

A reappraisal of indirect choroidal rupture using swept-source optical coherence tomography *in-vivo* pathology images in patients with blunt eye trauma

Anupriya Arthur, Nancy Magdalene Rajasekaran¹, Thomas Kuriakose

Purpose: To describe the *in-vivo* pathology of indirect choroidal rupture (ICR) in patients with recent ocular trauma using swept-source optical coherence tomography (SSOCT). **Methods:** Retrospective observational study of the presenting and follow-up OCT images of four consecutive Asian patients with blunt trauma presenting over a period of 6 months and review of OCT descriptions in the literature. **Results:** The three patients who presented within 2 weeks of injury showed a gap in the Bruch's membrane (BM)/retinal pigment epithelial complex at the site of the ICR. The distance of the gap ranged from 103 to 465 μm . Blood from associated tear in the choriocapillary layer dissected under the photoreceptor layer to lift it off the retinal pigment epithelium (RPE) layer. The Sattler's and Haller's layers of the choroid were not affected. Fibro-vascular proliferation occurring in the reparative phase bridges the gap in the BM, RPE complex. Late fibrous tissue proliferation extends into the Sattler's layer in the choroid and goes up to the outer nuclear layer in the retina disrupting its architecture. **Conclusion:** SSOCT is a useful tool to study the pathology of recent onset ICR as it is capable of imaging through blood. The primary injury in ICR seems to be a break in the Bruch's membrane. The RPE layer and choriocapillaries get disrupted secondary to the break in the BM as their basement membranes are part of the BM. Scarring during the reparative phase disrupts the adjacent layers of the choroid and retina.

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Choroid is a multifunctional layer of the eye.^[1] The vascular layer of the choroid is divided into the inner capillary layer, the middle Sattler's layer with medium vessels, and the outermost Haller's layer with large blood vessels. The Bruch's membrane (BM) a part of the choroid forms the innermost layer of the choroid. The BM is a penta-laminar structure consisting of the basement membrane of the retinal pigment epithelial cells on the inner aspect and the basement layer of the choriocapillaries on the outer side. The central layer is an elastic layer sandwiched by a collagen layer on either side.

Choroidal rupture was first described by Von Graefe in 1854 as a tear of the choroid, Bruch's membrane (BM), and the retinal pigment epithelium (RPE) due to blunt trauma of the eye.^[2] Choroidal rupture has been described as full thickness rupture of the choroid, with the white seen in the middle of the rupture being attributed to the sclera shining through.^[3] Choroidal ruptures are seen in 5% to 10% of patients with blunt trauma.^[4]

Choroidal ruptures can be direct or indirect. Direct rupture is parallel to the equator.^[5] Indirect choroidal rupture (ICR) due to a diffuse force is concentric to the disc.^[6] ICR are due to contrecoup effect at the opposite side of impact due to shock waves that traverse the eye and strike the posterior pole and constitute 80% of choroidal ruptures.^[7] Rupture could also be due to the stretch of the globe behind the equator due to pressure on the globe. The BM has poor elasticity and thus ruptures easily. As opposed to the BM, the high tensile strength of the sclera and the high elasticity of the neuro-sensory inner retina prevent their rupture.^[7,8]

The gold standard to study the pathology of ICR would be histopathology. A histopathological study on 47 cases of choroidal rupture does not mention the exact structures that were involved in the rupture and has focused on the inflammatory changes associated with it. In this study, in six patients, a choroidal rupture was seen extending from the RPE to the sclera, with the retina lying in apposition to the sclera. Since there were no signs of inflammation, the authors called it an artefact.^[9] A more recent study on an old choroidal rupture did not talk about the structures involved at the time of injury as the inflammatory and scarring process had distorted the anatomy.^[10] Histopathology studies on acute cases of ICR are not available.

Common investigations to evaluate choroidal ruptures *in-vivo* have been auto-fluorescence and fundus fluorescein angiograms.^[11] High-definition swept-source optical coherence tomography (SSOCT) now enables us to get cross-sections of the retina and choroid up to the sclera-choroidal junction *in vivo* much like histopathology sections and is now finding a place to study ICR both at the time of injury and for follow-up.^[12-14] OCT angiograms have a potential role in the diagnosis and follow-up of choroidal neovascularization in ICR.^[14,15] Authors have described two morphological types of rupture involving the RPE, BM, and choriocapillaries.^[8] Here, the extent of retino-choroidal involvement has not been described.

