INVITED REVIEW

Choroidal effusions- Types, presentations and management approaches: A narrative review

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ABSTRACT

Choroidal effusion is an abnormal accumulation of fluid or blood in the suprachoroidal space and encompass serous, hemorrhagic, and idiopathic (uveal effusion syndrome) variants. They remain a diagnostic and therapeutic challenge. Advances in imaging and surgical techniques have refined understanding of their pathogenesis, which is governed by intraocular fluid dynamics, Starling's law, and alterations in choroidal and scleral permeability. Serous effusions commonly occur after intraocular surgery, particularly glaucoma and cataract procedures, and may also be associated with inflammatory, vascular, neoplastic, and drug-induced causes. Hemorrhagic detachments, arising intraoperatively, postoperatively, after trauma, or spontaneously, particularly in eyes with systemic anticoagulation or fragile vasculature. Uveal effusion syndrome is rare, often linked with nanophthalmos or scleral abnormalities, and manifests as recurrent ciliochoroidal detachment with secondary exudative retinal detachment. Clinical features range from asymptomatic peripheral detachments to acute painful vision loss with expulsive hemorrhage. Diagnosis is largely clinical, aided by B-scan ultrasonography, ultrasound biomicroscopy, fluorescein and indocyanine angiography, and enhanced-depth OCT. Management is etiology-directed: many serous effusions resolve conservatively, while hemorrhagic detachments may necessitate timely drainage, vitreoretinal surgery, or adjuvant techniques such as viscoelastic-assisted drainage or perfluorocarbon liquids. Prognosis is generally favorable for serous effusions but guarded in massive hemorrhagic detachments, particularly with prolonged appositional choroidals or associated retinal detachment. This review consolidates current understanding of the types, pathophysiology, presentations, diagnostic tools, and management strategies for choroidal effusions, underscoring the importance of early recognition and tailored intervention.

Key Words: choroidal effusion, choroidal detachment, suprachoroidal hemorrhage, drainage

INTRODUCTION

Choroidal effusions or choroidal detachments (CDs), are the accumulation of fluid in the suprachoroidal space. They are broadly classified into two types of CDs – serous and hemorrhagic, depending on whether they contain transudate fluid or blood, respectively.[1] Idiopathic bilateral cases are described under an umbrella term called uveal effusion syndrome - they constitute a diagnosis of exclusion.[1] This article deals with all three broad types of effusions, their etiopathogenesis, risk factors, presentation, diagnosis, management, and prognosis.

1. A Historical perspective

Detachments of the choroid were first described by Knapp in 1868, when he enucleated an eye after cataract surgery, with the suspicion of a tumor.[2] Historically, they were often mistaken for tumours and enucleated, followed by a retrospective diagnosis of serous choroidal detachments

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(SCDs) on histopathology.[2] Due attention, however, has been paid to haemorrhagic detachments, with the oldest reported case dating back to 1700s. Vail, the first to successfully treat expulsive choroidal haemorrhage after cataract surgery, dramatically called it “bête noire”- the dreaded black beast.[3] Uveal effusion syndrome was first purported to be a distinct clinical entity by Schepens and Brockhurst in 1963.[4]

2. Anatomy of the choroid

The choroid is a pigmented, vascular connective tissue that lies between the sclera and the retina, forming the posterior part of the uvea. [5] From the retinal side towards the scleral side, the choroid comprises five sublayers:(a) Bruch’s membrane (b) the Choriocapillaris, (c) Sattler’s layer, (d) Haller’s layer, and (e) the Suprachoroidal layer, as seen in **Figure 1.** The choriocapillaris layer contains fenestrated capillaries, whereas the middle Sattler’s layer has medium-sized arterioles and venules, and the exterior most Haller’s layer has large diameter vessels.[5] While the Bruch’s membrane is thin and compact to permit nutrient and oxygen exchange with the outer retina, the suprachoroidal layer serves as a zone of transition, with its wavering thickness, encompassing several fibrous lamellae extending from the stroma of the choroid to the sclera.[6] These lamellae lie in close apposition to each other, but may become separated by fluid or blood in pathologic states.[6] Hence it is also called as the suprachoroidal space, because it acts as a potential space between the choroid and the sclera. Under physiological conditions, it contains approximately 10 microL of fluid.[9] When filled with fluid, the boundaries of the suprachoroidal space are the scleral spur anteriorly and the optic disc posteriorly. The choroid has firm attachments to the sclera at the ampullae of the vortex veins, and hence large choroidal detachments have a typical lobular appearance.[9] The attachments are more unyielding posteriorly than anteriorly, and nearly negligible between the ora and the equator, which is why CDs occur in the anterior part up to the equator.[10] Aqueous humor in the anterior chamber exits the eye via two pathways - either the trabecular meshwork or the uveoscleral. In the latter, it passes through the ciliary muscles to make way into the supraciliary and suprachoroidal spaces. Scleral outflow also accounts for a part of this pathway, directly or via the emissaries, the choroidal vessels, and then into the vortex veins.[11]

3. Pathophysiology of Choroidal Detachments

In the normal eye, the choroid and sclera lie in close apposition with each other due to the Intraocular Pressure and the arrangement of lamellar fibers.[7] In pathologic conditions that hamper the normal ocular fluid dynamics and pressure gradients, fluid collects in this potential space.[12] The presence of clear fluid in the suprachoroidal space is classified as choroidal effusion or Serous Choroidal Detachment or effusion, whereas the presence of

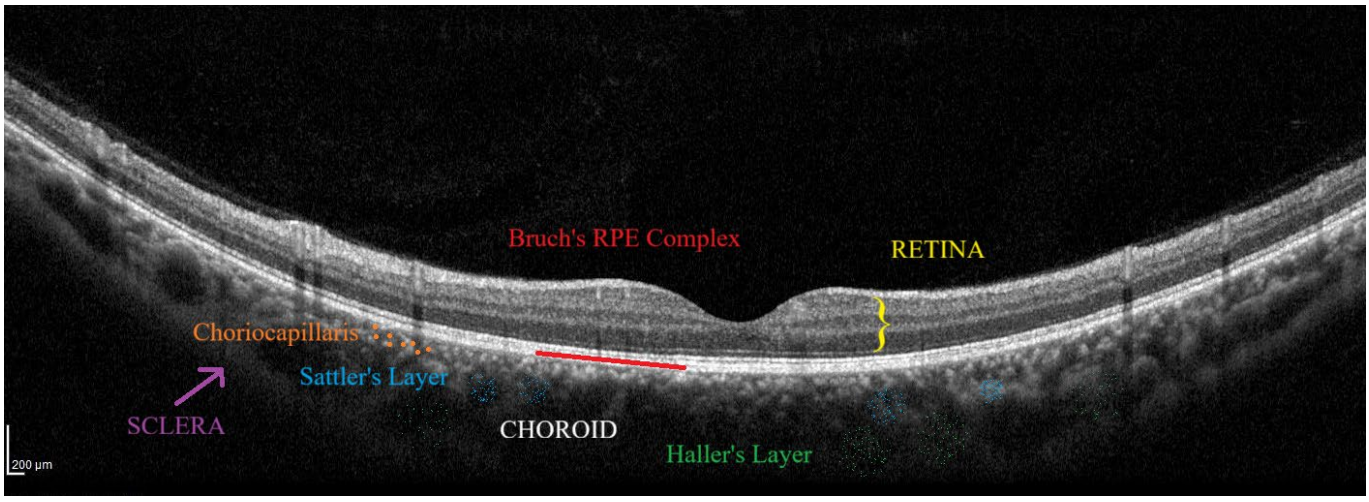

Figure 1. *EDI OCT (Enhanced Depth Imaging Optical Coherence Tomography) image of the left eye showing the layers of the choroid. From inner to outer side, the layers are as follows:(a) Bruch’s membrane (b)the Choriocapillaris, (c)Sattler’s layer, (d)Haller’s layer. The suprachoroidal layer is a potential space between the sclera and choroid.*

blood in this potential space is classified as Haemorrhagic Choroidal detachment or suprachoroidal hemorrhage.[1,19] The pathophysiology of these detachments is best explained by Starling's law,[13] and Starling's equation of capillary forces.[16] The Starling's law describes how differences in hydrostatic and colloid oncotic pressure govern fluid movements between blood and tissues.[15] Passage of small and medium-sized protein molecules through the isoporous capillary membrane is called transudation.[12] Capper and Leopold spoke of three forces that affect the choroidal coat and govern transudation.[13]

