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A Retrospective Case Series of Uveal Effusion Syndrome

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Disclosure
The authors declare no conflict of interest.

Précis: Uveal effusion syndrome is a rare condition caused by an underlying abnormality of the sclera resulting in the dysfunction of choroidal fluid dynamics. The clinical findings and management of affected eyes are presented.

Purpose: To describe the clinical findings and management of eyes affected by uveal effusion syndrome.

Methods: We retrospectively evaluated the charts of 13 eyes of 8 consecutive patients diagnosed with uveal effusion syndrome attending the ophthalmology unit of the University Hospitals Leuven, Belgium between 2007 and 2018. The presenting features, investigations, management and outcomes were analyzed for each case.

Results: Cataract surgery was the predisposing factor for uveal effusion in 6 eyes, 2 bilateral uveal effusions (4 eyes) were thought to be medication-induced and in 3 eyes the uveal effusion was described as idiopathic. Fundus examination of 5 out of 13 eyes showed bullous choroidal detachment, treated with pars plana vitrectomy with superotemporal sclerectomy or transscleral punction. Fundoscopy showed uveal effusion without serous retinal detachment in 3 eyes. Serous retinal detachment accompanied with uveal swelling was observed in 3 eyes and the 2 remaining eyes presented with uveal swelling only. The 8 non-bullous choroidal detachments were treated in a conservative way. A rapid resolution of subretinal fluid and uveal effusion was observed in all cases.

Conclusions: A conservative approach with acetazolamide treatment or just observation was used in our case series in choroidal detachment without substantial visual loss if over time slow improvement was documented. However further studies are needed to verify the effectiveness of the reported therapy.

Key words: Uveal effusion syndrome, choroidal effusion, hyperopia, serous retinal detachment, choroid.

Uveal effusion is an abnormal accumulation of transudative fluid from the choriocapillaris into the suprachoroidal space. This can result in a choroidal swelling, a choroidal detachment, subretinal fluid and a secondary serous retinal detachment and, finally degeneration of the retinal pigment epithelium (RPE). Uveal effusion is a clinical sign rather than a diagnosis. An uveal effusion due to hypotony, intraocular inflammation, an intraocular tumor and medication should be described as forms of secondary choroidal detachments. Other terms for this type of uveal effusion have been used conversely: choroidal effusion, ciliochoroidal effusion, ciliochoroidal detachment and choroidal detachment.[1]

In 1858 von Graefe[2], and in 1925 Verhoeff and Waite[3] described a spontaneous serous detachment of the choroid. Schepens and Brockhurst[4] defined the uveal effusion syndrome (UES) as a nanophthalmic disorder with a congenital scleral abnormality causing a thickened sclera which results in congestion of the choroidal venous system due to vortex vein compression.[5,6] Gass[7] and Jallow[8] hypothesized that, although the abnormality is congenital, ageing and hormonal changes result in a further impairment of the permeability of the sclera causing a decompensation and by consequence an idiopathic uveal effusion. Trelstad et al. found that the sclera showed histological and histochemical abnormalities.[9] A proliferation and migration of RPE cells in the subretinal space of patients diagnosed with uveal effusion syndrome was described by Forrester et al.[10] They proposed that the leopard-spot changes in the fundus of uveal effusion syndrome correlated to the foci of RPE proliferations and multi-layering.

Uyama et al. divided UES into three groups based on pathogenesis, axial length of the eye, and scleral thickness. Type 1 UES was a choroidal effusion in a nanophthalmic eye with an axial length less than 19 mm, high-grade hyperopia, and a thick sclera. Type 2 UES had a thick sclera but was not associated with nanophthalmos or hyperopia. Histologically type 1 and type 2 demonstrated abnormal sclera with disorganization of collagen fiber bundles and proteoglycandeposits in the matrix. In contrast, type 3 UES showed a normal axial length and a normal sclera. They postulated that a preoperative classification is essential for early surgical management.[11]

Brockhurst introduced the first treatment for UES.[6] He reported an effective surgical procedure for decompression of the vortex veins using sclerectomy. Since isolating the vortex veins is a rather difficult technique with substantial possible complications, Gass described good surgical results with sclerectomy and sclerostomy without decompression of the vortex veins.[7]

Case series regarding uveal effusion syndrome are sparse in literature, the clinical findings and subsequent management of affected eyes often vary. We describe the clinical findings of 8 consecutive patients diagnosed with UES and compare their management.

