Case Report

Hyponatremia: Case series with review of literature

Murthy M[1], Chatterjee S[2], Nigam R[3], Kosam D[4]

[1]Dr. Madhumita Murthy, Associate Professor, [2]Dr. S Chatterjee, Professor, [3]Dr. R Nigam, Associate Professor, [4]Dr. D. Kosam, Assistant Professor. All are affiliated with Department of Anesthesiology and Critical Care, Chhattisgarh Institute of Medical Sciences, Bilaspur, CG, India

Address for Correspondence: Dr Madhumita Murthy, E-mail: madhumitagarimella@gmail.com

Abstract

Hyponatremia is the most common fluid & electrolyte disorder prevalent in hospital settings, manifesting in form of wide spectrum of clinical symptoms resulting in increased morbidity and duration of hospital stay in critically ill patients. Here we are presenting three cases of diselectrolytemia which were later diagnosed to be cases of hyponatremia. The pathophysiology, clinical signs and symptoms along with recent guidelines for treatment of a case of hyponatremia is also discussed here in details.

Key words: Hyponatremia, Diselectrolytemia , Syndrome of inappropriate ADH secretion

Introduction

Serum sodium concentration <135 mmol/L is diagnosed as a case of hyponatremia. It is the most common disorder of body fluid and electrolyte balance with wide array of symptoms requiring an estimated 15-20% of emergency admissions to hospital [1].

Owing to its diverse presentations ranging from subtle to severe and at times life threatening, hyponatremia is therefore both common and important. For a long time, management of diselectrolytemia in general and hyponatremia in particular has remained institutional and speciality-centric. In the absence of common and holistic approach, the clinicians and the intensivists often had to face therapeutic dilemma, to be conservative or aggressive with management protocols. Abnormal serum sodium levels if corrected too slowly or too fast, can lead to irreversible neurological damage. Along with thorough understanding of the pathophysiology of hyponatremia, the fluid status of patient should also be corrected appropriately [2]. Features like gait disturbances, falls, concentration and cognitive deficits are common in these patients[3]. Osteoporosis is a frequent finding in cases of chronic hyponatremia and they often sustain bone fractures[4,5,6]. Whether these are concomitant findings with hyponatremia or are a result of renal or liver failure, is still uncertain, more often than not resulting in increased risk of terminating fatally[7,8,9].

Case-Reports

Manuscript received: 4th Mar 2015
Reviewed: 29th Mar 2015
Author Corrected: 5th Apr 2015
Accepted for Publication: 20th Apr 2015

1. A 40 year old lady was referred to our teaching hospital with complaints of high grade fever and considerable weakness for one week. There was no significant past medical history. Careful analysis of previous investigations and confirmation in our set-up, it was found that her electrolytes were deranged. Na^+ 125meq/L & K^+ 3.5 meq/L. Rest of the laboratory reports were normal. In the earlier hospital she had been administered with 3% NaCl solution. Gradually she developed numbness of limbs & later hemiparesis. It was diagnosed as a case of osmotic demyelination syndrome, secondary to rapid correction of serum Na^+. On admission, she was drowsy, had an attack of convulsions and was in respiratory distress. She was promptly treated for sepsis.

2. A 75 year old lady arrived at our teaching hospital in grave condition with fever and psychosis from a private health facility, where she had developed hemiplegia. The discharge summary and laboratory tests revealed previous hyponatremia which was rapidly corrected leading to this neurological sequaele. Despite critical care supports, she could not be revived.

