



REVIEW

Topical timolol in the treatment of monocular oscillopsia secondary to superior oblique myokymia: a review

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KEYWORDS

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Abstract Some reports have outlined many different treatment strategies for superior oblique myokymia (SOM) that attempt to reduce or eliminate patients' symptoms of monocular oscillopsia and/or diplopia. Most treatment strategies have focused solely on oral medications or invasive surgery. The following is a current and critical review of SOM along with its clinical findings/symptoms, demographics, theories of its pathogenesis, management/treatment options, new observations in SOM, as well as a case report to highlight a relatively novel idea for the treatment of SOM: topical timolol eye drops. It also highlights evidence of a new "localized theory" regarding topical β -blockers' mechanism of action in SOM compared to the previous systemic hypothesis proposed in 1994.

The case report shows a 29-year-old female patient who suffered with SOM symptoms for 8–10 years and then experienced a worsening of her symptoms shortly postpartum. The patient was prescribed topical timolol eye drops by the author in the affected eye based on one case report¹² from 1994, which completely eliminated her symptoms within 1–2 days of starting the treatment with any recurrence with the daily use of the drop at the time of this article. Given the robust effect in this case, topical timolol may be a potentially useful drug in the management of SOM given its affordability and safer side effect profile in comparison to the oral medications traditionally used in SOM.

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PALABRAS CLAVE

Mioquimia del oblicuo superior;
Oscilopsia;
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Nervio troclear;

Tratamiento de la oscilopsia monocular secundaria a la mioquimia del oblicuo superior: revisión y uso potencial de Timolol tópico

Resumen Los estudios reportados de casos de mioquimia del oblicuo superior (MOS) han puesto de manifiesto diferentes estrategias de tratamiento que tratan de reducir o eliminar los síntomas asociados de oscilopsia monocular y/o diplopía. Muchas estrategias de tratamiento se han centrado únicamente en medicaciones orales o procedimientos quirúrgicos invasivos. El presente artículo es una revisión crítica de la MOS en relación a sus hallazgos/síntomas

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Mioquimia del IV par craneal;
Mioquimia troclear

clínicos, datos demográficos, teorías de su patogénesis, opciones de manejo/tratamiento, y nuevas observaciones en cuanto a MOS, incluyendo la descripción de un caso que muestra una idea relativamente nueva de tratamiento del MOS: gotas oculares de Timolol tópico. También subraya la evidencia de una nueva "teoría localizada" referente al mecanismo de acción de los β -bloqueantes tópicos en la MOS, en comparación a la hipótesis sistémica previa propuesta en 1994.

El caso reportado hace referencia a una paciente de 29 años que padeció los síntomas de MOS durante 8-10 años, y experimentó un empeoramiento de dichos síntomas poco después del periodo post-parto. El autor prescribió a la paciente gotas de Timolol tópico en el ojo afectado, basándose en el reporte de un caso¹² de 1994, lo que eliminó completamente los síntomas a los 1-2 días de inicio del tratamiento, sin existir a fecha de este artículo ninguna recurrencia con el uso diario de esta medicación. Dado el sólido efecto de este caso, el Timolol tópico puede constituir un medicamento potencialmente útil para el tratamiento de la MOS, dada su asequibilidad y el perfil de seguridad de los efectos colaterales en comparación a las medicaciones orales tradicionalmente utilizadas en la MOS.

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Introduction

Superior oblique myokymia (SOM) is probably best described as an intermittent spontaneous rhythmic contraction of the superior oblique muscle presenting as rapid and small amplitude intorsions and depressions of one eye. SOM was first described by Duane in 1906 as a "unilateral rotary nystagmus"¹⁻⁷; however in 1970, Hoyt et al. coined the term "superior oblique myokymia" which is used most commonly today to describe this interesting condition.^{2,3} Another name often given to this condition, and probably considered more accurate, is "intermittent uniocular microtremor".^{4,6} Since 1970, there have been a moderate amount of articles, case reports, and studies performed to gain insight into how best to diagnose, treat, and manage this condition; however, the overall numbers of patients successfully treated with topical β -blockers/antagonists is very low overall in the ophthalmic literature.

