Acute Renal Failure in a Patient With Sheehan Syndrome and Rhabdomyolysis

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Keywords. hypothyroidism, acute kidney failure, rhabdomyolysis, Sheehan syndrome

We report a case of acute renal failure related to rhabdomyolysis in a patient with Sheehan syndrome, while other diseases that could cause rhabdomyolysis were excluded. The patient's kidney function completely recovered with 3 sessions of intermittent hemodialysis. After thyroxine replacement therapy, musculoskeletal symptoms disappeared and creatine kinase concentrations decreased. Steroid replacement therapy was also administered. The present case suggests that rhabdomyolysis could occur in a patient with Sheehan syndrome without other precipitating factors.

IJKD 2008;2:50-2
www.ijkd.org

Case Report

INTRODUCTION

Although muscle involvement is common in hypothyroidism,[1-3] as hypothyroid myopathy commonly manifests as fatigue, myalgia, stiffness, cramps, and delayed reflexes,[4-6] rhabdomyolysis is rare and only a few cases have been reported.[1,2-4-8] We report a case of acute renal failure in a 46-year-old woman with Sheehan syndrome due to rhabdomyolysis without any additional precipitating factors.

CASE REPORT

A 46-year-old woman was admitted to our hospital because of epigastric and right upper quadrant pain associated with nausea, vomiting, respiratory distress, loss of appetite, pain in the lower limbs, and oliguria. Anuria developed, subsequently. She had experienced severe progressive lower limb pain and weakness for 6 days prior to her admission. On past medical history, 10 years earlier in her last delivery of her 11th child, she had total hysterectomy due to uncontrolled postpartum hemorrhage. Afterwards, she has not have lactation.

On physical examination, her blood pressure was 100/60 mm Hg; pulse rate, 78/min; body temperature, 36.5ºC; and respiratory rate 32/min. She was pale and afebrile, but tachypenic with periorbital puffiness. No goiter was observed. She had no axillary or pubic hair, her skin was dry and coarse, and her breasts were atrophic. Heart examination revealed no abnormality, but she had rales in her lungs. Tenderness in the right upper quadrant and epigastric areas were present without rebound tenderness. Abdominal ultrasonography revealed normal liver, gall bladder, pancreas, and spleen. Her kidneys were normal in size, but exhibited increased echogenicity and corticomedulary differentiation. All her limb muscles were swollen with firm nonpitting edema, and severe stiffness and slight tenderness especially in the lower limbs were observed. No signs suggesting an associated systemic inflammatory disease were found. Her laboratory findings after admission are shown in the Table.

Diagnosis of rhabdomyolysis was established based on the severe myalgia; muscle weakness; marked elevation of serum levels of creatine kinase, lactate dehydrogenase, liver enzymes, and creatinine; 3+ blood on urine dipstick test; and the absence of red blood cells in urine microscopic examination. Myoglobinuria was confirmed by immunochromatography. Also, diagnosis of Sheehan syndrome was verified by the past medical history (severe vaginal bleeding during the last pregnancy resulting in total hysterectomy and no breastfeeding after delivery), symptoms and

Laboratory Findings on Admission in a Woman With Sheehan Syndrome and Rhabdomyolysis*

Laboratory Tests	Value	Reference Range
Serum urea, mg/dL	230	…
Serum creatinine, mg/dL	14	…
Serum sodium, mEq/dL	143	135 to 145
Serum potassium, mEq/dL	6.5	3.5 to 5.5
Serum calcium, mEq/dL	7.8	8.5 to 10.5
Serum phosphorus, mEq/dL	5.9	2.5 to 5
Serum uric acid, mg/dL	8	3 to 6
AST, IU/L	1204	20 to 40
ALT, IU/L	227	20 to 45
CPK, IU/L	57770	25 to 170
LDH, IU/L	3770	220 to 500
FT3, ng/mL	0.4	0.69 to 2.02
FT4, ng/mL	23	46 to 120
TSH, mIU/L	3.67	0.3 to 4.2
FSH, mIU/L	1.2	2 to 22
LH, mIU/L	1.1	2 to 22
Estradiol, pg/mL	18	30 to 400
Prolactin, ng/mL	1.3	2.8 to 29.2
Urine myoglobin	Positive	…
ANA	Negative	…
ANCA	Negative	…
PANCA	Negative	…
LE cell	Negative	…
C3	Normal	…
C4	Normal	…
CH50	Normal	…
ESR 1 hour	21	…
ESR 2 hour	49	…

*Ellipses are indicative of not applicable. AST indicates aspartate transaminase; ALT, alanine transaminase; CPK, creatine phosphokinase; LDH, lactate dehydrogenase: FT3, free tri-iodothyronine; FT4, free thyroxine; TSH, thyroid stimulating factor; FSH, follicle-stimulating factor; LH, luteinizing hormone; ANA, antinuclear antibody; ANCA, antineutrophil cytoplasmic antibody; PANCA, perinuclear antineutrophil cytoplasmic antibody; LE, lupus erythematosus; C3, complement 3; C4, complement 4; CH50, complement hemolytic 50; and ESR, erythrocyte sedimentation rate.

signs of hypothyroidism, and abnormal thyroid, hypothalamic, and ovarian function tests.

The patient underwent 3 sessions of hemodialysis and her kidney function gradually recovered after 2 weeks; her serum creatinine level decreased to 1.0 mg/dL at the time of discharge. Replacement therapy started with intravenous hydrocortisone, 100 mg every 6 hours, which was changed to oral prednisolone, 10 mg/d. Levothyroxin was commenced at a dose of 25 µg/d that gradually increased to 100 µg/d after 3 weeks. At 2-month follow-up, thyroid function tests normalized, but myalgia and muscle weakness remained. Four months later, myalgia and muscle weakness completely improved.

DISCUSSION

The present report describes a 46-year-old woman with Sheehan syndrome suffering from acute renal failure owing to rhabdomyolysis. Other known causes of rhabdomyolysis include collagen diseases (eg, poliomyelitis), ingestion of massive amounts of alcohol or drugs (eg, statins), vigorous exercise, trauma, infections, seizure, electrolyte disorders (eg, hyponatremia and hypokalemia).[7-9] In severe forms, rhabdomyolysis can become a life-threatening disorder, particularly when complicated by multiple-organ failure or acute renal failure.[1-3,8]

Muscle involvement is common in hypothyroidism,[1,5,6] and its myopathy is usually manifested with delayed relaxation of tendon jerks, proximal muscle weakness, muscle stiffness, myalgia, cramps, and occasional elevated levels of muscle enzymes[5,7,10]; however, rhabdomyolysis is quite rare.[1,5-8,10] The exact cause of rhabdomyolysis

in hypothyroidism is unclear, but both impaired glycogenolysis and impaired mitochondrial oxidative metabolism have been implicated.[1,2]

The present case represents rhabdomyolysis secondary to Sheehan syndrome in a developed stage that was manifested with acute renal failure. Although rhabdomyolysis is rare in hypothyroid patients,[1,2,4-8] it should be considered as an authentic cause of rhabdomyolysis. As a result, hypothyroidism must be considered in patients presenting with acute renal failure and elevated muscle enzymes.[4-6]

CONFLICT OF INTEREST

None declared.

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Received July 2007
Revised October 2007
Accepted October 2007