EDORIUM Journals

J Case Rep Images Obstet Gynecol 2022;8:100107Z08MS2022.
www.ijcriog.com

CASE REPORT PEER REVIEWED | OPEN ACCESS

Pseudohyponatremia in pregnancy

Marcos Sosa

ABSTRACT

Introduction: Ordering multiple laboratory tests has become commonplace in contemporary medical practice. Clinicians are sometimes surprised by abnormal test results that are unrelated to the chief complaint. Misguided attempts to correct abnormal lab values can potentially compromise patient safety. We report what we believe to be a rare occurrence of pseudohyponatremia complicating pregnancy.

Case Report: A 28-year-old woman with pregestational diabetes at 21 weeks gestation was admitted with a chief complaint of headache. An electrolyte panel revealed a serum sodium of 111 mEq/L. A repeat sodium level was unchanged at 111 mEq/L. The diagnosis of pseudohyponatremia was confirmed using direct potentiometry.

Conclusion: Pseudohyponatremia should be considered prior to initiating corrective measures for hyponatremia, especially in an asymptomatic patient.

Keywords: Direct potentiometry, Hyperlipidemia, Pregnancy, Pseudohyponatremia

How to cite this article

Sosa M. Pseudohyponatremia in pregnancy. J Case Rep Images Obstet Gynecol 2022;8:100107Z08MS2022.

Article ID: 100107Z08MS2022

****

Marcos Sosa[1], MD

Affiliation: [1]Department of Obstetrics and Gynecology, Atrium Health, Charlotte, NC, United States.

Corresponding Author: Marcos Sosa, MD, 4827 Camilla Dr, Charlotte, NC 28226, United States; Email: dr.marcos.sosa@gmail.com

Received: 08 August 2021
Accepted: 15 October 2021
Published: 07 February 2022

doi: 10.5348/100107Z08MS2022CR

INTRODUCTION

According to a recent report, hyponatremia is the most common electrolyte abnormality in hospitalized patients [1]. The mean age of hyponatremic individuals in that report was 67. This may partially explain why low sodium is less commonly encountered in pregnant women. Also, it must be remembered that serum sodium in pregnancy decreases physiologically by 3–5 mEq/L, necessitating adjustment of values used to define the normal range.

True hyponatremia, even when mild, is associated with increased mortality [1]. For this reason corrective measures are often undertaken immediately, as in our patient. However, unnecessary treatment of pseudohyponatremia can result in morbidity and mortality. The purpose of our case report is to remind clinicians to consider a series of easy steps to identify pseudohyponatremia before embarking on attempts to correct hyponatremia, especially in the setting of absent clinical symptoms.

CASE REPORT

A 28-year-old woman with pregestational type I diabetes, well controlled with a mix of insulin aspart and insulin NPH, at 21 weeks gestation presented to triage with a chief complaint of a throbbing headache for 12 hours. She suffered from chronic migraines and, when not pregnant, would take sumatriptan at symptom onset. However, she was concerned about the effect of sumatriptan on her fetus and declined to take the medication while pregnant. She was admitted to the hospital for management of a headache. Her blood pressure was normal at 112/68 and her urine dip was negative for protein. Her blood glucose level was 112 mg/dL. A routine electrolyte panel was ordered and revealed a serum sodium of 111 mEq/L. This was immediately repeated to rule out a laboratory error. The repeat sodium level was once again 111 mEq/L.

Oral fluids were restricted and intravenous normal saline was initiated at 300 mL/h while awaiting the repeat sodium evaluation. When the repeat electrolyte

panel showed no improvement in serum sodium, the administration of hypertonic saline was discussed, but not implemented. An osmolal gap was calculated and found to be elevated at 58 mOsm/kg of H_2O (see calculation below). A concern for possible ketoacidosis prompted an arterial blood gas evaluation. A normal pH of 7.41 reduced the concern for ketoacidosis. The diagnosis of pseudohyponatremia was entertained. A venous blood sample was analyzed on a blood gas machine. A blood gas machine uses a different methodology to assay serum sodium (direct potentiometry). The result was 138 mEq/L. Normal saline infusion and fluid restriction were discontinued. A serum cholesterol panel was obtained and revealed a total cholesterol of 1193 mg/dL. Further, a lipoprotein electrophoresis revealed a triglyceride level of 3140 mg/dL and markedly increased chylomicrons. A protein electrophoresis was normal.

