Suprachoroidal Hemorrhage: Risk Factors and Diagnostic and Treatment Options

by Dimosthenis Mantopoulos, MD, PhD; and Howard F. Fine, MD, MHSc

Suprachoroidal hemorrhage (SCH) is a clinical entity that was first reported in the literature more than 1 century ago. Nevertheless, its diagnosis is frequently stress-provoking, both for the patient experiencing deep and boring pain in the eye as well as for the doctor, who is sometimes uneasy about the exact etiology and clinical course of the condition. The prevalence of certain risk factors, such as the use of anticoagulants and aging of the population, are rapidly increasing, and for that exact reason the modern ophthalmologist should be prepared to promptly identify and efficiently manage this condition.

The proposed pathophysiology in the development of SCH implicates a combination of mechanisms, including acute ocular hypotony, uncontrolled systemic cardiovascular risk factors, fragile choroidal vasculature, elevated episcleral, and/or choroidal vascular pressure, as well as some degree of choroidal effusion.

The incidence of spontaneous SCH is rare but exact figures are unknown, whereas the risk of perioperative SCH varies significantly depending on the type of the ophthalmologic procedure. This risk is higher for patients undergoing glaucoma surgery (range: 0.15% to 6.1%) and vitreoretinal surgery (range: 0.17% to 1.9%) and lower in those having penetrating keratoplasty (range: 0.087% to 1.08%) or cataract surgery (range: 0.03% to 0.81%).

RISK FACTORS

Certain characteristics from the patient’s medical and ocular history increase the relative risk for SCH. The systemic risks include use of anticoagulant or anti-platelet medications, advanced age, uncontrolled hypertension, atherosclerosis, dia-
betes, general anesthesia, Valsalva-type maneuvers (including “bucking” under general anesthesia), and postoperative emesis.\textsuperscript{2,5,6} The ocular risk factors include acute drop of the intraocular pressure (IOP), history of glaucoma, axial myopia, aphakia, pseudophakia, ocular trauma, prolonged or complicated eye surgery (including vitreous loss during cataract surgery), history of SCH in the fellow eye, choroidal hemangioma, and retrobulbar anesthesia.\textsuperscript{4,7} During a vitrectomy, shaving the vitreous base with scleral depression followed by fluid-air exchange has been found to increase the risk of SCH.\textsuperscript{8} Interestingly, the use of epinephrine (rarely used nowadays), was previously considered protective against SCH.\textsuperscript{9}

**DIAGNOSIS**

SCH is frequently a clinical diagnosis. When it occurs intraoperatively during cataract or glaucoma surgery, changes in the red reflex first attract the surgeon’s attention. Additionally, the globe becomes hard, the anterior chamber shallows, and there can occur prolapse of intraocular contents. During vitrectomy, a retina specialist can directly visualize the SCH with its classic dark, dome-shaped appearance, often starting near the equator and progressing. Transillumination of the globe and identification of a shadow defect also helps in certain cases.

Diagnosing SCH in a new patient who comes to the office can be more challenging (Figure 1). Taking a good history with particular attention to the presence of pain, reviewing the pertinent risk factors, and performing a thorough eye examination are the first vital steps. In some cases, however, when the diagnosis remains unclear, other clinical entities must be ruled out. These include choroidal melanoma (Figure 2), congenital hypertrophy of retinal pigment epithelium (Figure 3), cavernous hemangioma (Figure 4), peripheral exudative hemorrhagic chorioretinopathy (Figure 5), melanocytoma, chorioretinal granuloma, choroidal effusion, and serous/exudative retinal detachment.

On examination, the clinician identifies one or more elevated, dark, dome-shaped lesions near the equator that frequently extend anteriorly or posteriorly. These lesions are frequently located close to the vortex ampullae. In equivocal cases, the gold standard is ultrasonography, which can provide important quantitative and qualitative information. On B-scan, clinicians can visualize the dome-shaped lesion (Figure 1A), often with hyperreflective internal nodules that correspond to blood clots. In the acute setting, when the blood clots are fresh and solid, the A-scan shows a double-peaked wide spike with medium-to-low irregular internal reflectivity. In subsequent visits, when the clots liquefy, the lesion becomes more homogeneous and hypo-echoic (Figure 1B). The absence of “intrinsic pulsation” on video capture argues against choroidal melanoma.\textsuperscript{4} On flu-
Occlusive angio-geometry, the retinal circulation overlying the SCH typically appears normal, which is not the case with choroidal tumors such as melanoma, where the FA can show “double circulation” as well as leakage or staining in the retina. Similarly, the indocyanine green angiogram is typically silent in SCH, whereas in choroidal tumors, it exhibits abnormal internal choroidal vessels. Other imaging modalities that can detect SCH but are not routinely performed are computed tomography and magnetic resonance imaging.