Symptoms of SOM typically include subjective visual disturbances such as spontaneous monocular diplopia, monocular oscillopsia, and quivering/jumping of the involved eye.^{2-9,13,14,22,23} One of its main differentiating points is its monocular nature.^{3-5,13,20,22,23,25} Signs usual involve a higher frequency (12–15 Hz), lower amplitude (3° – 6°), unilateral, intermittent cyclic, intorsion and/or depression of the involved eye which can be seen upon examination by the observer with mild to moderate magnification at the slit lamp.^{2-4,9,15,21-23} Occasionally, this cyclic intorting movement can also be seen during fundoscopic/ophthalmoscopic examination.^{2,21} The fast phase occurs during intorsion and in many cases the SOM can be increased by having the patient look in the direction of the superior oblique (down and in) which is most troublesome when a person is reading, driving, or with other visually demanding work.^{3,4,6,20-23} Many times a patient will know the exact maneuver that will trigger an attack of the ocular symptoms.⁴ These intermittent, recurrent episodes typically occur multiple times on a daily basis.^{5,6,13,22} Duration of symptoms vary dramatically from a few seconds to minutes; however, the majority of attacks seem to last 3–15 s.^{4-6,13,22,26} In some unfortunate cases, SOM may increase in duration until the symptoms are present

indefinitely, although this seems to be extremely uncommon.⁴ Eye movement recordings during these attacks have shown small vertical and torsional movements of the superior oblique muscle which are confined solely to the involved superior oblique muscle during each attack.^{2,3,13,20,23} Occasionally, an ipsilateral hyperphoria/tropia is present, which can be elicited via the alternating and unilateral cover tests, indicating an underaction of the superior oblique muscle,⁴ which can sometimes, but not always, be treated with a prismatic correction in the patient's habitual prescription using base down prism equal to the magnitude of the deviation over the hyper-deviated eye.^{8,22,23}

SOM appears to be independent of age as cases of teenagers through older patients in excess of 60 years of age have been reported.^{2,3} Females are reportedly about 2–3 times as likely as males to suffer from SOM.^{11,13,25} The average age of onset is estimated to be 34–37 years old.^{22,25} There appears to be a higher chance of involvement of the right superior oblique in comparison to the left; however, the underlying reason for this is currently unknown.²¹ Reported trigger factors and associations are numerous. Stress (mental or physical), fatigue, alcohol, caffeine, nicotine, and fluorescent lighting have all been reported as trigger factors.^{13,22,23,26,28} Of note, some reports outline that some patients have members of their families with a diagnosis of multiple sclerosis which raises the question of a hereditary nature or a relationship to multiple sclerosis, although this association is also unclear and only speculative at this point.^{2,3} A history of head trauma has been reported to be associated with SOM in many cases, although trauma is not a requirement for SOM.^{6,7,11,13,14,19,23,25} In those cases of SOM with a history of head trauma, the symptoms do not always start immediately after trauma but may occur many years later.^{6,7,11,13,14,19,23,25}

Correct diagnosis is most important in these patients as most of their symptoms, although troubling, can be difficult to detect during a routine eye exam because of their sporadic and intermittent nature.¹⁵ In most cases, a thorough case history and attentive listening to the patients' self-described symptoms can alert the medical professional and help to narrow down the differential diagnoses to the

possibility of SOM.^{13,16} As the diagnosis is mainly clinical in nature, attentive anterior segment examination by the eye professional is paramount as the unilateral, intermittent, low amplitude, high frequency cyclic, intorsion/rotary ocular movements are best recognized by focusing on the superior nasal or nasal conjunctival blood vessels during the slit lamp examination.^{2-4,13,15,16} Dynamic viewing is the best way to recognize these movements, as static viewing will not help elicit movements of the eye itself. It is important to differentiate between SOM and the more common, eyelid myokymia. Even with attentive investigation and high suspicion, it can be difficult at best to identify some cases other than with a good case history.¹⁵

