BRIEF COMMUNICATIONS

THE RAPID TRANSFORMATION OF HYPERTHYROIDISM TO HYPOTHYROIDISM COMPLICATED BY MYASTHENIA GRAVIS

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Myasthenia gravis (MG) is considered an autoimmune disease. Among the other autoimmune disorders that complicate MG, hyperthyroidism is the most frequently seen.¹ Concerning the effect of thyroid function on myasthenia, findings are conflicting. Some have reported that as the hyperthyroidism was treated there was a worsening of the MG^{2,3}; others have reported that the condition improved.^{1,4} We report a patient with hyperthyroidism that was rapidly transformed into hypothyroidism. Complications of MG occurred during this transformation.

A 25-year-old man had excessive sweating and palpitation since 1989 (when he was 20 years old). A physician noted the presence of a struma in October 1992. Laboratory studies in June 1993 revealed the following results: free triiodothyronine, 15.0 pg/ml (normal range, 2.5 to 6.0 pg/ml); free thyroxine, 3.0 mg/ml (normal range, 0.8 to 2.2 mg/ml); thyroid-stimulating hormone (TSH), 0.05 μ IU/ml (normal range, 0.3 to 3.9 μ IU/ml); thyroglobulin hemagglutination $\times 1600$ (normal, $< \times 1000$); and microsome hemagglutination $\times 6400$ (normal, $< \times 100$). A diagnosis of Graves' disease (hyperthyroidism) was confirmed, and treatment with 5 mg methimazole was initiated. Diplopia and ptosis were first noted in October 1993. Dysphagia and weakness in the extremities developed in January 1994. In March 1994, a diffuse, elastic, soft struma was palpated in the anterior neck region. Laboratory studies revealed the following results: free triiodothyronine, 3.4 pg/ml; free thyroxine, 0.57 ng/ml; TSH, 32 μ IU/ml; thyroglobulin hemagglutination, $\times 6400$; and microsome hemagglutination, $\times 25600$. TSH-binding inhibitory immunoglobulin was 12.2% (normal range, -10% to $+10\%$). Thyroid-stimulating antibodies was 240% (normal, $< 145\%$). Changes in results of thyroid function studies between June 1993 and this writing are shown in Table I. The anti-acetylcholine receptor antibody level was elevated to 3.1 nmol/L (normal, 0.2 nmol/L). Electromyography showed the waning phenomenon after continuous peripheral nerve stimulation typical of MG. Simultaneous occurrence of hypothyroidism and MG was diagnosed. After admission, methimazole treatment was discontinued, and replacement therapy with 25 mg thyroid extract was begun. When the patient attained euthyroid status, extended thymectomy was performed. Many lymphoid follicles, each with a germ center,

Table I. Changes in thyroid function by days of methimazole therapy

	-7*	30	60	90	150	180	270
Free T ₃ (pg/ml)	15.0	12.0	5.1	3.9	3.9	3.6	3.4
Free T ₄ (ng/ml)	3.0	3.83	1.52	0.96	0.96	0.96	0.57
TSH (μ IU/ml)	0.05	0.05	0.05	1.5	7.2	13	32

Normal values: Free T₃, 2.5-6.0 pg/ml; free T₄, 0.8-2.2 ng/dl; TSH, 0.3-3.9 μ IU/ml. MG symptoms were observed from day 120. T₃, triiodothyronine; T₄, thyroxine.

*Methimazole therapy was started on day 0. Day -7 thus represents 1 week before the start of therapy.

were observed histologically. These histologic findings were compatible with a diagnosis of MG. Symptoms caused by MG subsided after operation. Anti-acetylcholine receptor antibody, TSH-binding inhibitory immunoglobulin, and thyroid-stimulating antibody levels were reduced.

The relationship between hypothyroidism and MG has been studied by Feinberg, Underdahl, and Eaten.⁵ Drachman⁶ reported that aggravation of MG occurs in patients with both MG and euthyroid thyroid disease when function becomes either hyperthyroid or hypothyroid. Ours is a rare case in which the onset of MG coincided with the transformation of hyperthyroid to hypothyroid disease. It is difficult to assess the effect of thymectomy on thyroid function. TSH-binding inhibitory immunoglobulin and thyroid-stimulating antibody values clearly decreased after thymectomy. This observation suggests that the thymus also plays a role in the pathophysiology of autoimmune thyroid diseases.

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J THORAC CARDIOVASC SURG 1995;110:852

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0022-5223/95 \$5.00 + 0 12/54/62833