

## Epiretinal membrane profile on spectral domain optical coherence tomography in patients with uveitis

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**Purpose:** To study the epiretinal membrane (ERM) profile on the spectral-domain optical coherence tomography (SDOCT) in eyes with uveitis. **Methods:** In this prospective observational study, macula of uveitic eyes were evaluated by SDOCT (Cirrus, model 5000) for ERM. ERM was quantified (in microns) and were followed up along with the best-corrected visual acuity (BCVA) and treatment profile for 1 year. ERM morphology (focal, global, or mixed) and characteristics (thickness at fovea, maximum thickness, and location of maximum thickness in relation to fovea) were documented. Changes in altered foveal contour, cystoid macular edema (CME), and central foveal thickness were also noted. BCVA was noted when the inflammation subsided and it was correlated to specific ERM characteristics. SDOCT characteristics were compared in three treatment groups (no oral steroids, oral steroids with, and without immunomodulators). **Results:** Thirty-four eyes of 25 patients were evaluated. Mean logMAR BCVA decreased from 0.25 to 0.35 ( $P = 0.005$ ). Foveal involvement with ERM ( $P = 0.011$ ), lost foveal contour ( $P = 0.043$ ), and ellipsoid layer disruption ( $P = 0.017$ ) were associated significantly with reduced BCVA. Focal attachment of ERM was more commonly associated with CME ( $P = 0.03$ ). Median ERM thickness showed significant increase ( $P < 0.001$ ). Significant ERM progression from parafoveal to foveal ( $P = 0.02$ ), significant progression of the thickest area of ERM closer to fovea ( $P = 0.0006$ ) indicated a strong tendency of foveal involvement and this was correlated with worse BCVA ( $P = 0.009$ ,  $r = -0.44$ ). Oral steroids/immunomodulators showed no significant benefit on ERM progression. **Conclusion:** ERM progression in uveitis has a tendency to involve the fovea and is associated with significant vision loss, particularly in foveal ERM, focal attachment, and IS-OS disruption. Oral steroids and immunomodulators have no role in halting progression.

**Key words:** Epiretinal membrane, epiretinal membrane changes in spectral domain OCT, optical coherence tomography, uveitis

Epiretinal membrane (ERM) which is one of the sequelae of chronic uveitis contributes to the distance and near vision impairment and image distortions like metamorphopsia, micropsia, and occasional monocular diplopia.<sup>[1-3]</sup> Although ERMs are usually detected by fundus evaluation, detection sensitivity has been increased by the use of optical coherence tomography (OCT).<sup>[4]</sup> Spectral-domain (SD) OCT allows better visualization and improved ultrastructural evaluation of the pathological features of ERM and the underlying retinal changes. An earlier study reported mean thickness of ERM as  $61 \pm 28$  microns,<sup>[4]</sup> which is quite a wide range, however with SDOCT even thinner ERMs can be studied, generally in the range of 5-7 microns due to the higher resolution.<sup>[5-7]</sup> Although morphological SDOCT parameters have been correlated with visual prognosis in patients involved with idiopathic ERM, such observations have not extended to uveitic ERMs in the Indian population.<sup>[6-10]</sup> This study analyses the correlation of various characteristics of ERM secondary to uveitis with visual acuity, progression pattern, and the role of oral steroids and immunomodulators in the natural course of ERM progression.

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## Methods

In this prospective, observational study, uveitic patients presenting to the tertiary eye care center uvea out-patient department between June 2015 to May 2017 were included. Prior Institutional Review Board (IRB) approval was obtained and written informed consent forms were taken from all the parents. The study adhered to the tenets of the Declaration of Helsinki. Patients >18 years showing SDOCT supported diagnosis of ERM with either active uveitis on treatment or history of uveitis were included. Patients with coexisting retinal conditions, traumatic uveitis, prior eventful intraocular surgery, documented ERM before onset of uveitis, or any other non-uveitic pathology contributing to formation of ERM were excluded. Patients with dense cataract, non-dilating pupil, thick posterior capsular opacification or corneal opacity, and uncooperative patients were excluded due to poor-quality SDOCT images that prevented evaluation and quantification of the SDOCT

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data. Patients who underwent cataract surgery within the 12 months follow-up period were also excluded. Clinical data were collected at the baseline visit at the time of clinical diagnosis of ERM and at visits at 12 months follow up. The collected information included complete history, demographics, best-corrected visual acuity (BCVA), slit-lamp examination, and fundus evaluation. Retrospective analysis of severity of previous episodes of uveitis was done. Standardization for Uveitis Nomenclature (SUN) Working Group guidelines were used for uveitis anatomic classification and inflammation grading and activity.<sup>[11]</sup> Visual acuity was recorded at baseline and follow-up visits only when the eye was quiet or severity of residual inflammation was not deemed to be contributory to the decreased visual acuity and there were no other complications like complicated cataract or cystoid macular edema (CME).

