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Heavy Eye Syndrome With Asymmetric Ocular Surface Disease: Over a Decade of Follow-Up

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Introduction

Heavy Eye Syndrome (HES) is a strabismus disorder associated with high axial myopia and characterized by degeneration of the superior rectus–lateral rectus (SR–LR) band, resulting in displacement of the superior rectus and lateral rectus muscles and consequent ocular misalignment.[1-3] The condition may present with esotropia, hypotropia, and varying degrees of ocular motility limitation.[1,2,4,5] While the motility features of HES have been extensively described, its potential impact on the ocular surface has received limited attention.

Dry eye disease is multifactorial and may arise from tear film instability, inflammation, and mechanical factors such as altered eyelid–corneal interaction.[6–8] Reports highlighting long-term ocular surface involvement in HES are limited in the literature. Because HES occurs in eyes with marked axial elongation, these patients may also be susceptible to ocular surface symptoms and dry eye disease, consistent with observations in highly myopic eyes.[9] Although the exact mechanism remains uncertain, biomechanical and ocular surface factors may contribute to tear film dysfunction in individuals with this syndrome.

We report a young patient with HES who presented with asymmetric severe dry eye symptoms, predominantly affecting the eye with greater axial elongation, and who was followed for approximately 11.5 years with detailed clinical, imaging, and ocular surface assessments.

Case Report

A 23-year-old male was referred with complaints of blurred vision, itching, tearing, and redness predominantly affecting the left eye. Best-corrected visual acuity (BCVA) was 20/25 in the right eye and 20/100 in the left eye.

Anterior segment examination demonstrated minimal bilateral papillary conjunctival reaction, diffuse punctate epithelial erosions more pronounced in the left eye, and reduced tear meniscus height. Tear film break-up time was less than 3 seconds bilaterally, with more severe staining and symptoms in the left eye, consistent with asymmetric dry eye disease with marked aqueous deficiency in the left eye. In addition, Schirmer I testing was performed without topical anesthesia, and Schirmer II testing was performed according to standard methodology with nasal stimulation. Test values were of 10 mm and 7 mm, respectively, in the right eye and of 3 mm and 1 mm, respectively, in the left eye.

Ocular alignment assessment revealed esotropia and hypotropia of the left eye. Prism alternate cover testing demonstrated 20 prism diopters (PD) of esotropia at distance, 12 PD of esotropia at near, and 12 PD of left hypotropia at both distance and near. Axial length measurements showed marked anisometropia (right eye: 25.61 mm; left eye: 32.05 mm). Axial and coronal magnetic resonance imaging demonstrated inferomedial globe displacement in the left eye, with nasal displacement of the superior rectus and inferior displacement of the lateral rectus muscle, supporting the diagnosis of HES (Figures 1 and 2). Secondary neurological causes were excluded.[2] The patient had no history of contact lens wear, ocular surgery, allergy, atopy, or use of medications known to affect tear production. Although minimal papillary conjunctival reaction was present on slit-lamp examination, there were no clinical features suggestive of allergic ocular disease and no Horner–Trantas dots were observed. Eyelid position and function were clinically normal. Family history was negative for autoimmune disease. Autoimmune serologic testing was unremarkable. Anti-SSA/Ro, anti-SSB/La, HLA-

B27, and HLA-B51 testing were negative, reducing the likelihood of alternative systemic causes contributing to the severity and asymmetry of the ocular surface disease.

Fundus examination revealed a myopic fundus in the left eye, including a tilted optic disc, peripapillary atrophy, and retinal thinning.

