

Decompression Retinopathy After Phacoemulsification For Traumatic Cataract With Pupillary Block

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Abstract – Blunt trauma may cause traumatic cataracts severe enough to cause pupillary block. A dislocated lens from the coup-counter coup mechanism may also result in secondary angle closure and raised IOP. Therapies are to relieve narrow angles from the lens-iris apposition by performing cataract surgery. However, sudden reduction of IOP may result in extensive retinal hemorrhages as the globe decompresses. Nevertheless, 85% of patients with decompression retinopathy have no complaint. We present a case of decompression retinopathy after phacoemulsification. A 47-year-old man who works as a wallpaper assembler was hit by his own hand while working. He complained of hazy vision. Initial visit revealed high IOP treated with 3 topical antiglaucoma. A month later, the patient was referred to our center. His VA (visual acuity) at presentation was 3/60, IOP of 40 mmHg and closed angle. The anterior chamber was shallow with stellate cataract but no phacodonesis. He underwent an uneventful cataract surgery. Postoperatively, the IOP remained high and decompression retinopathy was seen. He underwent augmented trabeculectomy to further control his IOP. Decompression retinopathy resolved without significant visual loss. VA at final follow-up was 6/18 with IOP of 12. Risk of developing decompression retinopathy exist particularly when IOP is not adequately lowered prior to surgery. It causes rupture of the retinal microvasculature, especially in defective vessels autoregulation. IOP should be sufficiently reduced prior to surgery to prevent decompression retinopathy or other more sinister complications. Reducing the IOP in a stepwise manner is essential.

Keywords – traumatic cataract, decompression retinopathy, phacoemulsification.

1. Introduction

Ocular blunt trauma is often found in groups of middle-aged male within the working communities. Approximately 1.6 to 19 million people become blind due to trauma regardless of its causes, with most having initial visual acuity of <6/60.[1,3] The age group varies in different studies varying between 15-25 years and more than 70 years old, although the average age was around 40 years old.[1,2]

High intraocular pressure (IOP) after contusion happened because of alteration in aqueous dynamics either from pupillary block or angle closure. Furthermore a coup-counter coup phenomenon induces sudden alteration of the intraocular microvascular autoregulation.[5] The coup-counter coup phenomenon also causes traumatic cataract, commonly in a stellate shaped cataract, lens subluxation, lens swelling causing phacomorphic glaucoma or release of

lens protein into anterior chamber (AC) causing phacoantigenic glaucoma.[4,6] The initial symptoms and findings is related to trauma itself which are dull aching pain with blurred vision, hyphema/microhyphema with or without angle recess, and shallow AC. Hyphema itself may cause high IOP. Patient's prognosis and the therapy are related to the mechanism of trauma, the initial visual acuity findings, the extent of damage if its including lens subluxation, posterior capsule rupture, or retinal detachment (reported in 10-20% of patients with blunt trauma)[4]. Blood ocular barrier may be breached, releasing more plasma protein content as a form of extracellular edema, and further increase the IOP.[5,6]

The retina status of direct eye blunt-trauma could experience damage from vascular factors and inflammatory factors, the blood-ocular barrier (zonula occludens of retina vessels and endothelium, RPE-Bruch membrane zonula adherens and desmosom) is abruptly damaged thus render it to increase the permeability resulting in ganglion cell loss.[5]A proponent was commonly established that an inflammation with vascular permeability changes increase intraocular protein content and put the cataract surgical attempt more risky from insufficient pupil dilatation, post-operative risk of cystoid macular edema, and sudden IOP changes. IOP drop abruptly on failure of blood retina barrier might cause rare decompression retinopathy with white spot on the center of pre-retinal, intraretinal bleeding post-operatively.

2. Case Report

47 years old male had right eye (RE) blunt trauma after accidentally hitting his eye with his own hand while assembling paper walls. Immediately after the blow, he complained of blurred vision, redness, and pain. He was diagnosed to have microhyphema and secondary high IOP. He was treated with steroid eye drop, T Acetazolamide 250 mg q.i.d. and three topical IOP-lowering agents. However, the IOP did not reduced after 22 days and the patient was unable to tolerate the carbonic anhydrase eye drop.

At the first presentation to us, the RE visual acuity was 3/60 ph 6/36, LE was 6/6, RE conjunctiva was injected. The cornea was slightly hazy, the anterior chamber was shallow with cells 2+, and stellate-shaped cataractous lens but no phacodonesis. The RE IOP was 40 mmHg. Fundus examination was limited by the cataract and AC reaction, however, the optic disc (OD) could be seen with cup/disc ratio of 0.3. There was no retinal detachment or Berlin's edema. The patient's topical IOP-lowering medications were changed but he was counselled for cataract surgery should the IOP remained high.

