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# Case Report Metoprolol-induced hyperkalemia – A case report

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# ABSTRACT

Hyperkalemia is a condition that increased serum potassium levels, which can lead to life-threatening cardiac conditions. A 59-year-old female patient admitted to an emergency medicine ward with complaints of gradually progressive retrosternal chest pain. She was a known case of diabetes mellitus (DM), hypertension, and ischemic heart disease with a positive family history of DM and hypertension. She has treated with tablet metoprolol 12.5 mg twice daily from 20 days. On examination, the patient was restless due to unstable angina BP in the range of 110/70–180/90 mm of Hg, PR range 84–86 bpm, and SPO2 – 98%. Laboratory investigation revealed that the HbA1c was 7.19, and mean blood glucose of the past 90 days was in average control. Ultrasonography shows the Grade I renal parenchymal disease. The serum blood sugar level was elevated. Serum troponin I was 0.91 ng/ml. Ultrasonography abdomen was normal. Electro cardiogram: Sinus tachycardia suspected left inferior hemiblock, poor R-wave progression, inverted T- wave, and slide ST segments elevation and 2D-echocardiogram: IHD and RWMA at rest (basal inferior moderate left ventricle dysfunction). On hospital admission, the patient was treated with antiplatelet agents, anticoagulant, insulin, anti-ischemic agents, hypolipidemic agents, and potassium binder resins and diuretics. Patients with diabetes and kidney dysfunction have a higher risk of hyperkalemia in concomitants therapy with beta-blockers, so the health care workers should be aware of life-threatening events due to hyperkalemia secondary to beta-blockers. This case-report adds the evidence on the electrolyte related adverse drug reactions due to the beta-blockers.

Keywords: Beta-blockers, Hypertension, Adverse drug reaction, Hyperkalemia, Diabetes mellitus

# INTRODUCTION

Adverse drug reactions (ADRs) are hurdles in the management of illnesses. They increased the unwanted effects of drugs, increase the cost of therapy, challenge the safety of the therapy, and increase the length of hospitalization.<sup>[1-3]</sup> ADRs can be prevented to a large extent by timely diagnosis.<sup>[4,5]</sup> The magnitude of the adverse effect may range from a minor effect to even a life-threatening condition.<sup>[5,6]</sup> The serious life-threatening condition is secondary to elevated serum potassium is linked with cardiac dysfunctions. Medication cases hyperkalemia as primary or contributors in 35-75% of hospitalized patients but secondary to beta-adrenergic receptors blockers are relatively uncommon events occurring in 1-5% of hospitalized patients.<sup>[7,8]</sup> Beta-blockers are one kind of drug which can use for symptomatic relief of angina and prevention of ischemic events. The patient was prescribed with beta-blocker and developed the ADR of electrolyte imbalance. This ADR is completely preventable. Hence, we tried to report the ADR of metoprolol-induced hyperkalemia.

# Methodology

Informed consent was taken from the patient before collecting the information. The data in the patient case sheet and laboratory investigation reports were assessed. The causality assessment was done by the WHO scale and Naranjo's scales. The case study was done ethically according to the declaration of Helsinki by maintaining the subject confidentiality.

## **CASE REPORT**

A 59-year-old female patient admitted to a female emergency medicine ward with complaints of chest pain since night, gradually progressive in nature at the retrosternal location, constrictor types associated with sweating, and history of one episode of vomiting. She was known case of diabetes mellitus (DM) for 10 years, with hypertension for 5 years, and ischemic heart disease for 3 years and having a positive family of hypertension and DM in mother and three sisters. She does not have any recent evidence of drugs and diets that elevate serum potassium level, but from 4 September 2019, she was treated with tablet metoprolol 12.5 mg twice daily.

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#### Examination and causality assessment

The patient was not stable due to unstable angina BP in the range of 110/70-180/90 mm of Hg, PR range 84–86 bpm, and SPO<sub>2</sub> – 98%. The following score was obtained after applying the above scales for observed suspected ADR [Table 1]. The scales were used on the patient, and the respective scores were obtained by assessing the ADR. Causality was established based on the scores obtained using various standard scales. Re-challenging was not done as it is not required as per WHO-UMC for probable drugs. On analysis, the reaction was Bizarre type (Type-B) with a moderate severity of level 7 (a). The reaction can probably be prevented.

#### Laboratory investigation

The patient HbA1c was 7.19, and the mean blood glucose of the past 90 days was 159.5 mg/dl (in average control). Serum blood sugar level was in the range of from 384 to 200mg/dl from the day of admission to the day of discharge. Serum troponin I was 0.91 ng/ml (reference – <0.10). The cardiologist and cardiothoracic surgeon referenced the case as unstable angina. Chronic kidney disease and acute cardiorenal syndrome were ruled out during differential diagnosis. The serum potassium was elevated after the drugs, as referenced in Table 2.

## **Radiological investigation**

Ultrasonography abdomen: The report showed the Grade I renal parenchymal disease. There were no changes observed in the renal echotexture.

Electrocardiogram: Sinus tachycardia suspected left inferior hemiblock, poor R-wave progression, inverted T-wave, and slide ST segments elevation.

2D-Echocardiogram: IHD and RWMA at rest (basal inferior moderate left ventricle dysfunction).