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Department of Eye, Christian Medical College, Vellore, Tamil Nadu, India, ¹Department of Ophthalmology, The Queen Elizabeth Hospital, Adelaide, Australia

Correspondence to: Dr. Thomas Kuriakose, Department of Eye, Christian Medical College, Schell Campus, Arni Road, Vellore - 632 001, Tamil Nadu, India. E-mail: kuriakoset@gmail.com

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In the acute phase, choroidal ruptures may not be seen clinically because of the presence of sub-retinal blood, vitreous hemorrhage, anterior segment bleed, or inflammation.^[16,17] Once media clears, the typical feature is a whitish curvilinear lesion concentric with the optic disc, with the concavity toward the disc.^[18] With OCT, it is possible at times to make out the choroidal rupture even in the presence of sub-retinal blood.^[13] There is very little literature that has looked into the OCT scans of fresh cases of ICR to study the exact structural damage.

The aim of this article is to reappraise the clinical entity of ICR by studying the descriptions of this entity in the literature along with OCT structural changes noted in our series of patients.

Methods

This was a retrospective observational study on four consecutive patients seen in a tertiary care institution between February 2017 and August 2017. Three patients with acute trauma were followed up for over one month. Institutional Review Board and Ethics Committee Clearance were obtained - IRB: 11539 [Retro] dated: 26.09.2018.

A detailed history was taken regarding nature and circumstance of injury. Visual acuity was tested with the Snellen's chart. All patients underwent a thorough ophthalmological examination with Slit lamp and Goldman's applanation tonometry (GAT). Fundus examination was done with a slit lamp using a 90 D lens and indirect ophthalmoscope with a 20 D lens. All patients had a SSOCT (Topcon's DRI OCT Triton Plus which has a lateral resolution of 20 μm and an optical in-depth resolution of 8 μm and a scan rate of 100000 A scans per second) done with line scans through the site of suspected choroidal rupture. Digital fundus photograph was also taken with the same machine. Two patients consented to have an OCT angiogram (OCTA) done. The measurements were taken using the measuring tool of the machine. The average of three readings was taken as the width of the break. All measurements were taken by one investigator.

In the three patients who presented within 2 weeks of the injury, a repeat scan was done after a month.

Results

Four boys between the ages of 7 and 14 years presented to us during the study period. Three of them presented within 2 weeks of the injury and one after 5 months. There was only one contact with the child who presented late. Others were followed up for over a month.

Case 1

A 13-year-old boy presented with 5 months old injury to the left eye. Examination of the left eye showed iris sphincter tears with angle recession. A curvilinear stellate yellowish gray lesion was seen just nasal to the fovea with associated gray-white lesion at the fovea [Fig. 1a]. The right eye was normal.

SSOCT showed hyper-reflective, disorganized tissue involving RPE/BM complex, ellipsoid layer, external limiting membrane (ELM), and outer nuclear layer. This presumed scar tissue also involved the choriocapillary layer and the Sattler's layer. The Haller's layer was only minimally involved [Fig. 1b]. Scar tissue was also seen extending on the surface of the RPE away from the presumed site of rupture. The yellowish gray lesion seen at the posterior pole seemed to correspond to the areas where OCT showed the hyper-reflective scar tissue. No evidence of a full thickness BM or choroidal discontinuity could be made out.

OCT angiogram (OCTA) showed the inner retinal capillaries to be intact. The outer retinal capillary layer showed some

decreased vascular marking in the area of the scar. At the level of the photoreceptor and RPE, there was a discontinuity in the tissue pattern corresponding to the scar. Surrounding this discontinuity, there were new vessels. At the level of the choriocapillaries besides discontinuity in the vascular pattern due to scar tissue, there seemed to be increased vascularity along the rupture site itself [Fig. 1c].

Case 2

A 14-year-old boy presented immediately after injury to the left eye. Examination of the left eye showed a total hyphema which cleared in seven days and vision improved to 6/9. Fundus examination revealed whitish yellow curvilinear lesion and sub-retinal blood [Fig. 2a]. The right eye was normal.

SSOCT done seven days post injury showed discontinuity in the RPE/BM complex [Fig. 2b] in one area, and there was blood lifting the ellipsoid and ELM layers. The ellipsoid layer was intact over the area but appeared less defined and thicker compared to adjacent ellipsoid layer. The defect in RPE/BM complex was 195 μm . Except for the innermost layers of the choriocapillaries, the outer choriocapillaries and the outer two layers of the choroidal vasculature seemed to be intact. In another area with sub-retinal blood, there was a gap in the RPE/BM complex measuring about 454 μm [Fig. 2c]. One edge of the defect was shadowed by the retinal blood vessel over the region.

