

Unusual Presentation of Hyponatremia: Is it Water, is it medications, is it both or more.

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Abstract

Introduction

Case reports on multifactorial causes and atypical presentations of hyponatremia are infrequent. We present a case of multifactorial hyponatremia in a middle aged gentleman presenting with neurological deficits.

Case Presentation

A 55 year old Caucasian man with history of alcohol abuse, Hypertension and Type 2 Diabetes Mellitus presented with one day history of multiple falls, altered mentation, slurred speech and unilateral weakness. Patient drinks 4-6 beers/day and has recently been consuming around 2 gallons of water/day. Home medications were Hydrochlorothiazide, Lisinopril, Nifedipine, Dapagliflozin and Venlafaxine.

Patient was afebrile, pulse 95/min, blood pressure 112/54 mmHg, respiratory rate 22/min and O2 saturation 97% on room air.

On examination patient was euvolemic, encephalopathic with slurred incoherent speech. Rest of the exam was benign.

Labs were significant for Sodium(Na) 108 (UrineNa13), chloride 69, glucose 127, Osmolality 218 (Urine Osmolality 374), Cortisol 19, Thyroid Stimulating Hormone 1.3, Creatinine 1.2 (UrineCr 65.8), Blood urea nitrogen 27, Serum alcohol <10. Computer tomography Angiogram Head and neck and Electro-encephalogram(EEG) were unrevealing.

Diagnosis

Differentials for his neurological manifestations included Cerebrovascular-accident (CVA), seizure, Hyponatremia, infectious/metabolic encephalopathy and alcohol withdrawal.

CVA was ruled out, EEG for epileptiform discharges and infectious workup were negative. Signs and sympathetic symptoms of alcohol withdrawal were absent. Uremia, Hyperammonemia or carbon-dioxide narcosis were ruled out.

Hyponatremia workup revealed a conflicting picture with low Urine Na suggestive of polydipsia or Thiazide induced Hyponatremia but the High Urine Osmolality suggestive of Syndrome of Inappropriate ADH (SIADH) (table 1).

This patient had multiple contributory factors for hyponatremia including medications Venlafaxine (SNRI) and thiazide diuretics, poor nutrition and polydipsia.

Management

Venlafaxine and thiazide were discontinued. Hypertonic (3%) Saline was started until serum Na reached 121mmol/L which improved the confusion and focal neurological deficits. Mental status returned to baseline by day 4. The table shows trend in Sodium levels (fig.1).

Learning Objectives

1. Identify that Hyponatremia though is a common occurrence in elderly, it is important to consider the diagnosis and recognize its neurological manifestations in younger patients.
2. Implement an in-depth evaluation to diagnose a multifactorial etiology of hyponatremia where the routine algorithmic evaluation of hyponatremia utilizing the volume status and urine osmolality to ascertain one particular etiology might not be appropriate
3. Recognize that goal of treatment with Hypertonic saline is the resolution of acute neurological manifestations than a normalization of sodium levels

Tables and/or Figures

Table 1. Urine Sodium and Osmolality levels in Hyponatremia based on etiology

Hyponatremia Etiology	Urine Sodium (Na)	Urine Osmolality
Thiazide diuretic Induced*	< 25 (Usually ranges close to 20)	> 100
Hypovolemia	< 25	< 100
SIADH*	> 40	> 100
Venlafaxine Induced*	> 40	> 100
<u>Potomania*</u>	< 10	< 100
<u>Polydypsia*</u>	< 10	< 100

* Potential cause/contributor to the hyponatremia seen in our patient

Figure 1. Sodium trend during the hospitalization