All cases underwent SDOCT [Cirrus 5000 HD-OCT (Carl Zeiss Meditec, Dublin, California, USA)] and  $6 \times 6$  mm area was scanned with raster pattern and was interpreted by single examiner. The measurements were quantified by the built in automated calipers [Fig. 1a-c]. ERM was diagnosed exclusively on SDOCT findings: hyper-reflective signal at inner retinal surface and evidence of contractility including any distortion, corrugation of foveal contour.

ERM location was classified as foveal and parafoveal at baseline and we looked into any changes from baseline at 12 months. The central circle with a diameter of  $1000 \mu$  centered on foveal center was defined as the foveal area. The middle circle with a diameter of  $3000 \mu$  was defined as foveal plus parafoveal area, and the areas encompassed in the larger circle with a diameter of  $6000 \mu$  was defined as the entire macular area. Central ERM thickness (microns), maximum ERM thickness (microns), and location of maximum thickness (microns from fovea) were recorded at baseline and 12 months follow up. The attachment pattern of ERM to inner retinal layer was classified as focal, global, or mixed [Fig. 2].<sup>[12]</sup> ERM was labeled as focal when there were presence of clear spaces in between pinpoint attachment of ERM to inner retinal surface within the central 500 microns [Fig. 2a-c and h] and global [Fig. 2d and e] where there was near absence of the same. Mixed Mixed [Fig. 2f and g] ERM where both the types were present in about a 50–50 ratio.

Central subfield thickness (CST) – defined as the average retinal thickness in the area enclosed in a  $1000 \mu$  diameter circle centered at the center of fovea. The CST at baseline and 12 months was recorded for all eyes that were scanned. Visual acuity was recorded at baseline, 6, and 12 months follow-up. Visual acuity and OCT details were recorded when inflammation was under control.

Management of uveitis was based on therapeutic approaches encompassing specific treatment for the underlying systemic condition combined with topical and systemic corticosteroids. Immunomodulators were added in cases where intraocular inflammation was not controlled by corticosteroids and as corticosteroids-sparing agents. Treatment was aimed such that there was no or very minimal intraocular inflammation.

### Statistics

Statistical analysis was done using non-parametric tests and the SPSS software (version 20.0, SPSS 20, IBM, Armonk, NY, United States of America). Continuous data were analyzed using the Chi-square test and Kruksal–Wallis test. For analysis of serial

changes, Wilcoxon signed-rank test was used. Mann–Whitney U test was used for comparison of non-continuous/ordinal data. Linear regression analysis with calculation of the Spearman's correlation was done for identifying ERM characteristics associated with poor visual outcome.

## Results

Thirty-four eyes of 25 patients were included in the study. Mean patient age was 41.4 years (range 26–70 years). Eleven (44%) patients were males and 14 (66%) were females. Nine (36%) patients had bilateral ERM, while 16 had unilateral ERM. Fifteen eyes (44%) had anterior uveitis, 7 (21%) intermediate uveitis, 2 posterior uveitis (6%), 9 (26%) panuveitis, and 1 (3%) sclerouveitis. Uveitis was idiopathic in 12 (35%) eyes, while 8 eyes (24%) had VKH, 5 (15%) HLA-B27-associated uveitis, 4 (12%) TB-associated uveitis, and 5 (14%) had other causes including RA factor associated uveitis and Fuchs heterochromic iridocyclitis. Median duration of uveitis before OCT diagnosis of ERM was 12 months (mean  $18 \pm 15$  months, range 3–60). It was 23 months for anterior, 10 months for intermediate, and 8 months for posterior uveitis.

