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LETTER

Evaluation of the association between subconjunctival orbital fat prolapse and thyroid-associated orbitopathy

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Dear editor

I read with interest the article entitled "Subconjunctival orbital fat prolapse and thyroid associated orbitopathy: a clinical association" by Chatzistefanou et al.[1] The case-series study was undoubtedly well designed and conducted, in which the authors successfully revealed that subconjunctival orbital fat prolapse may occasionally be a predominant clinical manifestation of thyroid-associated orbitopathy (TAO) and suggested that the presence of subconjunctival orbital fat prolapse can alert the diagnosis of thyroid orbitopathy.

However, I would like to point out that the mechanism underlying the association between the two conditions is still unclear. Subconjunctival fat prolapse is usually caused by forward herniation of intraconal fat tissue due to dehiscence of tenon capsule precipitated by aging process or trauma.[2] In TAO, swelling and inflammation of orbital fat can lead to separation of the orbital septum from the capsulopalpebral fascia, which can cause fat prolapse into the orbit.[3] Previous studies showed that intraorbital inflammation in TAO can lead to apical crowding in orbit and intracranial fat prolapse.[4,5] Therefore, as the authors postulated, it would be plausible that elevated intraorbital pressure and increase in orbital fat in TAO can also allow anterior herniation of intraorbital fat.

Increased orbital fat and raised intraorbital pressure can also precipitate backward herniation of intraorbital fat into intracranial space and compressive optic neuropathy.[4,5] Thus, to support their postulate, it would be helpful to investigate intracranial fat herniation using orbital images, including computed tomography or magnetic resonance imaging, and to evaluate the optic nerve function using funduscopic examination, visual field testing, and color vision testing in patients with concurrent active TAO and subconjunctival fat prolapse. We believe that the presence of intracranial fat prolapse or compressive optic neuropathy may support their postulate.

Disclosure

The author reports no conflicts of interest in this communication.

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Dear editor

We appreciate Dr Han's comments and interest in our article. Intracranial herniation of intraorbital fat through the superior orbital fissure has been reported at an incidence of 19% in patients with thyroid ophthalmopathy in one study,[1] and its prevalence may vary between 24% and 82% in different studies[1–3] among patients with dysthyroid ophthalmopathy.

We reviewed again data from the patients' charts for symptoms and signs of compressive optic neuropathy upon presentation with subconjunctival fat prolapse.[4] There were no afferent pupillary defects or optic nerve head swelling or atrophy noted on fundoscopy in any of the patients reported in this series. Clinical suspicion for possible compressive optic neuropathy, prompting paraclinical investigation with visual field testing, had been raised for patient #2 who had a slight decrease in visual acuity to 0.9 in the right eye. Visual field testing by automated perimetry was within normal limits. Visual acuity improved to 1.0+ in the involved eye with a change in astigmatic correction.

We also reevaluated the magnetic resonance (MR) scans of patients in the applied MR sequences: 1) T1 fat suppression, 2) STIR, and 3) T2 spin echo. We did not highlight the presence of fat located intracranially, including patients 1, 2, and 3 who presented with active thyroid-associated orbitopathy and subconjunctival fat prolapse.

While the possible association between subconjunctival fat prolapse and intracranial fat prolapse may raise an intriguing question, intracranial fat prolapse extending posterior to the superior orbital fissure was not noted among patients in this series.

Disclosure

The authors report no conflicts of interest in this communication.

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