Severe hypernatremia in an adolescent male with anorexia nervosa

Kene Ebuka Maduemem, Comfort O. Adedokun

ABSTRACT

Introduction: Anorexia nervosa is a commonly encountered cause of severe underweight in pediatric settings. It is an eating disorder characterized by the inability to maintain a minimally normal weight, a devastating fear of weight gain, relentlessly dietary habits that prevent weight gain, and a disturbance in the way in which the body image is perceived. Hypernatremia is an unusual electrolyte imbalance in anorexia nervosa.

Case Report: This is a case of a 12-year-old male admitted with progressive weight loss following restrictive food intake for the preceding one year. He presented with severe hypernatremia. Correction of severe hypernatremia was successful after four to five days. This correction was slow but gradually achieved. There was no altered sensorium throughout admission which remains the worst nightmare of any managing team. Feeding protocol as per Junior MARSIPAN guidelines was adhered to. He made an excellent recovery and was discharged after 24 days of admission with a weight gain of 6.1 kg.

Conclusion: Anorexia nervosa is commonly encountered in pediatric settings and can cause potentially life-threatening physical and psychological complications. Hypernatremia is an uncommon metabolic abnormality; slow correction averts untoward side effects.
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Keywords: Anorexia nervosa, Hypernatremia, MARSIPAN guidelines

INTRODUCTION

Diagnostic and Statistical manual of Mental Disorders, Fifth edition (DSM-V), published in May 2013 revised the definition of anorexia nervosa from DSM-IV by removing amenorrhea as one of the criteria for diagnosis while focusing more on behaviors [1]. It is a commonly encountered cause of severe underweight in pediatric settings that can cause potentially life-threatening physical and psychological complications [2]. Hypernatremia is an unusual manifestation in anorexia nervosa. It is caused by net water loss (increased loss or decreased intake) or, rarely, sodium gain.

Herein, we describe a case of a 12-year-old boy admitted with progressive weight loss following restrictive food intake for the preceding one year. He presented with severe hypernatremia.
CASE REPORT

A 12-year-old boy was referred by his general physician on account of a week history of progressively worsening dizziness and lethargy. He had been refusing feeds for the last three months with dramatic weight loss. This is on a background of the desire to be ‘skinny’ for the preceding two years. There was a history of hiding food. Parents were suspicious of self-induced vomiting but never witnessed. No known use of laxatives or purgatives. Past medical history was insignificant. He has been a very well adolescent who gets on very well with siblings and friends. He looked up to Mo Farah (a British athlete) as his mentor.

He lived with parents and has two older siblings (16-year-old sister and 14-year-old brother). He was in 6th class and academically sound. He was very athletic: engages in at least one sporting activity per day during school year. No history suggestive of bullying in school. However, noted to have been called ‘fat’ by a classmate in 2nd class.

Examination revealed a cachectic, lethargic, pale looking and dehydrated adolescent male. Vital signs were: temperature 36.4 °C, heart rate 40/min, respiratory rate 15/min, supine blood pressure 108/66 mmHg, standing blood pressure 98/58 mmHg. Other significant findings were prominent ribs, scaphoid abdomen, dry, scaly skin, lanugo hair on arms, yellow palms. Weight was 32.4 kg, height 156.6 cm, body mass index (BMI) 13 kg/m².

A diagnosis of restrictive type anorexia nervosa was made. He was admitted for fluid resuscitation. Vital signs were continuously monitored with ECG monitor. Referral was sent to CAMHS, medical social work and dietician. He was strictly managed following the Junior MARSIPAN guidelines. There was a steady improvement in demeanor and weight.

Urgent electrocardiogram confirmed sinus bradycardia; heart rate of 38 beats per minute, normal QTc 420 ms (Figure 1). Admitting blood glucose was 8.2 mmol/L. Serum electrolytes and urea levels were deranged (sodium 182 mmol/L, potassium 2.7 mmol/L, chloride 143 mmol/L, urea 17.2 mmol/L). Creatinine: 82 umol/L. Calcium 2.42 mmol/L, Magnesium 1.28 mmol/L, phosphate 0.95 mmol/L. Hemoglobin 14.2 g/dl, White cell count 8.2 per μL (neutrophil 2.79, lymphocyte 4.92). C-reactive protein <0.2 mg/L. Venous blood gas: pH 7.38, pCO₂ 7.6, bicarbonate 33.7, base excess 8.6. Serum osmolality 369 mosm/kg. Serum ferritin 223 ng/ml, vitamin B12 650 ng/L, serum folate 5.8 ng/ml. Urine osmolality 1360 mosm/kg. Spot urine sodium 305 mmol/L. Thyroid function test was normal. Celiac serology was negative.