### ERM characteristics on SDOCT

At presentation, 65% eyes had foveal ERM and 35% had parafoveal ERM, while at the end of 1 year, 88% were foveal and 12% remained parafoveal. Eight eyes progressed from parafoveal to foveal in 1-year follow up [Table 1]. The median ERM thickness at fovea and the median maximum ERM thickness showed a statistically significant increase at 1-year follow up compared to baseline ( $P$  value  $<0.001$  each) [Table 1]. The median distance of point of maximum thickness from fovea also showed a significant decrease at 1-year follow up [Table 1].

Compared to baseline, 8 eyes (24%) developed CME with ERM ( $P = 0.038$ ), foveal contour was lost in 15 (44%) eyes ( $P < 0.001$ ) and 10 (29.4%) eyes developed Ellipsoid layer disruption in 1-year follow up. ( $P = 0.0006$ ). The mean central foveal thickness (CFT) changed from  $274 \mu \pm 44$  to  $303 \pm 47 \mu$  ( $P < 0.001$ ) in 1 year. CME was more commonly associated with presence of focal attachment of ERM at macula with or without combined global attachment [11 of 17 eyes (65%)] vs global attachment alone [4 of 17 eyes (24%)] ( $P = 0.03$ ). Altered foveal contour due to ERM was not significantly associated with CME ( $P = 0.22$ ).

Foveal involvement with ERM, lost foveal contour, and ellipsoid layer disruption were associated significantly with reduced visual acuity. [( $R = -0.44$ ,  $P = 0.009$ ), ( $R = 0.35$ ,  $P = 0.04$ ), and ( $R = -0.41$ ,  $P = 0.01$ ), respectively] [Table 2]. Focal attachment of ERM with or without global attachment had significantly worse visual acuity ( $P = 0.44$ ) but on linear regression analysis did not show significance [Table 2]. There was no significant difference in change in median ERM thickness at fovea and median maximum ERM thickness in patients treated without oral steroids vs those treated with oral steroids with or without immunomodulators [Table 3].

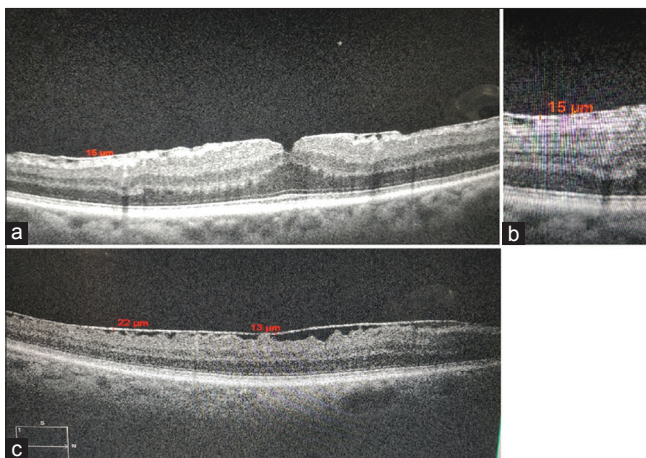
### Visual acuity

The mean logMAR distance visual acuity worsened from  $0.25 \pm 0.21$  to  $0.35 \pm 0.23$  ( $P = 0.005$ ) by 1-year follow up. All patients had N8 or better near vision at baseline. At 6-months follow up 18% patients dropped below N8, and at 1-year follow up 47% had near vision worse than N8 ( $P = 0.0001$ ).

**Table 1: Analysis of ERM characteristics on SDOCT**

ERM characteristic	Baseline	12 months	P
Location			
Foveal [n (%)]	22 (65)	30 (88)	0.02 <sup>SS</sup>
Parafoveal [n (%)]	12 (35)	4 (12)	
Type of attachment at macula			
Focal [n (%)]	10 (29)	11 (32)	0.48
Global [n (%)]	21 (62)	17 (50)	
Both focal + global [n (%)]	3 (9)	6 (18)	
CME due to traction*			
Present [n (%)]	7 (21)	15 (43)	0.038 <sup>SS</sup>
Absent [n (%)]	27 (79)	19 (57)	
Alteration in foveal contour*			
Yes [n (%)]	6 (18)	21 (62)	<0.001 <sup>SS</sup>
No [n (%)]	28 (82)	13 (38)	
Associated ellipsoid layer disruption			
Yes [n (%)]	1 (3)	11 (32)	0.001 <sup>SS</sup>
No [n (%)]	33 (97)	23 (68)	
ERM thickness fovea, $\mu$ median (range)	7 (0-11)	10 (6-17)	<0.001 <sup>S</sup>
Maximum ERM thickness ( $\mu$ ) median (range)	17 (14-22)	22 (18-28)	<0.001 <sup>S</sup>
Location of maximum ERM thickness ( $\mu$ from foveola) median (range)	1250 (778-1522)	870 (675-1190)	0.0006 <sup>S</sup>