Methods

This was a retrospective, single-center, observational study conducted in patients attending the ophthalmology unit of the University Hospitals Leuven between 2007 and 2018. We reviewed the charts of 13 eyes of 8 consecutive patients diagnosed with UES. Appropriate institutional review board and ethics approval was obtained for the study.

Inclusion criteria were defined as follows: (1) peripheral ciliochoroidal detachment on fundoscopy or ultrasound with or without the presence of a serous retinal detachment, (2) other causes of ciliochoroidal detachment such as hypotony, intraocular inflammation, intraocular tumor and rhegmatogenous retinal detachment were excluded.

Results

Relevant clinical features including age, sex, presenting features, best-corrected visual acuity (BCVA), refractive status, biomicroscopy, intraocular pressure (IOP), axial length (AXL), scleral thickness (ST), UES classification, treatment and final visual acuity and refractive error of all 8 reported cases are shown in Supplemental Table 1, Supplemental Digital Content 1, http://links.lww.com/IJG/A426.

In our case series, the mean age was 62 years (range, 34-77 years) and 4 patients were men (7 of the 13 eyes). Complaints of blurred vision with a myopic shift and anterior displacement of the lens-iris diaphragm was observed in 4 patients, 3 patients presented with symptoms of bilateral acute angle closure glaucoma (AACG),

one was referred with suspicion of an intraocular tumor but ultrasound examination showed a retinal detachment with choroidal swelling and no intraocular masses, another was referred with a serous retinal detachment after cataract surgery. We observed involvement of both eyes in 5 out of 8 patients. Fundus examination of 5 out of 13 eyes showed bullous choroidal detachment. Three eyes showed uveal effusion without serous retinal detachment, 3 eyes had serous retinal detachment accompanied with uveal swelling and 2 eyes presented with uveal swelling only. Leopard spot changes of chronic serous retinal detachment were seen in 2 patients (Fig. 1). B-scan ultrasonography was used to confirm the diagnosis in all cases (representative example in Fig. 2).

The uveal effusion was seemingly triggered by cataract surgery in 6 patients. Two uveal effusions were thought to be the result of the recent intake of medication: one patient underwent chemotherapy with oxaliplatin (Eloxatin; Sanofi; France) for her metastasized colon carcinoma and another had recently used topiramate (Topamax; Ortho-McNeil Pharmaceutical, United States) for migraine. 3 eyes were diagnosed with idiopathic uveal effusions.

In our case series 5 eyes with substantial visual loss were instantly treated with pars plana vitrectomy (PPV) to manage the retinal detachment in conjunction with internal drainage of subretinal fluid. After introduction of Perfluorocarbon Liquid (PFCL; DORC; the Netherlands), disappearance of the choroidal effusion was observed in a myopic patient with normal scleral thickness. The two other hyperopic patients with increased scleral thickness were treated with PPV in conjunction with a superotemporal sclerotomy or superotemporal transscleral puncture to reduce the choroidal effusion. Resolution of the subretinal fluid and choroidal effusion was rapid with a conservative treatment consisting of acetazolamide or observation in 8 out of 13 eyes reported here.

Discussion

UES is an extremely rare disease characterized by choroidal fluid collections, often associated with shifting subretinal fluid and subsequent retinal pigment epithelial changes. Causes of primary and secondary choroidal effusions are listed in Table 1 (re-adapted from Elagouz et al.[1] with addition of specific drug-induced choroidal effusions). When the etiology remains unknown, the term idiopathic UES is used.[1,8] In our case series, the uveal effusion was seemingly triggered by cataract surgery in 6 patients, choroidal effusion due to hypotony was excluded. Two uveal effusions were thought to secondary to the recent intake of medication, one patient underwent chemotherapy with oxaliplatin for her metastasized colon carcinoma, to our knowledge this was previously described with cisplatin therapy, another platinum-containing agent.[12] Another patient had recently used topiramate for his migraine. Craig et al. previously reported the mechanism of topiramate-induced acute-onset myopia and angle closure glaucoma.[13] Murphy et al. published a comprehensive review of the full spectrum of drugs implicated in drug-induced bilateral AACG through the uveal effusion mechanism using standardized criteria.[14]

Several hypotheses on the pathogenesis of UES exist. Schepens and Brockhurst[4] defined the UES as a nanophthalmic disorder with a congenital scleral abnormality causing a thickened sclera which results in congestion of the choroidal venous system.[5,6] The eyes of our patients did not fulfil the defined criteria for nanophthalmos and were classified as hyperopic, 2 patients had an increased scleral thickness.