1. The tissue pressure, or the intraocular pressure, acting against transudation of fluid from the vessels.
2. The intravascular pressure which promotes transudation.
3. The oncotic pressure, or plasma colloid osmotic pressure, is exerted by the plasma proteins, and draws fluid from the tissues into the vessels.[13]

The Starling equation is defined as: Net driving pressure = $Kf(Pc - Pi) - \sigma(\pi c - \pi i)$

where Kf = filtration co-efficient,

Pc = capillary hydrostatic pressure,

Pi = interstitial hydrostatic pressure,

σ = reflection co-efficient (correction factor),

πc = capillary oncotic pressure

and πi = interstitial oncotic pressure[2]

Capillary hydrostatic pressure favours the movement of fluid from the vessels to the extravascular compartment, while plasma osmotic pressure pulls fluid into the vascular tree,[13] as shown in **Figure 2**. Any process that promotes the movement of fluid from choroidal capillaries into the interstitial space, can lead to choroidal detachments.[1] Hypotony is the most essential and common element in the development of CDs.[17] It increases the pressure differential and favours transudation.[12] This mechanism in patients with raised episcleral venous pressure, those with already raised IOP and in intraoperative effusions that form rapidly, is that when the pressure difference is greater, it leads to "molecular sieving" due to accelerated filtration.[17] Quick filtration rate reduces the chance of large molecules entering and passing through the pores in the capillary membrane. However, in delayed and slow-forming choroidal effusions where the filtration rate is unrushed, relatively large molecules pass through.[18]

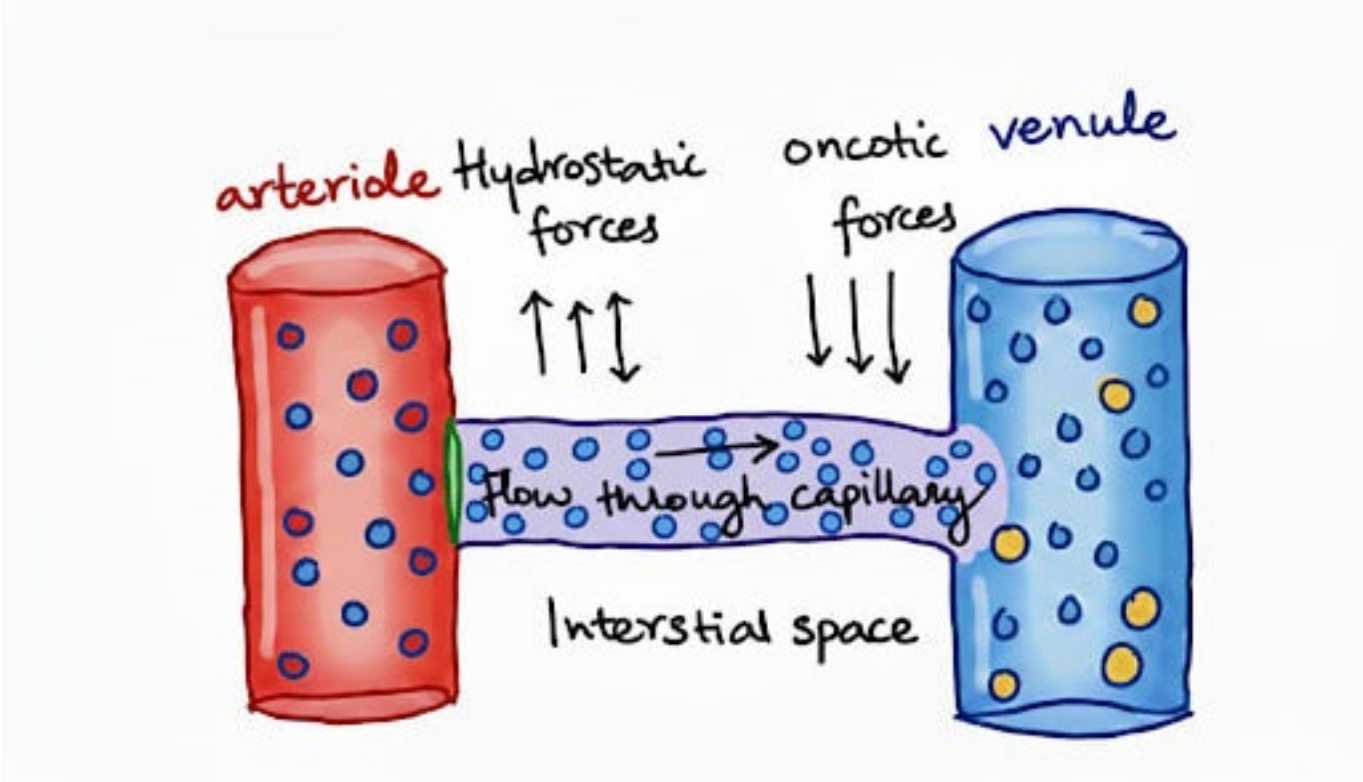

Figure 2. *The Starling's law of capillary forces. Capillary hydrostatic pressure pushes fluid into the interstial space, while the plasma oncotic pressure pulls fluid into the blood vessels.*

Rupture of capillaries causes the leakage of red blood cells into the suprachoroidal space, resulting in hemorrhagic detachments. They may also often start as innocuous serous choroidal effusions which then expand massively, causing the choroidal and ciliary vessels stretch and rupture.[12] While both the long end short posterior ciliary arteries can bleed, the former are more vulnerable because of short connections between the scleral exit and the outer choroid, which easily rupture during a detachment.[9]

Inflammation is another mechanism whereby the increase in permeability of the choroidal capillaries triggers the movement of blood and fluid from the capillaries and into the interstitial space.[12]

In UES, the primary pathology lies in the sclera rather than the choroid. Impeded transscleral intraocular fluid outflow compresses the vortex veins, leading to congestion of the choroidal veins, fluid accumulation and subsequent ciliochoroidal detachment. Long-standing SCDs in uveal effusion cause retinal pigment epithelium decompensation and failure of its pump mechanism, with subretinal fluid accumulation, leading to exudative retinal detachments.[4]

4. Etiology and Risk factors

While the detachments have been explained separately, it must be remembered that there can be significant overlap in the etiology, as often the hemorrhagic detachments may start as innocent serous effusions.[19] For instance, aging causes changes in the vessel wall with increased capillary permeability, which explains the significantly higher incidence of serous CDs and SCH in older patients.[9] The etiology of serous choroidal detachments has been listed in **Table 1.**

A. Serous Choroidal Detachments/ Effusions

a. Post Surgery:

SCDs are a known complication after any surgery that causes hypotony. Incidence reported after cataract surgery is 4.6%.[20] SCD can occur after glaucoma surgeries like trabeculectomy as well. It is attributed to overfiltration, bleb leak, impaired aqueous production due to antimetabolite toxicity or inflammation.[16] The documented rates of postoperative serous choroidal effusion have a wide range from 7.9 to 18.8% after trabeculectomy.[1] Intraoperative effusions are less common but have been reported in high-risk patients, for instance, those with raised EVP and Sturge Weber syndrome. [18] SCDs have also been reported after Ahmed glaucoma valve implantation[21] and cyclocryotherapy.[16]

b. Inflammation and other Ocular Disorders

SCDs have also been described in patients with posterior scleritis[16] and uveitis.[22] They are also known to occur in VKH and sympathetic ophthalmia.[16] Rhegmatogenous retinal detachments can be associated with serous choroidal detachments.[23] An important risk factor is prolonged duration of the RRD, leading to breakdown of the blood-retinal barrier with persistent low IOP and severe immune reaction. Other notable risk factors for SCD in RRD are multiple retinal breaks, pseudophakia, aphakia, and total RD. The prevalence of choroidal detachments among RRD ranges from 2% to 18.79%.[23]

c. Vascular

SCDs have been described in caroticocavernous fistulas (CCF). Low mean arterial pressure, high venous pressure, and subsequent tissue hypoxia are believed to be the cause