\*Attributable to traction due to ERM after inflammation control <sup>SS</sup>Chi-square test <sup>S</sup>Wilcoxon signed-rank test



**Figure 1:** Spectral-domain optical coherence tomography images showing few epiretinal membrane (ERM) characteristics measurements: (a) optical coherence tomography image section showing ERM thickness measurement with automated calipers. (b) Magnified image of (a). (c) ERM thickness at fovea and at point of maximum thickness using software calipers

Five (20%) patients had significant metamorphopsia to start with and at the end of 1-year follow up 80% had metamorphopsia disturbing their daily activities [Table 4]. There was significant negative correlation between the shift of point of maximum ERM thickness toward fovea and final visual acuity [Pearson correlation  $-0.3$  ( $P = 0.05$ )].

## Discussion

SDOCT is the best tool for study of ERM as it has an axial resolution of 5–7 microns. It can be used as a tool for not only detection and follow up of ERM, but also for morphological characterization and various analysis of

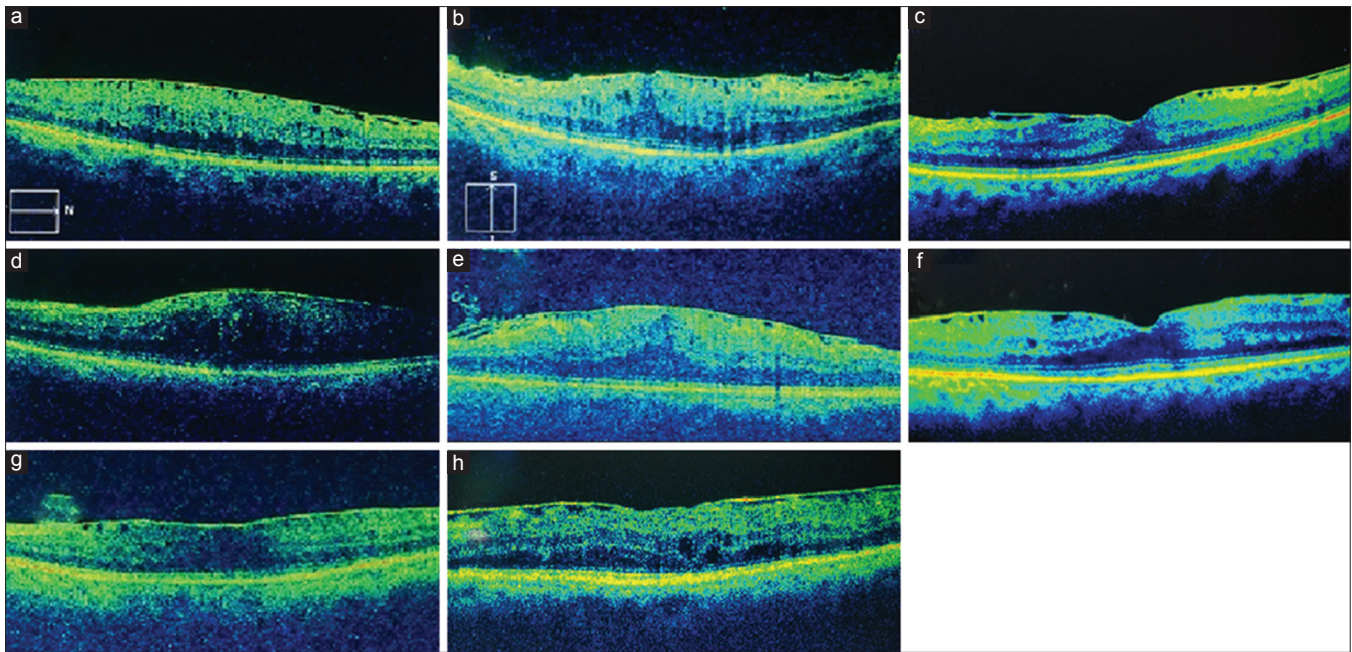
ERM. ERM formation is a known complication of ocular inflammation and uveitis patients may have poor visual acuity due to ERM even after inflammation control. ERM formation was observed to appear earlier in posterior and intermediate uveitis as expected due closer proximity of the inflammation to the retina.

