H181 Exercise-Associated Hyponatremia: An Update

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Learning Overview: After attending this presentation, attendees will better understand the critical nature of recognizing Exercise-Associated Hyponatremia (EAH) as a potentially fatal complication of overhydration and extreme exertional exercise that requires rapid diagnosis and treatment.

Impact on the Forensic Science Community: This presentation will impact the forensic science community by: (1) introducing attendees to this subset of acute hyponatremia that occurs exclusively in elite athletic populations; and (2) explaining the underlying physiology, the importance of rapid diagnosis and treatment, and the far-reaching implications of this condition for diagnostic and treatment algorithms developed for military training exercises.

EAH is a preventable condition associated with overconsumption of hypotonic fluids in a setting of extreme physical exertion that exceeds the renal capability to diurese.[1] EAH is seen in elite athletic populations, such as marathon runners, triathletes, and military training populations.[2] Long-held misconceptions regarding how much fluid intake is required during exercise exacerbates the problem of EAH.[3] The treatment of severe hyponatremia has traditionally focused on correcting the sodium deficit slowly to prevent the potentially lethal complication of central pontine myelinolysis. This traditional approach for the treatment of chronic hyponatremia does not apply to the current model of EAH in this population of elite athletes. The drop of serum sodium is due to water overconsumption during extreme exercise, can occur rapidly, and must also be corrected rapidly to avoid the fatal complication of irreversible cerebral edema, also termed Exercise-Associated Hyponatremic Encephalopathy (EAHE). Once recognized, the sodium deficiency must be corrected with 3% hypertonic saline as soon as possible. Rapid correction of the sodium deficits in acute exertional hyponatremia does not result in the classic complication of central pontine myelinolysis and avoids the complication of EAHE.[4] If treated per the traditional model in which the sodium deficit is corrected gradually, the use of 0.9% saline or other isotonic solutions in these patients can worsen volume overload, This leads to potentially fatal complications, such as pulmonary edema and EAHE.

The underlying physiology of EAH is hallmarked by the sodium-conserving actions of anti-diuretic hormone. These patients develop Syndrome Of Inappropriate Antidiuretic Hormone (SIADH) in which vasopressin is paradoxically released, likely as a sodium conservation measure.[5] This increase in vasopressin allows the renal tubules to conserve salt and also retain water, which prevents the usual diuresis from fluid resuscitation or continued overhydration. This inability to off-load excess fluid greatly predisposes these patients to pulmonary edema and ultimately irreversible cerebral edema.

This presentation reviews three fatal cases of EAH, examines the underlying physiology behind EAH, and reviews the diagnosis and treatment algorithms currently in development by the United States Army.[6]

Reference(s):

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2. Nutrition and Athletic Performance American College of Sports Medicine Joint Position Statement. March 2016.
3. Racinais et al. Consensus Recommendations on Training and Competing in the Heat. *BJSM* 2015;1164-1173.
4. Tien R. et al. Hyponatremic Encephalopathy: Is Central Pontine Myelinolysis a Component? *Am J Med* 1992 May; 92(5): 513-522.
5. Thomas C.P., *Syndrome of Inappropriate Antidiuretic Hormone Secretion*, updated 22 Apr 2017. http://emedicine.medscape.com/article/246650-overview#a4.
6. TB MED 507/AFPAM 48-152. *Heat Stress Control and Heat Casualty Management*. 7 March 2003.

Hyponatremia, Exercise, Electrolytes