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Is There a Link between 5α-Reductase Inhibitors and Hypoglycemia?

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5α-reductase inhibitors (5ARIs) have been an important treatment for benign prostate hypertrophy (BPH). Some have reported that 5ARIs might decrease the risk of type 2 diabetes mellitus (T2DM).[1] More recently, however, there has been growing evidence that these medications actually increase the risk of T2DM.[2] On the other hand, hypoglycemia associated with 5ARIs has not yet been reported. We recently experienced a case of hypoglycemia caused by finasteride treatment in a man without T2DM.

A 68-year-old man was admitted to the emergency department with recurrent hypoglycemia. Other than BPH, the patient had no other diseases. He had never been diagnosed with diabetes and had only taken 5 mg per day of finasteride beginning 3 months prior to admission. Before visiting the hospital, hypoglycemia accompanied by cold sweats and dizziness symptoms occurred and the patient measured his blood glucose by using his wife's self-monitoring blood glucose device even though he had regular meals. He reported that he had blood glucose below 55 mg/dL (range 40-55 mg/dL) over five times during 5 consecutive days.

The patient's vital signs were normal, but the level of blood glucose was 60 mg/dL, serum C-peptide was 5.85 ng/mL, insulin was 36.0 μU/mL, hemoglobin A1c was 5.5%, and the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) was 5.33. The other routine blood tests performed including thyroid function and urine analysis were all normal or negative. The anti-insulin antibody and anti-insulin receptor antibody tests were negative. A rapid adrenocorticotropic hormone test showed a normal adrenal gland response. A 72-hour fasting test was performed to establish the diagnosis of endogenous hyperinsulinemia. The results did not show a decreased glucose level below 55 mg/dL. Abdominal computed tomography (CT) scans showed no masses in the pancreas or other organs.

Taking finasteride was stopped immediately after hospitalization. Since then, no additional hypoglycemia has occurred. Laboratory data showed levels of C-peptide 0.54 ng/mL, insulin 1.98 μU/mL, and blood glucose 84 mg/dL on the day before his discharge. The patient has experienced no symptoms related to hypoglycemia in the past 5 months.

After excluding other causes, we concluded that the hypoglycemia had been induced by finasteride. The proposed mechanism was that 5ARIs could elevate blood glucose level via increasing insulin resistance. This class of medication prevents the conversion of testosterone to the more active 5α-dihydrotestosterone and reduces androgen-dependent prostate growth.[3] That induces features of androgen deficiency, which can lead to insulin resistance.[4] In individuals with insulin resistance, hypoglycemia as the first presentation of insulin resistance can be possible.[5] Indeed, we observed an initially elevated HOMA-IR level that indicates insulin resistance in our patient.

To the best of our knowledge, there are no reports of hypoglycemia following finasteride use. In addition to increasing the risk of incident T2DM, it should be also considered that hypoglycemia could occur in patients using 5ARIs. The further study focusing on the association between 5ARIs and insulin resistance and/or sensitivity to such will be needed.

CONFLICT OF INTEREST STATEMENT

None declared.

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