DISCUSSION

Hyponatremia is the most common electrolyte abnormality in hospitalized patients [1]. Morbidity and mortality associated with hyponatremia often leads clinicians to initiate measures to correct the serum sodium. Overly aggressive management in hyponatremic patients can lead to long-term neurological complications including central pontine myelinolysis. Pseudohyponatremia, a falsely low serum sodium, must be included in the differential diagnosis of a patient with a low serum sodium. We offer three recommendations to establish the diagnosis of pseudohyponatremia.

The first step is to measure the serum osmolality. The difference between the measured osmolality and the calculated osmolality is defined as the osmolal gap (contrast with anion gap) [2]. Calculated osmolality is determined by the following formula:

Calc Osm = 2X[Na] + BUN/2.8 + Glucose/18

The patient's serum osmolality was 290 mOsm/kg of H_2O. Her sodium level was 111 mEq/L, BUN 9 mg/dL, and glucose 112 mg/dL. Her calculated osmolality was 231 mOsm/kg of H_2O, giving her an osmolal gap of 58 mOsm/kg of H_2O. This elevated osmolal gap (normal less than 10 mOsm/kg H_2O), along with her failure to respond to corrective measures, led us to suspect pseudohyponatremia.

The second step is to use an alternative method of measuring serum sodium. Most modern laboratories use indirect potentiometry to measure sodium. This technique involves dilution and an assumption regarding the nonaqueous fractional components of serum. Details regarding this methodology are provided in a reference below [3]. Most blood gas machines use the method of direct potentiometry. Employing this assay in our case yielded a "true" sodium value of 138 mEq/L, confirming a diagnosis of pseudohyponatremia.

The final step is to determine the cause of the falsely low sodium. Most often it is hyperlipidemia (elevated chylomicrons, or triglycerides, or both), severe hyperproteinemia, or paraproteinemia. Our patient had very high chylomicrons and triglycerides.

In conclusion, we recommend consideration of pseudohyponatremia before any attempt to correct low serum sodium. Even though hyponatremia is a potentially dangerous condition, inappropriate management of pseudohyponatremia can lead to adverse outcomes.

CONCLUSION

Hyponatremia is a common laboratory finding. Severe hyponatremia in the absence of symptoms should compel the clinician to consider pseudohyponatremia as a diagnosis. Treating pseudohyponatremia with fluid restriction and hypertonic saline could be disastrous. Compare the serum osmolality to the calculated osmolality. A value greater than 10 mOsm/kg H_2O should prompt the clinician to consider pseudohyponatremia and related causes. Elevated serum proteins and lipids may be causes of pseudohyponatremia. Laboratory assessment of serum samples using a machine that performs evaluations via direct potentiometry, as compared to indirect potentiometry, may assist in the diagnosis.

REFERENCES

1. Waikar SS, Mount DB, Curhan GC. Mortality after hospitalization with mild, moderate, and severe hyponatremia. Am J Med 2009;122(9):857–65.
2. Turchin A, Seifter JL, Seely EW. Clinical problem-solving. Mind the gap. N Engl J Med 2003;349(15):1465–9.
3. Fortgens P, Pillay TS. Pseudohyponatremia revisited: A modern-day pitfall. Arch Pathol Lab Med 2011;135(4):516–9.

****

Author Contributions

Marcos Sosa – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Guarantor of Submission

The corresponding author is the guarantor of submission.

Source of Support

None.

Consent Statement

Written informed consent was obtained from the patient for publication of this article.

Conflict of Interest

Author declares no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.

Copyright