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Hypothyroidism Presenting as Reversible Renal Impairment

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ABSTRACT

Certain studies have shown pathophysiology of diminished renal function in hypothyroidism. Aetiology is due to direct effect of thyroid hormone on kidney, and also the hemodynamic effects play an important role.

Key words: Thyroid hormones, 99mTcDTPA, Cockcroft–Gault equation.

Introduction

Renal impairment is a common in daily clinical. Thyroid hormones regulate cellular functions. T3 and T4 influence serum creatinine levels. Hypothyroidism is common and can cause renal impairment, which is reversible. Hypothyroidism is undercooked cause of renal impairment. The classical clinical symptoms may be absent even in severe hypothyroidism. Patients with hypothyroidism should be clarified about elevated serum creatinine which might represent true renal impairment i.e., reduced GFR or simply increased generation and tubular secretion of creatinine, therefore further analysis by isotope GFR studies should be done. Here, we report a case of reversible renal impairment secondary to hypothyroidism.

Case Report

A 60-year-old man came with complaints of lethargy and muscle aches. No urinary complaints. He had a HR 56 beats/min, BP of 130/80 mmHg, pallor, dry skin, puffiness around eyes, on-pitting edema and slow-relaxing ankle reflexes. Thyroid gland not palpable. Investigations included haemoglobin 8 g/dL,microcytic hypochromic anaemia, serum urea 65 mg/dL, creatinine 2.7 mg/dL, estimated GFR (eGFR) 27mL/min/1.73 m2 by Cockcroft–Gault equation, sodium 137 mEq/L, potassium 4.1 mEq/L, chloride 101mEq/L, calcium 8.7mg/dL, phosphorous 3.1mg/dL, uric acid 6.2 mg/dL, protein 7.1gm/dL, albumin 4.2 gm/dL, bilirubin 0.7 mg/dL, aspartate aminotransferase 80 IU/L, alanine aminotransferase 30 IU/L, alkaline phosphatase 77 U/L, random blood sugar 90 mg/dL, cholesterol 422 mg/dL, triglycerides 757 mg/dL serum creatine phosphokinase 271 U/L.

Urine examination had 6–8 pus cells and culture was sterile. Urinary myoglobin was not detected and 24-hour urine protein levels were 80 mg. Ultrasound evaluation showed left kidney 9.7 3.7cm and right kidney 9 3.6 cm and normal patent renal arteries on both sides. 99mTcDTPA renal scan showed severely compromised cortical function with adequate clearance of left kidney and compromised cortical function with adequate clearance of right kidney. Patient was found to be hypothyroid and his thyroid profile was: thyroid-stimulating hormone (TSH) 400 mIU/L, free T3 1.8pg/mL, free T4 (FT4) 0.87, ant thyroid peroxidase antibodies 4550 IU/mL.

The patient was put on 100 microgram of levothyroxine daily and advised to follow-up. After 2 month follow up the serum creatinine had normalized. After six months of THRT, the patient became asymptomatic and had the following test results: haemoglobin 12 g/urea 24 mg/dL, creatinine 0.87mg/dL, eGFR of 87 mL/ min/1.73 m2 by Cockcroft–Gault equation, serum cholesterol 165 mg/dL, triglycerides 154 mg/dL and a normal thyroid function (TSH 3 mIU/L

and FT4 1.0 ng/dL. A repeat 99mTcDTPA renal scan revealed a mildly compromised cortical function with adequate clearance of left kidney and adequate cortical function with adequate clearance of right kidney, showing a remarkable recovery of renal function with THRT.

Discussion

In this case, the initial finding of renal impairment led to further investigations, which led to the diagnosis of hypothyroidism and THRT brought about recovery of renal function (Sanjay Vikrant *et al.,* 2013). The patient's presentation anemia, hypercholesterolemia and raised transaminases were perhaps clues to the underlying diagnosis to our patient. Primary hypothyroidism is associated with elevation of serum creatinine usually reversible. This increase is observed in more than half of adults with hypothyroidism. In kidney, it is involving renal growth and development, renal hemodynamic and sodium and water homeostasis.GFR is also influenced by thyroid dysfunction. Hypothyroidism associated kidney dysfunction seems to be related with decline in thyroid levels rather than thyroid autoimmunity (Montenegro *et al.,* 1996; Suher *et al.,* 2005).

The pathophysiology of impaired renal function in hypothyroidism is multifactorial. Among all mechanisms, direct effects of TH on cardiovascular system lead to lower cardiac output and renal blood flow which results in reduction of GFR, hyperlipidaemia and indirect effects through paracrine. Primary hypothyroidism is associated with reduction of GFR and RBF which becomes normal after levothyroxine administration. Similarly, the TH can be normalized with replacement therapy in hypothyroid patients with chronic kidney disease which improve GFR. Hypothyroid myopathy usually has myalgia, rhabdomyolysis which leads to acute kidney injury is rare complication of hypothyroidism. THTR for primary hypothyroidism leads to significant improvement of renal function in chronic kidney disease. It increases eGFR by about 35% in CKD patients.

Conclusion

Therefore, patients with renal impairment of unknown cause should undergo thyroid function tests as part of routine investigation. It is worth to see thyroid function in known CKD patients and appropriate THRT to correct reversible renal impairment due to hypothyroid.

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Study significance: It is worth to see thyroid function in known CKD patients and appropriate THRT to correct reversible renal impairment due to hypothyroid

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