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# Multiple parafoveal retinal detachment in myopic tractional maculopathy

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## Abstract:

This study investigates the occurrence of multiple parafoveal retinal detachments (RDs) in myopic traction maculopathy (MTM), emphasizing the atypical extrafoveal involvement compared to central foveal detachment commonly observed. Patient 1, a 46-year-old male, exhibited MTM with retinoschisis and four small subretinal fluid (SRF) pockets inferior to the fovea, accompanied by a hyperautofluorescent vitelliform deposit. Patient 2, a 43-year-old male, reported ring-shaped dim vision in the left eye, displaying MTM with six stable SRF pockets surrounding the fovea. These SRF pockets maintained a stable size ranging from 173 to 1140  $\mu\text{m}$  in diameter over the course of a 5-year follow-up period. Interestingly, fluorescein and indocyanine angiography showed no leakage or hyperpermeability in both cases. The study highlights the necessity for a comprehensive exploration of extrafoveal RDs in MTM, challenging conventional expectations. The mechanism of these persistent extrafoveal detachments associated with myopic schisis was unknown. The findings prompt further research to unravel the intricate mechanisms of this rarely reported phenomenon, emphasizing the importance of expanding our understanding of extrafoveal manifestations in eyes with MTM.

## Keywords:

Myopic traction maculopathy, retinal detachment, subretinal fluid

## Introduction

Myopic tractional maculopathy (MTM) is a sight-threatening complication commonly observed in individuals with high myopia, characterized by the pathological tractional forces exerted on the macula.<sup>[1]</sup> It was first identified on optical coherence tomography (OCT) by Takano and Kishi and termed “myopic foveoschisis,”<sup>[2]</sup> Panozzo and Mercanti later proposed the term MTM to describe the macular damage caused by traction forces.<sup>[3]</sup> Studies found that the traction mechanisms causing MTM are diverse, it can arise from the epiretinal membrane or residual focal vitreoretinal adhesion combined with posterior staphyloma and progressive scleral stretching in myopic eyes.<sup>[3-6]</sup> Eyes with MTM can progress from retinoschisis, foveal retinal detachment (RD), to full-thickness macular hole (MH), and

even total RD during the follow-up period, which may require surgical intervention in the early stage to prevent the consequent development.<sup>[7-9]</sup>

Central foveal detachment is a well-documented complication that can accompany MTM. This is attributed to tractional forces exerted within the staphyloma, leading to the separation of the sensory retina from the retinal pigment epithelium (RPE).<sup>[10-13]</sup> In addition, chorioretinal atrophy prevalent in high myopic eyes may compromise the adhesion between these two layers.<sup>[14]</sup> Shimada *et al.* described the progression from macular retinoschisis to foveal detachment in four stages and observed that an outer lamellar hole formation may predispose the retina to detachment.<sup>[8]</sup> Although vitrectomy may be performed in some cases to prevent subsequent MH, research has also demonstrated that foveal detachment in these eyes typically presents as shallow and is associated with preserved vision.

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This may be attributed to sufficient oxygen and nutrient diffusion from the choriocapillaris to the photoreceptors, allowing for partial survival and functional maintenance of the photoreceptors despite the detachment.<sup>[6]</sup> Although foveal detachment has been extensively investigated in numerous studies in MTM, none have reported on the occurrence of multiple parafoveal RD as observed in the cases described here. This finding challenges the conventional understanding of the disease, emphasizing the complexity of the vitreous-retina interface in high myopic eyes and the need for a nuanced exploration of its precise mechanisms.

## Case Report

Patient 1, a 46-year-old man with high myopia (axial length 30.26 mm), presented with metamorphopsia in the left eye. Best-corrected visual acuity (BCVA) was 6/7.5 in the right eye and 6/6 in the left eye. Myopic refractive error was -13 diopters (D) in both eyes. Both eyes were phakic and had not undergone any ocular surgery previously. Fundus examination and en face OCT at the outer retinal level exhibited MTM with retinoschisis and four small subretinal fluid (SRF) pockets packed inferior to the fovea with hyperautofluorescence vitelliform deposit at the lower margin of the pockets in the left eye. Similar findings were observed in the right eye, with two SRF pockets [Figure 1]. However, unlike the left eye, these pockets on his contralateral eye were associated with marked inner retina traction with schisis. Eplerenone 50 mg QD was attempted for 3 months; however, there was no significant improvement in the clinical presentation. The number of SRF pockets has remained constant over the past 5 years.