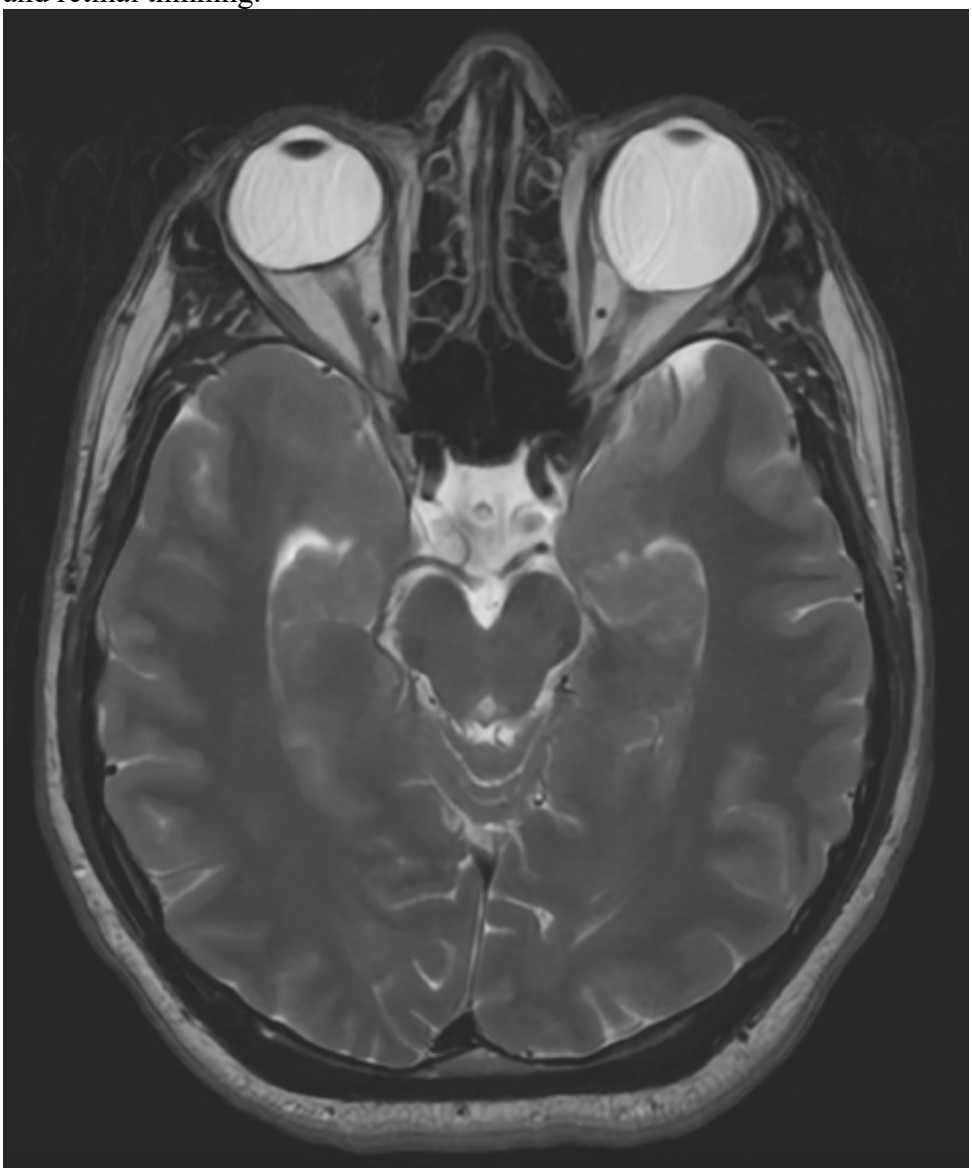

Figure 1. Axial T2-weighted magnetic resonance imaging scan showing significant axial elongation of the left eye (32.05 mm) compared to the right eye (25.61 mm), consistent with high axial myopia.

