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Case Report

Hyponatremia, a Constant Clinical Challenge: Euvolemic Hyponatremia Secondary to Solute Loss

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Abstract

A 42-year-old female male with deceased donor kidney transplantation presented with a month old history of diarrhea and serum sodium of 116 meq/L. Her weight had been unchanged over the past 6 months. Patient had maintained adequate hydration of 2-3 liters per day despite diarrhea as per previous instructions.On presentation, she was euvolemic and non-orthostatic and rest of the examination was unremarkable. The SIADH which was initially suspected was ruled out due to remarkably low urine osmolality (62 mOsm/Kg). She had no history of alcoholism, malnutrition, or psychogenic polydipsia. A diagnosis of euvolemic hyponatremia was made and cause was attributed to solute depletion from chronic diarrhea while she maintained liberal free water intake. Serum sodium normalized with intravenous normal saline followed by high salt diet. The case highlights that patients with diarrhea may present with euvolemic hyponatremia instead of hypovolemic hyponatremia if adequate oral free water intake is maintained.

Keywords: Hyponatremia; Kidney transplantation

Introduction

Solute loss as in diarrhea usually results in hypovolemic hyponatremia. We present a patient who developed euvolemic hyponatremia secondary to diarrhea as she maintained an adequate free water intake.

Case Presentation

A 42 year old Hispanic female with a history of kidney transplant a year ago was evaluated for diarrhea for the past 4 weeks. Her immunosuppression consisted of mycophenolate 720 mg oral twice daily, tacrolimus 5 mg oral twice daily, and prednisone 5 mg oral daily. Patient had her mycophenolate dose cut down and subsequently stopped 2 weeks prior to presentation due to continuous diarrhea. The initial stool studies ordered at that visit were unremarkable. She was advised to maintain adequate hydration. At her current visit, she was afebrile with blood pressure 128/68 mmHg, pulse 61/minute, respiratory rate 18/min, weight 122.6 lbs (55.6 kg), and BMI 21.7. Her weight had been unchanged over the past 6 months. She was euvolemic and non-orthostatic and rest of the examination was unremarkable. Laboratory data revealed: Serum Na- 116 mEq/L, K- 4.0 mEq/L, Cl- 83 mEq/L, HCO3-24 mmol/L, Glucose- 80 mg/dL, BUN- 8 mg/dL, Creatinine- 0.9 mg/dL, Calcium- 8.2 mg/dL, Albumin- 3.8 g/dL, Phosphorus- 2.7 mg/dL, Magnesium- 1.2 mg/dL, and tacrolimus trough level 9.4 mg/dl. Patient's serum Na was normal at 138 mEq/L a month ago. She denied excessive thirst but admitted drinking approximately 2-3 liters of water every day. Further work up revealed measured serum osmolality of 239 mOsm/kg, TSH-0.208 μIU/mL, Free T4- 1.4 ng/dL, random serum cortisol- 7.5μg/dL, urine osmolality- 62 mOsm/Kg, Urine Na < 19 mEq, Urine K < 7.5 mEq, Urine Cl <3 mEq, a 24-hour urine volume of 1.5 l, and 24-hour urine osmolar load (= urine osmolality x 24-hour urine volume) of 93 mOsm. The SIADH which was initially suspected was ruled out due to remarkably low urine osmolality. She had no psychiatric disease and was not taking any anti-depressants or anti-psychotic medications. A diagnosis of euvolemic hyponatremia was made and cause was attributed to solute depletion from chronic diarrhea while maintaining liberal free water intake. Patient responded well to IV hydration with normal saline given initially and then switched to high salt diet with subsequent normalization of serum Na to 138 mEq/L. Diarrhea was later attributed to rotavirus infection and it responded to decrease in immunosuppression.

Discussion

Hyponatremia is of three types: Hypovolemic, Euvolemic and Hypervolemic. Of these, euvolemic hyponatremia is the most common and accounts for 60% of hyponatremia. SIADH is the most common cause of euvolemic hyponatremia [1,2]. Other causes of euvolemic hyponatremia are hypothyroidism, primary polydipsia, beer potomania, endurance exercise and adrenocorticotropin deficiency or idiosyncratic drug reactions with thiazide diuritics or ACE inhibitors.Euvolemia is a clinical diagnosis aided by good history and physical examination, low serum uric acid levels, a normal blood urea nitrogen– to-creatinine ratio, and spot urinary sodium greater than 20 mEq per L [3].

The three important causes of hyponatremia- SIADH, low solute intake, and hypovolemia may look alike on initial presentation but may be distinguished based on the clinical and laboratory features as mentioned in Table 1. SIADH is usually a diagnosis of exclusion made in the setting of euvolemic hyponatremia typically with high urine osmolality and high/normal urine Na+ [8]. In our patient, the SIADH was ruled out due to remarkably low urine osmolality and low urine Na+. Clinically, the patient was euvolemic despite ongoing diarrhea likely due to adequate oral free water intake. There was no history of low solute intake. Low dietary solute intake is common in

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Table 1: Causes of hyponatremia.

	SIADH	LOW SOLUTE INTAKE	HYPOVOLEMIC
CAUSES	Tumors, Central Nervous system Disorders, Pulmonary Diseases	Beer Potomania Malnutrition (Tea and Toast Syndrome)	Diuretic Use (commonly thiazides), Salt- wasting nephropathy, Mineral corticoid deficiency
Serum Na+	Low	Low	Low
Serum Osmolality	Low	Low	Low
Urine Osmolality	High	Low	High
Urine Output	Normal	Normal	Decreased
Urine Na+	Normal/High	Low	Low
Patients' Weight	No acute change	No acute change	Decreased

beer drinkers (beer potomania) and malnourished patients who have low protein and high water intake diet. They have a marked reduction in water excretory capacity due to low solute excretion and excess of free water ingestion [4-6]. Their diet has very little or no sodium, potassium or protein, therefore their total daily solute excretion is less than 250 mOsm and hyponatremia can occur if they have free water intake more than 4 l/day[1-7]. They have suppressed ADH levels and typically low urine sodium and low urine osmolality [9]. On a normal American diet, the daily urine osmolar load is approximately 10 mOsmol/Kg of body weight. In a healthy person, the minimum urine osmolality (given no circulating serum ADH) can be as low as 50 mOsm/Kg. In our patient, the daily urine osmolar load of 93 mOsm was lower than expected urine osmolar load (10 mOsm/Kg= 556 mOsm) allowing for only 1.5 L urine output which was lower than her oral free water intake of 2-3 l per day. Hence, she retained excess water, resulting in fall in serum Na+. Once, her solute loss was corrected with normal saline and salt pills, the hyponatremia was corrected.

In conclusion, solute loss (as in diarrhea) but with adequate oral water intake may present with clinical picture of euvolemic hyponatremia instead of hypovolemic hyponatremia. This presentation should be considered in the discussion of the causes of euvolemic hyponatremia.

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