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Hypothyroid Graves' ophthalmopathy: a case report

Abstract: Hypothyroid Graves' ophthalmopathy is a rare condition, present in about 3% of all cases. Since thyroid-stimulating antibodies could be detected in a majority of euthyroid and hypothyroid Graves' patients, the most probable explanation for unincreased thyroid function is a reduction of tissue capability to response to stimulation. We present a 57-yr-old man visited the hospital with signs and symptoms typical of hypothyroidism. Since TSH was 77 IU/ml, FT4 6.8 pmol/l and TPO Ab 4828 IU/ml, the treatment with 100 mcg/day T4 was started. Three months later, when euthyroid, he developed Graves' ophthalmopathy with slight proptosis, moderate palpebral edema, conjuctival injection and chemosis, reduction of visual acuity to 0.7, diplopia and secondary glaucoma. He had no palpable goiter and ultrasound revealed small (V 5 cm³), diffuse hypoechoic thyroid. Orbital computed tomography (CT) showed a pronounced enlargement of all extraocular muscles (9-15 mm). TSH receptor antibodies were 65 U/l. Patient was treated with two doses of 0.5 g intravenous methylprednisolone during three days, followed by oral prednisone 40 mg/day tapered to 10 mg/day in four weeks. Six courses of therapy were performed. There were no significant side effects during the treatment. A prompt improvement of visual acuity, intraocular pressure and inflammatory signs was noticed, but diplopia became permanent. Orbital CT revealed a significant reduction of all rectus muscles (2-10mm). TSH receptor antibodies were 10 U/l, TPO Ab 8603 IU/ml. He developed cataract on his left eye and refused extraocular muscle surgery since he lost diplopia. Conclusion: Hypothyroid Graves' disease reflects a subtle relations between destructive changes in the thyroid gland and autoimmune mechanisms involved in thyroid pathology.

Key words: Graves' ophthalmopathy, hypothyroidism