Table 1. *Etiology of Serous Choroidal detachments*

Post Surgery/ laser	Trabeculectomy, Cataract Surgery, Keratoplasty, Cyclocryotherapy, Ahmed Glaucoma Valve, Post Panretinal Photocoagulation, Post YAG Peripheral Iridotomy
Inflammation and other ocular disorders	Episcleritis, Scleritis, Vogt-Kayanagi-Harada disease, Sympathetic ophthalmia, Rhegmatogenous retinal Detachment
Neoplastic	Metastasis, Melanoma, Lymphoma
Vascular	Caroticocavernous fistula, Cavernous Sinus Thrombosis
Miscellaneous	Drug induced, Preecclampsia

of the altered choroidal circulation.[24] Choo et al described SCDs attributed to impaired vortex vein drainage in a patient with underlying cavernous sinus thrombosis.[25]

d. Drug-induced

Topical antiglaucoma medications have been documented to cause SCD in isolation or in the setting of postoperative hypotony.[16] They been reported to occur with multiple oral medications like topiramates and sulfonamides.[16] Intravenous Daratumumab, used in the treatment of multiple myeloma, can rarely cause SCD. [26] Golash and Almeida described SCD and exudative detachments with uveitis, occurring secondary to severe hypotony caused by the use of Pembrolizumab in a patient with metastatic melanoma. .[27] Drugs that cause choroidal detachments are listed in **Table 2.**

Table 2. *Drugs that have associations with Serous Choroidal detachments*

	Drugs
Topical	Cidofovir Dorzolamide Bimatoprost Latanoprost Timolol
Oral	Topiramate Indapamide Methazolamide Acetazolamide Mefanemic acid Alendronate Hydrochlorthiazide Chlorthalidone Sildenafil Bupropion Escitalopram Phendimetrazine
Intravenous	Daratumumab Pembrolizumab

e. Neoplastic

SCDs can occur in patients with lymphoma and carcinoma.[16]

f. Miscellaneous

Mathers and Moodie described a patient who developed ipsilateral recurrent SCD after undergoing multiple surgeries for ipsilateral maxillary sinusitis- the proposed mechanism was inflammatory edema spreading to the orbit; however, the SCDs continued to recur even when the sinusitis subsided.[28] SCDs have been reported in HIV positive patients.[16]

B. Hemorrhagic Choroidal Detachments/ Suprachoroidal Hemorrhage (SCH)

SCH can be classified according to its etiology as surgical, trauma-related, or spontaneous.[29,30]

a. Peri-operative SCH

Suprachoroidal hemorrhage during surgery may be acute intraoperative "ASCH" or delayed postoperative "DSCH"- 8 weeks is the cut-off to distinguish the two.[29] The former occur more often in cataract surgeries that have complications intraoperatively, and the later after surgeries for glaucoma.[30] The incidence of intraoperative SCH for any eye surgery is roughly 0.29%.[29] The major ocular, systemic and perioperative risk factors for SCH have been listed in **Table 3.**

Table 3. *Risk factors for Suprachoroidal Hemorrhage*

	Risk factors
Ocular	High myopia Raised episcleral venous pressure Glaucoma History of SCH in fellow eye Aphakia Pseudophakia Prior intraocular surgery Age related macular degeneration
Systemic	Elderly age Atherosclerosis Tachycardia Liver Disease Hypertension Blood dyscrasia or coagulation defect Diabetes mellitus
Surgical risk factors	Retrobulbar anaesthesia/ General anaesthesia Sudden drop in IOP Valsalva maneuvers such as sudden coughing Posterior Capsular Rupture Vitreous loss Prolonged surgical time Intraoperative raised blood pressure or pulse rate

A. Cataract Surgery

Although modern phacoemulsification employs minute and self-sealing incisions with a closed irrigation-aspiration system, SCH can still occur, especially if wound integrity is compromised. Sudden removal of the phaco handpiece from the eye in a poorly made surgical wound that is not self-sealing causes hypotony, triggering SCH. The incidence of SCH in phacoemulsification has been reported to be 0.013- 0.5.%.[29]

Other important risk factors in cataract surgery include surgical aphakia (lack of lens and zonular support permits greater stretching and separation of the uvea from the sclera), myopia (decreased scleral rigidity and fragile vasculature of the choroid).[9] Increase in the retro-orbital venous pressure after local anaesthesia can impede flux in the vortex veins and, therefore, contribute to stasis of blood in choroidal vessels with subsequent hemorrhage.[29]

B. Glaucoma surgery

Glaucoma surgery is the most common surgery related to SCH. Incidence varies from 0.7% to 6.1%. The incidence is twice as high in tube shunt procedures compared to trabeculectomy.[29]

C. Corneal surgery:

SCH can also occur during or after penetrating keratoplasty. The incidence of intraoperative or immediate postoperative SCH during PK has been reported as 0.05%. In contrast, it has only been rarely reported after lamellar keratoplasty.[29]

D.Vitreoretinal surgery

In vitreoretinal surgery, the incidence of SCH varies from 0.06 to 4.3%.. Preoperative rhegmatogenous retinal detachment and scleral buckling procedures constitute the major risk factors in this specific group of patients. Vitrectomized eyes are at increased risk due to the absence of vitreous support and an increased vulnerability to IOP fluctuation. [29]

b. Trauma-related SCH

Chen et al. categorized patients with choroidal injuries after ocular trauma and reported that massive suprachoroidal hemorrhage occurred in 4.0% of these patients, with an unfavorable outcome in 64.9% of those eyes.[31]

c. Spontaneous SCH

Spontaneous SCH is rare but has been reported with both ocular and systemic risk factors. Systemic anticoagulation is the most common risk factor associated with use of thrombolytic agents like reteplase and heparin, and anticoagulants like warfarin. It has been reported in a patient with type 1 chronic myeloid leukemia and lymphoplasmacytic lymphoma. Valsalva maneuvers can also cause spontaneous SCH.[32]

C. Uveal Effusion Syndrome

Uveal Effusion syndrome is synonymous with Idiopathic Ciliochoroidal Effusion. It is a rare condition and is diagnosed by exclusion.[33] Uyama et al. described three types of UES, as mentioned in **Table 4.**[4] Nanophthalmos is a significant risk factor in type 1 UES.[33] It is a condition with structurally normal eyes that have shortened anterior and posterior segments, with secondary thickening of choroid and sclera.[4]

5. Clinical Symptoms and Signs

A. Serous Choroidal Detachments/ Effusions

SCDs are asymptomatic or cause painless vision loss or visual field loss.[12] There can be folds in Descemet's membrane, with a shallow or flat AC.[17] The detachments are convex-shaped, have well-defined borders, and the normal

Table 4. *Types of Uveal Effusion Syndrome (Uyama et al)*

	Type 1 UES	Type 2 UES	Type 3 UES
	Nanophthalmic	Non Nanophthalmic	Non Nanophthalmic
Axial length	Short <20.5mm	Normal or Slightly decreased but >20mm	Normal or Slightly decreased but >20mm
Hypermetropia	Present	Absent	Absent
Scleropathy	Present	Present	Absent
Management	Surgery preferred	Surgery preferred	Medical Management

retinal vessels course over them. Since the separation of the ciliary body relaxes the zonules and increases curvature of the lens, and there is anterior movement of the iris lens diaphragm – this can cause myopia and angle closure glaucoma.[16] **Figure 3** shows the typical appearance and presentation of a serous detachment.