The IOP at the second visit remained high and the patient was prepared for cataract surgery under local anesthesia. A capsular tension ring was also prepared should the need arises. anesthesia with preparation of capsular tension ring.

| RE | LE |
|--------------------------------|---------------------------------|
| AXL : 22.95 mm ACD: 2.20 mm | AXL : 22.47 mm ACD : 2.56 mm |

| | |
|--|--|
| Lens Thickness: 4.63 mm CCT: 618 micrometer | Lens Thickness : 4.68 mm CCT : 571 micrometer |
|--|--|

Table 1. Cornea topography and biometry measurement of the patient

The biometry revealed a reduced AC depth in the RE although the lens thickness was similar to the other eye. His central corneal thickness was increased possibly due to a subclinical corneal edema from the chronically raised IOP.

Preoperatively, the IOP was still 40 mmHg and intravenous Acetazolamide 250 mg was given prior to surgery. Phacoemulsification was performed as the usual manner. There was no anterior capsular breach or zonulysis. Phacoemulsification setting were at low values. The intraocular lens was implanted in the bag without the need for a capsular tension ring.

On postoperative day 1, his visual acuity was 6/18 pinhole same, N18 . The cornea was slightly edematous but the IOP was 14 mmHg, There was 3+ AC reaction but the AC was deep with no hyphaema. Fundus examination revealed multiple pre-retinal hemorrhages along the superotemporal and inferotemporal arcade, but the macula was not involved.

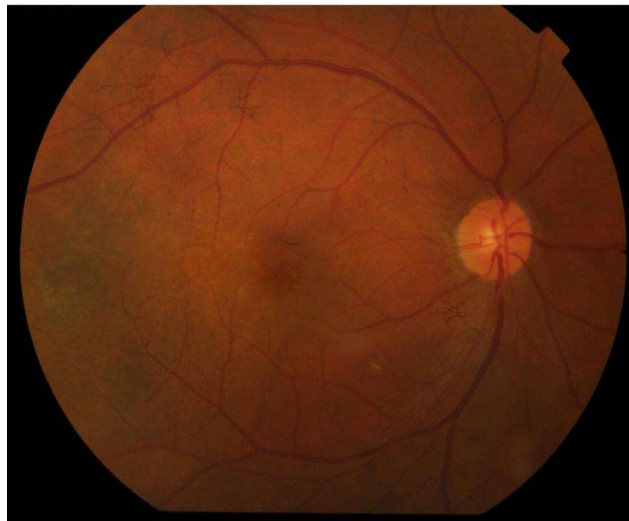


Figure 1. Flecks of deoxygenated blood from multiple hemorrhages along supero/inferotemporal arcades.

At subsequent visits, his IOP increased and was planned for augmented trabeculectomy with mitomycin C. Postoperatively vision 6/60 ph 6/24, bleb is formed with IOP 13, PC IOL

stable, and patient was discharged with no antiglaucoma medication. Final visual acuity was 6/18 refracted with +0.50 DS/-1.00 DC/X140 achieved 6/15⁺².

3. Discussion

The empirical evidence that middle-aged workers have higher prevalence of traumatic cataract provide opportunity to ophthalmologist in detecting what structures are involved in detail. Damages that happen is elicited from blunt object, shearing force, resulting in ganglion cell loss. Damages of the uveal structures and its identification prior the surgery is essential since the mechanism of traumatic cataract in this case is a shock wave that move the iris-lens diaphragm in a sudden manner (coup-counter coup mechanism) and blood aqueous barrier breach. The lens capsule acts as a barrier to ensure that the electrolyte in the lens is maintained at a certain level compared to the aqueous. The lens's sodium content is normally 10 mmol/L, and the potassium level is 120 mmol/L, higher compared to aqueous (38 mmol/L).

Blood ocular barrier breach contributes to inflammation, which may have happened since the initial trauma impact with the appearance of microhyphema. The inflammation was treated with steroid eye drops and cells was 2+ on the first clinic visit.[5,9-12] Following the breach in the aqueous barrier, polypeptide and inflammatory cells released will stimulate abnormal hyperplastic response of the lens epithelium, causing potassium loss and sodium influx. This increases the osmotic pressure in the lens and promotes water influx into the lens. These changes gives rise to the most pathognomonic sign of traumatic cataract which is either intumescent or stellate shaped cataract.