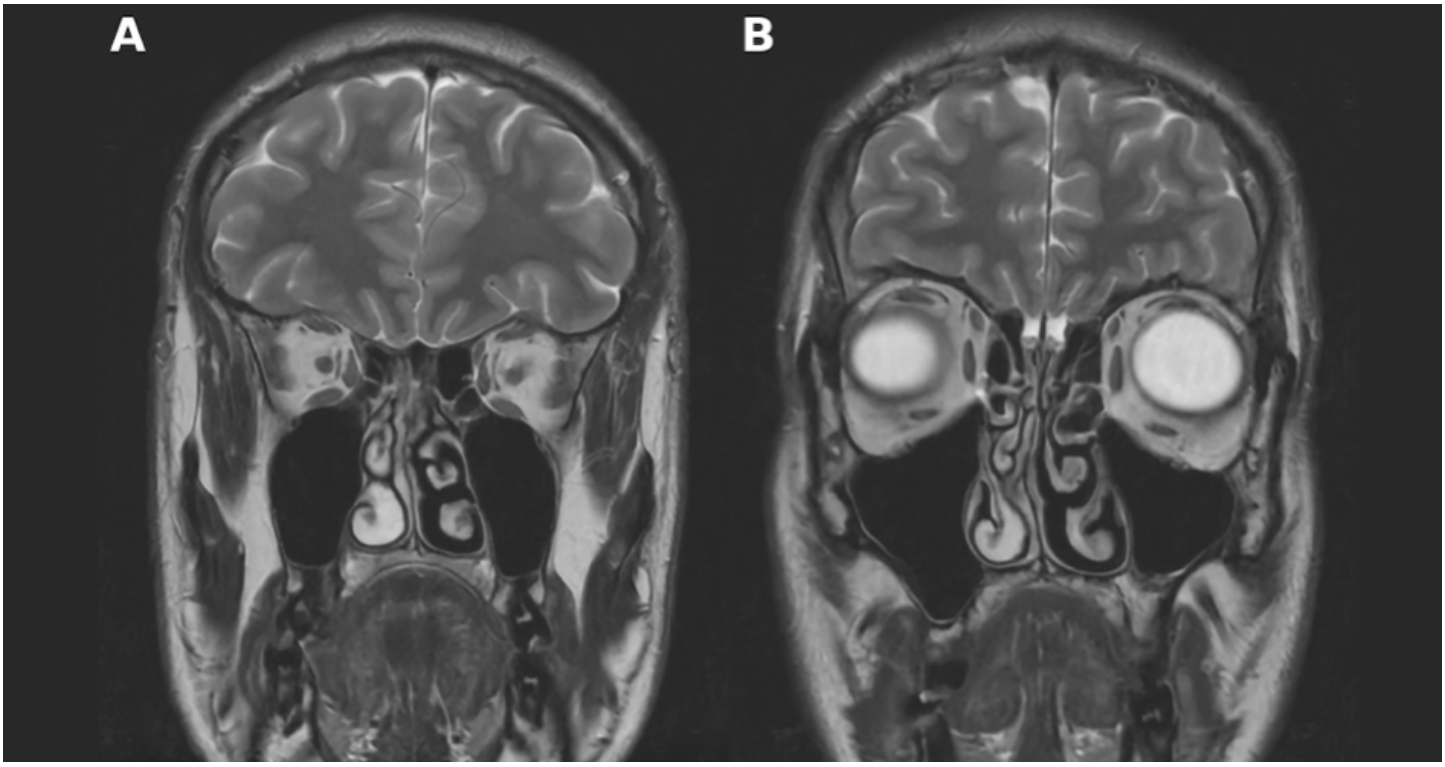

Figure 2. Coronal T2-weighted magnetic resonance imaging scans. A. Normal alignment of extraocular muscles in the unaffected right eye. B. In the affected left eye, nasal displacement of the superior rectus and inferior displacement of the lateral rectus are observed, supporting the diagnosis of Heavy Eye Syndrome.

The patient opted for conservative management consisting of bilateral topical cyclosporine 0.05% twice daily, preservative-free sodium hyaluronate 0.15% eye drops six times daily, and carbomer gel twice daily. Treatment was maintained continuously throughout the follow-up period. Over time, ocular alignment remained stable and visual acuity was preserved (BCVA: 20/25 in right eye, 20/100 in left eye). Corneal topography at the final follow-up demonstrated findings similar to those at baseline, with stable with-the-rule astigmatism in both eyes and no evidence of ectatic progression. Keratometric and pachymetric values, elevation maps, and D indices remained stable. Specular microscopy parameters showed no clinically significant changes (Table 1). Optical coherence tomography of the macula showed no new or progressive macular pathology and remained stable.

At the final follow-up visit, the patient reported marked symptomatic improvement. Although the left eye remained more susceptible than the right, ocular surface disease was clinically controlled. Punctate epithelial erosions were absent. Schirmer I test values improved to 14 mm in the right eye and 7 mm in the left eye, while Schirmer II test values improved to 15 mm and 5 mm, respectively. Tear film break-up time exceeded 15

seconds bilaterally. Nine-gaze photography demonstrated persistent esotropia and hypotropia in the left eye, consistent with the original motility abnormalities (Figure 3).

