Diplopia as the Sole Manifestation of Hyperthyroidism

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Abstract

Thyrotoxicosis can be associated with thyroid ophtalmopathy in up to 90% of patients. We describe a 62 year old healthy man who was presented with a history of diplopia for the past half year. His medical history revealed no other complaints and neither did his physical examination. Elevated levels of the thyroid function tests were found. Diplopia alone requires medical attention and leads to the diagnosis of autoimmune thyroid disease, because finding vertical (restrictive) diplopia is actually the appearance of this particular thyroid disease.

INTRODUCTION

Hyperthyroidism is a relatively common disorder. The major symptoms of thyrotoxicosis include palpitations, hyperactivity, anxiety, nervousness, heat intolerance, tremor, weight loss, diarrhea, disturbances of menstruation and sweating. Common signs of thyrotoxicosis include: weight loss despite increased appetite, tachycardia or atrial fibrillation, systolic hypertension, warm and smooth skin, fine tremor and muscle weakness. Younger patients tend to exhibit more sympathetic activation, such as anxiety, hyperactivity and tremor, while older patients have more cardiovascular symptoms, such as dyspnea and atrial fibrillation. The clinical manifestations of thyrotoxicosis do not always correlate with the extent of the biochemical abnormality [1].

Thyrotoxicosis can be associated with thyroid ophtalmopathy in up to 90% of patients. Sometimes, it is manifested only by periorbital edema, but it also can include conjunctival edema (chemosis), injection, poor lid closure, extraocular muscle dysfunction (diplopia), and proptosis. Only 5% develop severe ophthalmopathy, e.g., diplopia, visual-field deficits, blurred vision. A medical emergency occurs when the orbital edema causes optic nerve compression with early loss of color vision and orbit pain. Without treatment, continued pressure of the optic nerve can cause permanent vision loss [1, 2].

CASE DESCRIPTION

A 62 year old healthy man, married with two children was presented with a history of diplopia for the past half year. He felt it only in certain circumstances such us: after a long time on the computer, after prolonged reading, looking up in some angles of the view. His medical history revealed no other complaints and neither did his physical examination. Neurological examination was normal. Complete blood count, blood chemistry, thyroid function tests and acetylcholine antibodies were performed. Elevated levels of the thyroid function tests (Free T3-6.8pg/ml, Free T4 -1.7ng/dl and TSH < 0.05ulU/ml; Normal Blood Levels: TSH- 0.39-4.0ulU/ml, Free T3 - 2.3-4.2pg/ml, Free T4 - 0.8-1.5ng/dl) were found. The patient denied again any signs of hyperfunction of the thyroid gland (nervousness, anxiety, diarrhea, weight loss, tachycardia etc.), not one had been noticed. The interpretation of the results of orbital Computerized Tomography (CT) and CT of the brain was controversial. The presence of some mass in the left eye had been reported. A following Magnetic Resonance Imaging (MRI) revealed the thickening of the inferior and lateral rectus muscles suggesting thyroid ophthalmopathy. The patient was treated by Methimazole and consulted by neurophthalmologist. At this stage the diplopia was decided to be followed up only. If necessary it would be treated appropriately.

COMMENT

Diplopia is a visual symptom in which a single object is perceived by the visual cortex as two objects rather than one. Disorders associated with this condition include refractive errors, strabismus, oculomotor nerve diseases, trochlear nerve diseases, abducens nerve diseases and diseases of the brain stem and occipital lobe.

When evaluating a patient with a complaint of double vision,

it is important to distinguish monocular versus binocular diplopia, which are differentiated by asking the patient to cover each eye separately. In the setting of binocular double vision, one of the two images disappears when either eye is covered, because diplopia is the result of ocular misalignment. On the other hand, monocular double vision resolves when the affected eye is covered, but remains when the opposite eye is occluded. Causes of monocular diplopia include cataract, refractive error, and retinal disease, which can be managed accordingly by an ophthalmologist [$_3$]. Binocular vertical diplopia may be due to supranuclear processes, ocular motor nerve dysfunction, neuromuscular junction disease, diseases of eye muscle, mechanical processes causing vertical eye misalignment, and even retinal disease [$_4$].

Diplopia without obvious exophthalmos, known to be present in up to 17% of all cases, is caused by infiltrative endocrine ophthalmopathy due to ocular muscle imbalance and by mechanical orbital restriction of the extraocular muscle and not by an innervational defect.

We understand how the orbital changes and the symptoms of endocrine ophthalmopathy develop. The symptoms of thyroid eye disease depend on intensity of acute inflammatory reactions and its severity (extent of anatomical, functional, and cosmetic features). As the disease progresses the acute inflammation recedes, but signs and symptoms improve only partially because of the residual fibrosis and scarring of the orbital contents [$_5$].

Medical therapy of diplopia, caused by hyperthyroidism, attempts to shrink the swollen tissues with steroids, radiotherapy or cyclosporine. Surgery makes more space available for the swollen tissues and can be modified for each patient. Furthermore, rehabilitation for diplopia can be managed with prisms or extraocular muscle surgery($_6$). Cases of spontaneous resolution of diplopia are known as well.

Diplopia alone requires medical attention and leads to the diagnosis of autoimmune thyroid disease, because finding vertical (restrictive) diplopia is actually the appearance of this particular thyroid disease.

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