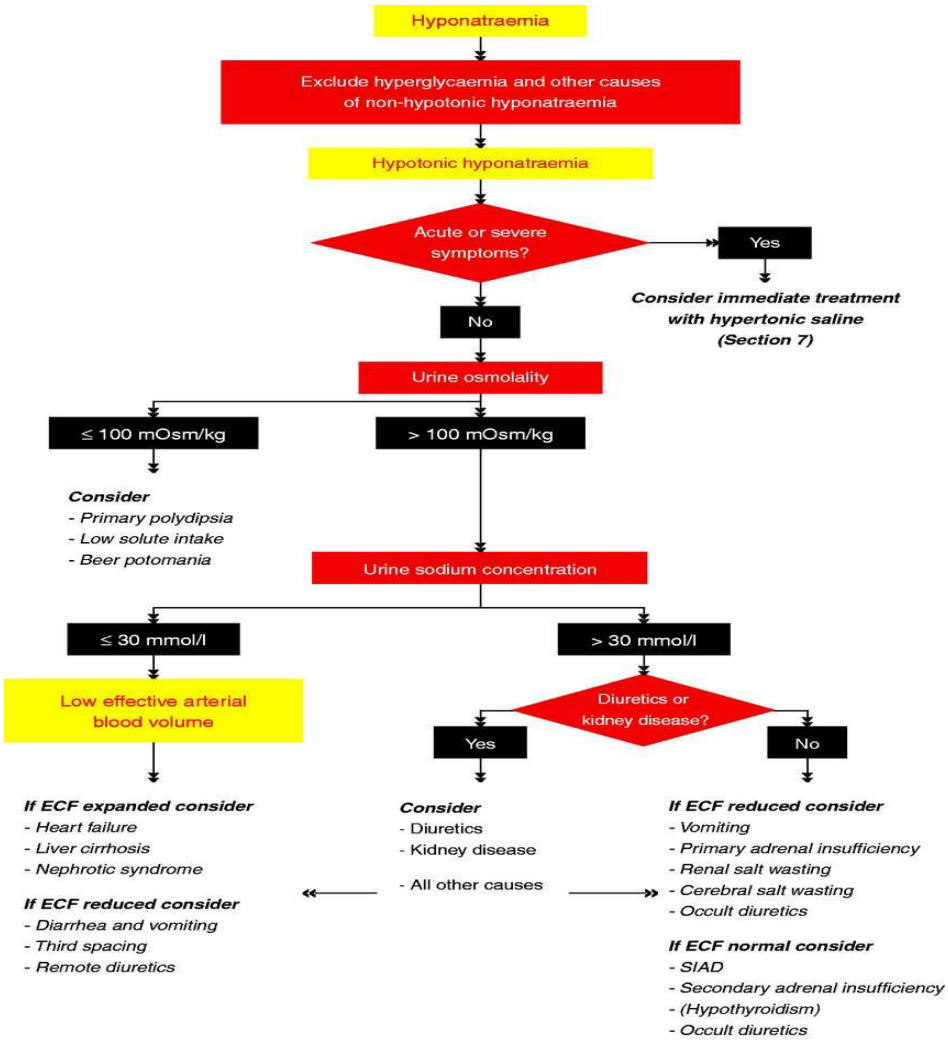
3. An 80 year old man, who had been on antihypertensive for last 20 years was admitted in our health set-up . He had developed bilateral pedal edema, which was overcorrected with diuretics elsewhere. Suddenly on day of admission, he became drowsy, delirious and developed hemiparesis and was promptly referred. Upon investigation, he was found to be hyponatremic, with serum Na^+ 112 meq/L while serum K^+ was normal. He was given 3% NaCl intravenous over 4 hours, followed by 0.9% NaCl. By the end of day, his serum Na^+ rose to

120 meq/L, & his condition improved. Next day he was administered with 0.9% NaCl. At the end of day two, his serum Na^+ became normal. He was successfully managed without any neurological complication.

Pathophysiology of hyponatremia [10]

Normal serum sodium concentration is in the range of 135-145 meq/L. Any value below135 meq/L is labelled as hyponatremia.

Table 1- Algorithm for the diagnosis of hyponatremia[10]



Classification of hyponatremia :

1. According to cause –
   a) Hypovolemic hyponatremia – Total body water (TBW) as well as total body Na^+ both decrease,
   Example – Gastrointestinal losses, renal sodium loss.
   b) Euvolemic hyponatremia – TBW increases while total body sodium is normal.
   Example – Hypothyroidism, SIADH
   c) Hypervolemic hyponatremia – Both TBW & total body sodium increases.
   Example – heart failure, liver failure.
   d) Redistributive hyponatremia – like in hyperglycemia & after administration of mannitol, there is shifting of water from intracellular to extracellular compartment. TBW & serum Na remain unchanged.
   e) Pseudohyponatremia – Due to hyperlipidemia or excess protein in blood, serum sodium appears low.

