

Acute Kidney Injury Secondary to Hypothyroidism-Induced Rhabdomyolysis

Hipotroidiye Bağlı Kas Yıkımı Sonrası Gelişen Akut Böbrek Hasarı

ABSTRACT

Acute renal impairment due to rhabdomyolysis in patients with hypothyroidism is quite rare. We describe a case of acute kidney injury due to hypothyroidism-induced rhabdomyolysis in a 47-year-old man who was under levothyroxine medication. Physical and laboratory findings were consistent with hypothyroidism, acute kidney injury, and rhabdomyolysis. Laboratory findings showed acute kidney injury, rhabdomyolysis and hypothyroidism. There was no obvious factor causing rhabdomyolysis except hypothyroidism.

KEY WORDS: Hypothyroidism, Rhabdomyolysis, Acute kidney injury

ÖZ

Hipotroidiye bağlı kas yıkımı ve buna bağlı gelişen akut böbrek hasarı nadirdir. 47 yaşında erkek hastada levotiroksin tedavisi altında iken hipotroidiye sekonder gelişen kas yıkımına bağlı ortaya çıkan akut böbrek hasarı olgumuzu sunuyoruz. Fizik muayene bulguları hipotroidi kliniği ile uyumlu idi. Laboratuvar bulguları akut böbrek hasarı, kas yıkımı ve hipotroidiyi doğruladı. Olgumuzda hipotroidi haricinde kas yıkımı yapacak başka bir sebep yoktu.

ANAHTAR SÖZCÜKLER: Hipotroidi, Kas yıkımı, Akut böbrek hasarı

CASE REPORT

A 47 year old man presented to our emergency service because of back pain and general muscle weakness. He had a five-month history of hypothyroidism and hypoparathyroidism due to total thyroidectomy. The patient was using levothyroxine, calcitriol, and calcium carbonate/vitamin D3 as medication. He denied any form of strenuous activity.

Physical examination revealed a body temperature of 36°C, pulse rate 60/min, and blood pressure of 110/60 mmHg. Neurological examination revealed decreased deep tendon reflexes. No evidence of dehydration was found.

Laboratory findings on admission (Table I) were as follows: Urinalysis showed blood reaction (+++) at the dipstick test without erythrocytes on microscopic examination,

compatible with myoglobinuria. Blood urea nitrogen (BUN) was 18 mg/dL, creatinine 2.1 mg/dL, creatine kinase (CK) 4807 IU/L (normal up to 195), SGOT 101 u/L, SGPT 61 u/l, LDH 732 U/L (150-500), Ca 7.3 mg/dl, P 3.4 mg/dl, FT3 1.49 µIU/ml (1.57-4.71), FT4 0.15 µIU/ml (0.89-1.76), TSH 81.46 µIU/mL (0.5-5.5), PTH 2.6 pg/mL, triglyceride 1244 mg/dL (40-180), total cholesterol 312 mg/dL, HDL cholesterol 48 mg/dL, and LDL cholesterol 125 mg/dL. Electromyogram showed a normal pattern. Renal ultrasonography was normal. There was no obvious factor causing of rhabdomyolysis except hypothyroidism.

Intravenous hydration with saline and alkalization of urine was begun soon after admission. The levothyroxine sodium dose was increased to 150 µg/day. Doses of calcitriol and calcium supplementation were also increased because of the hypocalcemia.

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Received : 01.01.2016

Accepted : 05.02.2016

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Table 1: Laboratory data of our patient.

	On admission	On day 4	After discharge	Normal range
BUN mg/dL	18	12	11	5-25
Creatinine mg/dL	2.1	1.6	1.4	0.6-1.2
CK IU/L	4807	----	424	24-195
LDH U/L	732		352	150-450
SGOT u/L	101	----	----	5-40
SGPT u/L	61	----	----	5-40
TSH μ IU/mL	81.46	----	20.4	0.5-5.5
PTH pg/mL	2.6	----	---	19.8-74.9
Triglyceride mg/dL	1244	---	541	40-180

BUN and creatinine were decreased to 12 and 1.3 respectively by the 4th day of treatment. As the hyperlipidemia was due to hypothyroidism, antihyperlipidemic treatment was not used. Interestingly, improvement of CK levels was rather slow. While the treatment was continuing, the patient was discharged at his own request. At the latest outpatient follow-up, BUN, Creatinine, TSH, CK and triglyceride levels were 11 mg/dL, 1.4 mg/dL, 20.4 μ IU/mL, 424 IU/L and 541 mg/dl, respectively.

DISCUSSION

Rhabdomyolysis is a potentially life-threatening syndrome characterized by muscle necrosis and release of intracellular contents to circulation (1). Hypothyroidism, though rare, should be considered a definite and authentic cause of rhabdomyolysis. The exact cause of rhabdomyolysis in hypothyroidism remains unclear. The common pathogenesis of all etiologies of rhabdomyolysis is related to the direct sarcolemmic injury or depletion of ATP within myocytes, leading to an increase in free calcium ions in the sarcoplasm (2-4). An association between severe hypothyroidism and acute kidney injury (AKI) is rare. Patients with severe and undiagnosed or undertreated hypothyroidism are more likely to develop complications such as rhabdomyolysis (5). On the other hand, hypothyroidism may aggravate renal ischemia due to low cardiac output, increased systemic and renal vascular resistance, and reduced glomerular filtration rate (6), and this condition makes the kidneys sensitive to nephrotoxic agents and alterations of renal haemodynamic events. Accordingly, a significant correlation between thyroid stimulating hormone and serum creatinine has been found; the greater the impairment in thyroid function, the more commonly renal failure occurred (7). Measurement of creatinine kinase levels is considered the gold standard for diagnosing rhabdomyolysis and CK values are generally considered predictive of the likelihood of developing AKI; a level of 5000 U/L or greater has been linked to development of renal failure

(8). In our case, the diagnoses of rhabdomyolysis and AKI were based on laboratory results of elevated serum CK and creatinine levels. The positivity of blood reaction in the urinary dipstick test without microscopic hematuria confirmed rhabdomyolysis. As the cause of rhabdomyolysis, disorder such as collagen disease (e.g. polymyositis), infection, strenuous exercise, trauma, and ingestion of massive alcohol were excluded. The CK levels of our patient was 4807 IU/L and decline of CK remained quite slow with appropriate medical therapy. As the hyperlipidemia was thought to be caused by hypothyroidism, antihyperlipidemic agents were not started. At the twenty-fifth day treatment, the CK, total cholesterol and triglyceride levels are still higher than normal; 3286 IU/L, 273 mg/dL and 521 mg/dL, respectively. Although the electromyographic findings in hypothyroidism are extremely variable, normal and myopathic patterns are usually observed (9). Electromyographic findings were normal in the present case.

In conclusion, the diagnosis of hypothyroidism should be considered in patients presenting with acute kidney injury and elevated muscle enzymes in the absence of other causes of rhabdomyolysis. Adequate therapy with thyroid hormone and intravenous fluid should be started in such patients. However, complete recovery may take a long time.

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