Invited Review

Management of Myopic Maculopathy: A Review

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Abstract

Myopia, including pathologic myopia, has seen a significant increase in prevalence in recent years. It is a significant cause of irreversible vision loss worldwide and prediction models demonstrate the substantial future impact on the population. With increased awareness and research, it is possible to prevent blindness on a large scale in the younger, productive age group affected by myopic maculopathy (MM). The vision-threatening manifestations of pathologic myopia include myopic choroidal neovascularization, macular atrophy, maculoschisis, macular hole, and retinal detachment. Myopic traction maculopathy (MTM) is a progressive manifestation of pathologic myopia and its treatment includes pars plana vitrectomy, macular buckle, or a combination. In this article we aim to review the diagnosis, clinical characteristics, and treatment of MM with an emphasis on recent developments in the surgical management of MTM. We discuss commercially available macular buckles, along with potential advantages to the use of macular buckle in MM. We review the new MTM staging system and its role in determining surgical management of these complex cases.

Keywords: Myopic maculopathy, macular buckle, myopic traction maculopathy, myopia, maculopathy

Cite this article as: Anderson WJ, Akduman L. Management of Myopic Maculopathy: A Review. Turk J Ophthalmol 2023;53:307-312

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Received: 07.05.2023 **Accepted:** 24.07.2023

DOI: 10.4274/tjo.galenos.2023.59844

Introduction

Myopia, including pathologic myopia, has seen a significant increase in prevalence in recent years. Prediction models suggest that by 2050, about 50% of the global population will have myopia and nearly 10% will have high myopia.[1] Unfortunately, with current trends, it is predicted that 33.7 million people will experience vision impairments and 18.5 million people will become blind due to myopic maculopathy (MM).[2] The impacts will be especially felt in Asian countries, where myopia is more prevalent than in the United States.[3] This makes the management of myopia progression, particularly in childhood and adolescence, a topic of significant concern. In this article we will provide an overview of MM, with a focus on the latest trends in the surgical management of myopic traction maculopathy (MTM).

Definition of Myopia and Pathologic Myopia

Myopia is defined as a spherical equivalent of ≤-0.50 diopter (D), when ocular accommodation is relaxed.[4] Pathologic myopia occurs in eyes with an axial length ≥26.5 mm, refraction ≤-6.0 D, with concurrent structural changes observed in the retina.[4,5] MM refers to any anatomical changes that occur in the macula of myopic eyes, primarily attributed to elongation of the axial length. When these anatomical changes progress over time, the term "progressive myopic maculopathy" is used. However, it is worth noting that most eyes diagnosed with MM were not born with it. In reality, most cases of MM have already been progressing at varying rates, shapes, and forms throughout the individual's lifetime.

Epidemiology

The prevalence of MM varies among different ethnicities and populations. Rates have been reported between 1-4%, with higher rates of around 8-10% in Asian countries.[6,7,8,9] A recent meta-analysis revealed that myopic patients have an increased risk of MM, especially those with high myopia (<-6.00 D) (odds ratio: 845.08).[8] However, the study also found that moderate myopia (-3.00 to -6.00 D) (odds ratio: 72.74) and even low

myopia (-0.5 to -3.00 D) (odds ratio: 13.57) were associated with increased risk of MM.[8]

Natural Course

It is well known that higher diopters of myopia and increasing axial length are directly correlated with an increased risk of developing pathologic myopia in later years.[10] Although high axial length may not be the only explanation, it is the most obvious and associated factor in pathologic myopia (similar to elevated intraocular pressure in glaucoma). Although the rate of myopia progression can slow down or stop in adult years, studies have shown that pathologic myopia can develop and worsen later in life. In fact, one study reports that 40.6% of patients progressed significantly to visual impairment within 12.7 years after the age of 40.[11]

In a case series by Sonne et al.[12], eyes with MM had an increased axial length after cataract surgery, averaging 1.32 mm over 13 years after their surgeries, resulting in reduced vision from 20/30 to 20/200 during the follow-up period. The maculopathy presented as choroidal neovascularization (CNV), maculoschisis, or macular atrophy.