OCT done through the same area as in Fig. 2b 6 weeks later showed that gap in the RPE/BM complex was bridged with hyper-reflective reparative tissue and this tissue had incorporated the ellipsoid layer, ELM, and part of the outer nuclear layer in it and disrupted them. Some tissue proliferation was seen at the choriocapillary level too [Fig. 2d].

Case 3

A 11-year-old boy presented with a 12-day old injury to the left eye. Examination revealed traumatic uveitis and mydriasis in the left eye. Fundus examination of the left eye showed a curved yellowish gray sub-retinal lesion at the macula involving the fovea [Fig. 3a]. Minimal sub-retinal bleed and a resolving vitreous hemorrhage were also noted. The right eye was normal.

SSOCT scan done at presentation showed a break in the RPE/BM complex measuring 465 μm at the fovea. There was hyper reflective tissue extending from the outer nuclear layer of the retina to the choriocapillary layer. Ellipsoid and ELM layer could not be made out clearly [Fig. 3b].

Two weeks later when the inflammation cleared, the vision improved to 6/18 from 6/60 in the left eye. Seven weeks after injury, the vision dropped to 6/36 and the SSOCT showed fibrous tissue invading all the neuro-retinal layers at the foveola [Fig. 3c]. In one area, fibrous tissue was seen extending into the vitreous cavity too [Fig. 3d]. There were a few cystic areas seen in the nuclear layers of the retina. OCTA showed normal inner and outer retinal layer vascularization. The tissue pattern at the level of the RPE was absent in the area of the rupture with no new vessels. At the level of the choriocapillaries, there were vessels in the scar tissue [Fig. 3e].

Case 4

A 7-year old boy presented 1 day after injury to the right eye. The left was normal. The right eye vision was 6/18. There were abrasions on the cornea. Anterior chamber had cells of 1+ and flare of 2+. Fundus examination showed a double humped sub-foveal bleed. The retina over the macula looked edematous clinically. OCT done at presentation showed blood between the RPE and ellipsoid layer. The ellipsoid and ELM layer over the blood lost its clear distinction and looked thickened due to

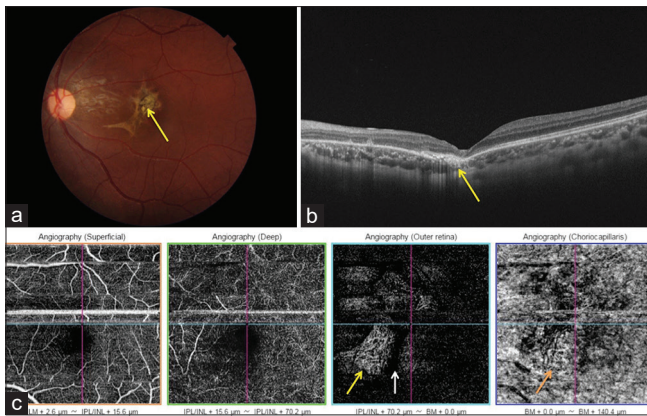


Figure 1: (a) Fundus photo of the left eye posterior pole showing sub-retinal scar and hyperpigmentation at the macula, involving the fovea (yellow arrow). (b) OCT line scan through the site of choroidal rupture showing hyper-reflective scar tissue (yellow arrow) incorporating all the layers from the nuclear layer of the retina to the Haller's layer of the choroid. (c) OCT angiogram with intact superficial and deep capillary layer. Discontinuity in the tissue pattern is seen at the level of the RPE (white arrow). New vessels associated with the scar tissue seen at the RPE level (yellow arrow) and choriocapillary level (orange arrow)

