Rhabdomyolysis in a Patient with Hypothyroidism

GURCAN KISAKOL, RECEP TUNC* AND AHMET KAYA

Department of Internal Medicine, Division of Endocrinology, Meram Medical Faculty, Selcuk University, Konya 42080, Turkey
*Department of Internal Medicine, Division of Rheumatology, Meram Medical Faculty, Selcuk University, Konya 42080, Turkey

Abstract. We describe a case of rhabdomyolysis associating hypothyroidism. Hypothyroidism frequently leads to myalgias, muscle stiffness, cramps and sometimes elevated levels of muscle enzymes, but rhabdomyolysis is quite rare. This report describes a case of rhabdomyolysis associating hypothyroidism in a 19-year-old man. Muscle enzyme levels were typical of rhabdomyolysis. Muscle biopsy and electromyographic findings were compatible with hypothyroid myopathy. Muscle functions completely recovered with levothyroxine therapy. The present case represents rhabdomyolysis secondary to undiagnosed hypothyroidism in a developed stage which manifests itself with rhabdomyolysis.

Key words: Hypothyroidism, Muscle enzymes, Rhabdomyolysis

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CLINICAL, usually moderate, myopathy frequently develops in patients with hypothyroidism and may reveal thyroid dysfunction. Biological signs of myositis, essentially a moderate rise in creatine phosphokinase plasma levels, are often found [1]. Myopathy is most often limited to myalgias, muscle stiffness, cramps and sometimes elevated levels of muscle enzymes. On the other hand, rhabdomyolysis is very rare.

We report a case of rhabdomyolysis associated with hypothyroidism in a 19-year-old man. The patient had proximal muscle weakness and tenderness, markedly raised muscle enzymes that normalized with thyroid replacement therapy. Hypothyroidism, though rare, should be considered as a definite cause of rhabdomyolysis.

Case report

A 19-year-old man admitted to the outpatient clinic of Rheumatology of Meram School of Medicine of Selcuk University with complaints of swelling, prominently of the lips and the extremities, severe myalgia and proximal muscle weakness of the extremities with difficulty in dressing and climbing stairs. He denied any form of strenuous activity and his complaints developed gradually, persisting for four months.

On examination, he was pale, afebrile, had peri-orbital puffiness and lip swelling and no goitre. All the limbs were swollen and pitting. He had proximal muscle weakness in both thighs (4/5), pelvic girdles (4/5), and shoulders (3/5). Relaxation of deep tendon reflexes was grossly delayed. Other systemic examinations were normal.

Investigations revealed: Hemoglobin 13.1 g/dl, total leucocyte count 5.6 × 10⁶/L with 90% polymorphs, serum Na 135 mEq/l, K 3.9 mEq/l, urea 28 mg/dl, creatinin 0.9 mg/dl, random blood glucose 96 mg/dl. Serum muscle enzymes were markedly elevated: creatinin phosphokinase 10210 IU/l (normal up to 170), creatine kinase-MM (CK-MM) 9350 IU/l (normal up to 20), LDH 985 IU/l (150–500), aspartate transaminase 388 IU/l, alanine transaminase 196 IU/l. Antinuclear antibody with immunofluorescence microscopy was negative. Urinary analysis showed moderate blood with dipstick, but on microscopic examination there were no erythrocytes. Therefore, we accepted this
finding to be compatible with myoglobinuria. Electromyogram (EMG) revealed polyphasic action potentials consistent with myopathy. Biopsy from the right quadriceps muscle was performed on the fifth day of admission. Examination of muscle biopsy specimens in the Pathology Department revealed findings consistent with rhabdomyolysis (Fig. 1 and 2). As the clinical and laboratory findings suggested hypothyroidism, serum free triiodothyronine and thyroxine were studied: free T3: 1 pg/ml (1.5–4.5), free T4: <0.2 ng/dl (0.8–1.9), TSH: >75 μU/ml (0.4–4). Both serum antimicrosomal antibody and anti-thyroglobulin antibody were abnormally elevated. Findings were compatible with autoimmune thyroid disorder so the patient was transferred to the Endocrinology clinic. Ultrasonography of thyroid gland was diffusely hypoechoic and heterogenic. Scintigraphy revealed a thyroid tissue with low Tc-99m uptake. Replacement with thyroxine (T4) was started at 100 μg/day, increased to 150 μg/day after two weeks, resulting in normalization of thyroid functions in 8 weeks. His muscle power improved gradually and serum enzymes normalized over a 12-week period. Edema of the lips and extremities disappeared within 8 weeks. At six months follow-up all clinical and laboratory findings were normalized. Levels of serum muscle enzymes and thyroid function tests after admission are shown in Table 1.