Case report

A 29-year-old white female presented for examination with a complaint of monocular, intermittent oscillopsia in her right eye (OD) only, and intermittent vertical and diagonal diplopia. She reports that her symptoms began 8–10 years earlier, but that she had noticed a significant worsening over the previous 10 weeks since she had returned to work from maternity leave. She reported that multiple episodes of these visual disturbances occurred unpredictably on a daily basis. The episodes of oscillopsia seemed to worsen when reading or working on a computer. She noticed that if she tilted her head slightly to the left and/or tilted her chin up so she was looking through inferior gaze, her symptoms seemed to improve most days; however, these maneuvers did not always eliminate/lessen her symptoms. She denied any history of previous or current headaches. She denied having any current or previous systemic issues and denied being on any medications or eye drops altogether. She also denied any history of surgeries and/or trauma to the eyes or head. Family medical and ocular history was positive only for glaucoma in her mother. She denied any history of multiple sclerosis or any other neurological disorders in her family. The patient denied alcohol, tobacco, nor substance abuse. She did report use of caffeine on a daily basis. She also denied having any drug/medical allergies.

Entering Snellen visual acuities were 20/20 in the right eye (OD) and 20/20 in the left eye (OS) without correction. Pupils were equal, round, and reactive to light in both eyes in both light and dark conditions with no afferent pupillary defect in either eye. Extraocular motilities were full range of motion in all diagnostic action fields in both eyes (OU). Confrontation visual fields, color vision, and stereoacuity testing were likewise within normal limits OU. Unilateral and alternating cover testing failed to elicit an ocular misalignment as the patient was orthophoric at distance and near without correction, with no evidence of vertical misalignment upon testing. The remaining external and dilated fundus exams were also within normal limits in each eye. Intraocular pressures measured via Goldman tonometry were 18 mmHg OU.

The patient was asked to return in a few days for a more in-depth examination/workup of her eyes which included: additional cranial nerve testing (V1, V2, V3, and VII), formal threshold Humphrey SITA threshold 24-2 visual field testing, ocular coherence tomography (OCT) of the optic nerves and maculas, fundus photos, vertical/horizontal subjective

Maddox rod testing in all 9 diagnostic action fields, Park's 3-Step tests, vertical fusion amplitudes, blood pressure, and marginal reflect distances (MRD1 and MRD2). All above testing returned completely within normal limits in both eyes.

Fortunately, at this visit under the biomicroscope, there were noticeable intermittent, yet rhythmic intorsions, and subtle depressions of the patient's OD. These intorsions were of low amplitude, yet moderate to high frequency. No such response would be found in the OS, which was consistent with the patient's self-reported case history of monocular oscillopsia of the OD. Lastly, the following blood tests were ordered to help rule out any possible underlying known causes of ocular motility disorders: complete blood count (CBC) with differential, elevated sedimentation rate (ESR), antinuclear antibody (ANA), reactive plasma regain (RPR), thyroid panel (TSH, T3, T4), and a myasthenia gravis panel including anti-acetylcholine receptor antibodies. All labwork returned within normal limits except the ANA titer, which was reported as 'positive' by the laboratory with a 1:40 ratio. Subsequently, given the marginally increased ANA titers, double stranded DNA (ds-DNA) titers were also ordered; however, these ds-DNA titers were negative. Given the fact, that ANA titers can be slightly elevated in about 15% of normal individuals, it was assumed the increased ANA was non-specific/non-contributory in this case and may have simply been a variant of normal.³¹

Due to the recent worsening of the patient's symptoms, the patient was referred to our local neurology clinic for further evaluation and neuro-imaging given the possibility of ominous causes such as space occupying lesions, multiple sclerosis, strokes, and arterio-venous anomalies in other reported cases of SOM.^{13,14,16,18,22,29,30} Similar to our examination, the neurologist found no neurological deficits on his exam; however, given the patient's symptoms he agreed that neuro-imaging was required to rule out any possible ominous disorders. Resultant magnetic resonance imaging (MRI) and magnetic resonance angiograms (MRA) of the brain and brainstem returned within normal limits. Therefore, the patient was diagnosed with idiopathic superior oblique myokymia. Given the fact that a few case reports in the literature highlight relief of symptoms in some patients affected by SOM with the use of β -blockers, such as propranolol and/or topical betaxolol,¹² the patient in this case study was prescribed topical timolol ophthalmic solution (0.5%) on a trial basis to be used one drop twice per day in her OD and told to return for follow up in 4–6 weeks. A follow-up phone call four weeks later revealed the patient had 100% relief of her SOM with the simple use of topical timolol 0.5% only once per day in the OD. She reported that after only 1–2 days of use the signs/symptoms of dizziness and oscillopsia completely disappeared and she had no recurrences since starting the timolol drops in her OD. At another follow up phone call two months later the patient reported her symptoms were still completely stable with no recurrences. She was asked to stop the timolol drops in her affected eye (OD) and to start the drops in the opposite eye (OS) to see if any effect could be found. Within two days of attempting to do this, her symptoms returned completely in her OD to pre-treatment levels. She promptly re-started the drops again in the OD and within one day her symptoms disappeared completely again and she has had no further recurrences since. Given her very satisfactory treatment and drastic

