Rhabdomyolysis Accompanying Thyroid Crisis: An Autopsy Case Report

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Rhabdomyolysis is occasionally associated with metabolic disorders such as diabetic coma, severe electrolyte disturbances and myxedema coma. We describe rhabdomyolysis accompanying thyroid crisis. A 50-year-old man with Graves’ disease developed rhabdomyolysis, congestive heart failure and hepatic failure during the course of thyroid crisis and then died of acute renal failure. Postmortem examination revealed rhabdomyolysis in the cardiac and psoas muscles, old myocardial infarction, hepatic centrilobular necrosis, renal cortical necrosis, and follicular hyperplasia in the thyroid. Circulatory collapse and dehydration under excessive hypermetabolic state presumably suppressed the source of energy and oxygen for muscle cells, leading to cellular damage.

Key words: thyrotoxicosis, thyroid storm, skeletal muscle injury, acute renal failure

Introduction

Nontraumatic rhabdomyolysis has been reported to be occasionally associated with metabolic abnormalities such as diabetic coma, severe electrolyte disturbances and myxedema coma (1–9). Recently, a patient with rhabdomyolysis accompanying thyroid crisis died of resultant acute renal failure. This case is presented here along with the autopsy findings.

Case Report

A 50-year-old man was urgently admitted from his place of work to our clinic on September 3, 1990, in a state of unconsciousness, hypersweating, tachycardia and hyperpnea. He complained of fever and diarrhea several days before admission. He also had a past history of Graves’ disease untreated for 3 years after cessation of a 6-year period of antithyroid drug therapy. On physical examination, his consciousness was turbid and irritable; the skin was moist and slightly icteric. His temperature was 38.8°C, blood pressure 110/80 mmHg, pulse rate 180 beats/min and regular; the respiratory rate was 36 times/min. His eyes were exophthalmic. The thyroid gland was moderately enlarged, firm and smooth. Cardiac systolic murmur (2/6) was audible in the apex. The remainder of the physical examination was unremarkable except for hepatomegaly (3 cm below left costal margin). A chest roentgenogram showed globular cardiac enlargement and mild pulmonary congestion. Electrocardiograms revealed sinus tachycardia, left ventricular hypertrophy and existence of old myocardial infarction. The enlargement and congestion of the liver were obtained on an echogram.

On laboratory examination (Table 1), serum total bilirubin, GOT, GPT, LDH and alkaline phosphatase were high and serum total cholesterol and heparplastin titer were low. Serum electrolytes were within normal limits except for slight increases in uric acid and BUN. Serum myoglobin was normal. The other data of laboratory examination were normal except for increases in white blood cell count and hematocrit. The urinalysis revealed no abnormalities. On endocrinological examination (Table 2), marked increases in serum thyroid hormone levels were found with high titers in thyrotropin receptor antibodies. Serum TSH was low. Plasma renin activity, plasma aldosterone and plasma cortisol were markedly high, but plasma catecholamines were normal.

Combined treatments of MMI, lugol, β-blocker and hydrocortisone with fluid transfusion were immediately initiated after the confirmation of thyroid crisis (Fig. 1). His blood pressure remained below 90/50 mmHg and the pulse rate high. On the next day of admission, serum GOT, GPT and LDH abruptly increased to 11,860 U/l,
6,140 U/l and 10,390 U/l, respectively, with marked increases in serum CPK (924 U/l) and serum myoglobin (2,429 ng/ml). These parameters gradually decreased to normal on the sixth day. Serum myoglobin also decreased to 13.0 ng/ml. BUN and serum creatinine were gradually increased to 158 mg/dl and 5.3 mg/dl, respectively, on the sixth day. He died of acute renal failure with DIC (platelet cell count, 4.4 x 104; FDP, 40 μg/ml; fibrinogen, 152 mg/dl) in the morning of the seventh day.

Autopsy findings showed diffuse enlargement of the thyroid with cuboidal epithelial cells in macro- and microfollicles and no lymphocytic infiltration (Fig. 2). Massive anteroseptal myocardial necrosis and aortic valvular stenosis were found with transmural ischemia in the subendocardium of left ventricle. Further findings were: massive centrilobular necrosis and congestion in the liver, moderate renal cortical necrosis with multiple microthromboses, and necrosis with inflammatory cell reaction, accumulation of phagocytes and vacuolization in the cardiac and psoas muscles (Fig. 3).

**Discussion**

The present patient developed acute rhabdomyolysis with abrupt increases in serum and urinary myoglobins, serum CPK, and serum GOT, GPT and LDH levels...
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Fig. 2. Histological finding of thyroid gland showing cuboidal epithelial cells in micro and macrofollicles (HE stain, ×200).

Fig. 3. Rhabdomyolysis in psoas muscle; inflammatory cell reaction, accumulation of phagocytes and vacuolization (HE stain, ×400).

during the course of thyroid crisis. Shortly after the development of rhabdomyolysis he died of acute renal failure with DIC. The autopsy examination revealed diffuse follicular hyperplasia in the thyroid, old myocardial infarction with subendocardial ischemia, massive centrilobular necrosis and congestion in the liver, acute renal cortical necrosis with multiple fibrinogenic microthromboses, and rhabdomyolysis in the cardiac and psoas muscles. Acute cortical necrosis with multiple fibrinogenic microthromboses may have been induced by DIC.

Rhabdomyolysis is defined as a syndrome resulting from skeletal muscle injury with the release of muscle cell contents into plasma (1). Although the most common etiologic factors of rhabdomyolysis are alcohol and drug abuse, muscle compression and generalized seizures (1, 2), rhabdomyolysis may be occasionally caused by metabolic disorders such as non-ketotic hyperosmolar diabetic coma (3), diabetic ketoacidosis (4, 5), hypopotassemia (6), hypo- or hypernatremia (1), hypophosphatemia (7), hypothyroidism with or without renal failure (8, 9). However, to our knowledge this is the first case of development of rhabdomyolysis during thyroid crisis.

The mechanism by which thyroid crisis caused non-traumatic rhabdomyolysis in the present patient remains unclarified. The present patient was not associated with known etiologic factors such as alcohol and drug abuse, hypopotassemia, hypo- or hypernatremia and hypophosphatemia. The plasma glucose level also was normal. The present patient, however, had circulatory collapse and dehydration, as indicated by his physical findings, and a rise of hematocrit, plasma renin activity and plasma aldosterone, under an excessive hypermetabolic state. Such severe circumstances presumably result in the loss of an important source of energy and oxygen for muscle cells, leading to cellular damage.

References