B. Hemorrhagic Choroidal Detachments/ Suprachoroidal Hemorrhage (SCH)

SCH that occurs during the intraoperative period presents with sudden shallowing of the AC, accompanied by a rapid rise in IOP after the initial hypotony. A black shadow with loss of red reflex is seen, and if not immediately managed, can lead to a catastrophic expulsive hemorrhage with prolapse of intraocular contents from the surgical wound site. Severe pain is present due to stretching of the long ciliary nerves.[29] Delayed onset SCH usually presents around one week after surgery- the patient complains of sudden painful vision loss, and nausea and vomiting secondary to raised IOP. If there is sufficient media clarity, dark brown convex elevations can be seen on a retinal examination,[29] as shown in **Figure 4**. Spontaneous SCH is rare and presents with similar symptoms, shallow AC, and a dilated and fixed pupil.[29,32]

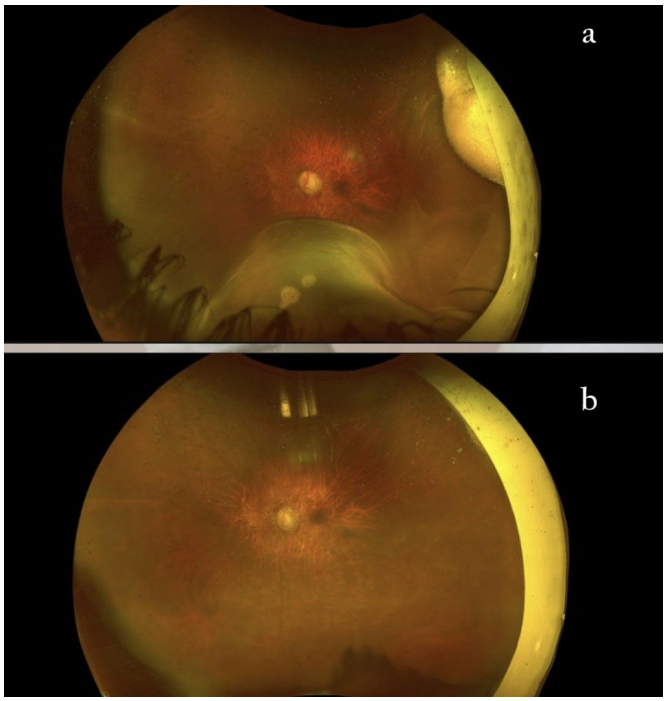

Figure 3.*(a) Color Optos fundus photograph of the Left Eye of a 6 year old male patient with bilateral congenital glaucoma who underwent combined Trabeculotomy and Trabeculectomy in the Left Eye. He developed serous choroidal detachment (black arrow) in the left eye 7 days after surgery, with an intraocular pressure of 4 mmHg. As there was no bleb leak or overfiltration, he was managed conservatively with oral and topical steroids. (b) The same patient 2 months after surgery. The choroidals resolved leaving behind areas of pigmentation (black arrow). The IOP was 14mmHg at this visit.*

C. Uveal Effusion Syndrome

UES usually affects young adult males and presents with bilateral annular ciliochoroidal detachment. High hypermetropia is seen if nanophthalmos is present. Anterior segment may show mildly dilated episcleral vessels, blood in Schlemm/s canal, and mild vitreous inflammation. The ora serrata can be seen without scleral depression. Shifting fluid sign and exudative detachments may be present. Leopard spots due to pigment clumps in the fundus are seen in long-standing cases.[33] **Figure 5** shows the typical presentation of UES.

6. Diagnosis

Choroidal detachments are a clinical diagnosis, but imaging may help in hazy or opaque media, to look for resolution, any other underlying pathology, an associated retinal detachment and to differentiate between the nature of the detachment.[9]

a. Ultrasound B scan

Choroidal detachments are classically described as "smooth, convex, dome-shaped elevations." They lack mobility on dynamic B scan, unlike the aftermovements seen in a PVD.[34] The CDs may be restricted to one or more sectors, demarcated by attachments that correspond to vortex vein insertions.[9] The space within the dome is hypoechoic or hyperechoic, depending on whether the CD is serous or haemorrhagic, respectively. *'Kissing choroidals*" may be seen if they are large enough to appose each other. [16] The corresponding A-scan shows a steeply rising, double-peaked, wide spike, characteristic of choroidal detachment, with lower reflective spikes in the suprachoroidal space in SCH, indicating clotted blood.[9] The differences between Choroidal detachment, Retinal Detachment and Posterior Vitreous Detachments on B scan are listed in **Table 5.**