TREATMENT OPTIONS

The management of SCH remains controversial. For most ophthalmologic procedures, outside the field of vitreoretinal surgery, an expulsive intraoperative SCH is an indication for urgent change of the surgical plan. Prompt closure of the surgical incisions followed by temporary increase of the IOP with digital compression on the globe might tamponade the expansion of the hemorrhage. If uveal prolapse has already occurred, urgent repositioning of the tissue into the intraocular space with careful injection of viscoelastic should be attempted. Placement of an anterior chamber infusion has been advocated as one possible intervention. The role of posterior sclerotomy in the acute setting remains debatable.

For a retina specialist who notices the SCH during pars plana vitrectomy (PPV), increasing the infusion

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**Figure 2.** Fundus photographs (A, B) of a 77-year-old female with choroidal melanoma. The patient presented with slowly progressive, painless vision loss. The exam was significant for subretinal fluid and exudates (yellow arrow), as well as a dome-shaped choroidal lesion temporally (blue arrow). (C) The A-scan revealed the low internal reflectivity that is classic in choroidal melanoma.

**Figure 3.** Fundus photograph of a dark, clinically flat, congenital hypertrophy of the retinal pigment epithelium. Note the sharply demarcated margins and central lacunae.
pressure to 60 mm Hg to 80 mm Hg or even higher, based on the patient’s blood pressure (BP), should be the first step. Having the anesthesiologist check and potentially address an elevated BP can prevent progression of the SCH postoperatively. Prophylactic antiemetics may help. If the SCH is small and the case non-elective (eg, retinal detachment), the vitrectomy may be completed with caution.4

The role of delayed surgical management remains controversial. Even though some authors have reported good results with early intervention, the consensus is that the surgical management should be delayed until the blood liquefies, a process that typically takes at least 7 to 14 days.13-15 Use of topical steroids, cycloplegics, ocular antihypertensives, and oral analgesics might help. During subsequent visits, ultrasound is used to assess the size of the SCH and its degree of liquefaction. A decision for urgent surgical management is usually reserved for appositional (“kissing”) or massive SCH. Prior to potential surgery, the patient, together with the primary care physician, must optimize the management of system-

Figure 4. Fundus photograph of a cavernous hemangioma in a 42-year-old asymptomatic patient. Note the cluster of grape-like, thin-walled sacular angiomatous lesions (A) with the classic leakage on fluorescein angiography (B).

Figure 5. Peripheral exudative hemorrhagic chorioretinopathy (arrow) in an asymptomatic 92-year-old male with end-stage age-related macular degeneration (A). The A-scan showed a relatively flat subretinal lesion with high internal reflectivity (B).
operative risk factors. An honest conversation between the patient and the surgeon is key in order to set realistic expectations.

Careful planning of the surgical steps prior to the actual procedure is vital. An anterior chamber maintainer (eg, Lewicky) is preferred in order to avoid iatrogenic damage to the retina and the choroid. Alternatively, a 6-mm infusion canula through the pars plana and distal to the quadrant of SCH is an option. The classic approach is to perform a conjunctival peritomy, and then create a 2-mm to 3-mm radial, full-thickness sclerotomy 4 mm posterior to the limbus in the quadrant of the most bullish SCH. Alternatively, nowadays, trocar/cannula systems can also be employed in a transconjunctival fashion by inserting the trocar/cannula in an angled fashion and withdrawing the trocar to allow blood to drain from the cannula. Gentle external massage on the adjacent sclera can facilitate drainage. The fluid that is released typically appears dark red mixed with blood clots. PPV with high infusion pressure and injection of perfluorocarbon liquid into the vitreous cavity has been reported to assist the mechanical displacement of the blood and expedite the drainage through the previously created sclerotomies. Some degree of residual SCH is common and should be deemed acceptable. The postoperative anatomic and visual recovery is usually slow.

SCH, perioperative or not, is a relatively uncommon condition. Nevertheless, given that it is frequently a devastating and vision-threatening complication, the retinal specialist should feel competent managing it. This skillset includes not only appropriate medical and surgical decision-making, but also carefully managing patient and family expectations — something that is part of the art of being a surgeon.

REFERENCES

7. Taylor DM. Expulsive hemorrhage: some observations and com-