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Case Report

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HYPONATREMIA: A CORRECTION DILEMMA

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ABSTRACT

Hyponatremia is a major problem associated with heart failure, with serum sodium concentration lower than 136 mmol/L. Hyponatremia has been reported as a risk factor for increased morbidity and mortality in patients with chronic heart failure. This case study, A 72-year-old female was admitted with the complaints of severe breathing difficulty and tiredness of 1-week duration. She had a past medical history of Diabetes mellitus and Hypertension. In last 2 years, she was suffering from DM with Nephropathy, Dilated Cardiomyopathy with Left Ventricular Dysfunction. The patient had a medication history of T.Amlodipine for hypertension and it may cause pedal edema and T.Losartan Potassium + Hydrochlorothiazide may cause hyponatremia. On admission, the patient has hypervolemic hyponatremia, after 2 days of treatment with Inj. Furosemide becomes euvolemic. Later the patient shows skin triggers which concluded hypovolemic hyponatremia and treated with inj. Furosemide and 3% hypertonic solution, it increases cardiac output through an increase in effective blood circulation volume, improve inappropriate elevation of AVP and enhance effects of diuretics. In conclusion, managing patient with heart failure associated with frequent hyponatremia is a difficult task which requires careful assessment and balancing of patient's volemic condition and serum sodium level by properly adjusting the dose of dietary sodium, diuretic therapy along with fluid intake.

KEYWORDS: Hyponatremia, Heart failure, Serum sodium.

INTRODUCTION

Hyponatremia is defined as a serum sodium concentration lower than 136 mmol/L.[1] The most common classification of hyponatremia is based on volume status: hypovolemic (decreased total body water with a greater decrease in sodium level), euvolemic (normal total body water with a decrease in sodium level) and hypervolemic (increased total body water compared with sodium).[2] In 2010, Heart Failure Society of America guideline recommends a sodium intake of <2 g/day in patients with heart failure.[3]

Hyponatremia has been reported as a risk factor for increased morbidity and mortality in patients with chronic heart failure. Therefore, aggressive correction of hyponatremia may have favorable effects on heart failure complicated by hyponatremia. It may increase cardiac output through an increase in effective blood circulation volume, improve inappropriate elevation of AVP and enhance effects of diuretics.[3] Clinical cardiac toxicity associated with hyponatremia has not been previously described, although in patients with congestive heart failure the frequency of ventricular premature beats has been shown to correlate with the severity of hyponatremia.[4]

CASE REPORT

A 72-year-old female was admitted with the complaints of severe breathing difficulty and tiredness of 1-week duration. She had a past medical history of Diabetes mellitus (DM) and Hypertension (HTN) 8years. In last 2 years, she was suffering from DM with Nephropathy, Dilated Cardiomyopathy with Left Ventricular Dysfunction. For that she was taking the following medication T.Gliclazide + Metformin (80mg+500mg, twice daily), T.Amlodipine (5mg, twice daily) and T.Losartan Potassium + Hydrochlorothiazide (12.5mg+50mg, once daily). On physical examination, her blood pressure was found to be 110/70mmHg and pulse 92beats/minutes. Also noted bilateral basal crepitations on auscultation and pedal edema.

The first day of admission, the report showed Serum Sodium 128mmol/L, Serum Pottasium 5.3mmol/L, Blood urea 42mg/dl, Serum Creatinine 1.6mg/dl and Random Blood Glucose 246mg/dl. The RBC Count was 2.98 million/cumm, Hb 8.6gm/dl, HCT 25.5%, RDW 14.1%, Polymorphs 82% and Lymphocytes 11%. The Echocardiogram report revealed dilated Left Ventricle (LV), Global LV hypokinesia, Moderate to severe LV dysfunction, moderate PHTR, Ejection fraction 30%, severe mitral regurgitation.