Clinical manifestations of MM (Table 1) present as a progressive disease and include tessellated fundus, lacquer cracks, macular atrophy, CNV, maculoschisis, macular hole, posterior staphyloma, and posterior pole retinal detachment.[5]

Prevention of Pathologic Myopia

Myopia progression is most rapid during childhood and adolescence, making this an ideal time to consider interventions to prevent high myopia. Spectacles, bifocal and multifocal contact lenses, orthokeratology lenses, and atropine have all been studied as potential interventions to slow myopia progression in childhood, with topical atropine showing the most promise in recent studies.[13,14,15,16] While the exact mechanism of atropine's effect on axial elongation is still unclear, it appears to be unrelated to accommodation effects. A recent meta-analysis demonstrated that low-dose 0.01% atropine is as effective as 1% atropine but with fewer side effects such as pupil dilation, loss of accommodation, and near vision blur.[17] Wei et al.[18] found that low-dose atropine resulted in a relative reduction of approximately 34% in myopia progression in children over a 1-year period. Low-dose atropine may also be preferred due to its relatively minimal rebound effect when treatment is stopped, and it is typically better tolerated by children compared to wearing contact lenses. Ongoing research is focused on developing interventions that can slow or halt the progression of myopia and prevents its pathologic consequences.

Table 1. Types of myopic maculopathy
Staphyloma
Macular atrophy
Lacquer cracks
Choroidal neovascularization, which may lead to disciform scar ("Fuchs spots")
Posterior pole retinal detachment

Manifestations of Myopic Maculopathy

Clinical characteristics of MM include tessellated fundus, lacquer cracks, and macular atrophy with later stages resulting in CNV and MTM. In 2015, Ohno-Matsui et al.[19] proposed an international classification system that classifies MM into categories with "+" signs indicative of features that may predispose to central vision loss (Table 2).

One of the initial clinical manifestations of MM is a tessellated fundus, which results from thinning of the retinal pigment epithelium (RPE) and reduced pigmentation. This leads to prominent choroidal vasculature, typically seen along the macula and arcades, and is associated with reduced choroidal thickness.[20] As the disease progresses, diffuse chorioretinal atrophy may develop, characterized by a yellowish-white appearance which usually first appears around the optic disc and macula. Patchy choroidal atrophy may also develop, with demarcated areas of gray-white lesions secondary to choriocapillaris dropout and subsequent RPE loss. While it is rare to experience central vision loss from chorioretinal atrophy in MM, it can occur in late stages of the disease.[19] Additionally, lacquer cracks are a common finding in the posterior fundus early in the disease course and are seen as yellow lines in a branching pattern that represent a rupture in the RPE, Bruch's membrane, choriocapillaris complex.[21] These cracks may result in subretinal hemorrhage, which can resolve without intervention. However, lacquer cracks are a known precursor to myopic CNV, which can be a more significant threat to vision. CNV may spontaneously regress, leading to atrophy and dark pigmented scars known as Fuchs spots from proliferating RPE cells surrounding the regressed CNV.

Posterior staphyloma, defined as an outpouching of the wall of the eye that has a radius of curvature less than the surrounding curvature of the wall of the eye, is commonly associated with MM, particularly at the posterior pole.[22] If present, it likely plays a role in the development of MTM due to progressive thinning and mechanical damage to the retina.[23]

Myopic Choroidal Neovascularization

Pathogenesis of myopic CNV

Myopic CNV is one of the most serious vision-threatening complications of pathologic myopia, affecting approximately 5-11% of patients and often resulting in sudden vision loss. Individuals with myopic CNV in one eye are at increased risk of developing CNV in the fellow eye, with a 35% chance over

Table 2. Classification of myopic maculopathy[18]	
Classification	**Clinical manifestations**
Category 0	No macular lesions
Category 1	Tessellated fundus
Category 2	Diffuse chorioretinal atrophy
Category 3	Patchy chorioretinal atrophy
Category 4	Macular atrophy
"Plus signs"	Lacquer cracks, choroidal neovascularization, Fuchs spots

an 8-year period demonstrated by Ohno-Matsui et al.[24] Myopic CNV typically arises from lacquer cracks and is classified as a type 2 CNV that enters from beneath Bruch's membrane as defects arise from the expanding scleral wall and subsequent thinning of the retina.

