Acute Kidney Injury secondary to severe hypothyroidism

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Abstract

We report a case of a middle-aged gentleman who was found to have deranged renal function. On further evaluation, he was diagnosed to have severe hypothyroidism for the first time. The Acute kidney injury was unexplained and recovered over the period of next few weeks, as hypothyroidism was treated with oral thyroxine supplements.

Introduction

Thyroid hormones have a variety of effects on the normal functioning of kidneys. Thyroid and kidney function derangements can have effect on each other. Acute renal dysfunction due to severe hypothyroidism is a rare entity with a few case reports in the literature.

Case report

A 39-year-old male patient, accountant by profession, presented to our clinic with symptoms of weakness, progressive lethargy, intolerance to cold and myalgia since last two months. He did not have any associated fever, arthralgia, abdominal pain or any urinary symptoms. His past medical history was unremarkable. Patient was not on any regular medications and had not taken any treatment for his new symptoms. There was no family history of renal or endocrine disorders.

On physical examination, his pulse rate was 76/min, Respiratory rate was 16/min and Blood pressure was 116/76 mm Hg. General Examination showed pallor. There was no edema or goiter. Systemic examination was essentially normal.

Laboratory exam showed Hemoglobin was 11.1 gm/dl, rest of the hemogram was normal. His renal parameters were deranged with Serum creatinine of 1.42 mg/dl (0.73-1.1 mg/dl); however, Urea level was normal, 5.7 mmol/L (3.2-7.4). Other related parameters were: Sodium: 138 mmol/L (135–145), Potassium: 3.8 mmol/L (3.5–5.1), Calcium 2.41mmol/L (2.1-2.6) and Uric acid of 480 umol/L (210–420). Liver function tests were normal except for elevated AST of 99 U/L (<35). 24-hour urine Creatinine clearance was 59 ml/min. Urinalysis showed no evidence of blood or protein and no casts on microscopy. The spot Urine albumin to creatinine ratio was normal (3.43mg/g) (Normal < 30). The creatinine kinase (CPK total) was elevated at 823 U/L (0–200). Renal ultrasound showed normal kidneys bilaterally and no urinary tract abnormalities or obstruction. Thyroid function tests revealed a markedly elevated TSH of 635 mU/L (0.4–4.4) with Free T3 of 1.15 pmol/L (3.1-6.8) and Free T4 of 1.03 pmol/L (12–22). TPO antibodies were positive: 186.3 IU/ml (0–34). Lipid profile was deranged with total Cholesterol of 338 mg/dl (< 200) LDL of 259 mg/dl (< 130) Triglycerides of 165 mg/dl. Ultrasound of the neck showed an enlarged thyroid gland with heterogeneous echotexture; TIRADS 2. A diagnosis of autoimmune thyroiditis with Acute kidney injury was made. The patient was commenced on thyroxine replacement (1.3 mcg/kg/day). Over the next 6 weeks there was significant improvement in his clinical status as well as his laboratory parameters.

Currently the patient is doing well on oral thyroxine supplements.

Table 1: Laboratory parameters over 6 weeks

	Week 0	Week 2	Week 4	Week 6
TSH(0.4–4.4mU/L)	635	150	--	12.25
Creatinine (0.73-1.1 mg/dl)	1.42	1.2	1.15	0.9
ALT (SGOT) (< 35U/L)	99	--	28	--
Hemoglobin (13-17 gm/dl)	11.1	--	--	12.7

Discussion

Thyroid hormones influence renal development, kidney structure, renal hemodynamics, GFR, the function of many transport systems along the nephron, and sodium and water homeostasis[1]. Effects of hypothyroidism and hyperthyroidism on kidney function are the result of direct renal effects, as well as systemic hemodynamic, metabolic, and cardiovascular effects. Fortunately, most of the renal manifestations of thyroid disorders, which are clinically most significant with hypothyroidism, are reversible with treatment[1].

A few case reports document increased levels of serum creatinine with hypothyroidism in humans. Elevation of levels of serum creatinine can occur within as little as 2 weeks of significant hypothyroidism. These levels typically normalize rapidly with thyroid hormone replacement after short periods of hypothyroidism, but slower and incomplete recovery has been noted with more prolonged periods of severe hypothyroidism[2-4]. 52Cr-EDTA clearance studies have confirmed that changes in levels of serum creatinine in patients with thyroid disorders do reflect actual changes in GFR[5]. These changes in GFR are likely due to a number of factors such as the effects of thyroid hormones on Cardiac output, systemic vascular resistance, renal blood flow, Nitric oxide synthetase activity as well as effect on RAAS pathway.

While the association between severe hypothyroidism and acute Kidney Injury is rare, clinicians need to be aware that hypothyroidism is not only a recognized cause of de novo renal failure but can also precipitate deterioration in patients with stable chronic renal failure. There are reports of improvement in renal failure following treatment of hypothyroidism in these patients[6].

Epidemiologic data also suggests that patients with advanced chronic renal dysfunction (Chronic Kidney Disease stages 4 and 5) may have an increased risk of hypothyroidism compared to the general population. This is often sub clinical and is therefore important to detect, especially in the elderly, as the condition is easily treatable[7-8].

Conclusion

We report a case of Acute kidney injury secondary to severe hypothyroidism, which reversed completely in 6 weeks with appropriate thyroxine supplementation. Knowledge of the association between thyroid dysfunction and renal impairment is important for the clinician; and while the association is relatively rare, hypothyroidism should be considered as a cause of renal impairment especially in the context of patients with no other clear explanation for their renal dysfunction.

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