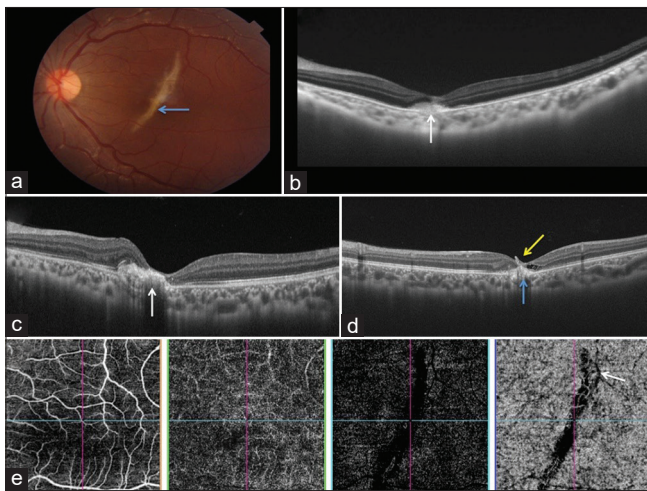


Figure 3: (a) Fundus picture of left eye showing a curvilinear yellowish gray sub-retinal lesion at the posterior pole involving the fovea (blue arrow). (b) OCT line scan through the curvilinear line near the fovea showing a gap in the RPE/BM complex 12 days after the injury. The ellipsoid layer and the ELM cannot be seen separately from the sub-retinal tissue. (c) OCT line scan 7 weeks after injury showing scar tissue (white arrow) bridging the RPE defect and incorporating almost the entire thickness of the foveal retina and the choriocapillary layer. (d) OCT vertical line scan 7 weeks after injury showing scar tissue (blue arrow) incorporating the entire thickness of the fovea with some scar tissue extending into the vitreous cavity (yellow arrow). (e) OCTA of left eye showed normal superficial and deep retinal vascular layers. The tissue pattern at the level of the RPE (outer retina) was absent in the area of the rupture with no new vessels. At the level of the choriocapillaries, there were vessels in scar tissue. (white arrow)

edema. OCT line scan showed two areas of breaks in the RPE/BM complex measuring 103 and 185 μm [Fig. 4a pre]. One week later since the sub-macular blood persisted, he underwent pars plana vitrectomy, sub retinal tissue plasminogen activator injection, and SF6 gas replacement of the vitreous cavity using the technique described previously.^[19] A month later the vision improved to 6/9. The fundus picture and OCT line scan after

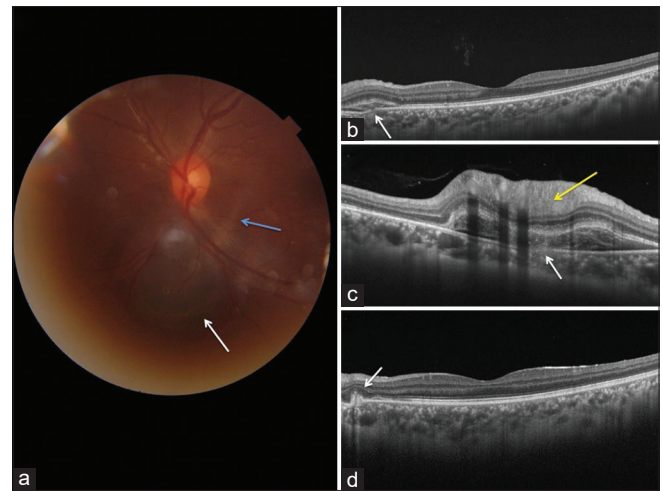


Figure 2: (a) Fundus photo 7 days after injury. White arrow showing large subretinal hemorrhage and blue arrow showing the yellow curvilinear choroidal rupture site. (b) Line OCT showing a gap in the RPE/Bruch's membrane layer (white arrow) with elevation of the neurosensory retina due to subretinal bleed. The gap corresponds to the blue arrowed portion of the curvilinear yellow line in Figure 2a. The ellipsoid layer and ELM layer can be seen above the blood collection. (c) OCT line scan through an area with obvious sub-retinal bleed in the acute phase showing a gap in the RPE/BM complex (white arrow). The sub-retinal bleed is seen elevating ellipsoid layer and ELM layers and there is thickening of the overlying retina (yellow arrow). (d) OCT line scan over the same area as in Figure 2b (white arrow) showing scarring and incorporation of the ellipsoid layer and ELM in the scar tissue