Based on investigation the patient had hypervolemic hyponatremia. she was treated with Inj. Furosemide 40 mg twice daily, T.Clopidogrel 75mg once daily, T.Isosorbide Dinitrate 5mg thrice daily, T.Carvedilol 3.125mg twice daily, T.Pantoprazole 40mg once daily, Inj.Human Insulin 30/70 (10^u-0-6^u) for 3 days. Patient has clinically improved and was discharged with T.Clopidogrel (75mg, once daily), T.Isosorbide Dinitrate (5mg, thrice daily), T.Carvedilol (3.125mg, twice daily), T.Pantoprazole (40mg, once daily), Inj. Human Insulin 30/70 (10^u-0-6^u) and T.Tolvaptan (15mg, once daily).

After 2 days, the patient came back to the hospital with the complaints of dyspnea, orthopnea. Arterial blood gas report shows that PO_2 42.5mmHg, PCO_2 30.66mmHg and lab investigation showed that Random Blood Glucose was 351mg/dl. The Serum Pottasium 4.7mmol/L, Serum Sodium 126mmol/L which indicates the patient persists euvolemic hyponatremia. Inj. Furosemide 20 mg IV Stat and Inj. Morphine 3mg IV Stat given for acute pulmonary edema. Later, her BP was lowered to 90/60 mmHg and started on Inj. Dopamine (2.5mcg/kg/min).

During her stay in the hospital, she had signs of dehydration and serum sodium level was 122mmol/L, it concludes hypovolemic hyponatremia. She was treated with IV 3% hypertonic saline as well as normal saline to improve the hydration along with other medications such as T.Clopidogrel (75mg, once daily), T.Isosorbide Dinitrate (5mg, thrice daily). And T.Valsartan (40mg, once daily), T.Ivabradine (5mg, twice daily) was given for 1st three days of treatment, later it has been stopped. Then T.Carvediol (3.125mg, twice daily) reduced to its half dose.

Eventually, serum sodium was increased to 131mmol/L, Random Blood Glucose 206 mg/dl. By correcting the hyponatremia patient improved symptomatically and later discharged in a stable condition.

DISCUSSION

Heart failure is one of the most common chronic medical conditions in the developed world. It is characterized by neurohormonal activation of multiple systems that can lead to clinical deterioration and significant morbidity and mortality. Hyponatremia is due to inappropriate and continued vasopressin activity despite hypo osmolality and volume overload.[5] In the specific hyponatremic patient, ascribing conduction defect to hyponatremia is usually complicated by other coexisting conditions which may have a direct deleterious effect on the conduction system; primary cardiac disease, diuretic, and antiarrhythmic drugs, or other electrolyte abnormalities.[4] CHF causes a decrease in cardiac output and circulating blood volume, which in turn triggers a compensatory response aimed at preserving blood pressure. This stimulates the body to retain both water and sodium. In addition, in CHF sympathetic stimulation is increased, causing renal vasoconstriction.[6]

The patient had a medication history of T.Amlodipine 5mg for hypertension and it may cause pedal edema and T.Losartan Potassium + Hydrochlorothiazide (12.5mg+50mg) may cause hyponatremia. CCB-related edema is caused by preferential arteriolar or precapillary dilation without commensurate dilation in the venous or postcapillary circulation.In addition, the reflex rise in precapillary resistance that ordinarily occurs with upright posture is effectively blocked by CCBs.[7] Hyponatremia with losartan is a chance occurrence; it may be explained by the hypothesis that AT1 receptor inhibition causes an angiotensin II-mediated decrease in renal tubular sodium reabsorption and reduced aldosterone release, resulting in hyperkalemia and hyponatremia. Additionally, in the elderly patient with the comorbid condition such as diabetes mellitus, losartan alone or in combination with a thiazide diuretic may cause such hyponatremia.[8] Thiazide diuretics such as hydrochlorothiazide reduce plasma volume by increasing sodium excretion.[9] Thiazide diuretics act by inhibiting reabsorption of Na+ and Cl- from the distal convoluted tubule by blocking the thiazide-sensitive Na+/Cl- cotransporter.[10]

