Retinal vein occlusion following vitrectomy for rhegmatogenous retinal detachment: a case report

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Introduction
Ocular vascular occlusions after intraocular surgery are rare complications. The causative factors are difficult to identify specially after vitreoretinal procedures. Vascular occlusions are more frequent when a cardiovascular disease coexists. We present a case where a person presented with branch retinal vein occlusion (BRVO) and cystoid macular oedema (CME) after vitrectomy for rhegmatogenous retinal detachment (RRD) without any systemic predisposing factors.

Case report
A 26-year-old male presented with blurred vision in his left eye for 1 month. Distant best-corrected visual acuity (BCVA) of the left eye was counting fingers at 3 m. Fundus examination revealed total RRD in his left eye with horseshoe tears at the periphery. The patient underwent routine work-up before surgery excluding all possible coexisting cardiovascular risk factors. Patient underwent routine 23G vitrectomy with belt buckling and silicone oil tamponade under peribulbar anaesthesia. SRF was drained through a retinotomy. The postoperative period was uneventful except that on the fifth postoperative day, few retinal haemorrhages were noted around superotemporal vessels. In his follow-up visit, after 6 weeks, BCVA in his left eye was 6/24 with an intraocular pressure of 12 mmHg. Fundus examination of the left eye showed an attached retina with sclerosed vessels in superotemporal quadrant along with retinal haemorrhages and cystic changes at macula suggestive of a superotemporal BRVO with CME. No signs of inflammation were seen in either of the eye.

Fundus fluorescein angiography (FFA) revealed a wide area of non-perfused retina (red arrow) before surgery excluding all possible coexisting cardiovascular risk factors. Patient underwent routine 23G vitrectomy with belt buckling and silicone oil tamponade under peribulbar anaesthesia. SRF was drained through a retinotomy. The postoperative period was uneventful except that on the fifth postoperative day, few retinal haemorrhages were noted around superotemporal vessels. In his follow-up visit, after 6 weeks, BCVA in his left eye was 6/24 with an intraocular pressure of 12 mmHg. Fundus examination of the left eye showed an attached retina with sclerosed vessels in superotemporal quadrant along with retinal haemorrhages and cystic changes at macula suggestive of a superotemporal BRVO with CME. No signs of inflammation were seen in either of the eye.

FFA showing nonperfused retina (red arrow), retinotomy site (yellow arrow), patelloid leakage at macula (blue arrow).

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near the retinotomy site (yellow arrow) along with sclerosed vessels with a petalloid pattern dye leak present at fovea (blue arrow) suggestive of CME, which was confirmed by the optical coherence tomography (OCT) (Figures 1 and 2). Further systemic investigations failed to find a systemic cause. The patient was advised topical non-steroidal anti-inflammatory eye drop and was asked to review after 1 month. The OCT after 2 months showed non-resolution of macular oedema (Figure 3). He subsequently underwent silicone oil removal with an intravitreal injection of triamcinolone acetonide (2 mg) for non-resolving cystoid macular oedema.

Discussion
The primary mechanisms for BRVO are mechanical compression of vein at arterio-venous crossing by anterior rigid artery, degenerative changes of vessel wall and/or abnormal haematological factors.¹

The main cause of impaired vision in BRVO is due to CME. CME occurs in 30% of eyes with BRVO.¹ CME can be differentiated by FFA by typical petalloid leakage pattern from cystoid degeneration which does not show leakage in FFA.

There are reported incidents of vascular occlusion after retrobulbar anaesthesia. The cause of such an episode attributed to reduction of retrobulbar blood flow velocities after retrobulbar anaesthesia.⁴ Drug-induced vasoconstriction plays a role in the decrease in ocular blood flow after peribulbar anaesthesia as does increase IOP which is probably caused by the mechanical effect of the injected volume in the orbit.⁴ Although the retinal vessels has a wide range of autoregulation under change in perfusion pressure, the autoregulation

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Figure 2  OCT at 6 weeks.

Figure 3  OCT at 2 months.
gets compromised in diseased states like diabetic retinopathy and glaucoma. And, in these situations, even a small reduction in ocular perfusion pressure, as induced by an increase in intraocular pressure, may reduce ocular blood flow. Similarly, high fluctuation of intraocular pressure is seen during vitrectomies. An increase in intraocular pressure occurs during fluid gas exchange due to a forward displacement of the lens-iris diaphragm resulting in anterior chamber flattening and secondary angle-closure glaucoma. Anterior chamber flattening can also occur due to ciliary body oedema and choroidal swelling caused by extensive laser or cryocoagulation.

A direct vascular trauma with endolaser burn or mechanical trauma could also be a possible reason for the BRVO in our case as is evident the location of vein occlusion close to endolaser burns and the retinotomy site.

**Conclusion**

Surgical manipulations causing decreased retinal and choroidal blood flow as well as pressure changes and mechanical damage may act as triggering mechanisms for retinal vein occlusions in such cases.

Vascular occlusion after intraocular surgery may occur without any associated cardiovascular disease. Affected patients should undergo systemic evaluation to rule out possible underlying systemic diseases and should be treated conventionally.

**References**


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