clinical presentation. An acute disturbance causes symptoms at a higher serum sodium level compared with a chronic change. Acute versus chronic is important to discern as it affects the treatment and complications.

Treatment of hyponatraemia varies depending on the acuity, the severity of symptoms, the serum sodium level, and the patient's volume status. The goal in treating symptomatic hyponatraemia is to maintain tissue perfusion and decrease cerebral oedema while avoiding the complications of treatment. Chronic hyponatraemia is corrected more slowly than acute hyponatraemia to avoid the possibility of central pontine myelinolysis. Acute hyponatraemia can be corrected more rapidly with little chance of central nervous system complications. The rate of correction for acute hyponatraemia may be as high as 2 mmol/l per hour, while treatment of chronic hyponatraemia must limit the rise to 0.5 mmol/l per hour.[2]

Treatment of symptomatic hypervolaemic hyponatraemia involves water restriction, diuretic therapy, and administration of both normal and hypertonic saline. Water restriction is the first line of treatment, but cannot be used alone in patients with severe symptoms. Many authors state that hypertonic saline can be used when the patient exhibits severe symptoms or if the serum sodium level is less than 115–120 mmol/l. Hypertonic saline was not used in the case presented because the patient was otherwise healthy and responded well to diuresis.

Iatrogenic hypervolaemic hyponatraemia should be considered as a possible diagnosis in patients, especially athletes, presenting to the emergency department after the administration of intravenous fluids. The underlying cause of hypervolaemic hyponatraemia must be sought while initiating treatment. The severity of symptoms, duration of onset, fluid status, and the serum sodium level should be quickly determined to guide treatment. The treatment should consist of a combination of fluid restriction, diuretics, and either normal or hypertonic saline. Rapid identification and treatment of acute hyponatraemia can lead to decreased morbidity and mortality.

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