Initial rehydrating fluid and regime was 5% dextrose in 0.9% saline. Aim of correction of hypernatremia was 0.5 mmol/L/h or 12 mmol/L/day. Potassium chloride was added in the fluids (20 mmol/500 ml). Serum electrolytes were checked every 4–6 hours. Serum calcium, phosphate and magnesium were checked daily to prevent refeeding syndrome.

Serum sodium level decreased from 182 mmol/L to 174 mmol/L at the end of the first 24 hours of admission (drop of 8 mmol/L). This gradually and steadily decreased to baseline on day-5 of admission to 140 mmol/L (Figure 2).

Oral feeding was gradually initiated by dietician. Sodium level returned to normal on day-5 of admission.
This correction was gradual and painstaking. Intravenous fluid was completely weaned off by the end of the first week. Twice weekly weighing was adhered to.

Daily ECG showed gradual improvement of bradycardia. Dietician plan was based on refeeding meal with initial daily calorie intake of 400 kcal which was gradually increased to 900 kcal within four days. Oral fluid intake comprising milk only was successively increased from 500 ml to over 800 ml within three days. The caloric requirement for his age was worked out to be 2250 kcal/day. On discharge, his daily caloric intake was 2000–2200 kcal.

The patient made a remarkable recovery. He was discharged after 24 days of admission. Weight on discharge was 39.5 kg (weight gain of 6.1 kg), BMI of 16.1 kg/m². He has been reviewed severally on the assessment unit and making good progress. He has been subsequently discharged from CAMHS services.

**DISCUSSION**

Electrolyte imbalances associated with anorexia nervosa increase the morbidity level significantly. Hypokalemia is the most common electrolyte abnormality. Hyponatremia is often due to excessive water ingestion, but may also occur in chronic energy deprivation or diuretic misuse [3]. Hypernatremia can also be classified based on the intravascular volume status-euvolemia, hypervolemia and hypovolemia. Hypernatremia initially causes fluid movement out of the brain that leads to cerebral contraction and consequently manifests as altered sensorium.

Manifestations of hypernatremia vary from non-specific central nervous system symptoms such as nausea, vomiting, irritability and lethargy to confusion, seizures, myoclonic jerks, coma and even death. Severe symptoms are likely to occur with acute increase in plasma sodium levels or concentration >160 mmol/L [4]. Hypernatremia often is the result of several concurrent factors. The most prominent is fluid intake.

Our patient was restricting food intake including water. There was no history of polyuria or polydipsia to suggest a possible diabetes insipidus given the level of hypernatremia. There was no strong evidence of any diuretic abuse or any form of gastrointestinal fluid loss. In this case, severe hypernatremia was thought to be chronic than acute. Correction was done slowly over 4–5 days. The choice of fluid was normal saline with 5% dextrose as the patient was not drinking. The fluid was subsequently changed to 0.45% saline. The volume of fluid was reduced from full to 50% maintenance. There was a gradual drop in the serum sodium level (Figure 2).

In patients with hypernatremia that developed slowly, the sodium level should be corrected at a rate of 0.5 mEq/L/h, with no more than 10–12 mEq/L in 24 hours [5, 6]. The target sodium level should be 145 mEq/L.

However, no prospective studies completely validate such recommendations [7].

Liam et al. published a teaching case highlighting management guidelines for chronic hypernatremia with emphasis on slower correction rate [7]. Conversely, Alshayeb et al. published a clinical investigation highlighting the association of persistent hypernatremia with increased mortality [8]. Fang et al. concluded that sodium correction rate of >0.5 mEq/L/h is associated with the development of cerebral edema [9]. By contrast, Robertson and team did not find any association between the rate of decline in serum sodium and clinical outcome [10]. He was not particularly volume depleted evidenced by his stable vital signs. Appropriately high urine osmolality will point towards a non-renal cause of euvolemic hypernatremia such as isolated hypodipsia or increased insensible losses. The former would fit into this case. The rate of correction of the hypernatremia was relatively slow but less dangerous. Feeding protocol was as per MARSIPAN guidelines to avoid refeeding or underfeeding syndromes [11].

**CONCLUSION**

Anorexia nervosa is commonly encountered in pediatric settings and can cause potentially life-threatening physical and psychological complications.

The management of anorexia nervosa is based on guidelines published by the Royal College of Psychiatrists with an emphasis on multidisciplinary approach. The goal in managing hypernatremia is to correct the water deficit in a reasonable time frame while avoiding untoward side effects.

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**Author Contributions**

Kene Ebuka Maduemem – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Comfort O. Adedokun – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

**Guarantor**
The corresponding author is the guarantor of submission.

**Conflict of Interest**

Authors declare no conflict of interest.

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