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Rhabdomyolysis and Acute Renal Impairment in a Patient with Hypothyroidism: A Case Report

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Abstract

We report the case of a 38 year old with hypothyroidism who developed acute renal impairment with rhabdomyolysis after strenuous physical activity. The TFT report confirmed hypothyroidism. In this patient CK was elevated which favoured rhabdomyolysis and increased creatinine indicated Acute renal impairment. We started patient on aggressive hydration and thyroid medications. His hypertension was also controlled with Hydralazine and his condition improved. Renal dysfunction which is acute with rhabdomyolysis in hypothyroid patients is not common, and we hope that this article will add to the present literature and give researchers sufficient data to better explain the condition.

1. Introduction:

The link between hypothyroidism and the renal system is widely understood. It encompasses both histological and physiological alterations. Reduced glomerular filtration rate, decreased blood flow to kidneys and plasma flow, water and electrolyte imbalance, particularly low sodium, and changes in tubular absorption and secretion are the most significant physiological abnormalities. Hypothyroidism has a little influence on renal function in most individuals, and it is frequently missed in clinical practice. When thyroid replacement medication improves renal function, this link is frequently demonstrated. Hypothyroidism has a little influence on renal function in most individuals, and it is frequently missed in clinical practice. When thyroid replacement medication improves renal function, this link is frequently demonstrated.

Hypothyroidism has also been linked to muscle problems and other muscular symptoms, ranging from simple stiffness, fatigueness, weakness, or increased CPK to more significant clinical features like Hoffman's syndrome or rhabdomyolysis. Hypothyroidism can cause alterations in the histological section of muscle. Despite the fact that there is a link between muscle problems and hypothyroidism, rhabdomyolysis which is characterized by muscular necrosis and presence of myoglobin in urine is not common in hypothyroidism. The example of 38-yrs -old man with hypothyroidism who suffered renal impairment , acute in nature with rhabdomyolysis is described in this article. This case report combines a restricted group of case studies that have described hypothyroid acute renal impairment in the context of rhabdomyolysis.

2. Case Report

A 38-yrs-old man with acute renal impairment was moved from an outside facility to our hospital. The patient had Crohn's disease but no other relevant medical history, and he was not known to be on any medications. He has a 20-year smoking history and smokes 10 cigarettes each day. He has been a persistent drinker for the past 25 years, consuming 750 ml of Brandy every day. Patient was

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apparently normal 2 days prior to admission after which he presented with complaints of edema of both lower limb extremities and generalized weakness and muscle cramps. The patient had also complained of fatigueness, lack of sleep and constipation 2 months prior to admission. He also became intolerant to cold. Before admission, the patient noted significant increase in weight. Patient had no history of seizure episode, trauma, or fall recently but gives history of excessive gym work out. On presentation, the patient had body temperature of 36.4 C, PR of 68/minute, RR of 18, and BP of 150/90 mm Hg. He had BMI of 38.1 kg/m2. O/E ,patient was conscious, alert and oriented. An enlarged, rubbery thyroid was felt on palpation. Bilateral pitting pedal edema was noted. Lymphadenopathy was not noted. On auscultation ,chest was clear and no added sounds and S1/S2 heard with no murmurs. On palpation abdomen was soft and nontender. Motor or sensory impairment was not present. Blood tests showed a creatinine of 1.56 (normal: 0.7–1.3 mg/dL). His Glomerular filtration rate was 54 (normal: >60 mL/min/ 1.73 m2). Blood urea nitrogen was 16 (normal: 7–20 mg/dL). His CK was 7250(normal: 46–171 U/L). Na+ was 142 (normal: 135–145 mmol/L),K+ was 4.4 (normal: 3.5–5.1 mmol/L), and Cl- was 100 (normal: 96–106 mmol/L). Electrolytes in urine were measured. Fractional excretion of sodium was 0.88 suggestive of renal impairment due to prerenal cause. His TSH was 155.7 (normal: 0.4–4.2 lU/mL). FT4 was <0.2 (normal: 0.8–2.7 ng/dL). Thyroid peroxidase antibody was 160 (normal: <35 IU/mL).

LFT showed an AST of 200 (normal: <35 IU/L), ALT of 100 (normal: <45 IU/L). Fasting lipid profile showed cholesterol of 450 (normal: <200 mg/dL), HDL of 54(normal: 40-60 mg/dL), and TG of 320 (normal: <150 mg/dL). WBC count was 7200 (normal: 4000– 10000). ESR was 10 (normal: <20 mm/hr). C Reactive Protein was 0.1 (normal: <0.8 mg/dL). BNP was 36 (normal: <100 pg/mL). hb was 12.3 (normal:12–15 g/dL). Ferritin was 500 (normal: 20– 250ng/mL). Serum iron was 100 (normal:59–158 g/dL). TIBC was 333 (normal: 250– 425 g/dL). Urine analysis was not significant.

Thyronorm 125 mcg was prescribed, and vigorous hydration was recommended. The fluids were then gradually reduced. After a day of therapy, his creatinine level dropped to 1.35 mg/dL and his Creatinine kinase level dropped to 4500 U/L. Leg edema and muscular cramps began to fade after just one day of medication. The patient was given iv hydralazine 10 mg to reduce BP. To control his hypertension, he was prescribed hydralazine 25 mg 6^{th} hourly. Over the course of two days, his health steadily improved, hence patient was discharged .

