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Sagging Eye Syndrome--an Overlooked Diagnosis

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Abstract

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Background: Sagging Eye Syndrome (SES) is a relatively unknown cause for binocular distance diplopia. SES presents with an acquired comitant or non-comitant small angle esotropia and/or hypotropia commonly occurring in the elderly population. Inferior displacement of the lateral rectus secondary to age-related degeneration of orbital connective tissue and extraocular muscles appears to be responsible for the ocular misalignment. SES patients often present with new onset or progressively worsening distance diplopia, typically warranting neuroimaging. However, through understanding SES, eye care practitioners may identify these cases, avoiding unnecessary imaging studies. **Case Report:** A 90-year-old male presented with complaints of intermittent horizontal diplopia at distance despite being prescribed prism for presumed decompensating phoria at his most recent eye exam. The patient noted progressing diplopia which appeared worse in right gaze. Full binocular examination revealed a non-comitant esotropia worse at distance than near. External exam revealed prominent deep superior sulci, blepharoptosis, and orbital fat loss bilaterally. Due to the progressive nature and incomitancy of the diplopia, a neuro-ophthalmology consult was obtained, and the patient was diagnosed with SES. A clinical diagnosis was made based on history, adnexal features, and motility patterns distinct to SES, obviating the need for confirmatory imaging. **Conclusion:** Patients presenting with new onset diplopia secondary to undiagnosed SES may prompt expensive and time-consuming investigations. It is critical that eye care practitioners accurately recognize the signs, symptoms, and clinical features of SES to avoid unwarranted imaging and patient anxiety. This case report reviews the clinical presentation, exam findings, and distinct picture of SES required for diagnosis and necessary to differentiate this condition from more serious neurologic conditions. Treatment and management will be discussed.

Note: No identifiable health information was included in this case report. Written informed consent was obtained for patient images.

Keywords

Sagging Eye Syndrome, acquired diplopia, non-comitant esotropia, strabismus, extraocular muscles

INTRODUCTION

Sagging Eye Syndrome (SES) is an often overlooked cause for acquired distance diplopia, chronic or acute, most commonly occurring in the elderly population.[1] Sagging Eye Syndrome was first described as a new underlying etiology for divergence insufficiency esotropia in adults and identified as a clinically distinct entity from Heavy Eye Syndrome (HES) in 2009 by Rutar and Demer.[1] Sagging Eye Syndrome, which is characterized by an esotropia and/or hypotropia in a non-myopic, elderly patient, was proposed to contrast from HES, a condition with a clinically similar presentation, but associated with high axial myopia.[2]

Both SES and HES are defined by a clinical strabismus that closely correlates to the structural and anatomical changes seen within the orbit as evidenced by radiological imaging.[3] Despite both SES and HES having a similar pathogenesis, which is described by a stretching of connective tissue and displacement of orbital muscles, the anatomical etiology for the structural changes seems to be different.[2] In the case of HES, high axial myopia causes a superotemporal displacement of the enlarged globe resulting in a stretching of the intermuscular connective tissue band between the lateral rectus and superior rectus. This causes the lateral rectus to shift inferiorly and the superior rectus to shift medially. Additionally, the lateral rectus is typically seen on imaging to be tightly apposed to the myopic globe.[2] In SES, age-related degenerative changes are responsible for weakening the lateral rectus-superior rectus intermuscular support band, causing the lateral rectus to be displaced inferiorly by gravity.[3] Other characteristic radiologic findings of SES include a bowing and elongation of the rectus muscles away from the orbital center with centrifugal displacement and migration of orbital fat toward the globe.[2,3] Although all recti extraocular muscles are affected by age-related degeneration of orbital connective tissue, severe stretching of the lateral rectus-superior rectus band is responsible for the clinical presentation of SES.[3]