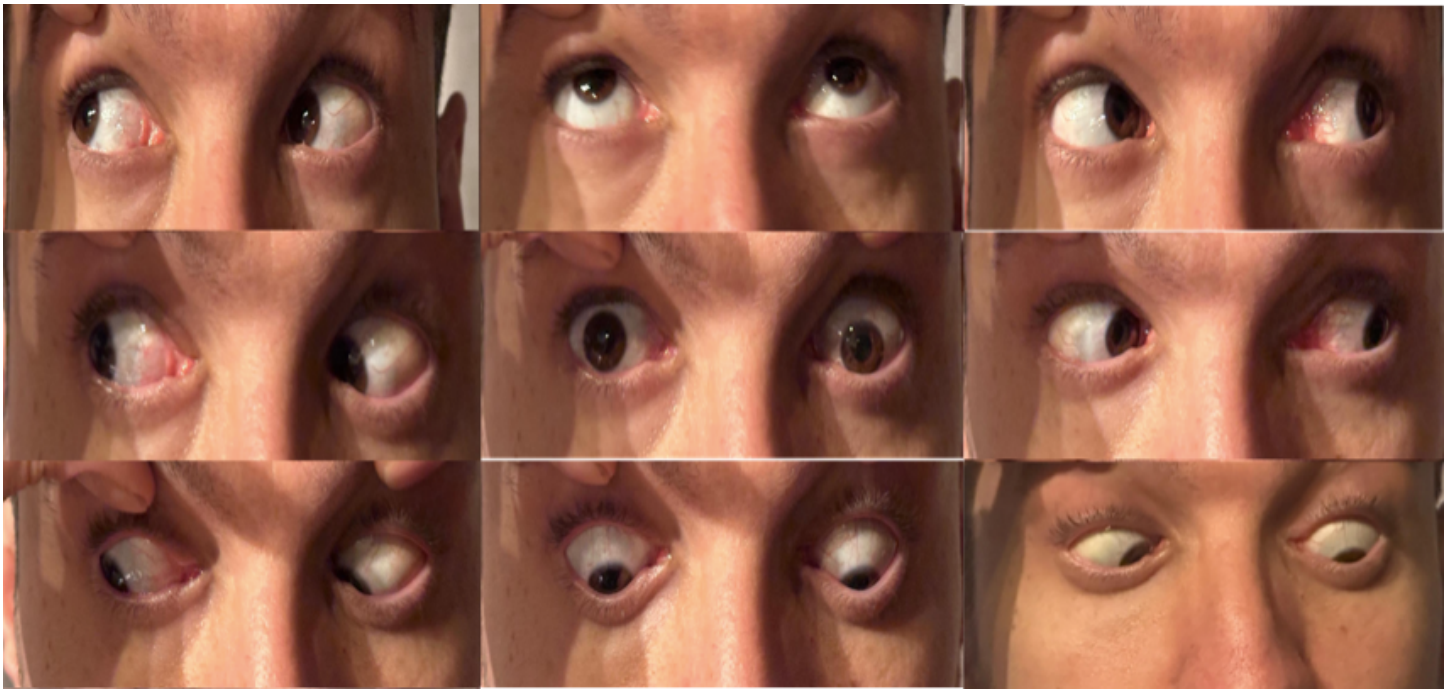

Figure 3. Nine-gaze ocular motility photographs demonstrating persistent left esotropia and hypotropia at the 10-year follow-up visit.

Discussion

Heavy Eye Syndrome is traditionally regarded as a motility disorder resulting from orbital mechanical alterations associated with high axial myopia.[1,2,4,5] However, this case suggests that HES may coexist with significant ocular surface involvement, particularly in the eye with greater axial elongation and globe displacement. Although our patient did not exhibit the profound limitation of ocular motility classically associated with myopic strabismus fixus, the orbital imaging findings and ocular alignment abnormalities were considered consistent with HES.

In this patient, ocular surface disease was clearly asymmetric at presentation and more severe in the left eye, paralleling the degree of axial elongation, ocular misalignment, and extraocular muscle displacement observed on magnetic resonance imaging. Markedly reduced Schirmer and tear break-up time values, together with prominent punctate epithelial erosions, were more pronounced in the affected eye, potentially suggesting a true laterality rather than nonspecific bilateral dry eye disease.

Despite persistent mechanical abnormalities and unchanged ocular alignment over the follow-up period, ocular surface symptoms and objective tear film parameters improved substantially with conservative therapy. Importantly, the substantial improvement in symptoms and ocular surface findings despite persistent esotropia and hypotropia suggests that ocular surface disease itself represented a major contributor to the patient's symptoms. Although both eyes demonstrated improvement, the left eye consistently remained the more vulnerable eye, possibly reflecting the ongoing mechanical predisposition. Axial elongation and associated

orbital anatomical alterations may contribute to a chronic environment favoring tear film instability, while appropriate anti-inflammatory and lubricating therapy can effectively control symptoms.

