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Case Study

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A CASE REPORT ON TELMISARTAN INDUCED HYPERKALEMIA

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ABSTRACT

Hyperkalemia is defined as a serum potassium concentration greater than approximately 5.0-5.5 mEq/L. Levels higher than 7mEq/L can lead to significant hemodynamic and neurologic consequences; levels exceeding 8.5mEq/L can cause respiratory paralysis or cardiac arrest. The main signs and symptoms are dyspnea, palpitations, chest pain, nausea or vomiting, paresthesias. The hyperkalemia can be diagnosed by monitoring the serum potassium, ECG, complete blood count, metabolic profile. Hyperkalemia can be managed by increasing the potassium excretion using a cation exchange resin or diuretics or by correct the source of excess potassium. Telmisartan is an angiotensin receptor blocker used to treat hypertension which acts by inhibiting vasoconstrictor and aldosterone -secreting effect of angiotensin II. The common adverse effects are hyponatremia, hyperkalemia, upper respiratory tract infection, back pain, cough, impotence, increased sweating, palpitations, tachycardia, rash, dermatitis. It is contraindicated to some other drugs like ramipril, aliskiren, elagolix.

KEYWORDS: Hyperkalemia, ARBs, Telmisartan, Angiotensin II.

INTRODUCTION

Telmisartan is in a class of medications called angiotensin II receptor antagonists. It works by blocking the action of certain natural substances that tighten the blood vessels, allowing the blood to flow more smoothly and the heart to pump more efficiently. Telmisartan is also used sometimes to treat heart failure (condition in which the heart is unable to pump enough blood to the rest of the body) and diabetic nephropathy (kidney disease in people with diabetes and high blood pressure).The common adverse effects are hyponatremia, hyperkalemia, upper respiratory tract infection, back pain, cough, impotence, increased sweating. Hyperkalemia is one of the common clinical problem that is most often due to acute or chronic kidney disease and/ or disorders or certaindrugs like beta blockers, ACE inhibitors, ARBs, potassium sparing diuretics and aldosterone antagonists. Redistributive hyperkalemia most commonly occurs in uncontrolled hyperglycemia (eg; diabetic ketoacidosis or hyperosmolar hyperglycemic state. The most serious manifestations of hyperkalemia are muscle weakness or paralysis, cardiac conduction abnormalities and cardiac arrhythmias. Hyperkalemia can be differentiated as

Mild- 5.5-6.0 mEq/L

Moderate-6.1-7 mEq/L

Severe ->7.0 mEq/L

CASE REPORT

A 73 year old male patient was presented to the ER with complaints of abdominal pain and fever with chills. MRI limited MRCP done on 29/5/2022 and reported as k/c/o acalculous cholecystitis with mild central IHBRD and dilated CBD. The patient had comorbidities like old CVA, Diabetes, dyslipidemia, hypertension, AKI on CKD and also patient had history of sepsis with MODS and surgical history of laparotomy and cholecystectomy. For the comorbidities he was on T. Clopidogrel 150 mg, T. Telmisartan 40 mg, T. Atorvastatin 10 mg, Inj. Human mixtard 20-0-25 units. After the radiological investigations open cholecystectomy planned. All the investigations except serum potassium was to be normal. The serum potassium was found to be as 5.9 mEq/L. Nephrology consultation were sought for surgery clearance as the patient had history of AKI on CKD. They advised to stop telmisartan 40 mg. After stopping telmisartan serum potassium were monitored and were found as nearby to normal level. For surgical prophylaxis patient was given with inj. cefoperazone sulbactam and anesthesia advices T. amlodipine, C. dutasteride and nebulization. At the time of discharge patient was found to be symptomatically better.

DISCUSSION

Hyperkalemia is an elevated level of potassium (K^+) in the blood.[1] Normal potassium levelsare between 3.5 and 5.0 mmol/L (3.5 and 5.0 mEq/L) with levels above 5.5 mmol/L defined as hyperkalemia. Typically

hyperkalemia does not cause symptoms.Common causes of hyperkalemia include kidney failure, hypoaldosteronism, and rhabdomyolysis. A number of medications can also cause high blood potassium including spironolactone, NSAIDs, and angiotensin converting enzyme inhibitors.The severity is divided into mild (5.5–5.9 mmol/L), moderate (6.0–6.4 mmol/L), and severe (>6.5 mmol/L).If the BUN and serum creatinine levels suggest kidney insufficiency, using the MDRD or CKD-EPI equation to determine the estimated glomerular filtration rate (eGFR) is recommended.[1] Chronic kidney disease alone generally will not cause hyperkalemia until the eGFR is less than 20-25 mL/min.

