Ocular decompression retinopathy after trabeculectomy with mitomycin-C for angle recession glaucoma

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A 45-year-old male presented with intractable glaucoma following 360-degree angle recession after blunt trauma. He underwent an uncomplicated trabeculectomy with mitomycin-C (MMC). Adequate precautions were taken to reduce the chances of sudden lowering of intraocular pressure (IOP). He did not have any intraoperative shallowing of the anterior chamber or postoperative hypotony, but still developed ocular decompression retinopathy. On detailed review of the previously reported cases we discovered that besides a large IOP drop after surgery, either the preoperative rise of IOP in all these cases was over a relatively short period or the course of their glaucomatous process was likely to have exposed them to intermittent spikes of high IOP. To our knowledge this factor has not been previously postulated in the pathophysiology of ocular decompression retinopathy. We illustrate this with a rare case of ocular decompression retinopathy after trabeculectomy with MMC for post-traumatic angle recession glaucoma.

Key words: Angle recession glaucoma, ocular decompression retinopathy, trabeculectomy with mitomycin-C

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During intraocular surgery an entry into the eye allows the intraocular pressure (IOP) to fall and equate with the atmospheric pressure. If this change is sudden and large then it can induce hemodynamic changes that may result in a rare clinical picture called ocular decompression retinopathy, first described by Fechtner et al. On detailed review of literature we discovered that the previously reported cases either had a significant rise of IOP over a relatively short period or the course of their glaucomatous process was likely to have exposed them to intermittent spikes of high IOP. This was then followed by a significant drop in IOP. We postulate that the rise of IOP over a short duration or in the form of high IOP spikes may have interfered with the autoregulation of retinal capillaries in these cases. We illustrate this with a rare case of ocular decompression retinopathy after mitomycin-C (MMC) augmented trabeculectomy.

Case Report

A 45-year-old Caucasian male was referred to our hospital eye service with raised IOP in his right eye following a blunt injury 12 months ago. He was experiencing blurred vision for the past six months but he did not seek any ophthalmic advice. His Snellen acuity was 20/30 in the right eye. The IOP was 55 mm Hg. Gonioscopy showed a 360-degree angle recession. He had traumatic mydriasis. The cup-disc ratio was 0.9 with inferior notching and he had a corresponding visual field defect. The left eye was normal. He did not have any other known ocular or current systemic co-morbidity and was not on any systemic medication. Maximum tolerable medical therapy (eye drops latanoprost 0.005% at night, apraclonidine 0.5% eye drops twice daily, acetazolamide slow-release capsule 250 mg twice daily given orally for two weeks) failed to bring his IOP below 42 mm Hg. At this stage he was referred to our glaucoma clinic.

A trabeculectomy with MMC was performed under general anesthesia with due precautions to avoid sudden lowering of IOP. Intravenous acetazolamide 500 mg was given with a slow intravenous infusion over five minutes. An oblique paracentesis was done to release aqueous very slowly. Two preplaced 10/0 nylon sutures, including one releasable suture, were used for the scleral flap. These were tied swiftly and tightly. The anterior chamber was maintained throughout the operation. The conjunctiva was sutured securely.

On the first day postoperatively a diffuse, functioning and non-leaking bleb was present. The IOP was 9 mm Hg. The visual acuity was 20/80. The optic disc was less cupped with a cup-disc ratio of 0.6 and the macula was clear and dry. There were multiple blot hemorrhages in the mid and far periphery in all four quadrants of the fundus and some of these had distinct white centers like Roth spots [Fig. 1]. Over the next six months he maintained a good bleb and an IOP of 12-14 mm Hg, his Snellen acuity improved to 20/30 and the hemorrhages gradually faded without sequelae.

Discussion

Decompression retinopathy is a rare complication of glaucoma filtration surgery in fit, young and phakic patients. It may rarely be seen after laser or medical treatment for acute glaucoma and valve implantation. Our patient had signs of decompression retinopathy similar to previously described cases. As the signs were present only on the operated eye it is unlikely to be a case of Valsalva retinopathy which is a bilateral condition. The absence of

![Figure 1: Some retinal hemorrhages in ocular decompression retinopathy had distinct white centers like Roth spots (arrow)](image)
retinal vascular tortuosity and dilatation, optic disc edema or hemorrhage on or around the disc ruled out a vascular occlusion.

Young individuals without hypertension or vasculopathy, tolerate the hemodynamic changes in choroidal vasculature very well.[1] However, if the autoregulation capacity of the retinal vasculature is impaired, then lowering of the IOP allows excessive blood flow through the capillary bed. This may lead to multiple focal leaks presenting as blot hemorrhages. These are repaired with a fibrin plug appearing as a white center in the deep hemorrhages. The hemorrhages may involve only the periphery[1] or may involve the macula,[1,2] and may later form an epiretinal membrane resulting in permanently reduced vision.[2]

On reviewing the previously reported cases we noted that in these patients either the IOP rose significantly over a relatively short period of time[3-10] or they were prone to intermittent spikes of high IOP due to the nature of their glaucomatous process.[1,8,10,11] In the first description of this condition by Fechtner et al., in 1992,[1] three out of four cases had juvenile open angle glaucoma (JOAG) and were aged 13-19 years at the time of surgery. Patients with JOAG are known to have spikes of high IOP despite maximum medical treatment.[1] Although the fourth case described by them originally had primary open angle glaucoma (POAG), he developed secondary acute rise of IOP immediately after a cataract surgery and developed decompression retinopathy after a trabeculectomy done 10 days later. Most other reported cases had rise of IOP over a short duration ranging from one day to a few months. These were cases of acute angle closure glaucoma with IOP rising over one to four days,[3-5] a case of neovascular glaucoma with IOP rising over three months,[6] a case of post-traumatic glaucoma with IOP rising over two months,[8] and case with secondary post surgical glaucoma with IOP rising over a few months.[8,9] Other cases were young patients,[7,8,11] and two of these had uveitis[7,11] therefore further increasing the possibility of being exposed to IOP spikes. Notably, none of these cases were of a typical adult onset POAG which characteristically has a slow built-up of IOP over years. Also, they could not be controlled medically to a safe IOP level before surgery.

The rise of IOP to a high level in a relatively short period of time, or high IOP spikes may contribute to impairment of the autoregulation of retinal vasculature. On lowering of the IOP the retinal vasculature gets overwhelmed by the increase in blood flow and this results in multiple hemorrhages. In our case the IOP rose to mid-fifties within months after the injury and decompression retinopathy occurred despite taking all precautions to minimize the large and sudden drop of IOP.

Ocular decompression retinopathy should be anticipated after glaucoma filtration procedure for cases with rise of IOP over a short duration or with possibility of spikes of IOP. Patients should be counselled about the risk of this complication. Precautions should be taken to reduce the risk of this complication. We medically lowered the IOP maximally before the surgery. A sudden drop of the IOP was avoided by a careful initial paracentesis, releasing the aqueous very slowly. The anterior chamber did not get shallow during the surgery. The scleral flap sutures were preplaced and tied swiftly. Releasable sutures were preferred as they can be tied tightly and released or adjusted later, if necessary. Despite all these precautions our patient developed a decompression retinopathy probably because the rise of IOP over a relatively short duration may have been a risk factor. This is supported by the above discussion of the previously published literature. Postoperatively, patients should be examined carefully for any signs of decompression retinopathy.

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