The inferomedial displacement of the globe observed on imaging may theoretically alter blink dynamics, tear distribution, and eyelid apposition, potentially contributing to ocular surface dysfunction. Similar mechanical factors have previously been implicated in ocular surface dysfunction and tear film instability.[6–8] Such mechanisms may lead to functional tear film impairment without permanent structural damage, as supported by the long-term stability of corneal topography, optical coherence tomography, and specular microscopy findings. The absence of progressive corneal or retinal pathology over a decade further supports a predominantly functional rather than structural process. Although tear film break-up time was reduced bilaterally at presentation, extensive evaluation failed to identify an underlying systemic autoimmune disorder or clinically significant exposure-related abnormality. The relationship between HES and ocular surface disease in this patient should be interpreted cautiously. Although the marked asymmetry of dry eye findings paralleled the degree of axial elongation, globe displacement, and ocular misalignment, a coincidental coexistence cannot be completely excluded. As this is a single case report, definitive causality between orbital mechanical alterations and ocular surface dysfunction cannot be established.

Axial elongation itself may represent an independent contributor to ocular surface instability and exposure-related changes. Hertel exophthalmometry, blink dynamics and Bell's phenomenon were not systematically documented at the initial presentation. However, there was no clinical evidence of lagophthalmos, exposure keratopathy, eyelid malposition, or blink-related abnormalities on examination. In addition, punctate epithelial erosions were not distributed in a pattern suggestive of exposure-related ocular surface disease. Therefore, it remains difficult to separate the effects of globe enlargement from those of HES-associated orbital alterations in a single patient.

The observed displacement of the superior rectus and lateral rectus muscles in the left eye is believed to arise secondary to degeneration or disruption of the superior rectus–lateral rectus (SR–LR) band, which is currently considered the principal anatomical abnormality underlying HES.[1,2]

While this case suggests a potential association between HES and dry eye disease, findings should be interpreted as being hypothesis-generating rather than direct causation. Severe ocular surface disease may be present in an eye with marked axial elongation and orbital anatomical alterations characteristic of HES, but it remains unclear whether HES is directly responsible for ocular surface changes.

Conclusion

In the present case, long-term conservative treatment was associated with symptomatic and objective improvement documented at final follow-up despite persistent ocular misalignment. The presence of ocular surface disease in highly myopic patients with strabismus and axial elongation may be suggestive of HES. Recognition and treatment of pre-existing dry eye disease may be helpful when planning strabismus surgery, as postoperative ocular surface symptoms and discomfort could otherwise be exacerbated. Further studies are needed to clarify the relationship between axial elongation, orbital anatomical alterations, and ocular surface dysfunction.

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Statement of Ethics

The patient gave written consent to publish the data. The report does not include personal information that could identify the patient directly or indirectly. All medical interventions have been carried out according to the latest protocols of therapy. Reporting and writing are all in compliance with the Declaration of Helsinki.

Conflict of Interest Statement

The authors declare no conflicts of interest related to this topic.

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Table 1. Longitudinal comparison of corneal topography and specular microscopy parameters at baseline and at final follow-up.

Parameter	Right Eye Baseline	Right Eye Final Follow-Up	Left Eye Baseline	Left Eye Final Follow-Up
Endothelial Cell Density (cells/mm²)	2,786	2,777	2,890	2,832
Coefficient of Variation (CV, %)	33	32	29	28
Hexagonal Cell % (HEX)	44	43	42	41
Central Corneal Thickness (µm)	575	574	556	554
Number of Cells Analyzed (#)	150	148	152	149
Keratometry (K1 / K2, D)	41.7 / 42.5	41.4 / 43.0	42.0 / 43.2	41.9 / 42.8
Steep Axis (°)	103.4	102.5	68.5	67.8
Thinnest Location (µm, x/y)	538 (−0.49, −0.56)	540 (−0.45, −0.60)	550 (+0.60, −0.13)	552 (+0.55, −0.10)
Front Elevation @ Thinnest (µm)	4	4	5	5
Back Elevation @ Thinnest (µm)	5	5	5	5
D Value	1.63	1.61	1.44	1.42