Management of myopic CNV

The current standard of care for myopic CNV is treatment with anti-vascular endothelial growth factor (anti-VEGF) agents. Bevacizumab (Avastin; Genentech Inc, San Francisco, CA, USA), ranibizumab (Lucentis; Genentech Inc), and aflibercept (Eylea; Regeneron, Tarrytown, NY, USA) have all shown short-term benefits in the management of myopic CNV.[25,26,27] In the RADIANCE trial, ranibizumab was found to have sustained improvement in best corrected visual acuity (BCVA) compared to photodynamic therapy at 12 months.[25] Aflibercept was also shown to be effective in the MYRROR study, which demonstrated a gain of 13.5 letters compared to a 3.9-letter gain in the sham control at 1-year follow up.[26] However, the use of anti-VEGF agents for myopic CNV should be considered judiciously, as several long-term studies have failed to show improvement in BCVA over a follow-up period of 5 years or more with bevacizumab, ranibizumab, and aflibercept.[27,28,29] This is due to the secondary chorioretinal atrophy which develops as a result of the CNV. There is some debate about whether anti-VEGF agents may worsen this atrophy by causing a degenerative effect on the RPE and choriocapillaris, which may be exacerbated in highly myopic eyes with an already extremely thin choroid.[30,31] It is important to note that myopic CNV behaves differently than CNV related to age-related macular degeneration, and these patients often require fewer injections to control the CNV.[5]

Recently, biosimilar agents have also been introduced into clinical practice. Of the anti-VEGF agents mentioned above, only ranibizumab (Lucentis 0.5 mg) and its biosimilar equivalents ranibizumab-nuna (Byooviz; Biogen, Cambridge, MA, USA) and ranibizumab-eqrn (Cimerlie; Coherus, Redwood City, CA, USA) are FDA-approved and on-label for treatment of myopic CNV.[32]

Myopic Traction Maculopathy

In 2004, Panozzo and Mercanti[33] introduced the term "myopic traction maculopathy" to describe various clinical changes associated with MM such as maculoschisis, retinal/foveal detachment, lamellar macular hole, and full thickness macular hole with or without retinal detachment. This led to the development of the MTM staging system (MSS) by Parolini et al.[34], which is currently the most widely used classification system for MTM. The MSS is based on optical coherence tomography (OCT) imaging and consists of four stages: Stage 1- inner/outer maculoschisis, Stage 2- predominantly outer maculoschisis, Stage 3- maculoschisis/macular detachment, and Stage 4- macular detachment. The foveal morphology is also described in stages: A- Normal foveal architecture, B- lamellar macular hole, and C- full-thickness macular hole.[35] A recent international validation study demonstrated good interobserver reliability of the new staging system.[34]

Pathogenesis of Myopic Traction Maculopathy

MTM and its sequelae can be seen as a progressive evolution of the same disease with multiple contributing factors. Incomplete posterior vitreous detachment, vitreomacular traction, and epiretinal membrane are pre-retinal factors that may exert centrifugal and tangential traction which contribute to the development of MTM.[36] Subretinal factors, such as progressive staphylomatous changes of the sclera, may also contribute to MTM by leading to retinal thinning and decreased blood supply, thereby weakening the adhesion forces between retinal layers. Forces perpendicular to the retinal plane, such as incomplete posterior vitreous detachment and progressive staphyloma, are more likely to contribute to progressive maculoschisis and Stage 1-4 MTM. On the other hand, tangential forces such as epiretinal membrane may be more responsible for the development of foveal changes and Stage A-C MTM. The evolution of MTM is a complex process with multiple overlapping factors contributing to the development and progression of the disease.

Management of Myopic Traction Maculopathy

The treatment of MTM can be addressed through either an ab interno or an ab externo approach. The timing and selection of surgical intervention depends on various factors such as the degree of visual impairment and the MSS classification. Typically, ab interno surgery is more effective in addressing pathology related to tangential centrifugal forces on the retina, while ab externo surgery may be more effective in addressing pathology secondary to perpendicular centrifugal forces. In severe cases of MTM, a combination or stepwise approach may be required to manage both components.

Macular Buckle

In the 1930s, the ab externo approach was first attempted by reinforcing the posterior sclera with materials such as fascia lata and donor sclera.[37,38] Schepens et al.[39] developed the first macular buckle technique in 1957, but it did not become common practice. Recently, there has been a renewed interest in macular buckling for MTM management. Several types of macular buckles have been developed and improved over time. Currently, commercially available macular buckles include the AJL macular buckle, T-shaped buckle, ando plombe, and adjustable macular buckle, although none are available in the United States.

Tanaka et al.[40] published a case series in 2005 on a T-shaped rod silicone plastic exoplant reinforced with titanium, which showed promising results. Parolini et al.[41] developed an L-shaped macular buckle using titanium and a soft sponge material which indents the macula and is sutured to the anterior sclera. This was further developed to utilize two optic fibers positioned in the head of the buckle to assist with macular buckle positioning at the fovea.