In SES, as the magnitude of the phoria/tropia increases over time with progressive deterioration and lateral rectus displacement, fusional divergence ranges are unable to adequately compensate, and diplopia typically ensues. Most often, patients with SES are elderly and present with complaints of acute-onset or progressively worsening intermittent distance diplopia without neurological symptoms.[4] Diplopia may worsen in both lateral gazes and is usually not present at near.[4] A clinical examination will often reveal a non-comitant small angle esotropia and/or hypotropia, with motility patterns showing limited supraduction but normal horizontal saccades.[3] Patients with SES show obvious adnexal changes recognizable on external examination and include a deepening of the superior sulcus, blepharoptosis, eyelid laxity, and high upper eyelid creases.[1] These

characteristic external features and motility patterns seen on examination are typically enough for diagnosis, and will usually aid in differentiating SES from other etiologies. Sagging Eye Syndrome is considered a clinical diagnosis and, in most cases, will not require confirmatory neuroimaging or further investigations.

Prior to the emergence of SES, acute or progressively worsening diplopia in the elderly population would, in many cases, warrant immediate lab and/or neuroimaging studies to rule out more ominous etiologies. As more SES cases have been reported, common clinical features have allowed eye care providers to more easily recognize and diagnose this condition. Clinical presentation, along with a sound binocular examination, can effectively rule out etiologies requiring neurological work-up and imaging, allowing optometrists to make a confident diagnosis. This case report will review clinical findings, work-up, and management for SES. Emphasis will be placed on the clinical presentation, examination findings, and distinct features of SES required for diagnosis and necessary to differentiate this condition from other differentials, including neurologic conditions.

CASE REPORT

A 90-year-old white male was referred by the Primary Care Optometry Clinic to the Acquired Brain Injury Eye Clinic at the Connecticut VA Healthcare System to address a recent worsening of diplopia. The patient presented with progressive intermittent distance diplopia that was worse in right gaze. Specifically, the patient most often noticed his diplopia while watching television or driving, and described an increase in diplopia when looking towards the right-side view mirror. He noted that the diplopia started approximately 20 months prior. He also reported that he had been prescribed prism glasses to address the diplopia at his last eye examination, 2 months after the diplopia began. The patient further admitted that although the new prism prescription helped slightly with the diplopia, it did not completely resolve it. He denied diplopia at near but admitted he spent very little time performing near tasks. Additionally, the patient denied any loss of vision, eye pain, fatigability, headache, facial numbness, dyspnea, dysphoria, dysphagia, abnormal gait, scalp tenderness, jaw claudication, fever, or any other neurological symptoms.

Incoming ocular history was notable for a left esotropia, pseudophakia of both eyes, posterior capsular opacification (right eye greater than left eye), and bilateral dry eyes. At his last eye examination, his left esotropia had been attributed to decompensation for which he was prescribed 10 prism diopters base out split between the two eyes for constant wear. At that examination, cover test at distance showed 18 prism diopters of left esotropia. The patient denied history of trauma, childhood strabismus, patching and/or vision therapy. Besides cataract extraction

in both eyes, the patient denied any other ocular surgeries. His medical history was remarkable for trigeminal neuralgia, spinal stenosis, hyperlipidemia, diabetes mellitus, chronic atrial fibrillation, and bladder cancer. His medications at the time of the exam were gabapentin 100 milligrams once per day, apixaban 2.5 milligrams twice per day, simvastatin 20 milligrams once per day, cyanocobalamin 1000 micrograms once per day, and a multivitamin tablet once per day.

Best-corrected visual acuities with his habitual prism prescription were 20/20- right eye with +0.75-1.75 x 085 and 5 prism diopters base out, and 20/25- left eye with +2.50-2.50 x 103 and 5 prism diopters base out. Pupils were equally round and reactive to light with no afferent pupillary defect. Extraocular motilities were full and unrestricted in all gazes with both eyes. Confrontation visual fields were full to finger counting in both eyes. Saccades and smooth pursuits were full and accurate in all directions with both eyes. Monocular ductions were full in the right and left eyes. Near point of convergence testing was within normal limits. The patient was not noted to have a habituated head turn or tilt. Distance cover test with his habitual prescription revealed 20 prism diopters of intermittent left esotropia, although the patient manifested the left esotropia approximately 90-95% of the examination duration. Near cover test with the habitual prescription revealed 5 prism diopters of intermittent left esotropia. Ocular alignment at distance was evaluated in 9 cardinal positions of gaze using a Maddox Rod with the patient's habitual distance prescription and without prism **(Table 1)**. Maddox Rod testing revealed a noncomitant esodeviation at distance that was worse in right gaze and down gaze. Additionally, a mild comitant right hyperdeviation was seen in all positions of gaze.