improvement in her symptoms she was told to continue the timolol 0.5% drops once per day in her OD and to return for follow up in 6–12 months to monitor her on-going ocular health and SOM symptoms.

Pathogenesis of SOM

Historically, hypotheses regarding the pathogenesis of SOM have been numerous from the time this condition was first reported. Originally, Hoyt et al. found phasic firing of abnormal units in the involved superior oblique muscle without corresponding inhibition in the inferior oblique muscle and therefore hypothesized that pathological changes in the membrane threshold of neurons in the trochlear nucleus itself was a possible explanation indicating that SOM is primarily a trochlear nucleus disorder.^{2–5} A second hypothesis is that tetanic bursts of action potentials may arise spontaneously in trochlear neurons or axons in an area of previous injury/trauma or inflammation.¹³ Along these lines, a third explanation may be that with trauma or inflammation a loss of some or many trochlear axons occurs with subsequent peripheral sprouting of remaining axons (neural aberrant regeneration) and may result in the development of a few large motor units in the superior oblique muscle in an attempt by the trochlear nerve to repair itself.^{6,13} This might result in the loss of fine voluntary motor control of the superior oblique muscle producing a microtremor of the eye. However, this fails to account for the intermittent nature of these abnormal eye movements.⁶

Most recently however, based on careful MRI imaging, the likely and most currently accepted theory regarding the mechanism of action of SOM is vascular compression of the trochlear root exit zone (REZ), usually from branches of the superior cerebellar or posterior cerebral arteries.^{18,21–23} This is based on two case reports of neuro-surgical microvascular decompression surgeries that resulted in complete elimination of SOM symptoms. This is very similar to the vascular compression mechanism of action believed to be caused by other microvascular contact syndromes such as glossopharyngeal neuralgia, hemifacial spasm, or trigeminal neuralgia.^{21–23} Thinly sliced images (1.0–2.0 mm versus 10.0 mm slices in conventional MRI) are best as this gives the investigation team the best-detailed views in order to detect subtle neuro-anatomical problems such as vascular compression of the trochlear nerve at its root exit zone (REZ).¹⁸ If the MRI-slices are too large then subtle vascular compression may be missed on review of the MRI. Vascular compression is defined on neuro-imaging as the absence of any detectable layer of cerebrospinal fluid between the trochlear nerve and an adjacent blood vessel, often a branch of the superior cerebellar or posterior cerebral artery.^{18,21}

Interestingly, a history of head trauma is often, but not always, found in patients with SOM,^{6,7,11,13,14,19,23,25} which raises the question of trauma being an explanation and/or alternate pathway for SOM other than the vascular compression theory. One study looked at the morphological structure of two patients (one with head trauma and the second with trauma iatrogenically induced to the trochlear nerve) with SOM via MRI and found that the affected superior obliques in both patients were statistically smaller in overall size/diameter than normal eyes. Although this study only

had two patients, the authors suggested that trauma/injury results in damage to the trochlear nucleus or its axons lead to possible atrophy of the superior oblique muscle.¹⁴ Then they suggest that resultant aberrant nerve regeneration causes a defective firing/contracting of the involved superior oblique, which may cause the symptoms related to SOM.¹⁴ Although the exact role of head trauma is currently unclear in SOM, it is this author's hypothesis that perhaps vascular compression and trauma are two separate causes of SOM with a final common pathway resulting in SOM symptoms in these patients. Perhaps the traumatic event in some patients actually causes the vascular compression of the REZ leading to SOM. However, it remains unclear at this point how trauma and vascular compression of the trochlear nerve's REZ are related, if at all, and this theory is currently nothing more than an interesting observation and idea by the author.