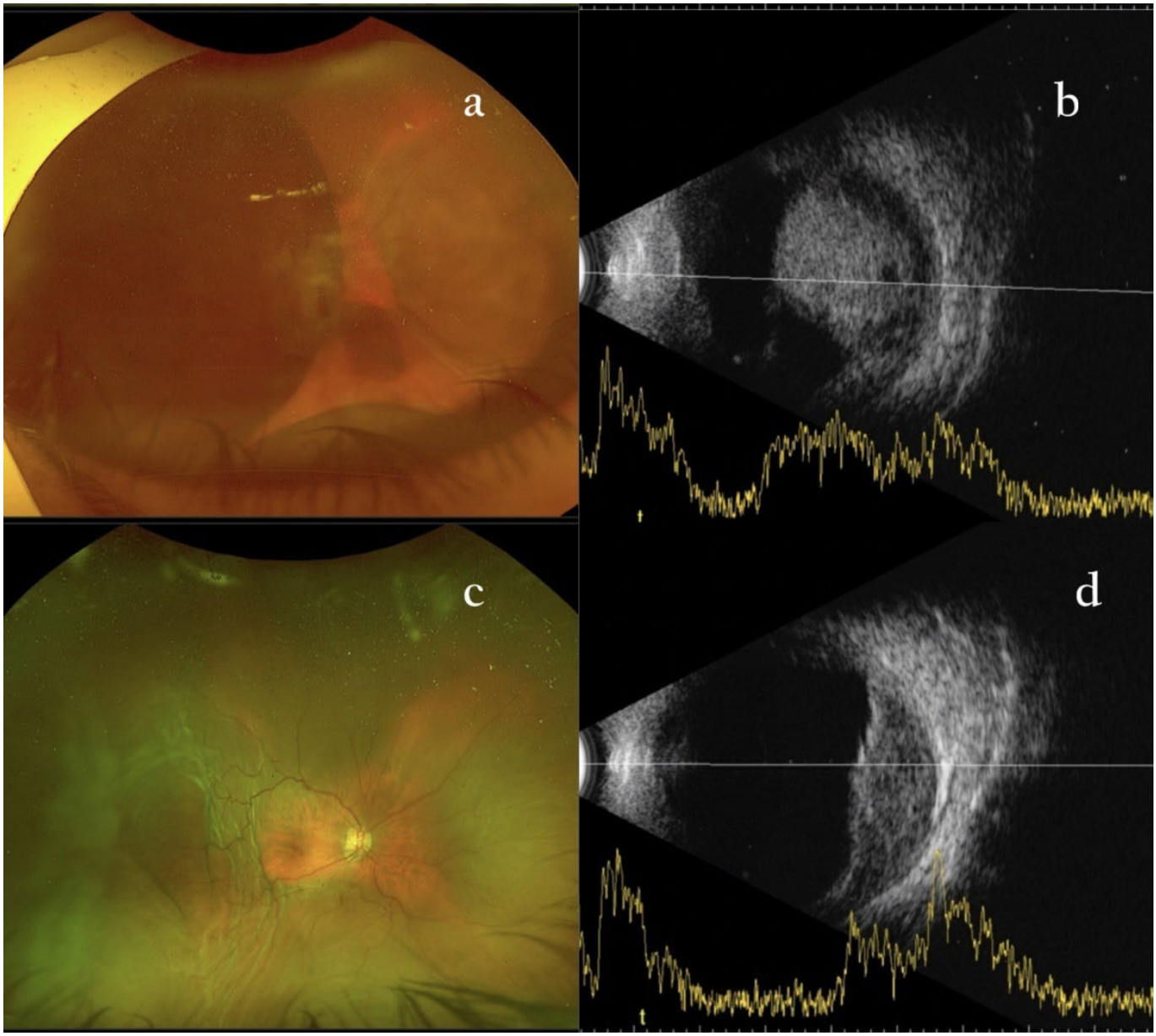

Figure 4 *shows (a) Optos Color Fundus Photography of the Right eye of an 80 year old female patient who developed acute intraoperative suprachoroidal hemorrhage during eventful cataract surgery with PCR. The patient was left aphakic and the incision was sutured. She was started on oral steroids, atropine and predforte. She had undergone Bilateral YAG Peripheral Iridotomy 2 months before the surgery in view of Bilateral Primary Closure . (b) B scan of the same patient showing massive hemorrhagic choroidals (c) Optos Color Fundus Photo shows resolving SCH with retinal folds, 1 month after the primary surgery(d) Corresponding B scan at the same visit shows resolving SCH, with decrease in its height*

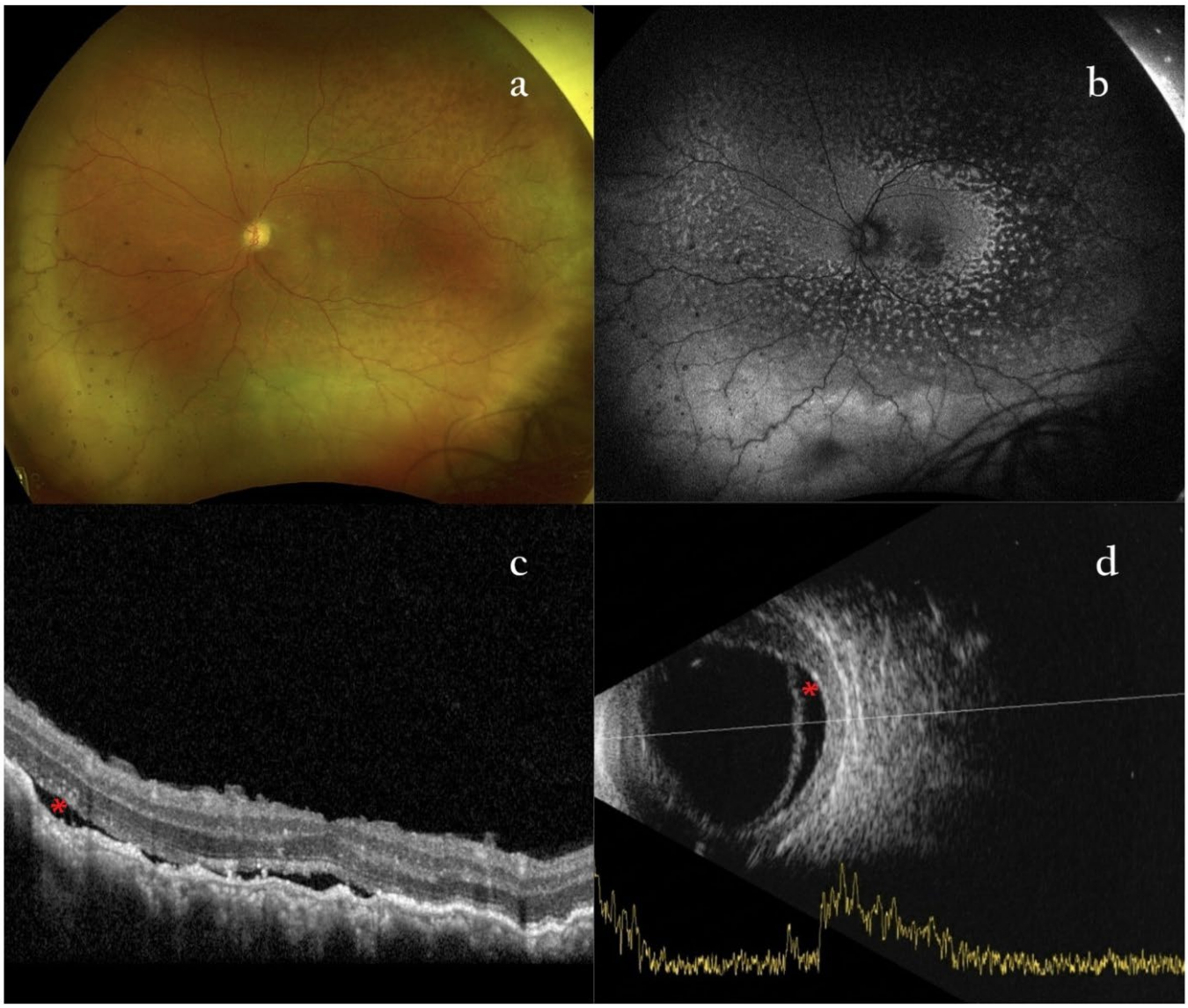

Figure 5 *shows (a) Optos Color Fundus Photography and Fundus Autofluoroscence of 54 year old male patient who presented with Uveal Effusion syndrome, showing characteristic leopard spots. (c) OCT image and (d) B scan showing exudative retinal detachment (red asterisks). The B scan also shows significant thickening of the choroid. The patient did not have nanophthalmos, and he was treated with systemic steroids, however he was lost to follow up.*

Table 5. *Differentiating Choroidal, Retinal and Posterior Vitreous Detachments on Ultrasonography*

	Choroidal Detachments	Retinal Detachments	Posterior Vitreous Detachment
Shape	Thick, Smooth, Convex, Dome shaped elevations	Thin, undulating membrane	Thin, irregular, mobile membrane
Mobility on kinetic scan	Immobile	Mobile	Highly mobile with aftermovements
Attachments	Attachment at scleral spur	Attachment at disc and ora serrata	Can have attachment at disc (Weiss ring)
Gain	Persists at low gain	Disappears at low gain	Disappears quickly at low gain
Location	Quadrantic, rarely kissing choroidals	Localised or Total	Free floating in the vitreous cavity

Different types of CDs and RD on B scan are shown in **Figure 6.**

Serial Ultrasound B scans can also be used to monitor clot liquefaction in haemorrhagic CD, as the highly irregular internal reflectivity progressively decreases with each visit, eventually appearing as low reflective, regular, mobile dots on kinetic B scan as the clot undergoes complete lysis in around 7-14days.[9,29] In UES, the sclera in type 1 and type 2 patients will demonstrate thickening, in addition to the ciliochoroidal effusions. B scan has been used pre and intraoperatively to identify areas of maximum thickening to target placement of sclerostomies used in the treatment.[33]

b. Computed Tomography

Haemorrhagic CDs have higher attenuation values compared to serous CDs[9,29]

c. Uveal Effusion Syndrome/ Idiopathic ciliochoroidal effusion syndrome

In UES, ICG-A shows diffuse granular hyperfluorescence in the early phase with dilated choroidal vessels. There is persistent hyperfluorescence in the late phase due to increased choroidal vessel permeability.[33]

d. Fundus Fluorescein Angiography

FA in UES shows areas of hypofluorescence within hyperfluorescence corresponding to leopard spots, without leakage.[33]

e. Optical Coherence Tomography

Enhanced Depth Imaging or EDI-OCT in UES may show areas of increased choroidal thickness. Focal RPE thickening can be seen in areas of leopard spots.[33]

