"MODIFIABLE HYPERKALEMIA WITH TIMELY RECOGNITION"-BETA BLOCKER INDUCED

ABSTRACT

This case report describes the rarest possibility of drug induced hyperkalemia with beta blockers (metoprolol). In medline citations only 2 cases were reported as metoprolol induced hyperkalemia in diabetes patients without renal insufficiency. In this case the patient is asymptomatic with T2DM, Sytemic Hypertension and with no known renal insufficiencies and the potassium value is ranging from 6 to 6.7 mmol/l. Evaluated and found no other abnormalities in laboratory investigations. Metoprolol succinate tablet is withdrawn the potassium value reaches to normal range in very short period

Keywords: metoprolol succinate, hyperkalemia, betablocker induced

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. The most important cause in india is diabetes with renal insufficiency. Other causes are due to increased potassium supplements , drug induced hyperkalemia(preferably drug to drug interactions and drug induced itself). 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.

CASE REPORT

A 63 year old lady with known history of T2DM, Dyslipidemia and Sytemic Hypertension came with asymptomatic hyperkalemia of 6.1 mmoles/ L . She was diabetic since 11 years with no features of renal insufficiency (serum creatinine : -1.01, ultrasound abdomen no abnormalities detected and albuminurea (+). All other electrolytes values were within normal limits. Diabetes she is on oral hypoglycemic agents and on insulins (Basal and bolus). Antihypertensives were

metoprolol succinate and cilnidipine. Since the patient is asymptomatic and no features of antihyperkalemic and arrythemic changes on electrocardiogram .Intiated on evaluation, along with antihyperkalemic medications.

Detailed history taken and nil suggestive of increased intake of potassium supplements .Blood presure values are on normal range with these medications and no history suggestive of decrease in urine output. Past history of palpitation and evaluated one year before with few atrial ectopics on electrocardiogram and normal echocardiography with good left ventricular function study. She was reviewed as outpatient basis frequently with antihyperkalemic measures and the elevated pottasium values started reducing pattern, since when the antihyperkalemic measures are withhelds the pattern of potassium raises again. 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 clinidipine for 1 week without antihyperkalemic measures. The repeat serum potassium level showed a decreasing pattern from the persistant levels of 6.5 to 4.8 and to 4 mmol/L in consecutive weeks.

DISCUSSION

Hyperkalemia induced by betablockers are very rare and it has several mechanisms (¹). The two main mechanisms are due to decrease in cellular uptake of potassium and suppression of aldosterone secretion from adrenal cortex. (^{2'3}) Diabetes and diabetes with renal failure cases has an increased rsk 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 (4,5). Only 1-5 % of patients occurred hyperkalemia which is predominantely induced by adrenergic beta blocker and more are common with patients on non selective beta blockers such as carvedilol, propranolol and labetalol (6,7,8). 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 (⁹).

CONCLUSION

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

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