2. According to biochemical severity –

a) Mild – 130 – 135 meq/L
b) Moderate – 125 – 129 meq/L
c) Severe – <125 meq/L

3. According to time of development of symptoms
   a) Acute - within 48 hrs.
   b) Chronic - documented to exist for at least 48 hrs.

4. According to symptoms -
   a) Moderately symptomatic: Nausea without vomiting, headache, confusion
   b) Severely symptomatic: vomiting, cardiorespiratory distress, seizures, Glassgow coma scale below 8, Abnormal and deep somnolence.

Diagnosis of hyponatremia[10,11] Table-1

If serum sodium is <135 meq/L, we look for serum osmolarity, (normal serum osmolarity is 275-290 mosm/l). Non-hypotonic causes of hyponatremia should be investigated. If serum osmolarity is less than 275 mosm/L, then the clinical fluid status of patient is observed. The two tests that have better diagnostic value than assessment of fluid status are urine osmolarity & urine sodium concentration [12].

- If urine osmolarity is <100 mOsm/kg -- indicates relative excess H_2O
- If urine osmolarity is >100 mOsm/kg -- Urine Na conc. is investigated
- If urine Na conc. is <30 mmol/L -- indicates contracted ECF.
- If urine Na conc. is >30 mmol/L -- assess ECF status & use of diuretics.

Other laboratory tests –
1. Serum urea concentration
2. Serum uric acid excretion
3. Fractional urea excretion
4. Plasma copeptin concentration

The fractional excretion of uric acid >12%, is the most diagnostic test to differentiate hyponatremia due to SIADH from non SIADH hyponatremia[12].

SIADH (Syndrome of inappropriate adrenal secretion) It is a diagnosis by exclusion, when urine osmolarity is >100 mOsm/kg, urine sodium conc. is >30 mmol/L with normal ECF status. Simultaneously, secondary adrenal insufficiency & hypothyroidism are ruled out [13,14].

Causes of SIADH include malignancy, pulmonary causes, drugs, Neurological disorders.

Table 2: Comparison between SIADH and cerebral salt wasting

	SIADH	**Cerebral salt wasting**
Serum urea concentration	Normal–low	Normal–high
Serum uric acid concentration	Low	Low
Urine volume	Normal–low	High
Urine sodium concentration	>30mmol/l	≫30mmol/l
Blood pressure	Normal	Normal–orthostatic hypotension
Central venous pressure	Normal	Low

Management of hyponatremia [10]

1. First hour management of severe symptoms regardless of whether hyponatremia is acute or chronic-
   1) IV infusion of 150 ml of 3% NS over 20 min.

   2. Serum Na^+ is investigated & above recommendation is repeated till there is a rise of 5 mmol/L of serum Na^+.
2. Follow- up by –
   1) Stopping 3% NS
   2) Maintaining with 0.9% NS
   3) Starting cause specific treatment.

4) Monitoring the serum sodium conc. – target being total of 10 mmol/L increase during 1st 24 hours & thereafter 8 mmol /L increase every 24 hours till the target level of 130 mmol/L is reached.

3. If serum Na conc. does not increase by 5 mmol/L after 1st hour treatment, continue 3% NS till there is an increase of 1 mmol / L / hr. of serum sodium.
4. If hypokalemia is accompanied with hyponatremia, correction of K^+ level will also increase serum Na conc.
5. For chronic hyponatremia – non-essential fluids & medications are stopped as they can contribute to hyponatremia. Fluid restriction is suggested to prevent further fluid overload in patients with expanded ECF.
6. In patients with SIADH – first line of treatment is restricting fluid intake and increasing solute intake with 0.25-0.5 g / kg / day of urea or a combination of low dose loop diuretic and oral NaCl.
7. For patients with reduced circulating volume – IV infusion with 0.9% NS is administered at the rate of 0.5 - 1 ml / kg / hour.

Effects of fast correction of serum sodium concentration[15,16]

Rapid correction of serum sodium can lead to osmotic demyelination syndrome. It is characterized by focal demyelination in the pons & extrapontine areas, associated with severe neurological sequaele. Patients with hypokalemia, female patient, alcoholics or with liver transplant are more prone to it. To reverse the symptoms of fast correction of serum sodium, an infusion of 10 ml/kg of electrolyte free water (glucose solution) is started over 1 hour under strict monitoring of urine output.

