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Case Report

A case report: Hypothyreosis complicated by rhabdomyolysis and acute renal injury

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ABSTRACT

Hypothyroidism is a common endocrine disorder characterized by the impaired function of the thyroid gland and reduced production of thyroid hormones. The clinical manifestations of hypothyroidism can vary significantly, ranging from asymptomatic presentations to those that are life-threatening. We present a unique case of acute renal failure attributed to rhabdomyolysis in conjunction with a severe form of hypothyroidism. Remarkably, the patient's condition improved completely following the administration of levothyroxine, resulting in the induction of a euthyroid state. This case underscores the importance of evaluating thyroid function in patients presenting with acute kidney failure, as it may play a critical role in clinical decision-making and management.

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INTRODUCTION

Hypothyroidism is one of the most prevalent endocrine disorders, presenting clinical manifestations that range from asymptomatic cases to generalized symptoms such as fatigue, malaise, excessive sleepiness, brittle nails, hair loss, and menstrual irregularities in women.[1] Most cases are diagnosed through biochemical markers, specifically elevated TSH and reduced fT4 levels, with treatment typically involving levothyroxine.[1] Nonetheless, there are rare instances where longstanding undiagnosed hypothyroidism can lead to acute kidney injury, with the underlying mechanisms remaining unclear. It is hypothesized that the deficiency of thyroid hormones adversely affects mitochondrial oxidation and glycogenolysis, thereby contributing to muscular atrophy. Additionally, this deficiency can lead to the deposition of glycosaminoglycans and the enlargement of type I muscle fibers, resulting in myopathy and increased fatigue. The lack of T4 may alter muscular permeability, facilitating

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the release of creatine kinase, which can accumulate and manifest as rhabdomyolysis—a recognized contributor to acute kidney injury.[2,3] We present a case of severe hypothyroidism with accompanying acute renal impairment that ultimately resolved completely following the administration of levothyroxine and the achievement of a euthyroid state.

CASE PRESENTATION

We present the case of a 64-year-old male patient who presented to the emergency department with a chief complaint of severe weakness, malaise, decreased appetite, jaundice, and bilateral lower extremity edema, which had developed over the preceding two weeks. The patient's medical history is significant for an acute myocardial infarction, for which he received stent placement and a diagnosis of hypertension, both of which were addressed approximately one year prior to his current presentation, on statin, antithrombotic and anticoagulant treatment. He denies having pre-existing chronic renal illness.The clinical examination of the patient revealed significant findings, including gray discolored skin, lower limb edema, and a notable lack of energy. Laboratory analyses demonstrated elevated levels of degradation products, with urea measured at 35 mmol/L (2.8-7.2mmol/L), creatinine at 675 μmol/L (50.4-110.5umol/L), sodium at 136 mmol/L (136-145mmol/L), potassium at 5.0 mmol/L (3.5-5.1mmol/L), calcium 2.0mmol/L (2.2-2.6mmol/L), phosphorus 1.7mmol/L (0.7-1.5mmol/L) and hepatic markers including AST at 1671 U/L (5-37U/L), ALT at 968 U/L (6-55U/L), CK at 4158 U/L (10-170U/L), CK MB at 683 U/L (7-30U/L), LDH at 1477 U/L (81-234U/L), and thyroid hormones fT4 at 0.08 ng/Dl (0.8-1.7ng/dL), fT3 0.63pg/mL (2.3-4.2pg/mL), TSH >150 μIU/mL (0.3-4.3uIU/mL), Anti-TPO >1300U/mL (<60U/mL), PTH 187pg/mL (12.0-65.0pg/mL). (Table 1).

These results indicate a severe case of hypothyroidism accompanied by rhabdomyolysis and acute kidney injury. Imaging studies, including abdominal and urogenital echotomography, returned average results. However, echotomography of the thyroid gland exhibited a heterogeneous structure with reduced dimensions and the presence of numerous fibrin threads, consistent with autoimmune thyroiditis, thereby corroborating the diagnosis of hypothyroidism. A comprehensive echocardiogram was performed per protocol, aorta with normal dimensions, AoV tricuspid with atheromatous altered annulus and cusps, opening preserved, Vmax 1.0m/s, maxPG 4mmHg, MV with fibrous altered cusps, mild to moderate mitral insufficiency, pseudonormal transmitral profile, dd type 2, TV - minor tricuspid insufficiency. Left ventricle has regular dimensions, no convincing aberrations in