Gass et al. proposed an alternative for the hydrostatic hypothesis as described above[7]. He postulated that a reduced scleral protein permeability could result in retained fluid in the suprachoroidal space. Bill et al. has shown that in healthy individuals, albumin leaves the eye mainly via a transscleral route.[15] For this reason the colloid osmotic pressure in the suprachoroidal space is effectively zero. If this transscleral route of albumin is somehow impaired, fluid could be retained in the suprachoroidal space. Histologic studies have shown reduced transscleral diffusion in the sclera of patients with UES caused by an abnormal accumulation of glycosaminoglycan-like material expanding the interfibrillary spaces.[9,16,17,18] A direct measurement of scleral permeability was performed by Jackson et al.[19] They tested transscleral diffusion of high-molecular weight molecules in scleral tissue from UES patients removed during surgery. Jackson et al. also tested the hypothesis that UES is the result of a reduced scleral hydraulic conductivity.[20] Their findings suggested that an increased

scleral thickness is unlikely to significantly impede the scleral permeability to water. Daniele and Schepens introduced a fourth hypothesis.[21] They reported two cases in which primary hypotony can provoke UES in an otherwise normal-sized eye with no morphologic abnormalities. Elagouz et al. commented on this hypothesis that most patients in UES have normal IOP.[1] The fifth hypothesis, put forward by Kumar et al., suggests that an increased permeability of the choroidal vasculature, caused by a non-specific choroidal inflammation, is the trigger for UES.[22] Finally, Elagouz et al. concluded that multiple factors can contribute to the pathogenesis of UES, although the relative contribution of each factor may vary in patients affected by UES.[1]

UES usually affects healthy individuals in middle age, with predominance among men, and is usually bilateral. In our case series, men were affected in 7 of the 13 eyes and 3 times the UES was bilateral. Patients frequently present with complaints of loss of visual field or acuity. A myopic shift with anterior displacement of the lens-iris diaphragm is frequently observed, as was the case in our case series in 4 patients. Slit-lamp biomicroscopy generally shows a shallow anterior chamber with or without angle closure. IOP is usually normal, but can also be elevated, mimicking AACG.[1] Areiter et al. recently reported the spectrum of angle closure, uveal effusion syndrome and nanopthalmos.[23] In our case series, 2 patients presented with the symptoms of AACG. This clinical presentation has previously been described.[12,13,24,25] The early signs of uveal effusion are thickening and engorgement of the choroid, ciliochoroidal detachment and secondary serous retinal detachment. An observation we made is that no real choroidal detachment was present in 3 patients. Rather, the choroid was swollen as diagnosed by ultrasonography. Other clinical findings may include mild dilatation of the episcleral vessels, blood in the Schlemm canal, and mild inflammation in the vitreous.[1] Classic late signs include changes in the RPE, called leopard spots, as were seen in our case series in 2 patients.[10] This pattern of pigment clumping in the fundus is typical but not specific, considering it can be a result of other conditions causing chronic choroidal elevation.[26]

UES is a diagnosis of exclusion and multiple other conditions have been mistaken for UES, therefore UES still represents a diagnostic challenge. The entire differential diagnosis of serous retinal detachment is shown in Table 2 (re-adapted from Elagouz et al.[1]).