Patient 2, a 43-year-old man with high myopia (axial length 31.41 mm), reported a ring-shaped dim vision around the center of his right eye. His BCVA was 6/15 in the right eye and 6/20 in the left eye. He also has a history of amblyopia in both eyes, which are pseudophakic following cataract surgery for congenital cataracts. En face OCT demonstrated macular tractional maculopathy (MTM) with 10 small SRF pockets surrounding the fovea in the right eye. Similar findings with four SRF pockets were found in the left eye with three SRF pockets [Figure 2]. Like patient 1, the SRF pockets in his contralateral eye were associated with prominent vascular traction and inner retinal schisis. The number and size of these SRF pockets remained stable over the 1-year follow-up.

Despite diligent investigation, fluorescein angiography (FA) and indocyanine green angiography (ICGA) did not reveal any signs of leakage, hyperpermeability, or myopic choroidal neovascularization in both patients. The mean choroidal thickness in areas of extrafoveal RD

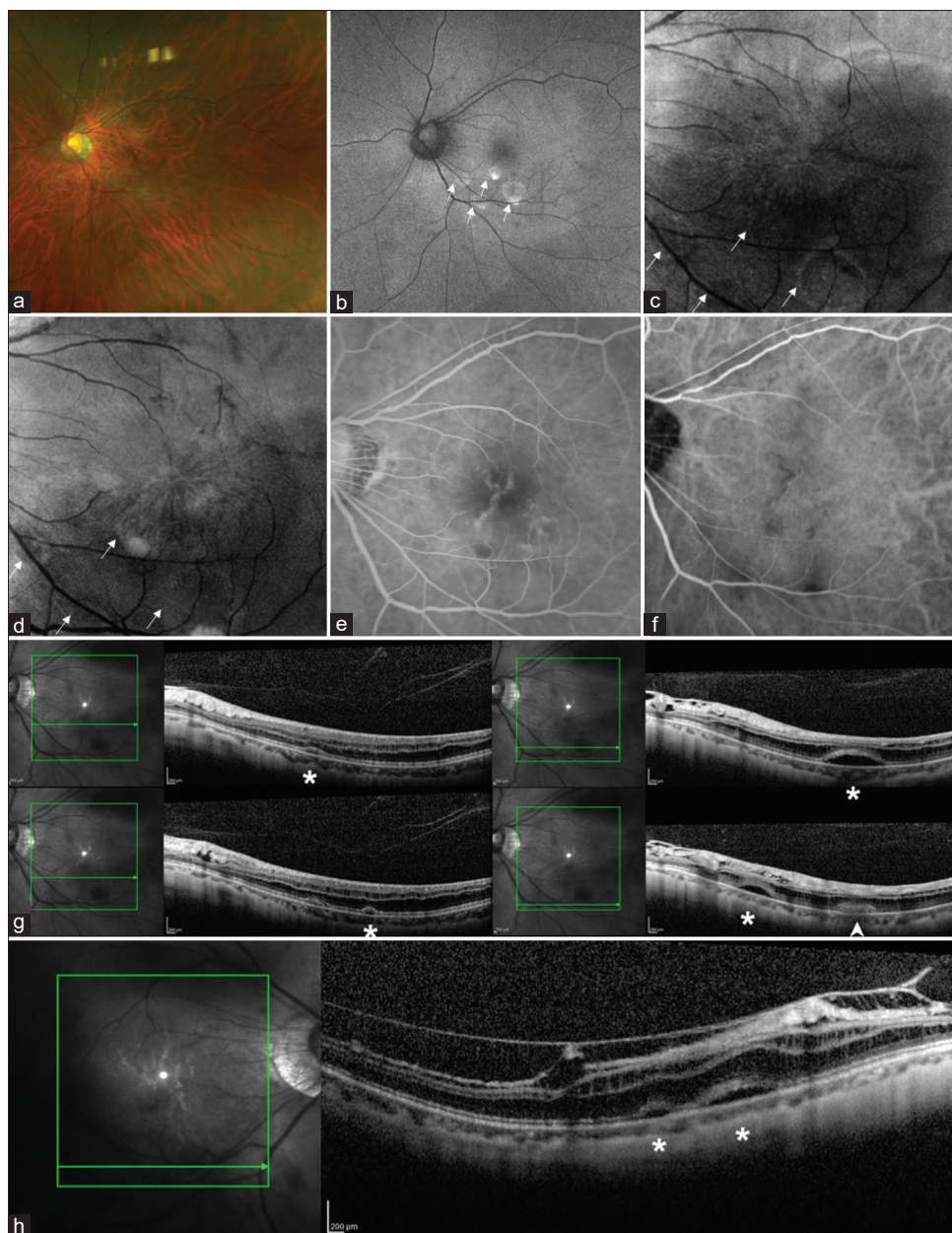
was  $108.1 \mu\text{m} \pm 15.8 \mu\text{m}$  in case 1 and  $92.2 \mu\text{m} \pm 12.7 \mu\text{m}$  in case 2, with no significant difference compared to areas without detachment. These SRF pockets maintained a stable size ranging from 173 to 1140  $\mu\text{m}$  in diameter over the course of a 1–5-year follow-up period. There was no development of an outer lamellar hole or increased thickness of schisis.

