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CASE REPORT

Hypothyroidism and associated acute kidney injury

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Abstract

Profound hypothyroidism can be a cause of unexplained acute renal failure. On correction of hypothyroidism renal dysfunction improves. There are reports of severe hypothyroidism causing rhabdomyolysis, elevated creatinine kinase and acute renal failure, but these are associated with other precipitating causes like exercise, trauma or statin use[1,2]. We report a case of gentleman who presented with profound hypothyroidism, elevated creatinine kinase and acute kidney injury whose renal function improved when his hypothyroidism was treated. There were no other causes of acute kidney injury.

Keywords: Hypothyroidism; Acute Kidney Injury; Renal dysfunction

Introduction

We report a case of hypothyroidism who presented with renal failure who improved after correction of his hypothyroidism. Association between renal failure and hypothyroidism is documented in previous case reports, but it's not found usually in textbooks. Electrolyte imbalance is seen in hypothyroidism, but associated renal impairment without underlying cause is rare[3].

Case report

A 37-year-old restaurant supervisor presented to OPD with generalized tiredness and myalgia for 2 weeks. He also noticed hoarseness of voice and puffiness of face. Since 2 weeks he has constitutional symptoms like low mood and lethargy with cramps in the legs. He had history of hypothyroidism for which he was on medications, but stopped since 1 month. History of hypertension for which he is on losartan 50 mg once daily. No history of NSAID intake. No history of dyspnea on exertion, cough and chest pain. He also denied any urinary symptoms like decreased urine output. He is nonsmoker. No surgeries in past and no history of allergies to any medications.

On examination his blood pressure is 130/78mmHg, pulse-64/min, normal respiratory rate, and he was afebrile. His saturation is 100% in room air. He is overweight with BMI of 29. He had puffiness of face and marked periorbital edema. Cardiovascular system examination was

normal with no added sounds or murmur and JVP was not raised. Respiratory examination was normal. Neurological and abdominal examination was unremarkable.

His CBC was normal, CK-MB 16.8 U/L(<25), Creatinine kinase is 1375 U/L(39-308), creatinine 1.37 mg/dL (0.7-1.2). Urea -30 mg/dL (11-38),TSH-175uIU (0.27-4.2), free t3 is 0.64pg/mL (2.2-4.4), Free T4 0.69ng/mL (0.93-1.7), Sodium-138 mmol/L, Potassium-4.48. His liver function tests were normal. TPO>100 his urine routine was normal.

He was started on oral thyroxine and intravenous fluids following which he became symptomatically better. His creatinine kinase, TSH and creatinine started decreasing, and he was discharged with levothyroxine 200 mcg once daily. On follow up after one month his TSH was 21 and creatinine 1.05 mg/dL. CPK was normal. His dose of levothyroxine dose was increased and was asked to come for follow up.

Discussion

Hypothyroidism is frequently associated with renal impairment but it is rarely determined to be underlying cause. Hence it's not routinely recommended while screening for patient with abnormal renal function [3]. Davis et al [4] demonstrated histological changes in kidney structure related to hypothyroidism when they exposed rats to ant thyroid drug aminotrizole (ATZ).

The study done by Kreisman & Hennessey [5] demonstrated in their study consistent reversible elevation in creatinine levels in patients with severe hypothyroidism. Hypothyroidism is not only cause of de novo renal failure, but also has been associated with worsening of renal dysfunction in patient with chronic kidney disease as mentioned by Nakahama H et al [6].

Our patient had elevated creatinine kinase, but there was no rhabdomyolysis and his creatinine kinase as well as renal dysfunction improved post hydration and correction of hypothyroidism. Hence, thyroid function test should be routinely incorporated while routinely screening patients with acute kidney injury and also to look for precipitating cause of chronic kidney disease.

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