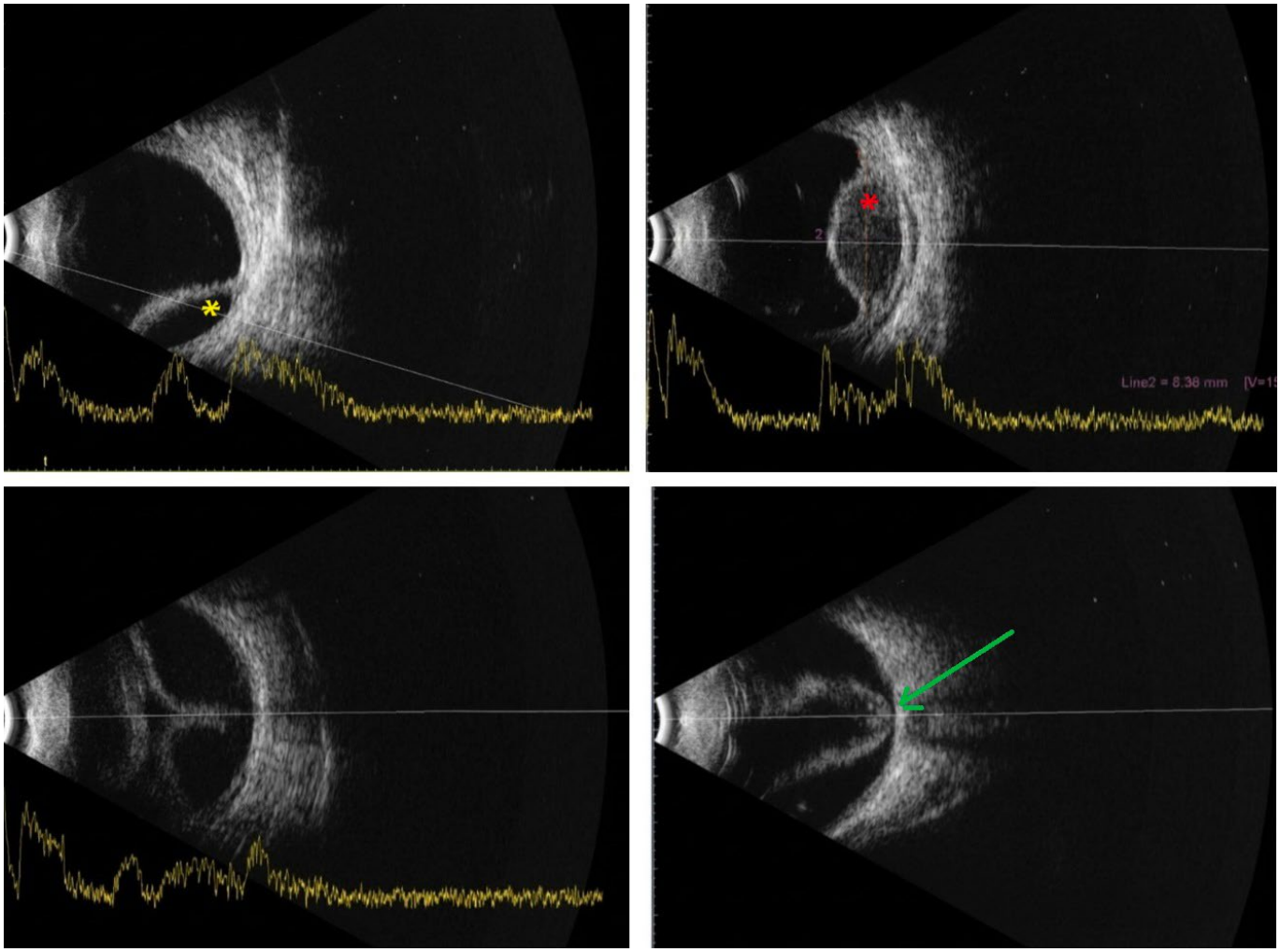

Figure 6. *B scan ultrasonography images showing (a) Serous choroidal detachment, with characteristic dome shape and hypoechoic space within the dome (yellow asterisk) (b) Hemorrhagic choroidal detachment with heterogenic areas and hyperechoic space within due to blood (red asterisk) (c) Kissing choroidal detachments apposing each other (d) Retinal detachment, with characteristic attachments at disc(green arrow) and ora serrata.*

7. Management

A. Serous Choroidal Detachments/ Effusions

The treatment is dependent on the etiology. Often, no treatment is needed for serous detachments other than observation.[2] Seidel's test should be done to rule out wound leak or bleb leak in operated patients, and if present, AC reformation or suturing needs to be performed.[1] Topical cycloplegics have been tried. Systemic steroids can be given - the justification for there is that uveal inflammation due to surgical trauma or otherwise, aggravates or precipitates choroidal detachment, and steroids counter the same.[10] Sclerotomy and surgical drainage of the fluid may be rarely needed, only in very large or non resolving serous effusions.[1] **Figure 7** shows the algorithm while managing serous effusions.

B. Hemorrhagic Choroidal Detachments/ Suprachoroidal Hemorrhage (SCH)

a. Intraoperative SCH

The first step in suspicion of intraoperative SCH is to suture all incisions and give digital pressure promptly. The eyelid speculum should be loosened or removed. Hyperosmotic agents like Intravenous mannitol can help lower the IOP. Reverse Trendelenburg position with head end elevation can decrease bleeding by decreasing flow to choroidal vessels. AC reformation with air or saline helps to prevent entrapment of vitreous in the wound. Immediate primary drainage sclerotomies, once popularised by Verhoeff, are no longer advocated.[9,29]

Medical management in postoperative period is chiefly directed at IOP control, with topical medications such as