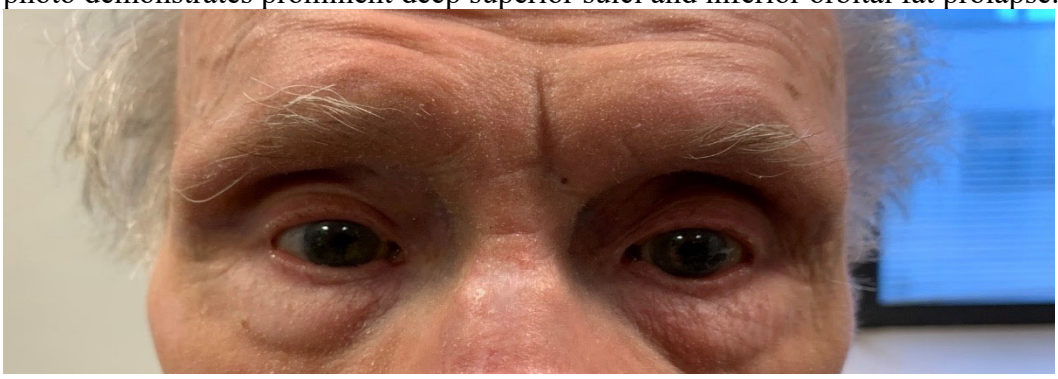
Table 1. 9-Position Maddox Rod at Distance

Right Gaze		Left Gaze
20 Esotropia 0.5-1.0 Right hypertropia	14 Esotropia 0.5-1.0 Right hypertropia	12 Esotropia 0.5-1.0 Right hypertropia
20-25 Esotropia 0.5-1.0 Right hypertropia	20 Esotropia 0.5-1.0 Right hypertropia	16 Esotropia 0.5-1.0 Right hypertropia
20 Esotropia 0.5-1.0 Right hypertropia	30-35 Esotropia 0.5-1.0 Right hypertropia	20 Esotropia 0.5-1.0 Right hypertropia

Distance vergence ranges were measured with the patient's distance prescription and without prism. Vergence range testing revealed base-in ranges of x/4/2 and base out ranges of x/8/6.

Anterior segment examination was significant for superior orbital fat atrophy (left eye greater than right eye), inferior orbital fat prolapse in both eyes, blepharoptosis in both eyes, and deep superior sulci left eye (greater than right eye) **(Figure 1)**. No proptosis, lid fatigability, or muscle weakness was observed. Margin-reflex distance measurements showed 3.5 millimeters/6 millimeters right eye and 3 millimeters/6 millimeters left eye. Conjunctiva, cornea, iris, anterior chamber, and angle findings were within normal limits in both eyes. Tonometry was 8 mm Hg in the right eye and 10 mm Hg in the left eye. Dilated examination revealed posterior chamber intraocular lenses in both eyes with posterior capsular opacification (greater in the right eye than the left eye), posterior vitreous detachments in both eyes, cup-to-disc ratios of .50 right eye and .45 left eye with healthy rim tissue in both eyes, 360 degrees of peripapillary atrophy (left eye greater than right eye), a few small hard drusen in the macula of both eyes, and reticular degeneration peripherally in both eyes.

Figure 1. External photo of patient.
Note: Patient raising eyebrows, thus blepharoptosis not manifested. However, photo demonstrates prominent deep superior sulci and inferior orbital fat prolapse.