Table 1. Laboratory parameters before and after treatment

Laboratory examination	0 Day	After treatment with levothyroxine and hemodialysis	After 2 weeks	Normal range
Urea	35mmol/L	21mmol/L	7.0mmol/L	2.8-7.2mmol/L
Creatinine	675umol/L	267umol/L	98umol/L	50.4-110umol/L
Sodium	136mmol/L	134mmol/L	136mmol/L	136-145mmol/L
Potassium	5mmol/L	4.9mmol/L	4.5mmol/L	3.5-5.1mmol/L
Calcium	2.0mmol/L	2.2mmol/L	2.3mmol/L	2.2-2.6mmol/l
Phosphorus	1.7mmol/L	0.9mmol/L	1.2mmol/L	0.7-1.5mmol/L
AST	1671U/L	28U/L	14U/L	5-37U/L
ALT	968U/L	69U/L	20U/L	6-55U/L
CK	4158U/L	377U/L	30U/L	10-170U/L
CK-MB	683U/L	38U/L	10U/L	7-30U/L
LDH	1477U/L	385U/L	150U/L	81-234U/L
TSH	>150uIU/mL	50.2uIU/mL	5.9uIU/mL	0.3-4.3uIU/mL
fT4	0.08ng/dL	0.1ng/dL	0.7ng/dL	0.8-1.7ng/dL
fT3	0.63pg/mL	0.75pg/mL	2.4pg/mL	2.3-4.2pg/mL
ANTI-TPO	>1300	>1300	>1300	<60U/Ml
PTH	187pg/mL			12.0-65.0pg/mL

Abbreviations: AST, aspartate aminotransferase; ALT, alanin transaminase; CK, creatine kinase, CK-MB, creatine kinase-MB; LDH, lactate dehydrogenase, TSH, thyroid-stimulating hormone; fT4, free thyroxine 4; fT3, triiodothyronine; ANTI-TPO, anti-thyroid peroxidase antibodies, PTH, parathyroid hormone

kinetics at rest, and normal systolic function. IVS and IAS are defect-free. Pericardium - echo-free area behind RV 5mm. Systolic stratification behind the LV. Given the diagnostic findings, there was a significant concern regarding acute kidney injury attributable to rhabdomyolysis, exacerbated by severe hypothyroidism. Consequently, the treatment regimen commenced with levothyroxine at a dosage of 100 mcg daily, corticosteroids at 40 mg twice daily, and rehydration therapy utilizing crystalloid solutions, complemented by diuretic therapy (furosemide 60 mg/daily) to facilitate diuresis. Although there was a noted clinical improvement, laboratory markers indicated a deterioration of degradation products. A recommendation for hemodialysis, consisting of four sessions, was made, reducing degradation products, hepatic markers, and creatine kinase levels (Table 1). Following a two-week therapeutic period, the patient continued to be monitored by an endocrinologist and a nephrologist. The dosage of levothyroxine was adjusted to 125 mcg based on TSH readings (Table 1). At the same time, the nephrologist suggested discontinuing further therapy as laboratory indicators, including degradation products and hepatic transaminases, completely normalized.

DISCUSSION

This case exemplifies severe autoimmune thyroiditis leading to acute kidney injury due to rhabdomyolysis, which necessitated a hemodialysis intervention—remarkably, initiating thyroid hormone replacement therapy led to the complete restoration of renal function. Cases such as this are sporadic, yet they have been documented in individuals experiencing myxedematous coma, a critical condition that calls for immediate diagnosis and treatment. The ramifications of hypothyroidism extend to nearly all major organ systems, with a particular focus on the cardiovascular system, where it is most extensively studied. Hypothyroidism is associated with increased vascular resistance, diminished cardiac output, and alterations in various parameters of cardiovascular contractility.[4] Furthermore, severe hypothyroidism negatively impacts renal function, specifically affecting glomerular and tubular function.[5] This occurs due to compromised cardiac output, which leads to renal hypoperfusion. Also contributing to elevated blood pressure, hypothyroidism induces increased peripheral vascular resistance, resulting in glycosaminoglycan accumulation within arterial walls, myopathy, and increased serum creatinine levels. In this patient, creatinine kinase levels exceeded 4000 U/L, indicating rhabdomyolysis.[6] The administration of levothyroxine replacement therapy ultimately resulted in the complete recovery from rhabdomyolysis and the associated acute renal injury.[7]

CONCLUSIONS

This case emphasizes the critical importance of evaluating thyroid hormone levels in patients presenting with acute kidney injury, particularly when prior renal disease is absent. Timely diagnosis and treatment can lead to significant improvements in renal function.

Conflict of Interest

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