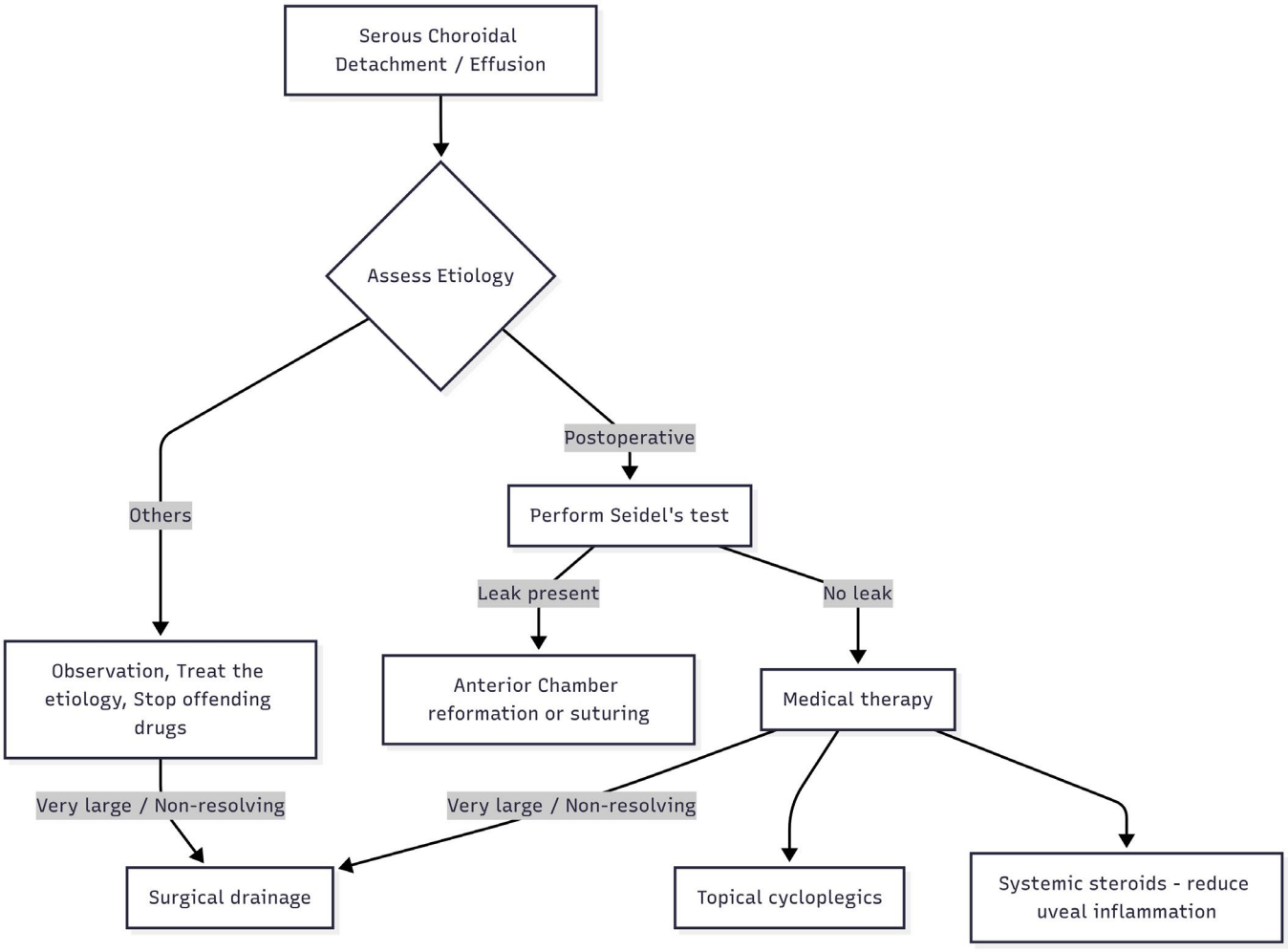

Figure 7. *Management Algorithm for Serous Choroidal Effusions*

aqueous suppressants and/or β-blockers, and oral carbonic anhydrase inhibitors.[29] Topical steroids are added to control inflammation, and topical cyclopegics to lessen the pain. Systemic steroids control inflammation and additionally aid in clot lysis[29] They also activate platelets and prevent rebleeding.[32] The use of aspirin and non-steroidal anti-inflammatory drugs (NSAIDs) is contraindicated, as they can worsen bleeding. [9,29]

Secondary surgical management is indicated in kissing choroidals with retinal apposition, large macula-involving SCH, uncontrolled IOP despite maximum medical management, severe eye pain, flat AC, lens dislocation, non-resolving vitreous hemorrhage, vitreous incarceration in the wound, and RD. [9,29,30] Surgical management may be either drainage procedures in isolation to evacuate the bleed, or in combination with vitreoretinal surgery for any associated conditions like VH or RD.[29] Management is summarised in **Figure 8**.

The timing of the second surgery is controversial. Waiting 10–14 days for the clot to lyse helps with better drainage.[9,29] However, early intervention has better outcomes in certain instances – for example, in kissing choroidals or in the presence of vitreous incarceration.[29]

1. Drainage Procedures :

The motives of surgery are to drain sufficient blood to prevent or relieve appositional choroidals, to lower or normalise IOP, reverse forward movement of the on lens-iris diaphragm and prevent or relieve angle closure. Uncomplicated SCH does not require additional VR surgery. A complete drainage of the blood may not be possible and is not mandated either (see the video: https://dergisi.org/retinavitreus/uploads/video/CD-drainage.mp4).[36] The surgery begins with a limited or 360-degree conjunctival peritomy, depending on the extent of SCH. The recti muscles ought to be isolated, to have view of all quadrants. Vortex veins should be identified and avoided, as the sclerotomies are to be made anterior to them. Drainage

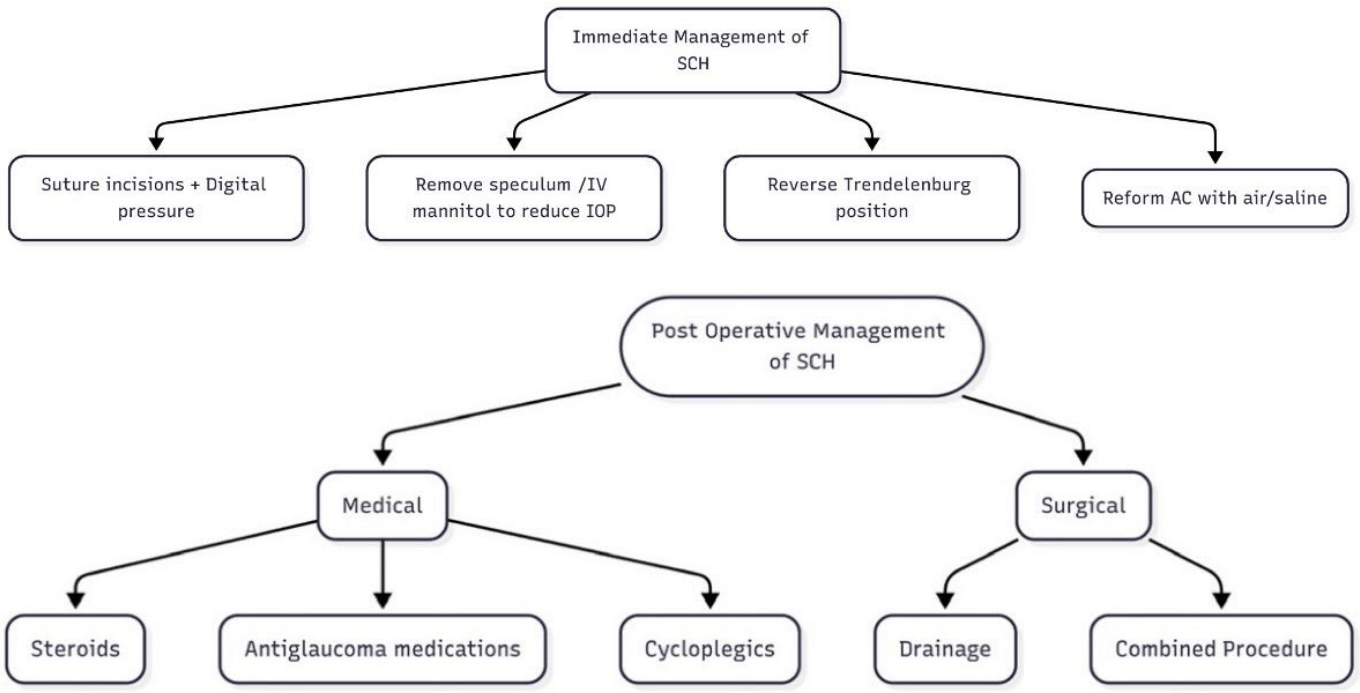

Figure 8. *Management Algorithm for Hemorrhagic Choroidal Effusions*

sclerotomies are usually of 1–2 mm in size and are made at a distance of 4–10 mm from the limbus, in a radial manner in the quadrant of the highest choroidal elevation.[29] IOP during the surgery is maintained using an AC maintainer or the placement of pars plana infusion cannula.[29,3] Following the full-thickness scleral incisions, dark blood gushes out- this process can be hastened by light pressure on the sclera. Sclerotomies may or may not require sutures.[36] Modified "posterior passive drainage," with multiple sclerotomies made at 10-15mm posterior to limbus, has been described by Boral and Agarwal, using 23 G or 25 G cannulas. [37] The advantages are minimal scarring and a more complete drainage; however, the process is slower and has limited ability to drain clotted blood.[37] The 23 or 25 G trocar cannulas can be used to perform transconjunctival sclerotomies as well. The trocar used to make the entry should be directed at a 15-20 degree angle, tangentially, so that full-thickness penetration does not occur and it reaches the suprachoroidal space.[36] When using valved cannulas, one can use forceps or vents to keep the valve open and inject vitreous substitutes, ensuring a speedy evacuation of blood.[29] The injection of gas, air, balanced salt solution, or even viscoelastic material to assist in the drainage of blood has been described.[9]