## Discussion

MTM is a condition often associated with high myopia, predisposed by pathological tractional forces on the macula. This study presents a distinctive scenario involving multiple parafoveal RDs in MTM, underscoring the unusual nature of extrafoveal involvement compared to the more common central foveal manifestations. Over a follow-up period of more than 5 years, the condition remained stable, aligning with descriptions of foveal RD documented in earlier studies.<sup>[1,7]</sup> The visual acuity remained stable and these extrafoveal separate pockets did not confluent or absorbed. The vitelliform deposits at the larger pockets of case 1 indicate a long-term sensory RD.

Several factors may contribute to the observed extrafoveal detachment in myopic traction maculopathy (MTM), involving two sets of forces: the preretinal and subretinal forces. Preretinal factors include the posterior vitreous cortex with vitreomacular traction, epiretinal membrane, and internal limiting membrane. These elements generate centrifugal and tangential forces that impact the macula. Subretinal forces include an increased axial length, posterior staphyloma, chorioretinal atrophy, and myopic macular degeneration.<sup>[15]</sup> Xiao *et al.* described the en face OCT features of extrafoveal retinoschisis of high myopia in 89 eyes and found that the posterior vitreous membrane frequently adhered to the retinal surface adjacent to major vessels or peripapillary areas.<sup>[16]</sup> The mechanical traction exerted by the vitreous on the parafoveal region plus vascular tenting, potentially exacerbated by posterior staphyloma in cases of high myopia, may promote retinoschisis and SRF accumulation. However, in their series, there were no parafoveal RD found under extrafoveal retinoschisis. In our two cases, the retina above the multiple extrafoveal RD did not show signs of vitreous membrane traction, epiretinal membrane, or increased inner schisis cavity or thickness in OCT. The morphology and location of the parafoveal SRF pockets are different from their contralateral eyes with the extrafoveal SRF pockets that are associated with vascular traction.

