Decompression Retinopathy After 25-Gauge Transconjunctival Sutureless Vitrectomy: Report of 2 Cases

Decompression retinopathy is a rare postoperative complication of eyes that undergo sudden ocular decompression. It has been reported mainly after glaucoma filtering surgery. The clinical appearance is characterized by multiple round intraretinal hemorrhages, some with white centers, located in the postequatorial retina.

Twenty-five–gauge transconjunctival sutureless vitrectomy (TSV-25) has recently been introduced for vitreoretinal surgery. It is performed through 3 self-sealing transconjunctival pars plana sclerotomies. Despite the small sclerotomy caliber, it has been demonstrated that some patients develop transient hypotony and even choroidal detachment in the immediate postoperative period.

We report 2 cases of ocular decompression retinopathy immediately after TSV-25 for retained lens fragments and elevated intraocular pressure (IOP) after complicated phacoemulsification.

Report of Cases. Case 1. A healthy 74-year-old white man was referred with retained lens fragments in the right eye after complicated phacoemulsification performed 20 days earlier. Visual acuity was counting fingers, with severe corneal edema, sulcus-fixated intraocular lens, IOP of 40 mm Hg despite maximum medical therapy, moderate vitritis, and retained lens fragments. Retinal examination results were otherwise unremarkable. Funduscopy results of the left eye were normal. A TSV-25 was performed under retrobulbar and peribulbar anesthesia without complications. On the first postoperative day, the IOP was 8 mm Hg. Fundus examination showed scattered superficial and deep intraretinal hemorrhages, with some white-centered hemorrhages, in the posterior pole and equatorial retina (Figure 1). There was no other retinal vascular finding consistent with vein occlusion, and no macular edema was noted on optical coherence tomography. One month later, the hemorrhages showed partial resolution. Six months postoperatively, the hemorrhage had completely resolved and visual acuity was 20/60 with persistent mild corneal edema.

Case 2. A healthy 68-year-old white man was referred with retained lens fragments in the right eye after complicated phacoemulsification performed 1 month earlier. Visual acuity was hand motions. Examination results were remarkable for severe corneal edema, IOP of 38 mm Hg despite maximum medical therapy, anterior uveitis, and a sulcus-fixated intraocular lens. Funduscopy revealed vitritis and multiple lens fragments but no intraretinal hemorrhages or retinal detachment. Examination results of the left eye were unremarkable. A TSV-25 was performed under retrobulbar and peribulbar anesthesia without complications. On the first

Figure 1. Color (A) and red-free (B) composite photos in case 1 showing scattered superficial and deep intraretinal hemorrhages, some with white dotted centers, on the posterior pole and beyond the vascular arcades.
postoperative day, the IOP was 4 mm Hg. Funduscopy revealed scattered superficial and deep intraretinal hemorrhages, some with white centers, in the posterior pole and equatorial retina. A residual cortical fragment was noted at the fovea (Figure 2). There was no other retinal vascular finding consistent with vein occlusion. One month postoperatively, the hemorrhages showed partial resolution and visual acuity was 20/60 (Figure 2).

Comment. Decompression retinopathy is a rare postoperative complication thought to arise in cases in which there is a sudden drop in IOP. More frequently, multiple intraretinal hemorrhages appear with central retinal vein occlusion, coagulopathies, and ocular ischemic syndrome. Both patients had no systemic diseases, and hemogram and coagulogram results were normal. The facts that no intraoperative retinal hemorrhages were noted, some round hemorrhages had white centers, and the IOP had a sudden drop immediately postoperatively called our attention to include decompression retinopathy in our differential diagnosis. Although fluorescein angiography was not performed, color fundus photographs showed no retinal venous dilation. In case 1, some venous branches were mildly tortuous (Figure 1), but this was not seen in case 2 (Figure 2). Carotid Doppler ultrasonography was not performed in these patients, but the lack of venous dilation, ocular pain, periphereral retinal hemorrhages, and retinal and optic disc neovessels as well as the fairly good visual recovery make the diagnosis of ocular ischemic syndrome less likely.

Case 1 had intraretinal white-centered hemorrhages that may correspond to fibrin and platelet aggregates. Pathobiologically, this can be explained by capillary rupture and subsequent physiologic repair. The large drop in IOP immediately postoperatively would correspond to an increase in arterial perfusion pressure that possibly overwhelmed the resistance of the capillary bed, causing their rupture. Additionally, both cases had moderate vitritis resulting from a breakdown of the inner blood-retinal barrier and increased retinal capillary fragility. Both patients had visual acuity recovery to 20/60 without any macular sequelae. Although not the rule, Fechtner et al\(^1\) have shown that most eyes recover good vision after decompression retinopathy.

Previously, Fuji et al\(^2\) and Rezende et al\(^3\) demonstrated the successful management of cataract complications, including cystoid macular edema and retained lens fragments, with TSV-25. Neither study included patients with preoperative uncontrolled ocular hypertension.

In conclusion, decompression retinopathy related to an immediate drop in IOP after TSV-25 can occur. Care should be taken to not leave the eye hypotonous at the end of the procedure, especially in eyes with high preoperative IOP. To our knowledge, this is the first report of decompression retinopathy after TSV-25.

Flávio A. Rezende, MD, PhD
Luiz Gustavo T. Regis, MD
Mônica Kickinger, MD
Simone Alcântara, MD

Correspondence: Dr Rezende, Centro de Estudos e Pesquisas Oculistas Associados, Rua Humaitá, 244/1202, Bloco 2, Humaitá, Rio de Janeiro RJ 22261-001, Brazil (frezende@bol.com).

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