Prism correction was evaluated over the patient's best corrected distance prescription in a trial frame. With 1 prism diopter base down right eye and 18 prism diopters base out, the patient reported single, comfortable vision. Updated distance spectacles were ordered as follows: +0.75-1.75 x 085 with 9 prism diopters base out and 1 prism diopter base down right eye, and +2.50-2.50 x 103 with 9 prism diopters base out left eye.

Based upon examination findings, the patient was diagnosed with divergence insufficiency. With the lack of neurological symptoms and a 2-year history of diplopia, emergent referral for imaging was not warranted. However, due to the incomitant esotropia and more recent progression of diplopia, it was determined to be medically prudent to obtain a neuro-ophthalmology consult. Neuro-ophthalmology diagnosed the patient with Sagging Eye Syndrome due to history, adnexal appearance, and clinical exam findings. The consulting provider agreed with our treatment plan of observation, and recommended against further neurological imaging at that time. The patient returned for follow-up 3 months later reporting single vision at distance and toward right gaze with his most recent spectacles. Furthermore, he denied any instances of diplopia or new onset symptoms. All examination findings remained stable.

DISCUSSION

In 2009, Rutar and Demer proposed a new clinical entity they referred to as Sagging Eye Syndrome, differentiating it from Heavy Eye Syndrome, a well described etiology for acquired strabismus found in high axial myopes.[1] While this alternative diagnosis shared similar clinical features and radiologic findings with HES, it was distinguished by its predisposition for the elderly and absence of axial myopia. Moving forward, this condition was primarily referred to as Sagging Eye Syndrome, although it has also been referenced in the literature as age-related distance esotropia,[5,6] divergence insufficiency esotropia,[7] and adult-onset chronic divergence insufficiency esotropia.[8] Sagging Eye Syndrome has been further characterized by the following clinical features: distance esotropia and/or hypotropia, average axial length, and extraocular muscle tissue degeneration causing displacement of the lateral rectus.[1]

The relative incidence of SES is likely underestimated due to the lack of widespread familiarity, causing it to be underdiagnosed. However, one retrospective observational case series involving patients over 40 years of age found the most common cause of diplopia to be SES (31.4%, n=945),[9] while Mittleman discovered SES present in 10.6% of adult strabismus cases over the age of sixty.[5] The average age of incidence reported in both studies was between 74-77 years old.[5,9] However, onset generally begins in the fifth decade or later in life, with a predilection for females.[5,6,8,10] Nevertheless, further investigations are still needed to better define and understand the demographic characteristics and risk factors for SES.

The typical clinical presentation of SES is a mid-70s female patient complaining of gradual or subacute intermittent horizontal diplopia at long distances such as while driving or in theaters.[7] Over time, the diplopia increases in frequency to become more constant and may start to occur with other distance tasks,

such as watching television. This progression in symptoms results from the inability of fusional divergence ranges to adequately compensate for the advancing binocular deviation. Patients usually deny diplopia at near,[7] but may report that the double vision is worse in both right and left lateral gazes.[4] Additionally, patients may have a prior history of blepharoplasty or similar cosmetic procedure to correct eyelid appearance.[3] Of note, patients with SES should not present with associated systemic, ocular, or neurological symptoms such as pain, headache, loss of vision, and other symptoms suggestive of serious underlying pathology.[3,4,11]

