Acute renal failure in two cases with hypothyroidism related rhabdomyolysis

Davut Akin, Sehmus Ozmen

ABSTRACT

Introduction: We describe two cases of acute renal failure due to rhabdomyolysis associated with hypothyroidism. Hypothyroidism usually leads to muscular disorders, otherwise rhabdomyolysis is quite rare.

Case Series: This case report describes two cases of acute renal failure due to rhabdomyolysis associated with hypothyroidism in a 72-year-old male and a 36-year-old female in whom muscle enzyme levels were typical of rhabdomyolysis. Other reasons of rhabdomyolysis were excluded. Renal functions were recovered in both cases after treatment.

Conclusion: Hypothyroidism must be considered in patients presenting with acute renal failure and elevated muscle enzymes even in absence of additional precipitating factor.
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Keywords: Acute Renal Failure (ARF), Hypothyroidism, Rhabdomyolysis

INTRODUCTION

Rhabdomyolysis is an injury of skeletal muscle resulting in leakage of cell content into bloodstream. Although muscular disorders are usual in hypothyroidism, rhabdomyolysis due to hypothyroidism is very rare and only a few cases have been reported [1–3]. Non-exertional and non-traumatic causes of rhabdomyolysis include drugs, toxins, infections, electrolyte abnormalities, endocrinopathies, inflammatory and myopathies [4]. Most of the cases of rhabdomyolysis due to hypothyroidism and acute renal failure (ARF) had additional precipitating factor [5]. Only a few cases without precipitating factor have been reported [3, 6–9].

We present two cases with ARF due to hypothyroidism associated rhabdomyolysis with no additional precipitating factor.

CASE SERIES

Case 1: A 72-year-old male was admitted to hospital because of confusion, decreased urine output and dark brown urine color for 10 days. A physical examination revealed dry skin and generalized body swelling. His blood pressure was 90/60 mmHg and his pulse rate was 60/min, with an oral temperature 37°C. Abnormal laboratory results were as follows: serum urea 217 mg/dL (10–45 mg/dL), creatinine 11.3 mg/dL (0.6–1.3 mg/dL), K 6.5 mmol/L (3.5–5.1 mmol/L), peak creatine kinase 6024 U/L (29–200 U/L), lactate dehydrogenase (LDH) 449 U/l (125–243 U/l), aspartate aminotransferase (AST) 449 U/l (125–243 U/l), aspartate aminotransferase (ALT) 24 U/L (10–35 U/L). Urine analysis revealed dark brown urine with a positive dipstick reaction for blood and granular casts. A history of hashimoto thyroiditis for 40 years was obtained from his medical records. He was not taking thyroxine replacement regularly, but hypothyroidism was detected. Serum free T3, free T4 and
TSH levels were 0.02 ng/dL (1.8-4.6 ng/dL), 0.065 ng/dL (0.8-1.8 ng/dL) and 100 μIU/mL (0.27-4.20 μIU/mL). He has high serum concentrations of antibodies to anti-microsomal AB and antithyroglobulin 500 U/mL (0-60 U/mL) and 390 U/mL (0-20 U/mL), respectively. Other laboratory tests were normal. Intravenous fluids and L-Thyroxin replacement (100 μg/day) was started. Two sessions of hemodialysis was performed because of uremic acidosis and mental changes. Renal ultrasonography was normal. He experienced a polyuria period after thyroxin replacement. Serum creatine kinase level was normalized in a week of treatment. The final serum creatinine was 1.1 mg/dL.

**Case 2:** A 36-year-old female admitted with myalgia and decreased daily urine. Her blood pressure 10/70 mmHg and rhythm was normal. Abnormal laboratory results were as follows: serum urea 189 mg/dL, creatinine 10 mg/dL, serum sodium 126 mmol/L, serum potassium 5 mmol/L, AST 337 U/L, ALT 82 U/L, peak CK 4267 U/L, LDH 804 U/L. Urine analysis revealed granular casts no RBC and a positive dipstick reaction for blood. She had a history of subtotal thyroidectomy due to multinodular goiter one year ago. She was not taking thyroxin replacement regularly. Thyroid hormone profile revealed a TSH 100 μIU/mL (0.27-4.20 μIU/mL), FT3 0.1 pmol/l (3.1-7.7 pmol/L), and FT4 2.2 pmol/L (9-18 pmol/L). She had low central venous pressure. Fluids (%0.09 NaCl 120 mL/hr) and bicarbonate infusion (4.2 mmol/hr) was administered intravenously. No hemodialysis was performed. Thyroxin replacement (100 μg/day) was started. Her final serum creatinine was 1.1 mg/dL, Serum creatine kinase returned normal level.

Both of the patients did not have a past medical history of diabetes, hypertension, or renal disease. Non-exertional and non-traumatic causes of rhabdomyolysis include drugs, toxins, infections, electrolyte abnormalities. Antinuclear antibodies (ANA), anti-Ro, anti-La, anti-Sm, anti-Jo-1 or anti-ribonucleoprotein (RNP) antibodies were negative. Other laboratory tests (such as lipid profile and blood sugar) were normal. Renal ultrasonography revealed normal sized kidneys with normal echogenicity. No additional precipitating factor both of patients could be related with rhabdomyolysis was detected.

**DISCUSSION**

This case series describes two patients suffering from rhabdomyolysis due to hypothyroidism, with no additional precipitating factor. Rhabdomyolysis was defined as creatine kinase levels above five times the upper limit of normal and renal findings in both cases. Although the main features of rhabdomyolysis are muscular symptoms and increased creatine kinase concentrations, it can become a life-threatening disorder when complicated by ARF. As a cause of rhabdomyolysis, disorders such as collagen disease (e.g., polymyositis), ingestion of massive alcohol, other agents, infection, and trauma were excluded in our cases from medical history.

The exact cause of rhabdomyolysis (abnormal glycogenolysis, mitochondrial oxidative metabolism, and triglyceride turnover, impair muscle function) in hypothyroidism remains unclear [10].

There is a wide variation in the clinical presentation of rhabdomyolysis. The classical triad of symptoms is muscle pain, weakness, and reddish-brown urine. However, these classical features are seen in fewer than 10% of the patients [4]. One of our patients also had muscle pain, weakness, and reddish-brown urine, but the other patient did not report a muscle pain and weakness probably because of confused mental status.

Rhabdomyolysis and ARF due to hypothyroidism is a rare entity. There are a few reported cases in literature [3, 6]. In most of these cases, rhabdomyolysis appears to have been precipitated by additional factors such as vigorous exercise [2, 5], trauma, anti-hyperlipidemic agents, or metabolic disorder. Although hyponatremia is a well-known aetiology that may cause rhabdomyolysis, hyponatremia level in our second case is not as severe as previous papers reporting hyponatremia as cause of rhabdomyolysis [11]. Therefore, hyponatremia may not be the main factor of rhabdomyolysis in our case. To our knowledge only 11 cases with ARF secondary to hypothyroidism-related rhabdomyolysis who had no additional precipitating factor have been reported in literature to date [3, 6-9].

**CONCLUSION**

In conclusion, hypothyroidism must be considered in patients presenting with acute renal failure and elevated muscle enzymes even in absence of additional precipitating factor.

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**Author Contributions**

Davut Akin – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Sehmus Ozmen – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

**Guarantor**

The corresponding author is the guarantor of submission.

**Conflict of Interest**

Authors declare no conflict of interest.
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