A possible subretinal mechanism underlying multiple RDs in MTM may be the compromised choroidal perfusion results from mechanical stretch forces.<sup>[17,18]</sup>



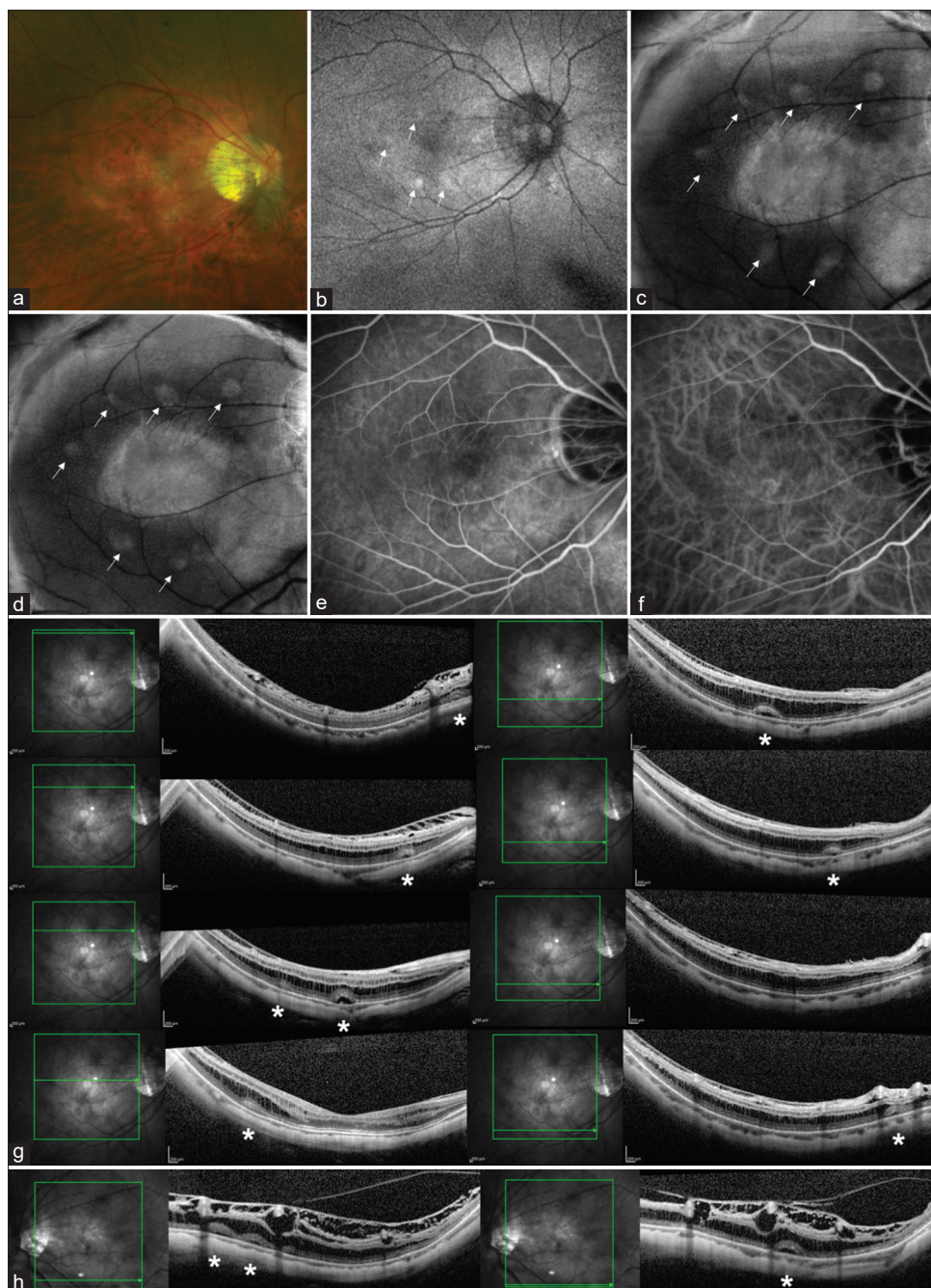
**Figure 1:** Patient 1 (a) Fundus color photograph showed peripapillary atrophy with tessellation. (b) Fundus autofluorescence showed subretinal fluid (SRF) pockets with hyper-autofluorescent vitelliform deposits (arrow). (c) En face optical coherence tomography (OCT) image of the left eye from 5 years ago revealed multiple persistent extrafoveal retinal detachments (RDs) (arrow). (d) An en face OCT image of the left eye showed persistent extrafoveal RDs. The number and size of these SRF pockets remained stable (arrow). (e) Late-phase fluorescein angiogram did not show any point of leakage or pigment tract. (f) Indocyanine green angiography did not detect neovascularization or hyperpermeability patches. (g) Serial OCT scan of the left eye from superior to inferior highlights the presence of extrafoveal RDs (asterisk) and vitelliform deposits (arrowhead). (h) Serial OCT scan of the right eye showed two SRF pockets outside the fovea (asterisk)