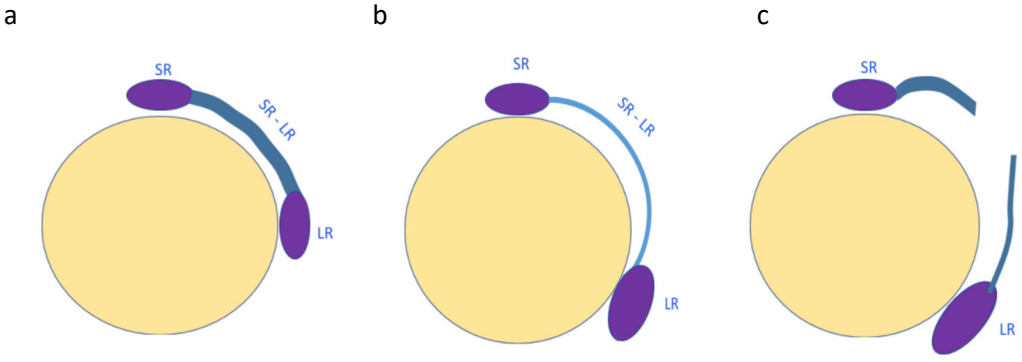
Sagging Eye Syndrome patients can be clinically identified by their distinct adnexal features in combination with specific examination findings **(Table 2)**. External examination will reveal characteristic findings of deep superior sulci, elevated upper eyelid creases, and blepharoptosis, all of which can be explained by age-related levator aponeurotic dehiscence.[11] Chaudhuri and Demer found 64% of SES patients had superior sulcus deformity, while 29% demonstrated elevated upper eyelid creases and blepharoptosis.[3] Additionally, generalized involutional orbital fat atrophy and inferior orbital fat prolapse are often observed. These prominent and distinct SES external features were demonstrated in this case report.

Table 2. Clinical findings of Sagging Eye Syndrome (SES)

Testing	**Sagging Eye Syndrome**
External/Slit lamp exam	Deep superior sulcus Elevated upper lid crease Blepharoptosis Orbital fat atrophy Inferior orbital fat prolapse
Extraocular muscles	Full or limited supraduction
Pupils	Pupils equal round and reactive to light without afferent pupillary defect
Alignment	Comitant or non-comitant esotropia and/or hypotropia at distance greater than near
Saccades	Accurate with normal velocity
Vergence	Reduced base in vergences at both distance and near, more so reduced at distance, with normal convergence

Age-related degenerative changes of the orbital connective tissue have been postulated to explain the clinical features seen in SES, including aponeurotic blepharoptosis and mechanical strabismus. Several studies have investigated the anatomical changes brought about by SES using magnetic resonance imaging.[2,3] These studies have concluded that SES results from a progressive elongation of the lateral rectus-superior rectus band, a ligament interconnecting the pulleys of the two extraocular muscles.[1,3,10] Moreover, Chaudhuri and Demer noted that although a 50% lengthening of an intact lateral rectus-superior rectus band was observed in non-strabismic patients, SES patients demonstrated a significant displacement of the lateral rectus pulley when compared to normal age-matched subjects.[3,4] It is the thinning, and ultimately rupture of this lateral rectus-superior rectus band that leads to the significant inferior sag of the lateral rectus pulley, resulting in the inferior displacement responsible for causing the esotropia and/or hypotropia clinically observed **(Figure 2)**. The presence and degree of the horizontal and/or vertical strabismus is likely related to whether the lateral rectus displacement is symmetrical or asymmetrical. A symmetrically inferior displacement of the lateral rectus between the two eyes will typically manifest purely as an esotropia.[3] In contrast, a cyclovertical strabismus, occurring either in isolation or in combination with an esotropia, is likely the result of a bilateral asymmetric inferior sag of the lateral rectus pulley.[3] Moreover, the eye that exhibits the greater amount of lateral rectus inferior displacement will likely demonstrate a hypotropia and excyclotropia compared to the fellow hypertropic eye.[3]

Figure 2. (a) Normal lateral rectus-superior rectus band and lateral rectus placement. (b) Thinned and lengthened lateral rectus-superior rectus band and inferior lateral rectus sag. (c) Ruptured lateral rectus-superior rectus band and inferior lateral rectus sag