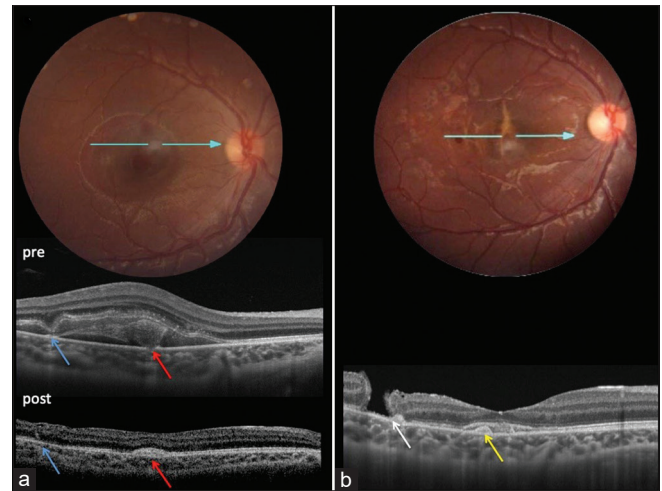


Figure 4: (a) Fundus color photo and line scan showing elevated retina with sub-retinal bleed over the fovea. The OCT line scan (pre) shows two areas of break in the RPE/BM complex (red and blue arrows). The raster OCT scan over the same area (post) after surgery to evacuate the sub-retinal blood shows some scar tissue (red and blue arrows). (b) Postoperative fundus photo and line scan show the retinotomy site and yellowish gray stellate lines under the retina. Scar tissue is seen between the photoreceptor layer and RPE (yellow arrow) corresponding to the yellowish gray area under the fovea. Full thickness neurosensory retinal defect is seen at the site of retinotomy. The RPE/BM complex break near the retinotomy site is plugged with scar tissue (white arrow)

surgery is shown in Fig. 4b. Clinically macular area showed multiple stellate lines under the retina that did not correspond to areas of choroidal rupture. The ELM layer and ellipsoid layer were continuous under the foveola. There was scar tissue

Table 1: Summary of the salient features of the cases

No	Age/ Sex	Duration of injury	Eye	P Vn F Vn	Mode of injury	BM, RPE tear gap	Layers involved in acute stage	Layers included in scar after a month
1	13/M	5 m	L	6/36 NA	Flying missile	NA	NA	RPE, BM, EL, ELM, ONL, CCL, SL, HL
2	14/M	1 d	R	HM 6/6	Fist	195 µm 454 µm	RPE, BM, CCL	RPE, BM, EL, ELM, ONL, CCL
3	11/M	12 d	L	6/60 6/36	Tennis ball	465 µm	RPE, BM, CCL	RPE, BM, EL, ELM, FTINL, VC
4	7/M	1 d	R	6/18 6/9	Flying missile	103 µm 185 µm	RPE, BM, CCL	RPE, BM, CCL

M=Male; m=Month; d=Day, R=Right, L=Left, P Vn=Presenting vision; F Vn=Follow-up vision; BM=Bruch's membrane, RPE=Retinal pigment epithelium, EL=Ellipsoid layer, ELM=External limiting membrane, ONL=Outer nuclear layer, CCL=Choriocapillary layer, SL=Sattler's layer, HL=Haller's layer. FTINL=Full thickness inner retinal layers, VC=Vitreous cavity; NA=Not applicable; HM=Hand movement

between the photoreceptor layer and RPE corresponding to the yellowish gray area under the fovea seen clinically. Full thickness neurosensory retinal defect was seen at the site of retinotomy. The RPE/BM complex break near the retinotomy was plugged with scar tissue. There were some cystic changes in the margin of the retinotomy site. At the site of the rupture corresponding to the area in Fig. 4a pre, there was scarring and closure of the BM break like in the previous two cases [Fig. 4a post].

Table 1 gives an overview of the cases presented. As has been noted above besides a break in the BM, the SSOCT showed blood between RPE and ellipsoid layer, and variable amounts of edema of the inner retinal layers in all the three acute cases. The gap in the BM ranged from 103 to 465 µm. The inner neuro-sensory retina and the Sattler's and Haller's layers of the choroid did not show any disruption in the acute stage.

OCT after a month of the injury showed scar tissue involving Sattler's layer, choriocapillary layer, BM, RPE, ellipsoid layer, ELM, and outer nuclear layer in all the cases.

Discussion

Ocular injuries worldwide lead to blindness in 1.6 million people.^[20] Majority of the injuries occur in males.^[21] Fortunately blunt ocular trauma is more common than open globe injury.^[22] Fundus examination of a patient with ICR reveals a curvilinear, crescentic, yellow or white line, widest at the center, and tapering toward the end. The rupture may not be clear initially; gradually it turns into a white streak with pigmented margins due to associated RPE hyperplasia.^[23] Although literature mentions that there is a rupture of the choroid, it is silent on the extent of the choroid involved. An SSOCT study by Pierro *et al.*^[23] on an acute case of ICR showed the disruption of RPE, BM complex, and ellipsoid zone with sub RPE blood.