Also historically, the vast majority of SOM cases have been idiopathic and overall benign in nature. However, there are case reports in the literature caused by more ominous causes, albeit much rarer, such as multiple sclerosis, cerebellar tumors, stroke, arterio-venous malformations, trauma, cerebellopontine arachnoid cysts, adrenoleucodystrophy (hereditary metabolic neurodegenerative disease), fistulas, hydrocephalus, iatrogenic, etc.^{13,14,16,18,22,29,30} Older researchers have advocated foregoing neuro-imaging secondary to the very uncommon yield of finding an underlying pathology;¹³ however, newer researchers suggest neuro-imaging for all new cases of SOM given the possibility of vascular compression and of the other possible ominous underlying neurological causes mentioned above, even though the ominous causes are much rarer.^{21,27}

Management/treatment options of SOM

After ruling out or identifying possible underlying causes mentioned earlier, management typically follows this methodical and logical treatment paradigm: observation, medical management, and surgical intervention.^{13–15,21} For patients who notice symptoms but yet can function adequately in their day-to-day lives, simple observation by the doctor and reassurance for the patient may be all that is required as long as rarer, more ominous causes are ruled out.²³

For patients whose symptoms are more troubling and whose symptoms interfere with their occupation or activities of daily living, medical intervention followed by surgical intervention is indicated.^{3,4} In some cases simple ground-in prism (base down over the hyper-deviated eye) in the patient's habitual spectacle prescription has provided relief of symptoms.⁸ Medical management historically has been almost exclusively the domain of oral drugs. Pharmacologic therapy is directed at diminishing the frequency of pathologic bursts of firing of the trochlear nerve; however, the mechanism of action (MOA) of these drugs in regards to SOM are not yet fully known, although they are believed to simply be an extension of their regular MOA.⁹ The original/prototypical drug used for SOM was carbamazepine,^{3–6,9,15,19} but with its use comes serious potential side effects such as leukopenia, acute renal failure, thromboembolism, and arrhythmias.^{3,4,26} Therefore,

other alternative oral medications have been compared, attempted, and investigated over the past 30+ years. In 2007, Williams et al. retrospectively studied a group of 20 individuals with SOM who were treated with different oral medications to determine which medication was best at relieving patients' symptoms. In their retrospective study, 90% (18 of 20) of SOM patients were started on 200 mg carbamazepine twice per day then titrated up to 200 mg four times per day which resulted in relief for 83% (15 of 20) of the patients.²⁵ Overall, 80% of patients treated medically experienced a positive response to some degree.²⁵ They noted that baclofen did not relieve the symptoms of any patients in the study, which has also been found in several other studies.^{5,25} Propranolol and phenytoin were two other medications attempted but with mixed results at best. Unfortunately, this study did not discuss on the efficacy of topical β -blockers, gabapentin, or memantine as none of the patients in the retrospective study were treated with any of these medications. Of the six patients in this study who discontinued medication secondary to side effects, all of them were on carbamazepine.²⁵

Subsequently, other oral medications have been tried and investigated by other case reports and studies in order to find a way to decrease symptoms and limit side effects at the same time, but some have had varying degrees of success. These oral medications have included: phenytoin, propranolol, baclofen, gabapentin, clonazepam, mirtazapine, and most recently memantine.^{7,10,13,15,19,21-26}

Surgical intervention is reserved only for those cases in which the patient is very symptomatic and topical and/or oral medications have failed to provide improvement/relief for the patient.^{3,4,27,28} Originally, the first surgical treatments attempted were tenotomy (complete severance) of the superior oblique tendon followed by recession (replacement of eye muscle at a different location on the globe designed to weaken the action of the involved muscle) or myectomy (removing a portion of the muscle belly to weaken its action) of the ipsilateral inferior oblique muscle.^{4,9,11,23,27} More recent studies/case reports appear to favor tenectomy (partial severance) of the superior oblique tendon,⁷ followed by myectomy (removing a portion of the muscle belly in order to weaken the muscle) of the ipsilateral inferior oblique muscle.^{11,13,23,27,28} Although this more recent approach of tenectomy followed by myectomy is typically very successful in eliminating oscillopsia, it can induce diplopia in downgaze secondary to iatrogenic trochlear nerve palsy and resultant hypertropia in 35% of patients, but this can usually be fixed with prism correction if needed.²⁸ Although surgery can be curative in some cases, some patients may still suffer with lingering oscillopsia or diplopia even after surgery. In some cases, additional surgery may be needed which may or may not result in elimination of the patient's symptoms.^{7,11} Therefore, a detailed explanation highlighting pros/cons, side effects, and risk of further surgery needs to be done with the patient. Some authors have also advocated the combination of superior oblique myectomy and resection of the trochlea via anterior orbitotomy if tenectomy fails from the first operation.^{9,27} Others have advocated a special hybrid tenectomy approach known as the Harada-Ito Procedure.¹⁵ In the Harada-Ito procedure, the anterior aspect of the superior oblique is recessed nasally to weaken the torsional function while not affecting