Brockhurst et al. described favorable surgical results with vortex vein decompression performed by scleral resection with sclerotomy.[6] Considering the isolation of vortex veins is a very complicated procedure and the decompression is technically difficult to perform without complications, such as vein rupture, other authors reported surgical techniques without vortex vein decompression. These procedures include performing scleral thinning procedures, called partial sclerectomies, with or without making scleral openings, known as sclerostomies or sclerotomies.[7,27,28,29,30] The scleral openings were made without considering the anatomical location of the choroidal effusion. Medical treatment including mydriatics, cycloplegic agents, and steroids before the standard recommended surgical approach has been described several times. However, the treatment is often not successful. A conservative therapy with nonsteroidal anti-inflammatory drugs has also been reported with rare success.[22] Kerstetter et al. hypothesized that prostaglandins analogues could increase the scleral permeability by increasing the uveoscleral outflow.[31] This hypothesis was confirmed by Weinreb et al.[32] Moldow et al. reported a positive clinical response in UES patients with acetazolamide.[33] In 2014, Derk et al. reported good results with a conservative therapy consisting of the combination of prostaglandins analogues and acetazolamide.[34] However, acetazolamide can also provoke an uveal effusion (Table 1) and should be anamnestically excluded as the causative factor. In our experience, acetazolamide is highly effective. If the UES worsens under therapy with acetazolamide, one should be aware of the potential side effect of this medication. Recently, Park and Lee confirmed that medical therapy can be of value in the treatment of UES before surgical treatment.[35] Shields et al reported in 2017 that oral, periocular, topical or a combination of corticosteroids provided control of UES in 95% of cases in 104 eyes with UES. They conclude that corticosteroids can be considered as a treatment, in the absence of nanophthalmos and/or scleral thickness abnormalities.[36] These patients were, according to the Uyama classification[11], classified as UES type 3 and surgical approaches

primarily involving the sclera would not seem to be necessary. It is interesting to note that resolution of the subretinal fluid and choroidal effusion was rapid with a conservative treatment in 8 out of 13 eyes reported here. Our case series presents a conservative approach with acetazolamide choroidal detachments may be considered in UES type 3 without substantial visual loss if no worsening or slow improvement can be documented on subsequent follow-up visits. Nevertheless, further case-series studies with UES patients are needed to determine the safety and effectiveness of this therapy.

In summary, we present the clinical findings and management of eyes affected by uveal effusion syndrome. UES is a rare disease usually associated with hyperopia and caused by an underlying abnormality of the sclera resulting in the dysfunction of choroidal fluid dynamics. It still represents a diagnostic challenge considering a ciliochoroidal effusion can be the consequence of numerous inflammatory and hydrostatic ocular conditions, therefore idiopathic UES is a diagnosis of exclusion. The condition is difficult to manage and it can result in severe visual loss due to chronic submacular fluid and secondary RPE changes.

Management of UES is complex and based on disease severity. A conservative approach with acetazolamide treatment or just observation was used in our case series in choroidal detachment without substantial visual loss if over time slow improvement was documented. Limitations include that effective subclassification was not possible considering not all patients had undergone the same diagnostic investigations and we did not conduct an internal nor a neurological examination, including Magnetic Resonance Imaging (MRI) of the brain and orbit, to rule out all possible inflammatory and hydrostatic causes of uveal effusion. Further case-series studies are needed to explore the various causative mechanisms of UES, and to determine if treatment can be tailored to a given patient.

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Figures/Table Legend

Figure 1 *Leopard Spots*
Figure 2 *Ultrasound B-Scan: annular bullous choroidal detachment*
Table 1 *Causes of Uveal Effusion*
Table 2 *Differential Diagnosis Serous Retinal Detachment*
Supplemental Table 1 *Demographic Characteristics*
Relevant clinical features including age, sex, best-corrected visual acuity (BCVA), refractive status, biomicroscopy, intraocular pressure (IOP), axial length (AXL), scleral thickness (ST), treatment, final visual acuity and refractive error of all 8 reported cases
Legend:
23G PPV: 23 gauge pars plana vitrectomy for retinal detachment, AXL: axial length (mm), CF: counting fingers, CT: computed tomography, F: female, IOP: intraocular pressure, IV: Intravenous, LP: licht perception, M: male, OD: oculus dexter, ODS: oculus dexter and sinister, OS: oculus sinister, RD: retinal detachment, SAC: secondary angle closure, ST: scleral thickness (posterior pole) obtained from A-scan associated with B-scan ultrasounds, > 1.7 mm noted as increased scleral thickness[38], SRD: serous retinal detachment, UES: Uveal Effusion Syndrome, UES type: Uyama classification[11]

Table 1. *Causes of Uveal Effusion*

Inflammatory

- Trauma and intraocular surgery
- Scleritis and infected scleral buckle
- Following cryotherapy and photocoagulation
- Chronic uveitis, pars planitis, Vogt-Koyanagi-Harada disease, and sympathetic ophthalmia

