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Original Research Paper

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STUDY OF HYPONATREMIA ASSOCIATED WITH MODERATE EXERCISE

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ABSTRACT Exercise associated hyponatremia is a major cause of collapse and confusion after prolonged exercise. Although initially it was attributed to salt loss, it is now predominantly due to excessive consumption of hypotonic fluids during and after the period of exertion and its associated diuresis suppression [1]. Other individual risks include female gender, slow running, NSAID use and excessive fluid ingestion. The weather conditions and advice given to the runners also have a major role in the incidence[2].

The objective of this case series is to view the presentation, treatment and response of those runners who presented with exercise-induced hyponatremia.

KEYWORDS :

INTRODUCTION

Hyponatremia is defined as the serum sodium concentration less than 135mmol/dl. Hyponatremic encephalopathy is a potentially fatal condition with cerebral oedema, vomiting, headache, confusion, seizures and pulmonary oedema[1,3]. It is almost most commonly related to SIADH. There have been several controversies regarding the correction of hyponatremia since too rapid a correction could cause cerebral demyelination. In EIH(Exercise Induced Hyponatremia) treatment recommendations are variable ranging from fluid restriction, 0.9% saline and hypertonic saline being used.

STUDY DESIGN

This was an observational case study at Sree Balaji Medical College. Patients who participated in marathon and presented to ER with altered mental state whose serum sodium concentration was less than 130mmol/dl were considered.

RESULT

A total of 15 patients were diagnosed with exercise-induced hyponatremia. 10 of them presented with confusion. There was no correlation between the running time and serum sodium concentration. All patients received 0.9%saline and 5 of them received 1.8%. 14 patients were symptomatically better the following morning while the others significantly improved.

DISCUSSION

EAH(Exercise Associated Hyponatremia) is dilutional hyponatremia due to excess water ingestion. Water and hypotonic fluids were freely available during the marathon. This was consistent with previous reports that describe that it commonly occurred in women.

In exercise-induced hyponatremia, it should be assumed that the serum sodium level was normal before the onset of the marathon and that the event of hyponatremia is acute allowing rapid correction without risking demyelination. The treatment regime used was rise in serum sodium concentration over 4 hrs from 0.25 to 2 mmol/l/hr. Despite receiving relatively hypertonic fluids, 2 patients demonstrated transient fall in their serum sodium levels.

The neurohormonal response to exercise is complicated with changes in circulating volume, arginine vasopressin, atrial natriuretic peptide and aldosterone being described. A study comparing renal handling of a free water load at rest found no difference in those subjects who had suffered EAH compared to control subjects who had not. Despite these individual variations in urine output and sodium loss, possibly related to hypervolemia and high ANP, that may have attributed to the difference seen.

Due to the relative slow correction of hyponatremia in our patients, none of them developed further complications and made complete recovery allowing rapid discharge. Although follow-up data was not much available, there were no such reports of complications.

CONCLUSION

In conclusion, the presentation of exercise-induced hyponatremia may be delayed and clinical deterioration after presentation has been described. Treatment is controversial but the use of 0.9% saline may not result in the rise in serum sodium concentration possibly due to ongoing absorption of hypotonic fluids and ongoing sodium loss. Presentation of EAH is therefore very important to treat and prevention of complications is very important.

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