the vertical function of the muscle.^{15,23} Still others have also advocated the injection of botulinum toxin in to the superior oblique muscles, but given the possibility of the injection also spreading to other ocular muscles such as the levator and superior rectus, this is not done as a first line treatment.⁹

In cases of structural brain/brainstem lesions or vascular compressions, neurosurgery is also sometimes, but very rarely, indicated to relieve symptoms or prevent possible life-threatening complications depending on the lesion's cause.^{16,17,30} In cases of vascular compression, microvascular decompression surgery (MDS) is performed. In MDS, the trochlear nerve is identified and followed to its root exit zone (REZ). Then, any vein or artery compressing the nerve is dissected free and Teflon pads are placed to prevent any further compression. Only two known patients have undergone this procedure; however, both procedures eliminated the SOM symptoms completely, but induced permanent trochlear nerve palsies in both patients which could be managed with prismatic corrections.^{17,23} The successfulness of this procedure in eliminating the patients' SOM symptoms is strong for the vascular compression theory of SOM. Overall, the bulk of the ophthalmic literature would agree with the viewpoint that invasive craniotomy surgical procedures should be justified only by the presence of intractable and absolutely unbearable symptoms.^{21,23} In the end, the decision for surgery regarding SOM of any kind should be based on the known etiology of the SOM and only after topical and oral medications have failed to yield substantial improvement in the patients' symptoms to the point that the patient cannot function appropriately in their day-to-day lives because of their debilitating symptoms.

Topical timolol as a novel treatment idea in SOM

Interestingly, in 1994, Bibby et al. described resolution of a patient's symptoms from SOM with the use of a topical β -blocker, betaxolol, a well-known glaucoma drug.¹² This was based on case reports of success in some patients with the use of betaxolol's parental/prototypical drug, propranolol, and one other study that reported some improvement with topical β -blockers.^{12,23} The exact mechanism of action regarding topical β -blockers in SOM is currently unknown; however, it is theorized that it is secondary to the weak membrane-stabilizing abilities of this class of drugs.¹² Bibby et al. hypothesized that enough betaxolol may be absorbed systemically through the ocular blood vessels to eliminate the SOM.¹² However, this author hypothesizes that the weak membrane-stabilizing properties of β -blockers do not necessarily work on a systemic level but rather locally on the trochlear nerve endings and superior oblique muscle tissues, which in turn slows down or eliminates the rhythmic contractions of the superior oblique muscle, much like β -blockers/antagonists do to cardiac muscle tissue in patients with hypertension. This "localized theory" is supported by the patient in this particular case study as her symptoms of SOM returned within two days of stopping the drops in the affected eye (OD) but also while using the drops in the same manner in the opposite eye (OS) as she had previously done in the right eye (OD). If the topical drops are, indeed,

absorbed systemically to a concentration high enough to elicit an effect, then switching the instillation of the drops between the two eyes would have, logically, also controlled the patient's symptoms. But in this case report, her symptoms of SOM returned in severity to pre-treatment levels and showed no benefit at all when the timolol was used in the contralateral eye. This would suggest that a localized effect with topical β -blockers at the trochlear nerve endings and/or superior oblique tissues themselves is responsible for the treatment effect, not systemically as Bibby et al. originally hypothesized.

