Hypothyroidism as a Cause of Rhabdomyolysis

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Abstract. We describe a patient presenting with muscular symptoms and rhabdomyolysis without any other precipitating factor, except primary hypothyroidism. After thyroxine replacement, musculoskeletal symptoms disappeared and creatine kinase concentrations decreased. Hypothyroidism is a rare cause of rhabdomyolysis, but should always be considered in a patient with an unexplained increase in creatine kinase concentrations.

Key words: Hypothyroidism, Rhabdomyolysis, Myopathy

ALTHOUGH muscle involvement is common in hypothyroidism, overt rhabdomyolysis is rare and only a few cases have been reported [1–6]. In most of them, a precipitating factor such as exercise [1], use of lipid-lowering drugs [2], or previous chronic renal failure [3] can be identified. We report a case of rhabdomyolysis, without any additional precipitating factor, in a patient subsequently diagnosed of primary hypothyroidism.

Case report

A 49 year-old man, born in Pakistan, was admitted to the hospital with a one-month history of tiredness, weight gain and progressive oedema. He referred myalgias, paresthesias in both hands and lower-limb weakness which had caused a reduction in everyday activity. He denied vigorous physical exercise, but worked as an ambulant salesman. His only medication consisted of ranitidine and aspirin.

He had no familial or prior personal history of thyroid disease. On physical examination, a normal heart-rate (80 beats/min), generalised nonpitting oedema, periorbital puffiness and diffuse goitre were found. No signs suggesting an associated systemic inflammatory disease were found. On admission, laboratory measurements revealed: creatine kinase (CK) 9332 U/l (Normal (N) <174), myoglobin 399 μg/l (N <70), LDH 1262 U/l, aldolase 34.9 U/l (N: 0–7.3), AST 136 U/l, ALT 84 U/l, triglycerides 2.24 mmol/l, total cholesterol 8.13 mmol/l and LDL cholesterol 5.71 mmol/l. Haematological tests, electrolytes and renal function were all normal at baseline. Thyroid function tests confirmed the diagnosis of hypothyroidism (see Table 1). The antimicrosomal antibody titre was 944 UI/ml (N <150).

Intravenous fluids were started immediately, and L-thyroxine replacement (100 μg/day) on the day after admission, leading to a progressive improvement of symptoms in a few days. On discharge, a week after admission, serum CK had decreased to 2297 U/l (see Table 1). After ten weeks on thyroxine replacement, the patient’s muscular symptoms had disappeared, CK concentrations had decreased to 257 U/l and thyrotropine concentrations were normal.

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Table 1. Main laboratory results on admission and during follow-up.

<table>
<thead>
<tr>
<th>Time since diagnosis</th>
<th>0 day</th>
<th>2nd day</th>
<th>7th day</th>
<th>5th wk</th>
<th>10th wk</th>
</tr>
</thead>
<tbody>
<tr>
<td>CK (U/l)</td>
<td>9332</td>
<td>4039</td>
<td>2297</td>
<td>404</td>
<td>257</td>
</tr>
<tr>
<td>AST (U/l)</td>
<td>136</td>
<td>115</td>
<td>22</td>
<td>24</td>
<td></td>
</tr>
<tr>
<td>Thyrotropin (mU/l)</td>
<td>147.7</td>
<td></td>
<td>2.10</td>
<td>2.0</td>
<td></td>
</tr>
<tr>
<td>Free thyroxine (pmol/l)</td>
<td>1.33</td>
<td></td>
<td>17.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thyroxine dose (ug/day)</td>
<td>0</td>
<td>100</td>
<td>100</td>
<td>150</td>
<td>150</td>
</tr>
<tr>
<td>Creatinine (umol/l)</td>
<td>107</td>
<td>121</td>
<td>126</td>
<td>99</td>
<td></td>
</tr>
</tbody>
</table>

0 day: on admission. wk: weeks. Normal reference levels: Thyrotropin: 0.25–5 mU/l, Free thyroxine: 10–22 pmol/l.

Discussion

The present case describes a patient suffering from rhabdomyolysis due to hypothyroidism, with no additional precipitating factor. Although the main features of rhabdomyolysis are muscular symptoms and increased CK concentrations, it can become a life-threatening disorder when complicated by acute tubular necrosis and renal failure. In most of the previously reported cases of rhabdomyolysis associated with hypothyroidism an aggravating factor has been identified. Although hypothyroidism is frequently accompanied by asymptomatic mild to moderate CK elevations, usually less than 10 times the upper normal limit, to our knowledge, only three previous cases of overt rhabdomyolysis have been ascribed to hypothyroidism alone [4–6].

The exact cause of rhabdomyolysis in hypothyroidism remains unclear, but both impaired glycogenolysis [1] and impaired mitochondrial oxidative metabolism [8] have been involved, and an autoimmune mechanism could be possible, as has been described for Graves' disease [9].

Important increases in CK concentrations can be seen in patients with hypothyroidism in the presence of a precipitating factor, vigorous exercise being the most common. It has actually been suggested that hypothyroidism should be suspected when a patient presents with rhabdomyolysis after exercise [1]. However, rhabdomyolysis can be due to exercise itself, and may cause increases in CK concentrations above 100,000 U/L [7]. Other known causes of rhabdomyolysis include inflammatory myopathies (e.g. polymyositis), congenital deficiency of muscular enzymes, trauma, infection, electrolytic disorders (hyponatremia, hypokalemia, hypercalcemia, hyperosmolar states), drugs and toxins (e.g. massive ingestion of alcohol), all of them absent in the present case.

In summary, although hypothyroidism is a rare cause of rhabdomyolysis, it should be suspected in patients presenting with muscle aches and high CK concentrations in the absence of other, more common causes of rhabdomyolysis. As soon as the diagnosis is made, thyroid hormone replacement should be started.

References
