

# Marathon Runner with Acute Hyponatremia: A Neurological Disorder

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## Abstract

We report the case of an athletic 49-years old female who has runned the 2011 Marathon of Paris who was addressed to the hospital for a confusion. The investigations revealed a cerebral edema complicating a severe hyponatremia secondary to an exercise-associated hyponatremia (EAH). Using 3% hypertonic saline solution the evolution the patient rapidly improves allowing discharge after 7 days. We then discuss the importance of EAH in long term efforts.

## Case Presentation

We report the case of an athletic 49-years old female with no medical records who has runned the 2011 Marathon of Paris (42.195 Km). In view of the marathon, the patient started to train a few months ahead as per two to three runs weekly. She completed the marathon within 5h30, with an average speed of 7.5 Km/h, on a warm day (27 degrees celsius). Along the run, she drunk 4L of mineral water (5 mg/L NaCl), and had some energy bars at the various feeding stations. Four hours after completion of the marathon, the patient felt dizzy and nauseous, with a strong asthenia and encompassed three vomiting periods associated with disorientation and confusion. She was then admitted to the Emergency Room 7 hours after the aforementioned symptoms occurred.

Upon admission, the patient was conscious but disoriented, clouded, with a Glasgow coma score of 13. She was afebrile and presented no circulatory failure. The neurological examination revealed of acute spread over of osteotendinous reflexes the four limbs, no systematic deficiencies and cutaneous plantar reflexes in flexion, isochoric and reactive pupils. Her body weight was 53.4 Kg (normal average body weight of 50 Kg).

The initial biological parameters were: natremia 121 mmol/L, chloremia 88mmol/L, protidemia 70 g/L, glycemia 8.5 mmol/L, kalemia 3.3 mmol/L, bicarbonates 18 mmol/L, blood urea nitrogen 3.7 mmol/L, creatinemia 68 µmol/L. The calculated osmolality was 260 mosm/kg and the measured osmolality was 246 mosm/kg. Arterial blood gases showed a respiratory alkalosis to compensate an increased plasmatic anion gap metabolic acidosis, with initial blood lactate 10.5 mmol/L. The initial collection of a urine sample showed an urinary osmolality of 489 mosm/L. We report a natriuresis and kaliuresis of 86 and 75mmol/L, respectively. The measure of the free cortisol was 1250 nmol/L at 8am, and 1350 nmol/L following the ACTH test, which therefore discharge form an adrenal origin with recovered hyponatremia.

The patient was then transferred to the Intensive Care Unit (ICU) for diagnosis and treatment. Four hours later, the patient had a generalized tonic-clonic seizure that resolved following 1mg clonazepam IV injection. The cerebral CT-scan showed a diffused supratentorial cerebral oedema. The patient was given a calculated bolus of isotonic saline as per the Adrogue formula. As no further clinical improvements followed, with a persistant hyponatremia of 121 mmol/L, the patient was given a treatment based on 3% hypertonic saline solution. Natremia was recorded and indicated 128 mmol/L and 136 mmol/L at 4 hours and 7 hours after beginning of the treatment, respectively. This biological improvement was associated with the resolution of the neurological perturbations.

The patient was discharge after 7 days following complete resolution of clinical and biological disorders.

## Discussion

The clinical and biological context is a physical Exercise-Associated Hyponatremia (EAH) as defined by a plasmatic sodium concentration below 135 mmol/L 24h following a prolonged physical activity [1,2]. Marathons attract plenty of non-professional runners like our patient who ran her first marathon at the age of 49. Risk factors in favor of EAH occurrence can be merge in two groups : i) runner condition-associated factor (female, low body weight, lack of training, slow runner, excessive drinking consumption during the run, and NSAIDs consumption), ii) environment-related factors (prolonged physical activity of over 4h, warm air temperature, drinks availability along the run [3].

This syndrome, first described in 1981 (1), is closely related to an evolution in high level sport practices (marathon, triathlon) and the concept of abundant hydration during the activity in order to reduce deshydration and hypernatremia risks [4]: an over drinking consumption during and after the run, and the related occurrence of a syndrome of inappropriate secretion of antidiuretic hormone (SIADH). The excessive drinking consumption during activity is the primary and critical risk factor associated to EAH occurrence [4,5]. Then, it has been shown that the risk to develop an EAH is correlated to an increase of body weight during the physical activity as observed in the present patient (+3,4 Kg). There are no indications of a thirst deregulation among runner, as thirst is a late adaptation mechanism in this particular regulation system [6]. Despite this, the hyperhydration during activity on its own can't resume hyponatremia. The kidneys can excrete 750 to 1500 ml/h of water in normal physiological conditions [7]. ADH is a hormone secreted by the hypothalamus, and is subject to stimulation upon low charges (1-2%) in the plasmatic osmolality [8]. In addition to the osmotic stimulation, other mechanisms inducing the central production of ADH have been described: nausea, vomiting organic stress, pain and endorphins, volemic status, central temperature increase or hypoxia [2,4,6-9]. The excess of ADH production is responsible for potentially serious hyponatremia as described by Schwartz and Bartter [10] and recognized as a key factor for the EAH occurrence [2,4,7,9,10]. Moreover, It has been suggested

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that cytokines release (IL6) could also be involved in the non osmotic stimulation of ADH [7,9]. A rapid decrease of natremia (<128 mmol/L) is correlated with the emergence of serious clinical alterations, notably neurological disorders following brain swelling (3), as described in the present patient. Patients associated with symptomatic EAH and subsequent serious neurological (confusion, vigilance disorders, coma, tonic-clonic seizures) or pulmonary (acute respiratory failure) disorders should imperatively be transferred in ICU with a repeated surveillance of the natremia. Acute hyponatremia treatment can benefit of a novel concept associated with a remarkable therapeutic efficiency [2,4,7,9,10]. Treatment of serious EAH conditions involves the administration of a 3% hypertonic saline solution at 1 ml/kg/h that is subsequently adjusted following natremia status. Increases of natremia to 1 mmol/L/h during the first 6h, 9 mmol/L during the first 24h and 18 mmol/L during the first 48h are accepted. Ideally, natremia should not evolve over 20-25 mmol/L during the first 48h [5,10]. The isotonic saline solution appears inefficient for the treatment of EAH as described in a previous study [4] and as observed in the present case, in which no noticeable positive biological response was detected following initial administration of saline.

The EAH prevention relies on information regarding hyperhydration risks during the run and the necessity to control water supplies in a range of 400 à 700 ml/h, depending on body weight and weather conditions [5]. Beltrami has described the historical link between overdrinking during a prolonged and intensive sport activity and the occurrence of hyponatremia. This overdrinking behavior seems to be supported by sport drinks selling companies... Moreover, these sport drinks (enriched solutions with mineral salts and ions, slightly less hypotonic than mineral water) do not prevent for EAH, neither are the sodium supplier by systematic per os intake [3].

## Conclusion

In summary, EAH is associated to a SIADH which the severity is

as high as is the hydric supply during and after the physical activity, occurring during or in the few hours following the activity. It could sometimes evolve as critical emergency situation with signs of neurological disorders, and the only efficient treatment consists of the administration of hypertonic saline solution. The prevention strategy consists in informing runners on good hydration practices during the run and the hyperhydration-associated risks.

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