Ocular alignment and motility examination for SES patients will usually show an esotropia and/or hypotropia. Affected patients may present with either one of these deviations in isolation, or demonstrate a combination of the two, as shown in this case report.[3] Sagging Eye Syndrome patients are more commonly found to have esotropia in the range of 10-20 prism diopters at distance.[12] At near, patients may demonstrate a significantly smaller magnitude of esotropia, esophoria, or orthophoria.[12] Additionally, the binocular deviation may be worse in lateral or downgaze, as observed in this particular case.[4,8] Examination may reveal inadequate compensating base in vergence ranges at distance with a sparing of fusional convergence.[7,13] The patient should demonstrate normal horizontal saccadic velocities.[3] Ductions will essentially be full despite the presence of an esotropia. However, some patients may show symmetrically limited supraduction with duction or version testing.[3] Lastly, SES patients displaying a hypotropia will often show excyclotorsion of the hypotropic eye.[3] The aforementioned clinical findings are important in establishing a diagnosis of SES and to aid in differentiating it from more concerning conditions.

Sagging Eye Syndrome is a clinical diagnosis made with positive findings, not a diagnosis of exclusion. This requires a thorough history and identification of distinct adnexal features and motility patterns on examination, similar to divergence insufficiency **(Table 2)**. As with any new onset diplopia in an older patient, it is critical to complete a comprehensive history and examination to exclude neurologic disorders. Diplopia presenting with clinical signs and symptoms of a divergence insufficiency can have benign etiologies, such as SES or HES, but can also present with neurologic disease. A thorough neuro-ophthalmic history is essential to rule out more concerning diagnoses and should include questions investigating systemic and neurologic symptoms **(Table 3)**. A history suggestive of a systemic or neurologic cause for divergence insufficiency and diplopia would not be consistent with SES and would warrant further investigation.

Table 3. Exam findings indicating possible emergent neurologic etiology for diplopia.[14]

Exam Component	Positive Findings
History	Headache, eye pain, vision loss, trauma, speech difficulty, dysphagia, shortness of breath, fever, unexplained weight loss, temporal scalp pain, jaw claudication, oscillopsia, muscle weakness, imbalance, gait abnormality, pulse synchronous tinnitus
Entrance testing	Decreased vision, afferent pupillary defect, anisocoria, non-reactive pupil, visual field deficit
Binocular testing	Motility restriction, nystagmus, multiple extraocular muscle/cranial nerve involvement
Ocular health exam	Ptosis, proptosis, optic nerve head edema

Both SES and the clinical presentation of divergence insufficiency may be mimicked by fourth and sixth nerve palsies, in addition to cerebellar disease, myasthenia gravis, thyroid eye disease, giant cell arteritis, and HES. However, these etiologies will not typically present with both the hallmark appearance and motility findings seen with SES. Significant limitations of gazes and slow saccades are distinct for paretic or paralytic strabismus. Unilateral or bilateral sixth nerve palsies, especially subtle bilateral sixth nerve palsies, can clinically mimic the presentation of SES.[15] Motility examination in neurological strabismus, such as a sixth nerve palsy, is differentiated from SES by showing restricted abduction of the affected eye(s) along with slow and inaccurate saccades.[16] Furthermore, the presence of papilledema, endpoint nystagmus, associated neurological symptoms, and acute onset are important features characteristic for bilateral sixth nerve palsies.[13,15] In this case report, the patient demonstrated full motilities in each eye, normal saccades, and lacked neurological symptoms consistent with sixth nerve

palsies. Sagging Eye Syndrome cases that exhibit only a vertical deviation may present in a similar fashion to a fourth nerve palsy. However, patients with a fourth nerve palsy will demonstrate excyclotorsion of the hypertropic eye,[17] as opposed to SES patients, who will exhibit excyclotorsion of the hypotropic eye.[3] Cerebellar disease can present with divergence insufficiency findings, although it is often associated with gaze evoked nystagmus and saccadic dysmetria.[18] Myasthenia gravis, thyroid eye disease, and giant cell arteritis may present with similar diplopic complaints to SES, though these will typically be accompanied by neurologic symptoms and demonstrate a wide variety of non-specific motility patterns.[19--21] All things considered, any atypical presentation or a presentation not entirely consistent with SES **(Table 2)** should prompt further investigation with a neuro-ophthalmology consult and/or neuroimaging.