The Akduman Myopia Support device (Figure 1) is a recently developed titanium macular buckle with several unique features that seem to make it advantageous.[42] Its concave supportive plate helps preserve the natural contour of the globe, possibly avoiding long-term foveal changes and any inhibition of the retina and choroidal circulation due to the indentation.[42]

Furthermore, its fixed stiffness and size create a fixed final axial length regardless of initial axial length unless adjusted for pseudophakic eyes where a rather limited indentation is desired. A recent case report demonstrated successful resolution of maculoschisis with improvement in refraction by 7.25 D.[42] This patient, whose preoperative OCT, postoperative OCT, and postoperative fundus photos are seen in Figure 2, had a reduction in axial length from 28.77 mm to 26.31 mm and also exhibited remarkable improvement in vision.[43] The surgery of this case can be reviewed at: https://eyetube.net/videos/titanium-macular-buckle-placement. The Akduman Myopia Support device was also reported to successfully close a recurrent MM hole.[42]

Recent developments in devices and surgical techniques have increased success rates and decreased complications with macular buckling. While intraoperative OCT is not yet widely available, it could be a valuable adjunct in macular buckle surgery. Given the increasing prevalence of MTM, it is important to be familiar with these techniques.

Vitrectomy

The ab-interno approach involves pars plana vitrectomy (PPV), with or without internal limiting membrane (ILM) peeling, and typically a gas tamponade. Vitrectomy in MM can be successful but has been associated with a high rate of recurrent retinal detachment, failure to close macular hole, and may induce iatrogenic macular holes during surgery. More recent studies have shown that ILM peeling and ILM flap improve success rates of macular hole closure in eyes with MTM.[44,45,46]

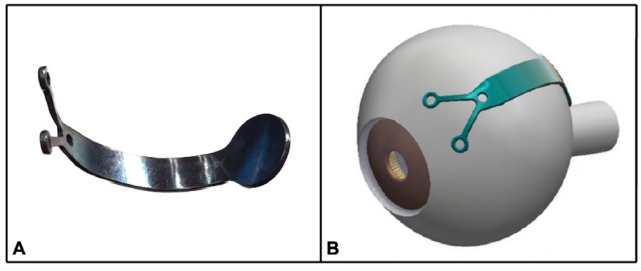

Figure 1. The Akduman Titanium Macular Buckle (A) and schematic representation of its position on the eye (B)

Surgical Decision Making

The choice of whether to use PPV, macular buckle, or a combination of both in the management of MTM depends on various factors. Parolini et al.[47] recently proposed new management guidelines for MTM based on the MSS in a retrospective review of the outcomes of PPV, macular buckle, or combined PPV and macular buckle in over 150 eyes with different stages of MTM. PPV was found to better address tangential centrifugal retina forces whereas macular buckle better addressed perpendicular centrifugal forces.

Early-stage maculoschisis can often be observed if vision is preserved and no significant epiretinal membrane is present. However, if mild maculoschisis is associated with worsening foveal pathology (lamellar macular hole, full-thickness macular hole), or epiretinal membrane, then PPV with ILM peeling or ILM flap and gas tamponade has high success rates in addressing these tangential forces. As maculoschisis and perpendicular forces worsen (Stages 2, 3, 4), then macular buckle becomes the preferred treatment. For Stages 2, 3, 4 (except those with full-thickness macular hole), macular buckle should be the initial treatment and PPV may be supplemented as a second surgery if foveal pathology progresses or does not resolve. If a full-thickness macular hole is present initially with Stage 2 or worse MTM, then a combination approach of PPV and macular buckle will likely be necessary to address both anterior/posterior and tangential forces.[47] Figure 3 provides an example of this combined surgical approach in Stage 4C MTM successfully managed with PPV, ILM peeling, and macular buckle by Parolini et al.[47]

As macular buckle techniques and devices continue to improve, it is expected to be utilized more frequently to address MTM. Macular buckle also addresses the underlying cause of progressive MTM, which is the increasing axial length of the eye. Additionally, it does not carry the risk of cataract progression, is reversible, and can also improve refraction in patients that likely are extremely near-sighted.