The corneal endothelium pump is also affected. The Na-K ATP pump is not sufficient to pump water out from the cornea and imbibe too much water causing cornea edema. In a more hazardous blunt trauma, the sudden spike of IOP could injure the trabecular meshwork, stresses the zonules, block aqueous flow and increases the IOP. Conversely, sudden trauma might also cause ciliary shutdown. The subluxation or dislocation of lens might happen, and iris-lens touch might also complicate the condition.

Blunt trauma is considered more hazardous than a simple open-wound trauma when its acute force generated diffusely reaches the posterior ocular structures and/or displacing the globe. By large, high IOP following blunt trauma affects the retina vessels. The retina vessels are in direct contact with the vitreous which can distribute and generate its buoyancy in acute trauma (retina vessels has its capacitance and resistance). Pressure on the retinal vessels affects the blood flow. The retina is unable to survive ischemia after 90-100 minutes.

High BP with arteriolar resistance may lead to pronounced changes on the retina vessels hemodynamic resulting in retinal hemorrhages. In our patient, his BP fluctuated from 120/70 pre-operatively to 155/100 on first 5 minutes of surgery and lower down to 122/71 mmHg. Some retinal hemorrhages were expected to occur after sudden lowering of the IOP when phacoemulsification started.[5,7-9]

Decompression retinopathy is common in young phakic patient when the IOP is dropped too fast. Furthermore, the retina hemorrhages (subretinal and intraretinal) are induced by retina vessels barotrauma that shed the glycocalyx on vessel's luminal and breakdown of tight junction of RPE-Bruch membrane. The consequences are increased hydrostatic pressure and more proteins pass through the blood retina barrier (BRB). Nitric Oxide is also secreted from shed glycocalyx and vasodilation happen, and a vicious cycle developed.[12]

Retinal bleeding and macula edema are possible end results of these pathophysiological processes. Whether retinal bleeding or macula edema happened depends on the nature of the injury and how severe the breach of blood retina barrier and intraocular structure are involved. Moreover, it is also subject to Starling Law's of retina vessels, which explains that this kind of edema can happen as hydrostatic pressure of the intraretinal vessel increase and protein extravasated into the retina causing increased retinal osmotic pressure and drive the plasma into retina. Extracellular edema in the retina might be shown on the OCT scan.

In this case report, IOP drop in a sudden manner in a state of blood retina barrier breach triggered multiple dot and blot hemorrhages as explained by the Starling Law. The appearance of multiple hemorrhages also indicate that the patient had retina autoregulation defect during and after cataract surgery (high BP with impaired arterial autoregulation). The mean ocular perfusion pressure was calculated to be 52 mmHg on first 5 minutes of surgery. MOPP then increased to 58 mmHg.

In this case, decompression retinopathy was seen 3 days after surgery. Some studies reported 1.5-2 days after surgery, and even on rare occasions, it may occur immediately after AC paracentesis. Decompressive in this case was asymptomatic and resolves spontaneously in few days. Macula edema did not happen in this case.

In traumatic cataract, the IOL calculation could be better recalculated in multiple attempts if the anterior lens capsule is compromised, or lens is too intumescent. Using immersion biometry, intumescent lens might dampen the light to calculate the true axial length. Using interferometry laser as an adjunct will provide surgeon several considerations of IOL Dioptric power chosen. This case yielded a slightly longer axial length and higher lens thickness using interferometry laser than the immersion technique.

Before phacoemulsification procedure, capsular tension ring was prepared. The anticipation of zonulysis was always in mind. Phacoemulsification in this case was also expected to lower IOP as a secondary outcome. As this patient had shallow AC, the narrow angle would be expected to open after phacoemulsification.[13,14]

On third clinic visit, despite strenuous attempts of lowering IOP and reducing inflammation, IOP was 32 mmHg. This condition could be contributed to the patient anterior chamber biometric value, especially Lens Vault (LV). LV is correlated to low of IOP afterwards (in a study the changes of non glaucomatous eye undergone phacoemulsification was 26.11 mm^3),

moreover some patients with narrow angle in other study also get better angle opening with respect in their measurable anterior chamber angle biometrics before and after cataract removal.[11,13,14]

IOP measurements and detailed anterior chamber depth evaluation is essentials in patient with traumatic cataract. The cataract status of patients will be better handled if emphasizing these factors along with expecting complication such as decompression retinopathy. In summary, decompression retinopathy should be informed when obtaining consent in eyes with high IOP undergoing along with its visual disabling sequelae.

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