The case described above was compatible with the expected clinical picture of Sagging Eye Syndrome. The patient displayed the classic adnexal features of SES described in Table 2 and demonstrated full and unrestricted ductions as well as full horizontal saccades with normal saccadic velocities on motility testing. However, he did have a larger than average esotropia at distance compared to the typical magnitude documented for SES in the literature. Additionally, although the patient did exhibit lateral and down gaze incomitance patterns with Maddox Rod testing, which is not unusual for SES patients, the esodeviation was worse in right gaze and significantly worse in downgaze, slightly atypical for SES. Due to some of these inconsistent features and the concern that SES can co-exist with a paralytic strabismus, the authors felt it was medically prudent to consult with neuro-ophthalmology. However, in hindsight, the patient's clinical examination revealed no abduction deficits, an absence of neurological symptoms, normal saccades, and classic adnexal features distinct for SES which were enough to confirm a diagnosis of Sagging Eye Syndrome.

The diagnosis that can more easily be confused with SES is HES due to the similar clinical presentation. Although HES has a similar motility pattern to SES, HES patients will have high axial myopia, are typically younger, are less likely to exhibit ptosis, and may have abduction motility deficits in addition to deficits of supraduction.[1] Differentiation of SES and HES can be more clinically challenging in the older population where both high myopia and involutional changes may coexist. However, discerning between these two diagnoses will not impact clinical management unless surgical intervention is warranted. In such cases, imaging will be required to confirm SES or HES diagnoses as the surgical technique will vary between the two conditions.[3]

Since patients with SES typically present with complaints of diplopia, the goal of treatment is to provide clear and single vision. This may be accomplished with the use of compensatory prisms or strabismus surgery.[4] Studies have shown that prism adaptation, ("eating prism"), in the elderly population is reduced

compared to younger populations, making prisms a viable option for treatment of binocular dysfunctions such as SES.[22] Due to the inherent risks of surgical procedures, prism correction is the preferred first-line treatment method for SES.[4] If prism correction cannot be tolerated, or the angle of the strabismus is large, patients can undergo surgical correction, specifically lateral rectus resection or medial rectus recession.[6-8,10] Both surgical techniques have been found to have equivalent efficacy, resulting in no postoperative diplopia or convergence insufficiency, although larger amounts of medial rectus recession are required compared to lateral rectus resection when treating SES.[10] In one study of 24 patients, both methods of surgical correction were compared, resulting in no cases demonstrating symptomatic diplopia or convergence insufficiency over the course of 8.5 to 40 months post-surgery.[10] Importantly, patients electing to have surgery require orbital imaging to determine the anatomic changes present. Since SES and HES can coexist and present with similar symptoms, pre-surgical high-resolution fast spin-echo T2-weighted coronal magnetic resonance imaging of the orbit is crucial to determine if lateral rectus sag is accompanied by normal globe placement indicating SES.[3] Comparatively, HES imaging will show superotemporal prolapse of the globe with the lateral rectus tightly apposed.[2,23] Surgical intervention is more often used to treat HES than SES. However, new surgical techniques, such as lateral rectus equatorial myopexy, are under investigation and may hold promise as future therapies.[8]

CONCLUSION

Sagging Eye Syndrome is a relatively unknown ocular condition associated with binocular distance diplopia. Sagging Eye Syndrome manifests in the elderly population as a comitant or non-comitant small angle esotropia and/or hypotropia and can prompt unnecessarily expensive and time-consuming investigations. However, SES is a clinical diagnosis that in most cases will not require confirmatory neuroimaging. With a thorough patient history and comprehensive clinical examination including binocular evaluation and motility testing, an astute clinician can recognize the distinct features of SES and rule out emergent etiologies of diplopia. Identification of SES as a mechanical cause for acquired adult horizontal and vertical strabismus has significant clinical implications. Recognizing SES is significant to the patient for avoiding unnecessary neuroimaging and emergent neurological consultation which can spare the financial and emotional burden that accompanies extensive neurological investigation.

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