THYROID-ASSOCIATED ORBITOPATHY WITH SUPERIOR OBLIQUE MUSCLE INVOLVEMENT: A CASE REPORT

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A 29-year-old male with a 5-year-history of hyperthyroidism complained of diplopia and proptosis. After subtotal thyroidectomy, he still had diplopia in a certain gaze. Computerized tomography showed inferior rectus muscle enlargement in the right eye and inferior rectus, medial rectus, and superior oblique muscle enlargement in the left eye. Ocular examination with the cover and uncover test revealed hyperphoria and exophoria in the left eye. The upward gaze of the right eye was more limited than that of the left eye. Since superior oblique muscle involvement in patients with thyroid orbitopathy is quite rare, we discuss its effect on ocular motility in patients with thyroid-associated orbitopathy.

Key Words: superior oblique muscle, thyroid-associated orbitopathy
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Thyroid-associated orbitopathy (TAO) is an organ-specific autoimmune disorder. Most patients with Graves’ hyperthyroidism have evidence of TAO [1]. TAO is characterized by enlargement of the extraocular muscles and an increase in retrobulbar fat [2]. Hypertrophy of extraocular muscles is mainly caused by increases in glycosaminoglycan production by orbital fibroblasts [3]. Cellular infiltration of interstitial tissues by lymphocytes, plasma cells, macrophages, and mast cells contributes to muscular fiber fibrosis and the development of restrictive myopathy and diplopia. The most commonly involved extraocular muscles are the inferior rectus (IR) and medial rectus (MR) muscles. The superior oblique (SO) muscle is much less often involved in TAO than other rectus muscles [1]. We report a man with TAO who had multiple extraocular muscle involvement, including the SO muscle. The mechanisms of abnormal eye position caused by multiple hypertrophic muscles, and the interaction of these muscles, are explored.

CASE PRESENTATION

A 29-year-old male patient had TAO and complained of double vision on a certain gaze. He was euthyroid. His best-corrected visual acuity was 20/20 in each eye. Lagophthalmus was noted in both eyes. Intraocular tension was 15 mmHg in the right eye and 16 mmHg in the left eye. The cover and uncover test revealed exophoria (30 prism diopter) and hyperphoria (15 prism diopter) at primary gaze in his left eye. Moderate motility limitation of the left eye on downward and left gaze and mild motility limitation on upward gaze were found on ocular examination. There was more severe limitation in the right eye on upward gaze than in the left eye (Figure 1).

Fundus examination was normal in both eyes. The Humphrey auto-perimetry examination was within normal limits. Orbital computerized tomography (CT) showed that the IR, MR, and SO muscles of the left eye and the IR muscle of the right eye were enlarged. The muscle tendons were not involved and other orbital structures were normal. Among the hypertrophic muscles, the IR muscle of the right eye was...
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the most hypertrophic on orbital CT scan (Figure 2). The diplopia bothered this patient by limiting the extraocular muscles in certain directions but not in the primary position. After initial oral prednisolone and later anti-inflammatory drug treatment, extraocular motility improved somewhat, although the diplopia still existed at the end of the vertical gaze.

**DISCUSSION**

Clinically appreciated restriction of ocular motility correlates very strongly with increased mean diameter of respective extraocular muscles ($p < 0.01$) [4]. Orbital CT scan is a useful tool to explain the restriction severity of ocular motility in TAO patients with multiple muscle involvement [4–9]. The prevalence of SO muscle hypertrophy in TAO has been reported as 4.9%, compared with 57% for IR muscle and 61.3% for MR muscle involvement; isolated SO muscle involvement in TAO has not been reported [4].

According to the orbital CT in our patient, both the SO and IR muscles were hypertrophic in the left eye. This suggests that the patient will have hypotropia in the left eye because the main actions of both muscles are depression, and the eye position will be much more hypotropic than if the IR muscle alone was involved. In contrast, the patient

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**Figure 1.** Extraocular motility. Top row: upward gaze is much more limited in the right eye than in the left eye. Center row: orthophoria in the primary position. Bottom row: downward gaze is much more limited in the left eye than in the right eye.

**Figure 2.** Orbital computerized tomography. (A) Muscle tendons are not involved and the other orbital structures are normal. (B) Coronal view shows that the inferior rectus (IR) muscle in the right eye is enlarged and the IR, medial rectus, and superior oblique muscles in the left eye are also enlarged.
had hyperphoria and exophoria only in the left eye at primary gaze. There are several possible explanations for this observation and the motility restriction in our patient. The right IR muscle was much more enlarged than the left IR muscle. It is possible that the total restriction effects of the IR muscle and the SO muscle in the left eye nearly equaled the hypertrophy of the IR muscle in the right eye in the primary position. Thus, the balance of depressor functions between both eyes meant that the patient had no diplopia or hypotropia of the left eye at primary gaze. The other possibility is that the hypertrophy of the SO muscle actually restricts the downward movement of the eyeball rather than reinforcing downward movement. On downward gaze, the right eye still behaved more downward than the left eye. This observation provides evidence that hypertrophy of the SO muscle actually restricts the downward movement of the eyeball rather than reinforcing downward movement. When the patient looked toward the right upper, upper, and left upper directions, the right eye was much more limited than the left eye. Since the main actions of the IR muscle are depression and extorsion, hypertrophy of the IR muscle in the right eye restricted the elevation function of the superior rectus muscle and the inferior oblique muscle more than that in the left eye. Because the IR muscle was much more hypertrophied, the restriction in the upward gaze was greater than that caused by both IR and SO hypertrophy of the fellow eye in this patient.

We report an interesting patient with TAO and SO muscle involvement. The restriction and motility effects of SO hypertrophy in patients with TAO are still not clear, so further case collection is necessary to clarify this issue.

References