Hydrostatic

- Hypotony and wound leak
- Hunter syndrome
- Dural arterio-venous fistula
- Drug-induced
    - Topiramate
    - Acetazolamide
    - Hydrochlorothiazide
    - Venlafaxine
    - Indapamide
    - Methazolamide
    - Bupropion
    - Cabergoline
    - XTC
    - Escitalopram
    - Flucloxacillin
    - Sulfasalazine
    - Oxaliplatin
- Primary angle closure glaucoma (PACG)

Uveal effusion syndrome (UES)

- Hypermetropic or nanophthalmic
- Idiopathic

Table 2. *Differential Diagnosis of Serous Retinal Detachment*
• Chronic central serous chorio-retinopathy (CSCR) • Posterior scleritis • Rhegmatogenous RD with uveal detachment • Multifocal choroiditis • Uveal melanoma • Metastatic tumor • Severe hypertensive choroidopathy • Vogt-Koyanagi-Harada disease • Systemic diseases (myxedema, multiple myeloma)

Supplemental Table 1. *Demographics and clinical features of patients with uveal effusion.*

Case	Age	Sex	Presenting features/ Referral	BCVA (Snellen)/ Refractive Error	IOP (mm Hg)	Clinical/imaging findings	AXL (mm)	ST (mm)	Diagnosis: UES type	SAC	SRD	Treatment	Outcome Final BCVA (Snellen)/ Refractive Error
1	77	F	OS painless decreased vision, myopic shift 2 weeks post phaco	OD 0.7 -0.25(-0.5x25°) OS 0.6 -3(-0.75x77°)	OD 20 OS 20	- Shallow anterior chambers, no wound leak - Annular peripheral choroidal effusion - Echo-B-scan: choroidal effusion 360°, normal scleral thickness, no T-sign	OD 21.43 OS 21.61 Hyperopia	OD 1.21 OS 1.36	Primary UES: UES type 3 Predisposing factor: cataract surgery	–	–	Oral acetazolamide 250 mg (1x/day)	Beginning resolution of uveal effusion after 1 week, full resolution after 1 month, no relapse. OD 0.7 -0.25(-0.5x25°) OS 0.8 -0.50(-0.50x80°)
2	76	M	OD Referral to rule out an intraocular tumor	OD 0.8 +0.25(-0.5x11°) OS 0.6 +0.50(-0.75x5°)	OD 9 OS 10	- Normal anterior chambers, peripheral serous RD - Echo-B-scan: flat serous RD 8-11 o'clock, no threatening of the	OD 22.04 OS 21.82 Hyperopia	OD 1.10 OS 1.23	Primary UES: UES type 3 Idiopathic	–	+	Watchful waiting	Serous retinal detachment and uveal swelling resolved after 3 months

						macula, choroidal swelling and no intraocular tumor							without need for any topical or systemic treatment. OD 0.8 +0.25(-0.5x11°) OS 0.6 +0.50(-0.75x5°)
3	76	F	ODS Myopic shift 1 month post phaco OD and 3 weeks post phaco OS	OD 0.9 -2.75(-0.75x87°) OS 0.8 -3.5(-0.5x78°)	OD 18 OS 15	- Very shallow anterior chambers, no wound leak - Bullous inferotemporal choroidal effusion - Leopard spot retinal changes	OD 21.75 OS 21.91 Hyperopia	OD 1.30 OS 1.21	Primary UES: UES type 3 Predisposing factor: cataract surgery	+	–	Oral acetazolamide 500 mg (3x/day) Topical atropine IV mannitol 15% 150cc	Total resolution of angle closure, choroidal detachments and subretinal fluid after 2 months, no recurrences. OD 0.9 -0.25(-0.75x90°) OS 0.9 -0.50(-0.25x80°)

4	66	M	Painless loss of vision	OD CF +0.50 (-	OD 17	- Normal depth	OD 20.55 OS 20.31	OD 1.8	Primary UES: UES	–	+	23G PPV RD + endolaser + Siluron 2000 oiltamponnade + superotemporal sclerotomy	Uveal effusion and serous