### Table 1. Muscle enzymes and thyroid function tests during eight-week follow-up

<table>
<thead>
<tr>
<th>Weeks</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPK (IU/L)</td>
<td>10210</td>
<td>926</td>
<td>338</td>
<td>205</td>
<td>175</td>
<td>168</td>
</tr>
<tr>
<td>LDH (IU/L)</td>
<td>985</td>
<td>604</td>
<td>579</td>
<td>503</td>
<td>389</td>
<td>507</td>
</tr>
<tr>
<td>AST (IU/L)</td>
<td>388</td>
<td>201</td>
<td>156</td>
<td>95</td>
<td>43</td>
<td>38</td>
</tr>
<tr>
<td>FT3 (pg/ml)</td>
<td>1.25</td>
<td>3.09</td>
<td>4.21</td>
<td>4.05</td>
<td>3.96</td>
<td></td>
</tr>
<tr>
<td>FT4 (ng/dl)</td>
<td>0.2</td>
<td>1.01</td>
<td>1.22</td>
<td>1.56</td>
<td>1.78</td>
<td>1.64</td>
</tr>
<tr>
<td>TSH (μU/ml)</td>
<td>148</td>
<td>18</td>
<td>3.7</td>
<td>1.6</td>
<td>2.01</td>
<td></td>
</tr>
</tbody>
</table>

**Discussion**

The present report describes a case of undiagnosed hypothyroidism in a developed stage which manifests itself with rhabdomyolysis. Onset was gradual and progressive that mimicked polymyositis. Diagnosis of rhabdomyolysis was based on the findings of severe myalgia, muscle weakness, marked elevation of serum CK, and histologic findings. As a cause of rhabdomyolysis, disorders such as collagen disease (e.g., polymyositis), ingestion of massive alcohol or other agents, infection, trauma, or congenital deficiency of muscular enzymes were excluded.

The clinical spectrum of hypothyroid myopathy is varied. Delayed relaxation of tendon jerks and proximal muscle weakness correlate with biochemical severity of hypothyroidism [1]. Rhabdomyolysis is, however, quite rare [2]. Myolysis in hypothyroidism is caused by changes in muscle fibres from fast twitching type II to slow twitching type I fibres, deposition of glycosaminoglycans, poor contractility of actin-myosin units, low myosin ATPase activity and low ATP turnover in skeletal muscle [3]. Absence of inflamma-
Rhabdomyolysis and Hypothyroidism

Rhabdomyolysis and hypothyroidism are both associated with muscle damage and weakness. The infiltrate with fiber necrosis is compatible with non-inflammatory disease, in our case with hypothyroid rhabdomyolysis. Thus, the muscle disorder in this patient which led to rhabdomyolysis was considered to be hypothyroid myopathy.

To our knowledge only nine cases of rhabdomyolysis due to hypothyroidism have been reported in English literature to date. Most of the cases were precipitated with exercise or trauma [4, 5]. Abnormal carbohydrate, protein and lipid metabolism of hypothyroid muscle might increase the rhabdomyolysis risk during exercise. Two others were associated with other factors combined with hypothyroidism like the use of anti-hyperlipidemic agents or toxic material [6]. On the contrary, our case is unique, in which there was no precipitating factor.

In conclusion, unexplained muscle weakness, high levels of muscle enzymes, findings of rhabdomyolysis in a young patient may be related to hypothyroid myopathy. Adequate therapy with thyroxine leads to complete recovery in such patients.

References