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Tenofovir Induced Hypokalaemia - A case report

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Tenofovir Induced Hypokalaemia - A case report

Abstract

Tenofovir, an important medication for HIV/AIDS, carries the risk of hypokalemia in patients receiving treatment. Factors such as dietary deficits, vomiting, or diarrhea further increase this risk. Tenofovir acts as a mitochondrial toxin by inhibiting the DNA polymerase gamma enzyme, crucial for mitochondrial DNA replication. Consequently, ATP generation is depleted through the aerobic pathway, leading to dysfunction in the proximal tubule. This dysfunction manifests as renal acidosis type 2, which involves reduced bicarbonate excretion by the proximal tubule, resulting in renal bicarbonate wasting and decreased serum bicarbonate levels. Consequently, the lumen of the distal nephron becomes negatively charged, leading to compensatory potassium secretion and ultimately causing severe hypokalemia. Understanding the mechanisms underlying tenofovir-induced hypokalemia is crucial for healthcare providers to monitor and manage electrolyte imbalances in patients undergoing tenofovir treatment.

Keywords

hypokalemia, tenofovir, HIV, Adverse Effect

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CLINICAL HISTORY

A 53-year-old lady presented with weakness and fatigue on and off since a few days. She is a known case of retroviral disease since 3 years on anti-retro viral medication - tenofovir + lamivudine + efavirenz.

She was also recently diagnosed to be having carcinoma of breast on the right side, locally advanced disease (cT4b N2 Mx) and underwent modified radical mastectomy with lymph node clearance. Later patient started receiving chemotherapy.

On investigation, it was found that her serum potassium level was low.

Past history: Initially patient was on long term Zidovudine based regimen. But since the patient developed pancytopenia, the ART regimen was changed to Tenofovir based regimen. Low serum potassium levels were seen throughout the chemotherapy cycle with repeated correction.

K/c/o of diabetes and hypertension since 2005 on treatment

CLINICAL EXAMINATION

General Physical Examination

Patient is moderately built and nourished, is alert, cooperative, and well oriented.

No pallor, no icterus, no clubbing, no cyanosis, no lymphadenopathy, no oedema.

Pulse: 86 beats per minute, measured in the right radial artery, normal in rate, rhythm and volume.

Peripheral pulses felt bilaterally.

BP: 130/85 mm of hg measured in right brachial artery in supine position

CVS: S1 and S2 heard, no murmurs.

RS: B/L NVBS, no added sounds

P/A: Soft and non-tender with no organomegaly

CNS: Conscious and oriented

INVESTIGATIONS

CBC:

- Hemoglobin: 12.8 g/dL; Hematocrit: 39.4%; RBC count: 3.37 cells/mcL; TLC: 6050 per cubic millimeter; Platelet count: 1.26 lakh/mcL

LFT:

- Bilirubin total: 0.78 mg/dL; Bilirubin direct: 0.41 mg/dL; Total proteins: 6.2 g/dL; Albumin: 2.6 g/dL; A/G ratio: 0.7; AST: 25 IU/L; ALT: 16 IU/L; Alkaline phosphatase: 113 IU/L

ABG:

- pH: 7.351; pCO2: 35.5 mmHg; pO2: 98.9 mmHg

Urine routine:

- Urine albumin: negative; Urine sugar: positive 4+

Urine microscopy:

- RBCs: nil; Pus cells: plenty; Epithelial cells: 1-2; Casts: nil; Crystals: nil

Initial serum level of potassium: 2.9 mmol/L (Normal value = 3.5-5 mmol/L)

Level of serum potassium at the time of discharge: 3.6 mmol/L

DIAGNOSIS:

The patient was diagnosed with tenofovir-induced hypokalemia.

TREATMENT:

In response to the diagnosis, the tenofovir medication was immediately stopped, and the patient's antiretroviral therapy (ART) regimen was changed to a non-tenofovir-based combination consisting of Abacavir, Lamivudine, and Dolutegravir. Additionally, potassium supplementation was introduced to address the hypokalemia.

OUTCOME:

Following the treatment adjustments, the patient's potassium levels showed improvement and gradually returned to normal range during the follow-up period. The changes in medication and the addition of potassium supplementation proved effective in resolving the tenofovir-induced hypokalemia.

DISCUSSION

Tenofovir is a medicine used for the treatment and/or prevention of HIV/AIDS. Patients undergoing treatment with tenofovir are susceptible to developing hypokalemia. The risk of hypokalemia increases in the presence of dietary deficits or increased loss through vomiting or diarrhea. (1)

Tenofovir functions as a mitochondrial toxin and works by inhibiting the DNA polymerase gamma enzyme, which is essential for mitochondrial DNA replication. Consequently, this leads to ATP generation depletion through the aerobic pathway, resulting in mitochondrial dysfunction in the proximal tubule. The manifestation of this dysfunction is observed as renal acidosis type 2. (2)

Renal tubular acidosis type 2 is characterized by a decrease in bicarbonate excretion by the proximal tubule, leading to renal bicarbonate wasting and a decrease in serum bicarbonate levels. These events result in a negative lumen of the distal nephron, which is compensated by an increased secretion of potassium, ultimately leading to severe hypokalemia. (3)

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