



“Modifiable Hyperkalemia with Timely Recognition”- Beta Blocker (Metoprolol succinate) induced

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Authors' contributions

This work was carried out in collaboration between both authors. Both authors read and approved the final manuscript.

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Case Study

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ABSTRACT

This case report describes the very rarest possibility of drug induced hyperkalemia with beta blockers. In literature search only 2 cases were reported as metoprolol succinate induced hyperkalemia in diabetes patients without renal insufficiency. As per FDA reports only 0.5 % patients have reported hyperkalemia induced by metoprolol [1]. Patient is diabetic and hypertensive. And she is on oral hypoglycemic agents, premixed insulin and on beta blockers along with calcium channel blocker. The potassium value was ranging from 6 to 6.7 mmol/l. Evaluated in detail and no other abnormalities noted in laboratory investigations. Once the offending drug metoprolol is withdrawn the potassium value reaches to normal range in very short period.

Keywords: Metoprolol succinate; hyperkalemia; betablocker induced.

1. INTRODUCTION

As per literature this is the first case report on Metoprolol induced hyperkalemia from India .The incidence of hyperkalemia is increasing in the day today clinical practice is most offently due to acute kidney injury or chronic kidney disease [2].

Other causes are increased potassium supplements [3], drug induced hyperkalemia (preferably drug to drug interactions and drug induced itself) [4]. In this case we are inhancing a very important rarest possibility of hyperkalemia which due to betablocker induced in a T2DM with no renal insufficiency.

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2. CASE REPORT

A 63 year old lady with known history of T2DM, Dyslipidemia and Systemic Hypertension came with asymptomatic hyperkalemia of 6.1 mmoles/L. She was known type 2 diabetes mellitus since 11 years with no features of renal insufficiency (serum creatinine : - 1.01 with normal creatinine clearance, ultrasound abdomen no abnormalities detected and albuminuria (+). All other electrolytes values were within normal limits serum magnesium, serum calcium. Diabetes she is on oral hypoglycemic agents and on insulins (Basal and bolus). Antihypertensives were metoprolol succinate and cilnidipine since 2 years. Since the patient is asymptomatic and no clinical features of antihyperkalemic and arrhythmic changes on electrocardiogram. Initiated on evaluation, along with antihyperkalemic medications.

Detailed history taken and nil suggestive of increased intake of potassium supplements. Blood pressure values are within normal range with these medications and no history suggestive of decreased urine output or features of renal failure. Past history of palpitation and evaluated one year before with few atrial ectopics on electrocardiogram and normal with echocardiography. Patient was evaluated on (OP) outpatient basis with routine serum potassium values, the potassium values decreased on antihyperkalemic measures and it started to persist to hyperkalemic range once the antihyperkalemic measures are withdrawn. The serum cortisol, ACTH, Aldosterone levels were within normal range. Finally the drug induced mechanism of betablockers suspected and discontinued metoprolol and increased the dose of cilnidipine for 1 week without antihyperkalemic measures. The repeat serum potassium level showed a decreasing pattern from the persistent levels of 6.5 to 4.8 and to 4 mmol/L in consecutive weeks.

3. DISCUSSION

Hyperkalemia induced by betablockers are very rare and it has several mechanisms [5]. The two main mechanisms are due to decrease in cellular uptake of potassium and suppression of aldosterone secretion from adrenal cortex [6,7]. Diabetes with renal failure cases has an increased risk for hyperkalemia. Hyperkalemia is also shown to be linked to propranolol-induced hyperkalemia, there is abrupt cell lysis with

propranolol induced type with the release of intracellular potassium [8,9]. Only 1-5 % of patients occurred hyperkalemia which is predominantly induced by adrenergic beta blocker and more are common with patients on non selective beta blockers such as carvedilol, propranolol and labetalol [10,11,12]. As per the study report of FDA 2018 evaluated the incidence of hyperkalemia in 24296 patients taking metoprolol succinate and found 287 patients with hyperkalemia [13]. Labetolol intravenous has reported hyperkalemia in CKD with hemodialysis and in preeclampsia patients [11]. Propranolol has antihyperkalemic properties and it has been also used in infants to treat hemangioma [14,15].

4. CONCLUSION

Patients with renal insufficiency and type 2 diabetes mellitus have more incidence of hyperkalemia. In this case patient is type 2 diabetes mellitus, hypertensive and with no renal insufficiency. The timely recognition of betablocker induced hyperkalemia can avoid unnecessary investigations and prevent severe complications like arrhythmias which may be fatal.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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