			2 months post phaco OS and 2 months later OD (4 months post phaco)	0.25x 34°) OS CF +0.25 (-0.25x 30°)	OS 16	anterior chambers - OS Peripheral choroidal effusion, macular folds, flat inferior serous RD; OD bullous inferior RD with choroidal effusion - Echo-B-scan: OS annular choroidal detachment, inferior serous RD; OD inferior choroidal	Hyperopia	9 OS 1.96	type 2 Predisposing factor: cataract surgery				retinal detachment resolved in both eyes. Silicon oil removal after 3 months, no relapse. OD 0.3 +0.50(-0.25x 35°) OS 0.6 +0.50(-0.50x 30°)

						detachment							
5	50	F	OS visual impairment and painful eye movements and serous RD 4 months post phaco	OD 0.9 -2(-0.75x 153°) OS 0.1 -1.75(-1x145°)	OD9 OS9	- Normal depth anterior chamber, no wound leak - Retinal folds, choroidal effusion - Echo-B-scan: choroidal effusion 360°, no T-sign - CT (head): no pathology	OD 25.26 OS 25.18 Myopia	OD 0.99 OS 1.12	Primary UES: UES type 3 Predisposing factor: cataract surgery	–	+	23G PPV RD + endolaser + Silicon oiltamponnade	Uveal effusion and serous retinal detachment resolved. Silicon oil removal after 3 months, no relapse. OD 0.9 -2(-0.75x 153°) OS 0.9 -1.75(-1x145°)
6	60	M	OS gradually progressive blurred vision and OD red painfu	OD 0.4 +1.25 OS LP +0.75	OD13 OS15	- Shallow anterior chamber - Bullous choroidal	OD 22.38 OS 22.17 Hyperopia	OD 1.78 OS 1.83	Primary UES: UES type 2 Idiopathic	–	+	OS 23G PPV RD + superotemporal sclerotomy OD 23 G PPV RD + superotemporal transscleral punction	Resolution of uveal effusion and serous retinal detachment, no relaps

			14 months later			effusion ODS - Leopard spot retinal changes OS - Echo-B-scan: choroidal effusion 360° and serous RD OS								e. OD 0.9 +1.75 OS 0.3 +2.0(-0.25x 7°)
7	58	F	ODS painfull eyes, blurred vision 1 day post chemotherapy (oxaliplatin)	OD 0.9 - 1.0 OS 0.9 - 1.0	OD 26 OS 27	- Very shallow anterior chamber - Retinal folds and serous RD - Echo-B-scan: choroidal swelling - CT (head): no pathol	OD 22.30 OS 22.42 Hyperopia	OD 1.14 OS 1.27	Secondary UES Trigger: Platinum containing chemotherapy (oxaliplatin)	+	+	IV acetazolamide 500 mg (3x/day) Topical steroids (4x/day) Topical atropine	Resolution of uveal swelling and serous retinal detachment within one week. Chemotherapy with oxaliplatin was paused. No recurrences. OD 0.9 +1.0 OS 0.9 +1.25

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8	34	M	ODS Acute angle closure glaucoma after topiramate use; myopic shift	OD 0.12 - 1.50 OS 0.7 - 1.50	OD 60 OD 60	- Very shallow anterior chamber - Choroidal effusion	OD 22.19 OS 22.07 Hyperopia	OD 1.25 OS 1.19	Secondary UES Trigger: Topiramate	+	–	Oral acetazolamide 125 mg (3x/day) Topical steroids (6x/day) and oral prednisone 80 mg	Resolution of angle closure with normalization of IOP within one week. No recurrences. OD 0.8 +0.75 OS 1.0 +1(-0.50x88°)
Total		M 4 (7 eyes) F 4 (6 eyes)				Cataract surgery as predisposing factor 6/13 eyes Drug induced 4/13 eyes Idiopathic 3/13 eyes				SAC 6/13 eyes	SRD 8/13 eyes	Conservative/Medical: 8/13 eyes Surgical: 5/13eyes (substantial visual loss at presentation)	

23G PPV: 23 gauge pars plana vitrectomy for retinal detachment, AXL: axial length (mm), CF: counting fingers, CT: computed tomography, F: female, IOP: intraocular pressure, IV: Intravenous, LP: light perception, M: male, OD: oculus dexter, ODS: oculus dexter and sinister, OS: oculus sinister, RD: retinal detachment, SAC: secondary angle closure, ST: scleral thickness (posterior pole) obtained from A -scan associated with B-scan ultrasounds, > 1.7 mm noted as increased scleral thickness[38], SRD: serous retinal detachment, UES: Uveal Effusion Syndrome, UES type: Uyama classification[11]