## Treatments

On hospital admission, the patient was treated with the following medications:

Tablet aspirin 150 mg orally once in a day continue, tablet clopidogrel 75 mg PO orally once in a day continue, tablet atorvastatin 40 mg once in a day at bedtime, tablet isosorbide dinitrate 5 mg sublingually whenever necessary, tablet telmisartan 40 mg+ amlodipine 5 mg once in a day in the day of admission only, injection tramadol 50 mg intramuscularly

| Table 1: Causality assessment scores or suspected ADR. |       |                |  |  |  |  |
|--|-------|----------------|--|--|--|--|
| Scales   | Score | Assessment     |  |  |  |  |
| WHO-UMC  | 6     | Probable       |  |  |  |  |
| Naranjo's  | 7     | Probable       |  |  |  |  |
| Karsch and Lasanga                                     | Moder | rate to severe |  |  |  |  |

whenever necessary, injection insulin human regular 30U-0-25U subcutaneous for 5 days, tablet metoprolol 25 mg orally for 2 days, injection calcium gluconate 10% 10 cc over 10 min three times in a day for 4 days, nebulization salbutamol 2 mg four-hourly per nasal for 5 days, tablet amlodipine 5 mg orally once in a day for the past 3 days and continue, powder calcium polystyrene sulfonate 15 g orally 3 times in a day for the past 3 days, injection insulin 60 international unit in 100 ml normal saline intravenous infusion for the past 3 days, and injection furosemide 40 mg intravenously past 2 days.

The patient was in low dose metoprolol 12.5 mg orally twice a day since 20 days, and after hospital admission of chief complaint of unstable angina, she was administered with one of the anti-ischemic drugs beta-adrenergic blocker metoprolol 25 mg twice in a day for 2 days, after administration of the double dose of metoprolol patient serum potassium elevated, as shown in Table 2. From the 3<sup>rd</sup> day of admission, the patient was prescribed with amlodipine 5 mg orally once in a day and continue. On the 3<sup>rd</sup> day, the patient was an intervention with potassium binding resin 15 g 3 times in a day for the 3 days, simultaneously, metoprolol was also stopped, which shows improvement in laboratory parameters, as shown in Table 2. Injection insulin also administered to improve glucose utilization and enhance intracellular entry of potassium by decreasing the serum osmolarity.

| Date of<br>hospital<br>admission | Metoprolol<br>dose | Serum<br>potassium<br>(mEq/L) | Serum<br>creatinine<br>(mg/dl) | Serum<br>urea<br>(mg/<br>dl) | Potassium<br>binder  |
|----------------------------------|--------------------|-------------------------------|--------------------------------|------------------------------|--|
| Day 1                            | 12.5 mg<br>25 mg   | 6.7<br>6.9                    | 2.1                            | 64                           | -  |
| Day 2                            | 25 mg              | 7.0                           | 2.2                            | 62                           | -  |
| Day 3                            | Stopped            | 4.5                           | 2.5                            | 65                           | Calcium<br>polystyrene<br>sulfonate<br>15 gm 3<br>times per<br>day |
| Day 4                            | -                  | 5.9                           | 1.8                            | 55                           | Calcium<br>polystyrene<br>sulfonate<br>15 gm 3<br>times per<br>day |
| Day 5                            | -                  | 5.3                           | 1.7                            | 50                           | Calcium<br>polystyrene<br>sulfonate<br>15 gm 3<br>times per<br>day |

Mg: Milligram, mEq: Milliequivalent, L: Liter, dl: Deciliter, gm: Gram

## Outcomes and discharge

After 5 days of hospital admission, the patient was discharged after improvement in clinical symptoms and serum potassium level, serum creatinine, and serum urea level with prescription of tablet amlodipine 5 mg once in a day and blood-thinning agents like tablet aspirin 150 mg once in a day, tablet clopidogrel 75 mg once in a day, antidyslipidemic agents like tablet atorvastatin 40 mg once in a day at bedtime, hypoglycemic agents like tablet voglibose 0.3 g orally once in a day, and injection insulin (Humalog Mix 50/50, 50% insulin lispro protamine + 50% insulin lispro injection) 35 units after breakfast in the morning and 35 unit after dinner at night.

## DISCUSSION

Beta-blockers induce hyperkalemia by various mechanisms such as suppression of aldosterone secretion from the adrenal cortex and a decrease in cellular uptake of potassium by betablocking.<sup>[9]</sup> Complete blocking of these receptors by betablocker leads to a decrease in voltage gate sodium-potassium adenosine triphosphate pump activity resulting in a decrease in cellular uptake of potassium. Patents comorbidities with renal failure, DM, or both have a higher incidence of hyperkalemia secondary to beta-blockers therapy than without risk factors.<sup>[10,11]</sup> The ADRs are the drug-related problems and can be prevented by designing the safest drug regimen.<sup>[12]</sup> The physician should update themselves with the current best evidence-based medicine and update themselves with the drug-related problems. The clinical pharmacists are in an excellent position to intervene in therapy and optimize the drug regimen most safely.<sup>[4]</sup>

#### **Pharmacist intervention**

Angiotensin receptor antagonists are to be used with caution in hyperkalemia and kidney dysfunctions.

Voglibose can be changed with other suitable anti-diabetic drugs to minimize the kidney-related ADRs.

## CONCLUSION

Our case portrays the probable hyperkalemia secondary to the metoprolol intervention. Even though patients with cardioselective beta-blockers are less risk of inducing hyperkalemia, but have higher incidences of hyperkalemia in patients with DM and renal dysfunction, and also among the patients receiving beta-blockers. Beta-blockers are one of highly utilized among the cardiovascular agents, and health worker should be made aware of life-threatening events of it and patients on beta-blockers should be closely monitored for serum potassium level, kidney function, and arrhythmia.

#### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

#### **Conflicts of interest**

There are no conflicts of interest.

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