When the choroid is unable to adequately supply oxygen and nutrients to the outer retina, the photoreceptor cells and other retinal layers may function abnormally. This vascular insufficiency undermines the structural integrity of the retina and reduces its adherence strength to the underlying pigment epithelium.<sup>[19]</sup> As a result, even minor additional stresses or vitreoretinal traction can precipitate RDs. Importantly, the impact of poor choroidal perfusion extends beyond a single anatomical location within the macula, predisposing the entire retina

to mechanical separations and, potentially, multiple extrafoveal RDs. However, the choroid thickness and choroidal blood perfusion in ICGA of our two patients did not show signs for flow insufficiency.

One common cause of serious RD (SRD) is central serous chorioretinopathy (CSC), a disorder characterized by the accumulation of fluid under the retina, frequently affecting the macula.<sup>[20,21]</sup> In CSC, fluid leakage through the RPE into the subretinal space leads to the formation





**Figure 2:** Patient 2 (a) Fundus color photograph showed peripapillary atrophy with tessellation. (b) Fundus autofluorescence showed multiple subretinal fluid (SRF) pockets with hyper-autofluorescent vitelliform deposits (arrow). (c) En face optical coherence tomography (OCT) image of the right eye from 1 year ago revealed multiple persistent parafoveal retinal detachments (RDs) (arrow). (d) En face OCT image at present revealed persistent parafoveal RDs similar in size (arrow). (e) Late-phase fluorescein angiogram did not show any point of leakage. (f) Indocyanine green angiography did not detect neovascularization or hyperpermeability patches. (g) Serial OCT scan of the right eye showed MTM with ten SRF pockets (asterisk). (h) Serial OCT scan of the left eye showed vitreomacular traction with three SRF pockets near lower vascular arcade (asterisk)

of a serous detachment. The mechanisms underlying the location of SRD involve disruptions in the normal functioning of the RPE, responsible for maintaining the integrity of the subretinal space. Changes in the permeability of choroidal or retinal vessels may also contribute to fluid leakage.<sup>[22,23]</sup> However, CSC, one of the diseases in the pachychoroid spectrum, is rarely reported in high myopia.<sup>[24]</sup> It is noteworthy that both FA and ICGA in the reported cases of this study showed no

signs of hyperpermeability or leakage or pachyvessels. Neither was choroidal thickening found in these two cases. This suggests that alternative mechanisms or factors may contribute to the observed SRD. In addition, in patient 1, eplerenone treatment was not effective. This discrepancy between the absence of detectable leakage on angiography and the persistent SRD highlights the need for a deeper understanding of the underlying pathophysiology.

In summary, this study highlights the need for a comprehensive investigation into persistent extrafoveal RDs in MTM. It further emphasizes the role of en face OCT and fundus autofluorescence in monitoring disease progression. The extrafoveal involvement challenges traditional expectations, prompting considerations of multifactorial factors such as vitreous dynamics, ocular anatomy, and chorioretinal perfusion in high myopic eyes. Future research is important to explore the complex mechanisms underlying this rarely-reported phenomenon in MTM.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patients have given their consent for their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

### Data availability statement

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

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Nil.

### Conflicts of interest

Prof. Shih-Jen Chen, an associate editor at Taiwan Journal of Ophthalmology, had no role in the peer review process of or decision to publish this article. The other authors declared no conflicts of interest in writing this paper.

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