

The Ubiquitous use and Side Effects of Diuretics

Shobana S and Hanifah M

Department of General Medicine, Mahatma Gandhi Medical College and Research Institute, India

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Abstract

Diuretics are some of the most frequently used medication in medicine and are widely used for therapy of hypertension and volume overloaded states as in liver failure, Nephrotic syndrome, congestive cardiac failure, etc. The acute use of Diuretics has several side effects like hypovolemia, hyponatremia, hypokalemia, hyperuricemia and dangerous side effects like metabolic alkalosis. Diuretic use account for about 90% of clinical case of metabolic alkalosis. We report a case who presented with clinical features of disorientation, hypoxia, hypercarbia, severe electrolyte imbalance, which was diagnosed as metabolic alkalosis after laboratory and clinical feature secondary to acute diuretic use for idiopathic pulmonary hypertension diagnosed outside and discharged without monitoring electrolytes. Metabolic alkalosis has many harmful side effects like cardiac arrhythmias, hypoventilation, seizures, tetany, and hepatic encephalopathy. This case highlights the importance of electrolyte monitoring while prescribing diuretics.

Keywords: Diuretics, hypokalemia, metabolic alkalosis, arrhythmias

Introduction

Diuretics is potentially used agent for edematous disorders. Its notorious use can lead to adverse complications like electrolyte imbalance and volume depletion. Recently a lot of patients are diuretic dependent⁽¹⁾⁽²⁾

Case Report

A 58 year old lady came to our hospital with a chief complaint of weakness followed by altered sensorium. Patient previously had history of breathlessness for 20 years aggravated since 15 days associated with pedal edema and decreased urine output. Who was admitted treated in private hospital with a diagnosis of idiopathic pulmonary hypertension with IV diuretics and discharged 5 days prior. No past history of diabetes, hypertension, bronchial asthma, tuberculosis, epilepsy, jaundice. No other significant past history. On examination Pt was not oriented to time, place, person, afebrile, Pallor present, no icterus, no cyanosis, no clubbing, no lymphadenopathy, Bilateral pitting pedal edema, Jugular venous pressure was raised, bloodpressure-120/80mmHG, Pulse rate-104/min, Temperature-98F, SPO2-88% @ RA 93% with 4% lit o2. On systemic examination patient had bilateral basal crepts and scattered wheeze with other systems being normal. Our initial provisional diagnosis was metabolic encephalopathy and corpulmonale. On laboratory

investigations blood electrolyte were potassium-1.2mg/dl, sodium-137md/dl, and chloride-87 mg/dl. Mild renal impairment was present (creat-1.2mg/dl, urea-36mg/dl). Urine sodium-109mg/dl, urine potassium-50mg/dl, urine chloride- 121mg/dl, urine ca -3.2mg/dl, serum calcium 5.6mg/dl. Blood gas showed PH-7.66 bicarbonate -66, pco2- 57, po2-48. HRCT showed no evidence of mosaic attenuation, no fibrous bands, no evidence of embolism. Echocardiography showed severe pulmonary hypertension pasp-75mmHg RA /RV dilated Mild concentric left ventricular failure. Final diagnosis of diuretic induced metabolic alkalosis with idiopathic pulmonary hypertension was made. Treatment given were Diuretics was stopped, K replacement was given with isotonic saline, low dose Spironolactone was initiated and patient was advised Sodium restriction. Over a period of treatment hypokalemia and hypochloremia corrected, renal parameters came to normal, metabolic alkalosis improved gradually, patient sensorium level improved.

Discussion

Loss of gastric acid (vomiting, NG drainage) and diuretic use account for 90% of clinical cases of metabolic alkalosis⁽³⁾.

Few common causes of metabolic alkalosis are:

A: addition of base to extracellular fluid (no ecf volume depletion Milk-alkali syndrome, Excessive NaHCO₃ intake,

Recovery phase from organic acidosis (excess regeneration of HCO_3), Massive blood transfusion (due metabolism of citrate)

B: chloride depletion (ecf volume depletion) Loss of acidic gastric juice, Diuretics, Post-hypercapnia, Excess faecal loss like villous adenoma),

C: potassium depletion (normal or increased ecf) Primary hyperaldosteronism, Cushing's syndrome, Secondary hyperaldosteronism, Some drugs (e.g. carbenoxolone), Kaliuretic diuretics, Excessive licorice intake (glycyrrhizic acid), Bartter's syndrome, Severe potassium depletion

D: other disorders- laxative abuse, severe hypoalbuminemia.

Complications

Excessive use of diuretics can lead to the following complications.

- 1) Volume depletion
- 2) Electrolyte imbalance
- 3) Acid base disorders

Clinical features of metabolic alkalosis

1. CNS-neuromuscular excitability leading to paresthesia, light headache, carpopedal spasm
2. CVS-hypotension and cardiac arrhythmias
3. Respiratory Abnormalities- compensatory hypoventilation causing hypoxia
4. OTHERS-weakness, muscle cramps, postural dizziness due to hypovolemia, muscle weakness and polyuria due to hypokalemia.

Chloruretic agents such as chlorothiazide, furosemide, and their congeners all directly produce the loss of chloride, sodium, and fluid in the urine. These losses, in turn, promote metabolic alkalosis by several possible mechanisms. (1) Diuretic-induced increases in sodium delivery to the distal nephron accelerate potassium and proton secretion. (2) ECF volume contraction stimulates renin and aldosterone secretion, which blunts sodium loss but accelerates the secretion of potassium and protons.

(3) Potassium depletion will independently augment bicarbonate reabsorption in the proximal tubule and (4) stimulate ammonia production, which, in turn, will increase urinary net acid excretion. Urinary losses of chloride exceed those for sodium and are associated with alkalosis even when potassium depletion is prevented⁽⁴⁾. Diuretics blocks Na and Cl channels, more Na is delivered to DCT, Na exchange with K under the effect of aldosterone, kaliuresis and hypokalemia ensues, depleted ECF and high aldosterone leads to hypokalemia, Hypokalemia augments renal ammoniogenesis. When K is high in the lumen, fewer NH_4 is reabsorbed because of the competition between NH_4 and K.

With hypokalemia more NH_4 is reabsorbed and then secreted as NH_3 combining with H and raising the HCO_3 in plasma⁽⁵⁾.

Conclusion

The association of diuretics and metabolic alkalosis should never be underestimated. Metabolic alkalosis should be kept in mind while using diuretics and early aggressive management will be lifesaving.

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