In the three acute cases we did SSOCT on, the rupture involved only the RPE/BM complex and the choriocapillary layer of the choroid. The outer Sattler's and Haller's layers were spared. Break in the RPE layer was the most consistent OCT feature seen, pointing to the possibility that for mechanical reasons, the structure that primarily gives way or stretches to an extent that it damages the adjacent structures is the BM. The break in the RPE and the choriocapillaries is secondary to the BM stretch. In one of our patients (case 4), there was an area where the BM appeared intact but the RPE continuity was not clear. Here, there was a dip in the ellipsoid/ELM complex probably due to lack of bleeding and a tissue adhesion due to the expansile force. It is the break in the choriocapillaries and BM that causes the sub-retinal bleeding. A definite area of break in the ellipsoid layer just over the RPE break was not observed in any of our cases. In the area where the blood was minimal at

the site of BM break, the ellipsoid layer and ELM were intact and elevated. The differential elasticity of the retina and the potential space between the RPE and the neuro-sensory layer appear to limit the damage to the outer layer of the retina at the time of the BM tear.

The healing of the BM rupture occurs with fibrovascular proliferation that forms a dense fibrous scar with variable degree of hyperplasia of the RPE.^[9] The vessels in the fibro-vascular tissue could be seen in the two patients with the OCTA. These vessels should not be mistaken for sub-retinal new vessels similar to that seen in age-related macular degeneration. As was demonstrated histo-pathologically^[9] in one of our cases too, the fibrous tissue was seen extending into the vitreous cavity. The second OCT done after a month in all the three acute patients showed progressive scarring with distortion of ellipsoid layer, ELM, RPE/Bruch's complex, and the choriocapillary layer. This scarring could reduce the vision if the injury involves the fovea.

Retinal layers inner to the outer plexiform layer (OPL) are not affected by ICR and this is confirmed by OCTA where the superficial and deep retinal capillary plexus are intact. The OCTA picture segmented at the avascular outer retina and the choriocapillary layer showed discontinuity suggesting a disruption of these layers. OCTA segmented at the outer layers of the choroid appeared normal. OCTA study done by Pierro *et al.* on an acute case also revealed a similar picture like ours.^[23]

Sub-retinal blood causes photoreceptor damage and can be demonstrated in electroretinograph (ERG) studies.^[24] Sub-retinal blood causes destruction of the photoreceptors and other retinal layers by a mixture of mechanisms.^[25-28] A similar mechanism could reduce vision in cases with sub-foveal bleed secondary to ICR. Although there are no randomized trials, early pars plana vitrectomy with sub-retinal tissue plasminogen activator (tPA) seems to be effective in clearing the sub-retinal blood.^[29]

One deduction that may be possible from this study is that the yellowish gray lines seen classically in ICR are not due to the rupture *per se* but are due to the dehemoglobinized blood and scarring that occurs in the area of rupture. In our cases, sub-retinal yellowish gray lines were seen in areas where there were no BM ruptures but had subretinal fibrosis/dehemoglobinized blood.

Shin *et al.* have shown that some patients with ICR in the posterior pole show complete healing of the RPE. They have attributed this healing to the higher density of RPE in the region and better reparative cytokines available in the posterior pole.^[30] It is also possible that these are the cases where BM itself do not rupture completely and the remaining central layers act as a scaffold on which the RPE can regrow.

In our small series, we did not see any type 2 ICR as described by Nair *et al.* in their series.^[8] However, this classification has been questioned.^[31]

There are several shortcomings in this study. The number of patients we studied are small. Our patients were all males between 7 to 14 years old. Older patients may respond differently to injuries due to differences in the elasticity and tissue strength. The patients being children could not be submitted to multiple scans to study the entire area of the lesions. The inflammatory response also differs with age. One has to be cautious about generalizing these findings to the rest of the population. The age of the traumatic lesions here is less than 6 months and long-term tissue modelling has not been studied.

Conclusion

SSOCT is a useful tool to study the pathology of indirect choroidal rupture (ICR). ICR in children can show disruption in the BM/RPE complex. In the acute stage, blood from the break in the choriocapillaries dissect into the sub-retinal space lifting the photoreceptor layer. The choroidal tear is partial and the outermost layer (Haller's layer) of the choroid was intact in all our cases. Dehemoglobinized blood and sub-retinal fibrosis contribute to the yellowish gray color in and around the rupture site. The fibrosis that occurs as part of the healing process can extend into the deeper choroidal layers and the more superficial layers of the retina. The role of sub-foveal blood drainage to reduce blood toxicity and scarring needs further evaluation.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/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.

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Department of Ophthalmology, CMC, Vellore.

Conflicts of interest

There are no conflicts of interest.

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