Although there are cases of idiopathic SOM resolving on its own over a period of months to years, this is unlikely to occur in this case report as the patient reported her symptoms had started over 8 years previous to her presentation to our clinic and seemed to be getting worse according to her case history, but stopped 1–2 days after starting her topical timolol eye drops. In addition to this, her symptoms returned after discontinuing the drops in her affected eye and completely stopped again when the drops were re-started in the affected eye. Although the number of cases that report actual improvement with topical β -blockers is sparse at best in the literature, topical β -blocker use is an interesting and relatively novel treatment idea that may lend relief to SOM sufferers as it did in this case report.

Potential new observations in SOM

A notable observation found by the author in the patient of this case report was that her symptoms seemed to worsen postpartum, which raises the question of whether pregnancy and/or hormones could be a trigger factor in SOM. Interestingly, in the author's review of the literature on SOM, other separate case reports over the years have only passively mentioned a return or start of patients' SOM symptoms shortly after becoming pregnant or shortly postpartum, but none ever ventured to say that one may be associated with the other.^{9,10,22} To the author's knowledge, no other studies have mentioned or discussed this possible link with SOM specifically either. If pregnancy is found to be a risk factor or is involved somehow with SOM, this may obviously help explain why females are affected by SOM much more commonly than males. A possible link between SOM and pregnancy/hormones is complete speculation on the part of the author at this time, but it may be an interesting point to investigate with other studies in the future as possible risk factors, associations, or triggers for SOM.

Lastly, trauma has been shown to be associated with SOM as well. Given that men tend to engage in higher risk activities than women, perhaps cases of SOM in men may be more trauma-related, and perhaps cases of SOM in women may be more hormone/pregnancy related, but they may share a final common pathway resulting in the symptoms of SOM. At this time, this is strictly an observation from the literature and is, again, complete speculation at this point on this author's part. Further studies will be necessary to reveal the underlying reason why women are more afflicted than men with SOM and if trauma, hormones, or pregnancy even play a role at all in the pathogenesis of SOM.

Conclusion

This case is interesting and important because it provides another example to the overall low reported numbers of SOM being successfully managed with only topical eye drops, notably a common topical β -blocker, timolol 0.5%. Although this is only one case report, SOM is a rare ocular condition at best. Given the overall rarity of SOM but relatively limited treatment options other than the standard battery of oral medications with the possibility of significant side effects, it would be ideal if a topical medication is available to treat SOM. Although, to the author's knowledge, there has not been any formal study investigating the total efficacy of topical timolol in the management of SOM in comparison to other medications used historically for SOM, it is the author's viewpoint that timolol is a very well understood drug in the eye care community and that it may provide a simple, safe, first-line treatment option in cases of SOM for some patients that should be evaluated in future studies. Admittedly, in this specific case, there was only one patient treated successfully with this drug, therefore the author cannot entirely omit the placebo effect so solid conclusions cannot be extracted about the real effect of this drug despite the drug's rather robust effect for the patient. More in-depth study is needed to conclude with any certainty whether topical timolol would indeed be an upgrade over previously tried oral medications.

In the event of medical management failure, surgery may be considered; however, it does have serious potential risks involved. Surgery should only be done in the event of absolutely unbearable symptoms and only after several topical and/or oral medications have been attempted. This methodical treatment approach (observation, medications, then surgery) allows, at least theoretically, the least risk of side effects from all medications. As most of these oral medications may be out of the comfort zones of most eye-care providers, an appropriate referral to a neurologist or neuro-ophthalmologist familiar with these medications may be necessary at the point of topical β -blocker failure.

In the acute setting, eyecare providers should be able to identify SOM in a timely manner and rule out the rarer, more ominous causes of SOM with neuro-imaging and bloodwork with either a neurologist or the patient's primary care provider. A history of head trauma, even years earlier should be sought by the examiner during the case history as this has a close association with SOM in some patients. Then, the eyecare provider should focus on managing the patient's symptoms with medications if an underlying disorder cannot be found. The patient in this case study has been symptom free for 4 months duration at the time of this paper with the use of daily topical timolol in her affected eye. Given her normal neuro-imaging studies and bloodwork, she will be followed closely and will be referred back to the neurologist for a trial of oral medications if her topical timolol eyedrops fail to provide continued relief of her symptoms.

Conflicts of interest

The author has no financial or any other conflict of interest to report.

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