About 32% of the uveitic eyes with ERM in our study showed a focal pattern of attachment to the underlying retina at 1 year. The focal pattern results in a more irregular contour of the underlying retina, explaining the cause of worse visual acuity. A previous study by Nazari *et al.* observed 50% of uveitic ERMs to be focal and reported focal attachment of ERM was an independent factor associated with a less favorable visual acuity in patients with uveitis.<sup>[12]</sup> Mori and associates also showed that approximately half of the patients with non-idiopathic ERM also have a focal attachment pattern to the retina.<sup>[13]</sup> As established in non-uveitic ERMs, focal ERM is associated with lower visual acuity in patients with uveitic ERMs as well.<sup>[9]</sup>

In our study, global ERMs showed a tendency to progress to focal type or developed a focal component. This could be due to the presence of histological components like – myofibroblasts, fibroblasts, lymphocytes, occasional macrophages in the ERMs, as well as associated inflammatory cytokines can lead to contraction of the ERMs, leading to retinal surface changes.<sup>[14,15]</sup>

Ellipsoid layer status on SDOCT is indicative of the anatomical integrity of the photoreceptors. Disrupted ellipsoid layer is associated with poor visual acuity after surgical removal of ERM.<sup>[9,16,17]</sup> As previously established, the physical forces of the contracting ERM may cause outer retinal damage as well in cases of idiopathic ERM but in uveitis patients the intraocular inflammatory milieu may also contribute to ellipsoid layer disruption.<sup>[18,19]</sup> Defects in the ellipsoid layer progressed significantly in our study, and these eyes had significantly





**Figure 2:** Spectral-domain optical coherence tomography images showing different configurations of epiretinal membrane (ERM): focal ERM (a-c); global ERM (d and e); mixed ERM (f and g); progression from mixed ERM (g) to focal pattern (h)

**Table 2: Correlation of visual acuity with spectral-domain optical coherence tomography characteristics of the epiretinal membrane in uveitis**

Parameter	Subgroups	No of eyes	Median LogMAR VA	Inter-quartile range	P*	Spearman's rank correlation
ERM location	Foveal	30	0.3	(0.2-0.6)	0.011	-0.44
	Parafoveal	4	0.15	(0.1-0.2)		P=0.009
ERM attachments <sup>§</sup>	Focal	11	0.3	(0.3-0.6)	0.044	-0.196
	Global	17	0.2	(0.2-0.3)		P=0.266
	Both	6	0.3	(0.3-0.3)		
Foveal contour	Normal	13	0.2	(0.1-0.3)	0.043	0.351
	Altered	21	0.3	(0.3-0.6)		P=0.042
Cystoid macular edema	Yes	15	0.3	(0.3-0.6)	0.067	-0.319
	No	19	0.2	(0.2-0.3)		P=0.066
IS-OS junction	Disrupted	11	0.3	(0.3-0.8)	0.017	-0.414
	Intact	23	0.2	(0.2-0.3)		P=0.015

\*Mann-Whitney U test; <sup>§</sup>Kruskal-Wallis test

poorer vision. The median ERM thickness at fovea and the median maximum ERM thickness showed a statistically significant increase from baseline to 1 year. The results of this study demonstrate that ERM thickness in the foveal area correlates significantly with vision. Thus, the foveal ERM thickness may be a useful parameter in future visual correlative studies involving uveitic ERM patients. The ability to predict progression pattern of uveitic ERMs may affect uveitis management strategies as well. The progression of significant number of parafoveal ERM to foveal and progression of the thickest point toward fovea, indicates a tendency of foveal involvement over time. This makes us believe in the possibility that uveitic ERM may begin developing from the peripheral macular areas, initially causing negligible visual symptoms and spread toward the fovea which gradually become symptomatic warranting imaging even in asymptomatic cases.

