A CASE OF CHRONIC THYROTOXIC MYOPATHY ASSOCIATED WITH MYELOPATHY

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Abstract: We present a 52-year-old woman with Graves' disease, who was characterized by having both chronic thyrotoxic myopathy and posterolateral myelopathy. Both symptoms began and progressed concomitantly. After the administration of methimazole for treatment of Graves' disease, myopathy improved, while myelopathy remained unchanged. Various possible disorders to cause myelopathy were sought. However, roentgenographic findings of entire spine X ray, myelogram, magnetic resonance imaging and cerebrospinal fluid examinations were all with negative results. The present data imply that rare manifestations associated with myelopathy in this patient may be due to thyrotoxicosis which is strongly linked to an abnormality in autoimmune mechanisms.

Key words: Graves' disease, Chronic thyrotoxic myopathy, Myelopathy

INTRODUCTION

Graves' disease is a multiorgan metabolic disorder with hyperthyroid state. It is well known that hyperthyroidism is associated with several neuromuscular disorders including acute thyrotoxic myopathy, chronic thyrotoxic myopathy, exophthalmic opthalmoplegia, myasthenia gravis and periodic paralysis\(^1\). We have recently experienced a patient with characteristic manifestations of myelopathy in addition to chronic thyrotoxic myopathy. Myelopathy results from a number of different causes such as vascular disease, demyelinating disease, viral infection, remote effects of malignancy and sarcoidosis\(^2,3\). Myelopathy is not listed among usual clinical neuromuscular complications of thyrotoxicosis. In the present paper, we report an interesting case showing hyperthyroidism associated with chronic thyrotoxic myopathy and myelopathy.

CASE REPORT

The patient was a 52-year-old woman. Since February, she had muscle weakness of all extremities. After March she had difficulty in walking, lost 10kg in weight despite good appetite for the past two weeks and was admitted to our hospital. Physical examination showed height 151cm, weight 46kg, blood pressure 120/60mmHg and pulse rate 92 per minute. She had neither tremor nor exophthalmos, though showing diffuse enlargement of thyroid gland. There were no abnormalities of chest and abdomen, and so in all cranial nerve functions. She showed a spastic and waddling gait with weakness and atrophy of proximal muscles of all extremities (Fig. 1).

Tonus of the upper limbs was normal, while it was spastic in the lower limbs. A jaw jerk was normal. Bilateral tendon reflexes of the upper extremities were symmetrically diminished, but those of the lower extremities were all hyperactive with positive Babinski and Chaddock signs. There were no fasciculations. Sensory functions were normal except for marked decrease in vibration sense in the lower limbs. Cerebellar function was normal. Normal laboratory data included serum alkaline phosphatase, creatine phosphokinase, myoglobin and potassium, although there was decrease in serum cholesterol (106mg/dl) and increase in serum creatine (1.42mg/dl) and aldolase (5.9IU/l/37\(^\circ\)C). Renal functions were all within normal limits. Chest X-ray and electrocardiogram showed...
Table 1  various parameters in the patient

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<th>6/26</th>
<th>7/23</th>
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<tbody>
<tr>
<td>TSH (μU/ml)</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>1.2</td>
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<td>free-T₄ (ng/dl)</td>
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<td>2.23</td>
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<tr>
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<td>30.1</td>
<td></td>
<td>15.0</td>
<td>−10.0</td>
<td>+10.0</td>
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<tr>
<td>MHA (×)</td>
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<td></td>
<td></td>
<td>400</td>
<td></td>
<td>&lt;100</td>
</tr>
<tr>
<td>THA (×)</td>
<td>(−)</td>
<td></td>
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<td>(−)</td>
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</tbody>
</table>

TSH : thyroid stimulating hormone, T₃ : triiodothyronine, T₄ : thyroxine, TRAb : anti-TSH receptor antibody, MHA : microsomal hemagglutination, THA : thyroid hemagglutination

DISCUSSION

This is a rare case of hyperthyroidism associated with posterolateral myelopathy and proximal myopathy. Hyperthyroidism can cause different types of deficits in neuromuscular systems. Chronic thyrotoxic myopathy is one of the most common disorders. In contrast, myelopathy is a rare neuromuscular complication of thyrotoxicosis. To our knowledge, so far, some cases with posterolateral myelopathy and pyramidal tract dysfunction have been described in hyperthyroid patients4-6). The present case showed obvious pyramidal tract dysfunctions of the lower limbs and marked decreases in vibration sense. The similar case with posterolateral myelopathy associated with thyrotoxicosis has been reported4). After the administration of methimazole, the myopathy improved in the present case, while posterolateral myelopathy remained unchanged. This is in agreement with the clinical manifestation reported by Melamed et al4) who showed incomplete recovery of neuromuscular disturbances following antithyroid treatment. Garcia et al also reported a case of corticospinal tract disease due to hyperthyroidism, a case showing residual hyperreflexia of the right lower extremity which was not affected by treatment with methimazole6). Various possible disorders associated with myelopathy were sought, but roentgenographic and laboratory findings

no significant abnormalities. Table 1 shows the values of thyroid functions with increase in free-T₄ and free-T₃ concentrations. Basal metabolic rate (BMR) was +26%. The titers of antimicrosomal antibody and anti TSH receptor antibody were elevated. Antithyroid antibody was negative. Thyroid scintigram using ¹²³I showed diffuse high uptake. ¹²³I thyroid uptake was 49% at 3hrs and 35% at 24hrs. These data were compatible with hyperthyroidism. Myogenic pattern consisting of low amplitude and short duration was shown by needle electromyography of quadriceps femoris muscles.

Conduction velocities and latencies of bilateral median, ulnar and peroneal nerves were normal. Roentgenograms of the entire spine, myelogram, magnetic resonance imaging and brain computed tomography showed no abnormalities. Serum vitamin B₁₂ and folic acid were within normal limits. Results of cerebrospinal fluid examination, including protein concent-
myopathy, myelopathy and Graves' disease

ranged within normal. These observations suggested that hyperthyroidism may be responsible for the posterolateral myelopathy in this patient. The pathophysiological mechanisms by which hyperthyroidism caused myelopathy remain unknown at present, but one possibility is that both thyrotoxicosis and posterolateral myelopathy shared a common cause affecting the different organs simultaneously. In our patient the titer of antimicrosomal antibody and anti-TSH receptor antibody were elevated, and posterolateral myelopathy may be related to autoimmunity in its pathogenesis, this concept being proposed by Melamed et al. Alternatively the other possibility is that thyrotoxicosis itself may directly cause myelopathy. Hyperthyroidism can cause varying metabolic abnormalities at many organs containing muscles and central nervous system. Mitochondrial abnormalities have been observed on electron microscopy in the muscle fibers of rats and humans with hyperthyroidism. It has been reported that the cellular dysfunctions were due to a direct effect of excessive thyroid hormone which resulted in the rapid decline in mitochondrial respiration and oxidative phosphorylation, and farther the intramuscular changes in water electrolytes and enzymes. Concerning this problem it must be clarified how thyroid hormone physiologically and pathologically acts on nerve cellular functions in the present case.

REFERENCES