Nadarajah et al described a technique where they used two full-thickness scleral flaps made at the equator in inferonasal and inferotemporal quadrants for drainage after 180-degree peritomy, followed by the injection of 100% perfluoropropane gas to help evacuate the SCH.[38] However, they suggested that the use of a non-expanding gas bubble or silicone oil is not helpful for passive drainage, as the positive pressure from these cannot be maintained once the hemorrhage drains.[3]

Kurup et al. performed passive choroidal drainage using sclerotomies made at 4mm and then injected viscoelastic into the posterior segment in patients with appositional SCH.[39] The viscoelastic was left behind inside at the end of the procedure, with reasonable view to the posterior pole but an IOP in the high 20s in the postoperative period.[39]

2. Combined Procedures – External Drainage and Vitreoretinal Surgery

For SCH complicated with vitreous hemorrhage, retinal incarceration, retinal detachment, or dislocated lens, combined procedures, ie, drainage of SCH, along with VR surgery, are indicated.[9,40] Before making the pars plana incisions for VR surgery, the drainage procedure must ideally be performed first, as the SCH causes anatomic distortion, and hence creation of ports may be problematic. During PPV, Perfluorocarbon liquids can be instilled in the vitreous cavity – they have a specific gravity higher than that of water and sink to the posterior pole and flatten the retina when injected in a patient in the supine position. Their maximum tamponading effect is also in the posterior direction, and hence they force out all other ocular fluids as well as hemorrhage out from the eye, from the anteriorly placed sclerotomies. The PFCL can be removed either via a fluid-fluid exchange or a liquid-air exchange.[9] After PFCL removal, and at the end of VR surgery, silicone oil can be used for endotamponade – it provides good visualisation during vitrectomy. It is anti-inflammatory and protective against proliferative vitreoretinopathy. It is also believed to maintain IOP in the long term, protective against the hypotony that usually exists in SCH patients. It also minimises the risk of rebleeding.[41]

Rizzo et al. described a technique in 9 patients, where they left the PFCL in situ during primary surgery, with face-down positioning, and performed a second surgery to remove it approximately two weeks later, during which they injected silicone oil.[42] At six months after the second surgery, they did not observe any recurrence, retinal detachment or PVR.[42]

Karaca et al described active drainage of the blood 25-gauge sclerotomies were made, and after initial passive drainage, a butterfly needle was inserted to the aspiration port of the vitrectomy suite, and the program was set to extrusion mode, ensuing active aspiration of the blood.[43] PPV was then performed, followed by Silicone oil or gas (C3F8 or SF6) injection as endotamponade after air–fluid and air–perfluorocarbon liquid exchange.[43]

Suprachoroidal Injection of t-PA

Recombinant t-PA increases plasminogen binding to the fibrin clot, speeds up clot lysis and has negligible systemic risks.[44] Fei et al described direct injection of t-PA into the suprachoroidal space.[45] They used alteplase and performed a combined procedure the next day. The choroid was attached during follow-up.[45]

Chai et al. used subtenon's urokinase injection, followed by a combined procedure, in a series of 4 patients with SCH, and obtained relatively good anatomical success [46].

C. Uveal Effusion Syndrome

Medical management:

A multitude or a combination of agents have been tried with variable success. Steroids and NSAIDs (both oral and topical) have been used as they counter the inflammation. PG analogues have been used, they act by decreasing the intraocular pressure. Additionally, PG analogues increase activity of scleral metalloproteinases and may reduce collagen levels, permitting greater fluid outflow through the abnormal sclera. Carbonic anhydrase inhibitors, such as Acetazolamide, have been used. The mechanism is that they inhibit the RPE pump mechanism and trigger outward flow; however, they have also been implicated in causing effusions and require close monitoring when used. The use of anti-VEGF agents in UES has been documented as well; however, the exact mechanism of their action in UES is unclear. Type 3 UES has shown limited or variable responses to surgical management, and as such, medical therapy is preferred in these patients.[33]

Surgical management :

The preferred surgical techniques are partial-thickness or full-thickness sclerotomies. Vortex vein decompression is no longer recommended due to risks of hemorrhage and vortex vein amputation. [33]

Quadrantic Partial Thickness sclerotomy

This procedure was first described by Gass and Johnson.[47] Rectangular, partial thickness sclerectomies of half to one third depth and size 5 X 7 mm, were made in each quadrant. The location was anterior to the equator and outside the meridian of each vortex vein, to avoid its intrascleral course. At the center of this sclerectomy bed, a linear sclerostomy (of size approximately 2 mm) was made.[47]

Extensive circumferential partial-thickness sclerectomy

Mansour et al performed 90% depth scleral windows, with excision performed over 3 and 1/4 quadrants.[48] They excluded the superior-temporal quadrant, not to damage the superior oblique.[48]

Full-thickness sclerotomy

Kong et al. performed full-thickness scleral incisions between the recti, at a distance of 6-8mm from the limbus.[49] The scleral flap was loosely sutured at two corners for loose approximation to permit drainage.[49]

Other modifications

ExPRESS Shunt has been used to shunt fluid in UES from the anterior chamber to the subconjunctival space. The use of mitomycin C has also been described - it helps reduce scarring of the sclerectomy bed.[33]

Prostaglandin analogues may be beneficial in UES, as they increase uveoscleral outflow. Derek et al reported the resolution of bilateral effusions in 3 patients with their use. [8] Targeted use of periocular steroids (eg., sub-tenon's triamcinolone) can be considered as another less invasive intervention for persistent UES, especially after filtration surgeries.[4]

8. Prognosis

SCDs usually follow a benign course, resolving with conservative therapy, with no significant reduction in best corrected visual acuity. [1]Large and persistent choroidal detachments can have immediate and long-lasting vision-threatening sequelae, especially when there is an associated hypotony and maculopathy.[50] Factors associated with poor prognosis in SCH are low preoperative visual acuity, kissing choroidals with prolonged apposition, vitreous incarceration, 360-degree SCH, and associated RD.[29] 30% of eyes with SCH eventually have no light perception if untreated.[36] Eyes with limited SCH have better outcomes.[30] Patients with nonspontaneous SCH and who received systemic steroids usually achieve greater improvement in logMAR VA and anatomic success.[34] Factors associated with favourable and unfavourable long term outcomes in SCH have been listed in **Table 6**.[14,34] In UES, after surgery, the resolution of subretinal and/or supraciliochoroidal fluid occurred within 6 months in 83% after one procedure and in 96% after two or more procedures. Recurrences are reported in 23% of patients after surgery - they may resolve spontaneously or require resurgery. [47].

Table 6. *Factors associated with Long term outcomes in suprachoroidal hemorrhage [14],[34]*	
Favourable	Unfavourable
Traumatic or Perioperative SCH	Spontaneous SCH
Limited SCH	Massive SCH
Treatment with Systemic Steroids	Kissing Choroidals
Non Corneal Precipitating surgery	
Post Pars plana vitrectomy tamponade with air, C3F8 or Silicone oil	

9. Take home message

Choroidal detachments have a wide and varied etiology. While serous detachments are often self-resolving, attention must be paid to the larger detachments, which can cause angle closure or turn hemorrhagic.[12,19] The haemorrhagic detachments may occur most commonly in the setting of surgery, but can also occur after trauma, or very rarely, spontaneously.[29,30] During surgery, if not immediately managed, they can cause an expulsive bleed. A second surgery, in addition to drainage procedures, is performed for complicated SCH. [9,40] Uveal effusion syndrome forms a rare, distinct entity and is an idiopathic condition with bilateral ciliochoroidal effusions. Both medical and surgical treatments have been described. Medical management is preferred in type 3 UES.[33]

10. Methods of literature search

A PubMed search was done, and all articles were retrieved using the keywords “choroidal detachment”, “choroidal effusion”, “suprachoroidal hemorrhage”, “uveal effusion”. Articles published in non-peer-reviewed journals and non-English language journals were excluded. The text of the residual articles was studied in detail to compile the present review.

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