Conclusion

Hyponatremia is most common electrolyte imbalance in clinical practice. It is having large spectrum of clinical presentation. High degree of suspecian is most important to make a diagnosis. Slow correction is always advised to avoid neurological complications.

Funding: Nil, **Conflict of interes**t: None initiated.
Permission from IRB: Yes

References

1. Pfennig CL Md, Slovis CM Md. Sodium Disorders In The Emergency Department: A Review Of Hyponatremia And Hypernatremia. *Emerg Med Pract*. Oct 2012; 14(10):1-26.
2. Hawkins RC. Age and gender as risk factors for hyponatremia and hypernatremia. *Clin Chim Acta*. Nov 2003; 337(1-2):169-72.
3. Renneboog B, Musch W, Vandemergel X, Manto MU & Decaux G. Mild chronic hyponatremia is associated with falls, unsteadiness, and attention deficits. *American Journal of Medicine* 2006 119 71.e1–71.e8. Gankam Kengne F, Andres C, Sattar L, Melot C & Decaux G. Mild hyponatremia and risk of fracture in the ambulatory elderly.*QJM: Monthly Journal of the Association of Physicians* 2008 ;101 :583–588.
4. Hoorn EJ, Rivadeneira F, vanMeurs JB, Ziere G, Stricker BH, Hofman A, Pols HA, Zietse R, Uitterlinden AG & Zillikens MC. Mild hyponatremia as a risk factor for fractures: the Rotterdam Study. *Journal of Bone and Mineral Research* 2011 ;26 :1822–1828.
5. Verbalis JG, Barsony J, Sugimura Y, Tian Y, Adams DJ, Carter EA & Resnick HE. Hyponatremia-induced osteoporosis. *Journal of Bone and Mineral Research* 2010 ;25 :554–563.
6. Liamis G, Rodenburg EM, Hofman A, Zietse R, Stricker BH & Hoorn EJ. Electrolyte disorders in community subjects: prevalence and risk factors. *American Journal of Medicine* 2013; 126: 256–263.
7. Wald R, Jaber BL, Price LL, Upadhyay A & Madias NE. Impact of hospital-associated hyponatremia on selected outcomes. *Archives of Internal Medicine* 2010; 170 :294–302.
8. Hoorn EJ & Zietse R. Hyponatremia and mortality: moving beyond associations. *American Journal of Kidney Diseases* 2013; 62 :139–149.
10. Spasovski G ,Vanholder R, Allolio B etal. Clinical practice guideline on diagnosis and treatment of hyponatraemia Eur J Endocrinol. 2014 Feb 25;170(3):G1-47.
11. Fauci AS, Kasper DL, Longo DL et al. in *Harrison's Principle of Internal Medicine* 2008; 17th edition vol. I : 274-287.
12. Fenske W, Maier SKG, Blechschmidt A, Allolio B & Stork S. Utility and limitations of the traditional diagnostic approach to hyponatremia: diagnostic study. *American Journal of Medicine* 2010; 123: 652–657.
13. Schwartz WB, Bennett W,Curelop S & Bartter FC. A syndrome of renal sodium loss and hyponatremia probably resulting from inappropriate secretion of

antidiuretic hormone. *American Journal of Medicine* 1957; 23**:** 529–542.

14. Ellison DH &Berl T. Clinical practice. The syndrome of inappropriate antidiuresis. *New England Journal of Medicine* 2007; 356: 2064–2072.

15. Verbalis JG, Goldsmith SR, Greenberg A, Schrier RW, Sterns RH. Hyponatremia treatment guidelines 2007: expert panel recommendations. *Am J Med*. Nov 2007; 120(11 Suppl 1):S1-21.

16. Murase T, Sugimura Y, Takefuji S, et al. Mechanisms and therapy of osmotic demyelination. *Am J Med*. Jul 2006; 119(7 Suppl 1):S69-73.

.....

How to cite this article?

Murthy M, Chatterjee S, Nigam R, Kosam D. Hyponatremia: Case series with review of literature. *Int J Med Res Rev* 2015;3(3):356-360. doi: 10.17511/ijmrr.2015.i3.064.

……..