FUROSEMIDE INDUCED SEVERE HYPOKALEMIA WITH ATRIAL FEBRILLATION (AF)- A CASE REPORT

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ABSTRACT

Introduction: Furosemide-induced hypokalemia, also termed “Pseudo-Bartter-Syndrome”, is found mainly in young females. Hypokalaemia is usually defined as a serum concentration of potassium <3.5 mmol/L. Normal range: [3.5- 5.1]. Case presentation: A 25- year-old female is a known case of Chronic rheumatic heart disease (CRHD) since 10 months came with the complaints of 2 episodes of loose motions, vomiting and palpitations since morning admitted in female medical ward of General Medicine department. Conclusion: This case shows that Furosemide-induced severe hypokalemia (Adverse drug reaction of Furosemide) may have leaded to cardiac arrhythmia, Atrial fibrillation.

KEYWORDS: Hypokalemia, Furosemide-induced, Chronic rheumatic heart disease (CRHD), Atrial fibrillation (AF).

INTRODUCTION

Hypokalaemia is usually defined as a serum concentration of potassium <3.5 mmol/L. Normal range: [3.5- 5.1] It can be classified as follows:[2]
Mild - 3.1 - 3.5 mmol/L
Moderate - 2.5 - 3.0 mmol/L
Severe - <2.5 mmol/L

It is probably the most common electrolyte abnormality affecting hospitalised patients. Most cases are mild with a serum potassium but in 5% of cases it is <3.0 mmol/L. Severe
hypokalaemia is even rarer. Importantly, even mild hypokalaemia can increase the incidence of cardiac arrhythmias.

**Diuretics**

Loop and thiazide diuretics are the most common cause of drug-induced hypokalaemia. Loop diuretics seem to produce less average fall of potassium concentration compared with thiazides. However, the potassium depletion is usually mild and severe hypokalaemia (less than 3.0mmol/l) is uncommon with diuretics. The prevalence of diuretic-induced hypokalaemia varies from a study to another and with the used dosage.

A recent study found that diuretic induced hypokalaemia was the most prevalent adverse drug reaction in an elderly hospitalised population both during hospitalisation and at the time of admission.

In a retrospective study, 33% of elderly patients taking furosemide alone were hypokalaemic as compared to 4% of those receiving furosemide in combination with potassium supplementation.

In a recent observational study, hypokalaemia occured in 8.5% of patients treated with low doses of thiazide diuretics but in only about 1% did the potassium level fall to less than 3 mmol/l. The risk of hypokalaemia with diuritic is dose dependent and is greater when dietary sodium intake is higher.

There was no significant association with age and no association with gender. Volume depletion caused by diuretics activates renin-angiotensin-aldosterone pathway. Therefore, increased production of aldosterone stimulates the reabsorption of sodium and water and promotes excretion of potassium via the secretory potassium channels in the collecting ducts leading to hypokalaemia.

**Mechanisms**

Drugs can decrease serum potassium concentration by two major mechanisms. Hypokalaemia may result from redistribution between extracellular and intracellular compartments, or an increased potassium loss through the gastrointestinal tract or kidney.
Complications

- Cardiac arrhythmias and sudden cardiac death.[8] (those with congestive cardiac failure, underlying ischaemic heart disease, and on digoxin or aggressive therapy for hyperglycaemia in diabetic ketoacidosis are most vulnerable).
- Muscle weakness, flaccid paralysis, rhabdomyolysis.

CASE PRESENTATION

A 25-year-old female is a known case of Chronic rheumatic heart disease (CRHD) since 10 months came with the complaints of 2 episodes of loose motions, vomiting and palpitations since morning admitted in female medical ward of General Medicine department.

Physical Examination

On examination, she was found to be conscious/coherent, afebrile, pulse rate 90 bpm, Heart $s_1 + s_2$ with irregular rhythm, lungs clear and abdominal examination revealed no abnormalities. Provisional diagnosis was done as Acute Gastroenteritis. Past medication history was found that patient using Tab.Furosemide 20mg since 15 days.

Laboratory Evaluation

Laboratory examination revealed a normal anion gap and normal chloride with severe hypokalemia. Complete blood picture, random blood sugar, blood urea nitrogen, and serum creatinine were within normal limits.

Treatment

Initially symptomatic treatment was initiated for GI complication and past medical history of CRHD ie., Intravenous administration of normal saline solution, Inj.Metronidazole 500mg/IV/TID, Tab.Sporolac/PO/TID Inj.Heparin 5000 U/IV/QID, Tab.Paracetomol 500mg/PO/TID, Inj.Furosemide 20mg/IV/BID, continued for three days and Tab.Digoxin 0.25mg/PO/OD for five days. Once the disease was confirmed that it is diuretic induced hypokalemia based upon serum electrolytes the treatment was targeted towards Hypokalemia, Syp.KCl 10ml/glass of water/Tid.

DISCUSSION

Furosemide-induced hypokalemia, also termed “Pseudo-Bartter-Syndrome”, is found mainly in young females.[10]
This patient with severe hypokalemia is interesting for the following reasons: It is extremely rare that serum potassium levels decrease below 2.5 mmol/l in case of Furosemide induced and that such low levels have been lead to cardiac arrests, as the patient had finally confirmed as Atrial fibrillation. Previous reports show that severe hypokalemia is associated with cardiac arrests and the necessity for cardiopulmonary resuscitation.\textsuperscript{[11]}

**Table no: 1 Serum Electrolyte findings**

<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>Sodium ($Na^+$)</td>
<td>135-145 mmol/L</td>
<td>134</td>
<td>137</td>
<td>139</td>
</tr>
<tr>
<td>Potassium ($K^+$)</td>
<td>3.5-5.1 mmol/L</td>
<td>2.7</td>
<td>2.2</td>
<td>3.3</td>
</tr>
<tr>
<td>Chloride ($Cl^-$)</td>
<td>95-110 mmol/L</td>
<td>108</td>
<td>109</td>
<td>111</td>
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</table>

In this case, the patient has been used Tab. Furosemide 20mg/PO/OD, which has been doubled after being admitted in hospital i.e., 20mg/po/BID. Which has worsen the condition have been concluded from the day-4 (second) serum $K^+$ levels has been drastically decreased after high dose administration of Furosemide. Then the corresponding diuritc (furosemide) has been stopped, as the condition has suspected has diuretic induced hypokalemia. After the drug has been subsided, serum $K^+$ levels were found to be normal in three days without any potassium supplements. Hence, this condition was finally diagnosed and confirmed as “Furosemide-induced Hypokalemia”.

**CONCLUSION**

This case shows that Furosemide-induced severe hypokalemia (Adverse drug reaction of Furosemide) may have leaded to cardiac arrhythmia, Atrial fibrillation in this case. Because hypokalemia always have the complications of cardiac arrest in a congestive heart disease (CHD) & CRHD patients and also in patients with Digoxin therapy.

Hence $K^+$ sparing diuretics like Spironolactone are more efficacious and more convenient in correcting hypokalemia than other potassium supplements, by which has been treated in this case or ACE inhibitors are the drug of choice when diuretics have been stopped.

**Authors’ contributions**

SA investigation of the patient, SA literature research, drafting of the manuscript, corresponding author. VA literature research, drafting of the manuscript. RB drafting of the manuscript. All authors read and approved the final manuscript.
REFERENCES