On the day of admission, the patient had pedal edema, acute pulmonary edema and hypervolemic hyponatremia. So she was under the treatment of Inj. Furosemide 40mg IV twice daily to help reduce swelling and water retention.[11] On discharge Inj. Furosemide has changed to T. Tolvaptan (15mg 1-0-0) since hypervolemic hyponatremia persists. It's a novel, orally active, selective, nonpeptide antagonist that blocks arginine vasopressin from binding to V_2 receptors of the distal nephron, induces the excretion of electrolyte-free water without changing the total level of electrolyte excretion. In patients with heart failure, Tolvaptan appears to decrease body weight and edema and increase serum sodium concentrations without adversely affecting serum electrolyte levels.[12]

Since the excess fluid has removed but the patient also persists euvolemic hyponatremia. Later the patient Serum sodium level 122mg/dl also occurs skin triggers; it concludes that she was having hypovolemic hyponatremia. She was treated with Inj. 3% NaCl 50ml/hr and T.Furosemide 40mg once daily and it increases cardiac output through an increase in effective blood circulation volume, improve inappropriate elevation of AVP and enhance effects of diuretics.[1] Further the sodium level comes to 131mg/dl.

CONCLUSION

This case study suggests that managing patient with heart failure associated with frequent hyponatremia is a difficult task which requires careful assessment and balancing of patient's volemic condition and serum sodium level by properly adjusting the dose of dietary sodium, diuretic therapy along with fluid intake.

REFERENCE

1. Theodosios D Filippatos and Moses S Elisaf, Hyponatremia in patients with heart failure. World Journal of Cardiology, 2013; 26: 5(9): 317–328.
2. Michael M. Braun and Craig H. Barstow, Diagnosis and Management of Sodium Disorders: Hyponatremia and Hypernatremia. AAFP Journal, 2015:1; 91(5): 299-307.
3. Daisuke Morisawa (MD) and Shinichi Hirotani, Combination of hypertonic saline and low dose furosemide is an effective treatment for refractory congestive heart failure with hyponatremia. Journal of Cardiology Cases, 2014; 9(5): 179-182.
4. Pawan Suri and Kaiser Habeeb, Hyponatremia Presenting as Cardiac Conduction Defect. JK Science, 2009; 11(2): 85-86.
5. Adam Romanovsky and Sean Bagshaw, Hyponatremia and Congestive Heart Failure: A Marker of Increased Mortality and a Target for Therapy. International Journal of Nephrology, 2011; 23: 732746.
6. William T Abhramm, Managing Heart Failure in Hyponatremia, 1(5).
7. Domenic A. Sica MD, Calcium Channel Blocker-Related Peripheral Edema: Can It Be Resolved?. The journal of clinical hypertension, 2003; 5(4): 291-295.
8. Saibal Das and Sanjib, A case of losartan-induced severe hyponatremia. Journal of Pharmacology and Pharmacotherapeutics. 2015; 6(4): 219-221.
9. Da-Rae Kim, M.D and Joo-Hee Cho, M.D, Severe Hyponatremia Associated with the Use of Angiotensin II Receptor Blocker/thiazide Combinations. Pub Med Central Canada., 2013; 11(2): 56–59.
10. Kyu Sig Hwang, M.D. and Gheun-Ho Kim, M.D. Thiazide-Induced Hyponatremia. PMC Journal, 2010; 8(1): 51–57.
11. James G. O'Brien, M.D. and Shobha A. Treatment of Edema. AFP Journal, 2005; 71: 2111-2118.
12. Robert W. Schrier, M.D., Peter Gross, M.D. Tolvaptan, a Selective Oral Vasopressin V2-Receptor Antagonist, for Hyponatremia. The New England Journal of Medicine, 2006; 355: 2099-2112.