Patient was kept on follow-up and on review after 6months, his creatinine decreased to 0.4 mg/dL and his Glomerular filtration rate was >60 (normal: >60 mL/min/1.73 m2). Thyroid stimulating hormone was 0.5 , his hb was 14 and AST level was 40.

3. Discussion

Thyroid and renal functions are intimately linked, and the interactions have been thoroughly documented . Renal physiology as well as kidney growth and development is very much dependent on thyroid hormones. Hypothyroidism can cause hyponatremia by affecting renal hemodynamics and causing water and salt retention. Renal impairment in hypothyroidism has a mysterious pathogenesis. Renal impairment is hypothesized to be caused by decreased cardiac output and increased renal and systemic vasoconstriction seen in hypothyroidism, which results in decreased blood flow to kidneys and plasma flow and a lower Glomerular filtration rate. Blood pressure changes seen during thyroid dysfunction are too minor to explain the changes observed seen in renal function, according to a research by Den Hollander et al.. Aside from hypothyroidism's effect on cardiac output, T3 has a direct effect on systemic vascular resistance, which can directly affect blood flow to kidneys, resulting in renal impairment .Furthermore, free T3 and T4 levels correlate with brain natriuretic peptide levels, which may influence cardiac output and Glomerular filtration rate. The impact of thyroid hormones on creatinine tubular secretion is one mechanism that might explain kidney involvement in hypothyroidism. In the sarcoplasmic reticulum of nephrons, T4 is known to modulate Na+/Ca2+ channels and Na+/K+-ATPase activity . Muscle involvement is another important route of renal dysfunction in hypothyroidism. Because creatinine generation in muscles is reliant on thyroid function, muscle problems and other muscular symptoms are

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prevalent in hypothyroidism. These muscle symptoms can vary from minor stiffness, weakness, or an increased CK level to more severe conditions like Hoffman's syndrome or rhabdomyolysis. Acute renal dysfunction caused by hypothyroid rhabdomyolysis is uncommon, with just a few case reports . This article contributes to that list by providing researchers with sufficient information to define the unusual incidence of acute renal dysfunction caused by rhabdomyolysis induced by hypothyroidism.

Seizures episodes, trauma, alcohol and drug intoxication, hard exercise, rhabdomyolysis due to drugs, and other factors are all linked to rhabdomyolysis. Rhabdomyolysis is a common occurrence; however it is generally asymptomatic, with just laboratory abnormalities. Rhabdomyolysis can cause electrolyte imbalances as well as acute renal failure in extreme situations. Hypovolemia, pyrexia, electrolyte imbalance, congenital muscular diseases, and hypothyroidism are just a few of the conditions that leads to more severe clinical symptoms in rhabdomyolysis.

As previously stated, hypothyroidism is an uncommon underlying cause of rhabdomyolysis that can both cause and worsen the condition. In our case, effort (excessive gym workout) produced rhabdomyolysis, which was aggravated by the patient's hypothyroidism, resulting in severe renal impairment. Severe rise of serum CK to 7250 U/L (reference: 46–171 U/L) revealed rhabdomyolysis in our instance. Although no clear cut-off point for rhabdomyolysis has been determined, a concentration of 5 times the upper limit of the normal value is usually recommended. However, serum CK levels will not correspond to the degree of renal failure . A creatinine level of 1.56 mg/dL (normal: 0.7–1.3 mg/dL) was discovered, pointing towards acute renal failure. Patients improved after initiating thyroid hormone replacement treatment in all the case studies that showed acute renal impairment owing to rhabdomyolysis caused by hypothyroidism . Cai and Tang described two individuals that needed blood purification as well. After initiating thyroid hormone replacement medication and intensive fluid replacement, patient's condition improved dramatically.

In our case patient reported bilateral edema in the lower legs, as well as some muscular pain. Thyroid hormones are thought to influence small channel permeability, causing albumin leakage from the vessels . Patients with hypothyroidism may have edema due to a decrease in renal plasma flow, Glomerular filtration rate, and free water clearance . Hyponatremia is found in hypothyroidism patients, but because our patient's blood sodium level was normal, hyponatremia was ruled out as the reason for rhabdomyolysis. Hypothyroidism can have a negative impact on the hematological system, resulting in Anemia. In individuals with overt and subclinical hypothyroidism, chronic illness anemia is the most common anemic type, according to the researchers. We should point out that the patient in this case had chronic anemia with increased Ferritin and normal TIBC, as well as an HGB of 12.3. When the patient was followed up four months after being discharged from the hospital, his HGB had reverted to normal.

TPO antibody levels were increased in our patient, indicating that his hypothyroidism was caused by persistent autoimmune thyroiditis. Chronic autoimmune thyroiditis is the most common cause of hypothyroidism, according to research.

4. Conclusions

We describe a case of acute renal dysfunction caused by rhabdomyolysis induced by hypothyroidism in this case report. Renal dysfunction which is acute with rhabdomyolysis in hypothyroid patients is not common, and we hope that this article will add to the present literature and give researchers sufficient data to better explain the condition.

We furthermore underline that individuals with acute kidney impairment which is unexplained and symptoms such as myalgia and widespread weakness, should have their thyroid status checked.

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