Depending on the clinical findings and the results of the above laboratory work, the following may be indicated:

- Glucose level - In patients with known or suspected diabetes mellitus
- Digoxin level - If the patient is on a digitalis medication
- Arterial or venous blood gas - If acidosis is suspected
- Urinalysis - If signs of kidney insufficiency without an already known cause are present (to look for evidence of glomerulonephritis)
- Serum cortisol and aldosterone levels - To check for mineralocorticoid deficiency when other causes are eliminated

ECG

- Serum uric acid and phosphorus tests - For tumor lysis syndrome
- Serum creatinine phosphokinase (CPK) and calcium measurements - Forrhabdomyolysis
- Urine myoglobin test - For crush injury or rhabdomyolysis; suspect if urinalysis reveals blood in the urine but no red blood cells are seen on urine microscopy
- Vital for assessing the physiologic significance of the hyperkalemia
- ECG findings generally correlate with the potassium level, but potentially life-threatening arrhythmias can occur without warning at almost any level of hyperkalemia
- In patients with organic heart disease and an abnormal baseline ECG,bradycardia may be the only new ECG abnormality
- ECG changes have a sequential progression, which roughly correlate with the potassium level, but with the caveats mentioned above.[2]

Early ECG changes of hyperkalemia, typically seen at a serum potassium level of 5.5-6.5mEq/L, include the following:

- Tall, peaked T waves with a narrow base, best seen in precordial leads[3]
- Shortened QT interval
- ST-segment depression

At a serum potassium level of 6.5-8.0 mEq/L, the ECG typically shows the following:

- Peaked T waves
- Prolonged PR interval
- Decreased or disappearing P wave
- Widening of the QRS
- Amplified R wave

At a serum potassium level higher than 8.0 mEq/L, the ECG shows the following:

- Absence of P waves
- Progressive QRS widening
- Intraventricular/fascicular/bundle branch blocks

The progressively widened QRS eventually merges with the T wave, forming a sine wave pattern. Ventricular fibrillation or asystole follows. The management for hyperkalemia are

➢ If the patient has only a moderate elevation in potassium level and no ECG abnormalities, treatment is as follows:

- Increase potassium excretion using a cation exchange resin or diuretics
- Correct the source of excess potassium (eg, increased intake or inhibitedexcretion)

➢ In patients with severe hyperkalemia, treatment is as follows:

- IV calcium to ameliorate cardiac toxicity, if present.
- Identify and remove sources of potassium intake.
- IV glucose and insulin infusion to enhance potassium uptake by cells.
- Correct severe metabolic acidosis with sodium bicarbonate.
- Consider beta-adrenergic agonist therapy (eg, nebulized albuterol, 10 mg, administered by a respiratory therapist); preferred over alkali therapy in patients with kidney failure.
- Increase potassium excretion by administering diuretics or gastrointestinal cation-exchange medications.
- Emergency dialysis for patients with potentially lethal hyperkalemia that is unresponsive to more conservative measures or with complete renal failure.

Medications for increasing potassium excretion include the following:

- IV saline and a loop diuretic (eg, furosemide), in patients with normal kidneyfunction
- An aldosterone analogue, such as 9-alpha fluorohydrocortisone acetate (Florinef), in patients with hyporeninemia or hypoaldosteronism or solid organ transplant patients with chronic hyperkalemia from calcineurin inhibitor use
- Potassium binders include cation exchange resins such as sodium polystyrene sulfonate (SPS; Kayexalate), patiromer, or sodium zirconium cyclosilicate (Lokelma); an SPS retention enema

may be used for hyperkalemic emergencies, oral products have slower onset of action, but may be considered for patients with advanced kidney failure who are not yet on dialysis or are transplant candidates.

Surgery is typically unnecessary, but the following scenarios may involve surgicalintervention:

- Patients with metabolic acidosis and consequent hyperkalemia due to ischemic gut: Exploration required
- Patients with hyperkalemia due to rhabdomyolysis: Surgical decompression of swollen ischemic muscle compartments may be needed
- Patients without end-stage renal disease who require hemodialysis for control of hyperkalemia: Placement of a hemodialysis catheter for emergent dialysis needed

CONCLUSION

Hyponatremia is one of the long term use by adverse event of angiotensin receptor blockers. Hence, physicians should evaluate the patient's condition inorder to prevent the progression of the adverse event. Although the patient gets recovered after stopping the offending agent and need follow up whether any recurrence.

CONFLICTS OF INTEREST

The authors have obtained the necessary patient consent forms where the patients have given their approval for participation in the investigation, followed by representation in the concerned article. The patients do understand that the authors will ensure that their identitieswon't be revealed.

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