Intraretinal fluid accumulation can occur due to osmotic imbalance: decreased clearance by the RPE or increased retinal vascular permeability due to inflammation (a parameter that is taken care in this study).<sup>[20]</sup> The vitreous is implicated in various ways for CME. The vitreous fibers adhere to the Müller cell end-feet and exert tractional forces onto the cells and activate vascular leakage.<sup>[21,22]</sup> Similar mechanism can be extended for ERM. ERM is reported in up to 70% of uveitic eyes complicated by CME, and its presence often is associated with a poor response to medical treatment and a poor chance for visual improvement.<sup>[23,24]</sup> Central macular thickness does not seem to be a reliable predictor of visual acuity in the presence of uveitic ERM. Although the tangential forces of a focally adherent ERM and intraocular inflammation both may contribute to the development of CME. In this study we removed that confounding factor by recording OCT parameters

**Table 3: Analysis of association between spectral-domain optical coherence tomography characteristics of the epiretinal membranes and treatment modalities for inflammation control in 1-year follow up**

Treatment modality	Median change in ERM thickness at fovea*( $\mu$ ) (range)	$P^{\S}$	Median change in maximum ERM thickness* ( $\mu$ )	$P^{\S}$
Without oral steroids ( $n=8$ )	5 (0-17)	0.3	6 (3-10)	0.27
Oral steroids ( $n=20$ )	5 (0-8)		5 (-4 to 24)	
Oral steroids + immunomodulators ( $n=6$ )	2.5 (0-6)		0.5 (-4 to 4)	

$^{\S}$ Kruskal-Wallis test \*At 12 months compared to baseline

**Table 4: Visual outcome analysis**

	Baseline	6 months	12 months	$P^*$
Distance vision				0.005
LogMAR BCVA (mean $\pm$ SD)	0.25 $\pm$ 0.21	0.28 $\pm$ 0.20	0.35 $\pm$ 0.23	
Near vision				
$\geq$ N8 or better [ $n$ (%)]	34 (100)	28 (82)	18 (53)	0.001
N10-N18	0	6 (18)	16 (47)	
<N18	0	0	0	
Metamorphopsia $n$ (%)	7 (20)	12 (35)	27 (80)	0.001

\*Chi-square test

when the eye was quiet or relatively quiet. In our study CME attributable to traction due to ERM (after inflammation was controlled) was more commonly associated with presence of focal attachment of ERM at macula compared to the global attachment alone, probably signifying non-uniform or greater traction. CME is not always a prerequisite for altered foveal contour. In our study, the presence of altered foveal contour was not always due to intraregional cystic spaces. Diffuse edema and tractional component of the ERM could have been the cause of altered contour.

In eyes with uveitis and ERM, there was significant reduction in BCVA in 1 year. Proportion of eyes with near vision of N8 are better dropped from 100% to 53% in 1 year, with 80% having metamorphopsia. Foveal involvement with ERM, lost foveal contour, and ellipsoid layer defect are associated significantly with reduced visual acuity. It is expected that ERMs involving the fovea would cause greater visual impairment because of greater sensitivity to small structural alterations.

Studies are required to establish the role of severity of uveitis with time taken for ERM formation, the only limitation being the non-availability of uniform scales of severity over the different anatomical subgroups. Detection of uveitic ERM in earlier phases and in the peripheral macula and aggressive control of inflammation did not halt the progression of ERM or prevent foveal involvement. Our study showed that despite inflammation control, the visual acuity loss was significant and the ERM thickness significantly increased. ERM thickness had no significant difference in the three treatment groups namely – no oral steroids, oral steroids, oral steroids and immunomodulators.

The side effects of long-term oral steroids are known in the medical literature and the cost of immunomodulators are high. The mere presence of an ERM should not prompt the treating

ophthalmologist to alter the ongoing anti-inflammatory therapy. One of the treatment goals in uveitis should be to control the inflammation before the development of ERM. Management of intraocular inflammation would probably delay the onset of ERM development. However, studies are required to validate it. Surgical intervention for ERM may be indicated in fovea involving focally attached ERMs as it is associated with higher incidence of CME and visual impairment. Limitations of the study include, a relatively small sample size, non-randomized design and in eyes with broad ERM attachment, the isolated ERM thickness could not be taken, a higher pseudo-thickness was used involving internal limiting membrane (ILM).

## Conclusion

In conclusion, this study gives better predictability of visual acuity changes with time based on characteristics of ERM on SDOCT in uveitis cases and may also serve as a guide in tailoring proper follow up and management. Larger studies with more follow up may be required for further validation of our results.

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## Conflicts of interest

There are no conflicts of interest.

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