Conclusion

With increased awareness and research, it is possible to prevent blindness on a large scale in the younger, productive age group affected by MM. The vision-threatening manifestations of pathologic myopia include myopic CNV, macular atrophy,

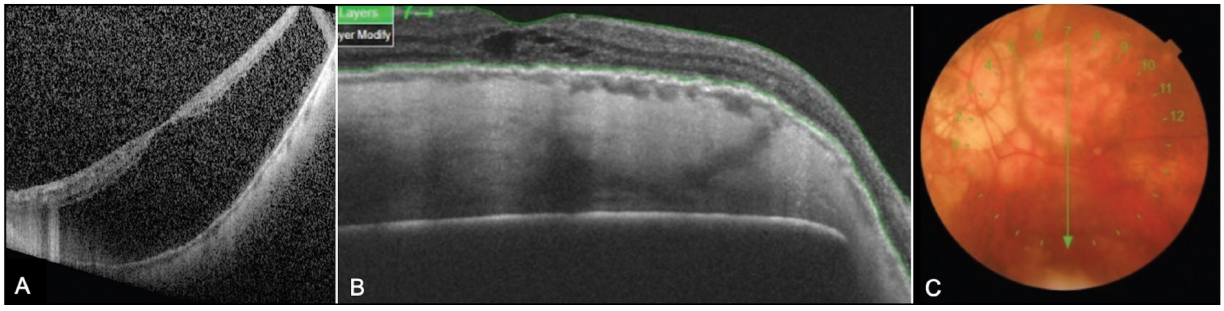

Figure 2. Preoperative optical coherence tomography image of a patient who underwent Akduman Titanium Macular Buckle placement (A). Postoperative photo demonstrates the indentation helping resolve the posterior pole retinal detachment reducing the axial length. No vitrectomy was performed (B). Postoperative fundus photo after Akduman Titanium Macular Buckle was placed with adequate indentation in the macula (C) (image courtesy of Retina Today)

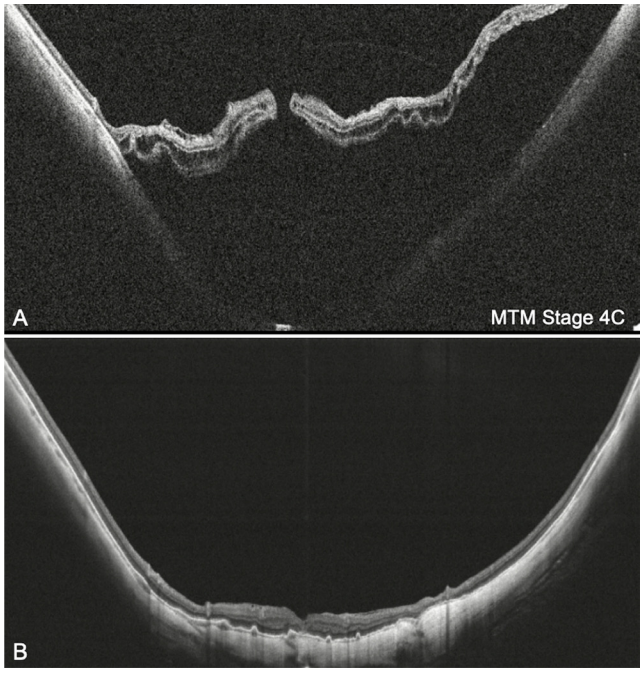

Figure 3. Severe myopic traction maculopathy Stage 4C with macular retinal detachment and full-thickness macular hole (A). Optical coherence tomography image after pars plana vitrectomy with internal limiting membrane peeling and macular buckle surgery performed by Parolini et al.[47] (B) (images courtesy of Barbara Parolini, MD)

maculoschisis, macular hole, and retinal detachment. While anti-VEGF therapy can improve short-term BCVA in myopic CNV, more studies are needed to assess its long-term benefits. MTM is a progressive manifestation of pathologic myopia and its treatment includes PPV, macular buckle, or a combination of both. The recently proposed MTM MSS provides a framework for approaching the surgical management of these cases. However, effectively treating vision-threatening manifestations of pathologic myopia such as CNV and MTM remains challenging. This highlights the importance of treating high axial length, the underlying cause of these pathologies, with myopia control in the early years and devices such as macular buckle to directly address axial length progression, which can ultimately prevent or delay vision loss.

Ethics

Peer-review: Externally and internally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: L.A., W.J.A., Concept: L.A., Design: L.A., Analysis or Interpretation: L.A., W.J.A., Literature Search: W.J.A., L.A., Writing: W.J.A., L.A.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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