

# Cystoid macular edema in complicated cataract surgery: A case report

Hatem Barhoom<sup>1</sup>, Sharanjeet-Kaur<sup>2</sup>, Sabri Kamarudin<sup>3</sup>

<sup>1</sup>Optometry department, Faculty of health science, Islamic University of Gaza, Gaza strip, Palestine

<sup>2</sup>Optometry Department, Faculty of Health Sciences, Universiti Kebangsaan Malaysia, Jalan Raja Muda, Kuala Lumpur, Malaysia

<sup>3</sup>Ophthalmology Clinic, Hospital Selayang, Leboh Raya Selayang – Kepong, 68100 Batu Caves, Selangor, Malaysia

## Email address:

[hbarhoom@iugaza.edu.ps](mailto:hbarhoom@iugaza.edu.ps) (H. Barhoom)

## To cite this article:

Hatem Barhoom, Sharanjeet-Kaur, Sabri Kamarudin. Cystoid Macular Edema in Complicated Cataract Surgery: A Case Report. *Science Journal of Clinical Medicine*. Vol. 3, No. 3, 2014, pp. 43-45. doi: 10.11648/j.sjcm.20140303.13

**Abstract:** One of the major risk factors for conversion from Phacoemulsification (Phaco) to Extracapsular Cataract Extraction (ECCE) is the Posterior capsule rupture. The capsule rupture or any cause leads to Vitreous Loss (VL) will develop tractional inflammation to the retina and Cystoid Macular Edema (CME) will occur. A 65 years old Chinese man had a history of left eye phaco converted to ECCE, anterior vitrectomy and Anterior Chamber Intra Ocular Lens (ACIOL) implant was done due to complication by inferior zonulolysis and VL. After 10 months he complained of dropped Best Corrected Visual Acuity (BCVA) in the operated eye and he was diagnosed to have CME. The diagnosis was done using Optical Coherence Tomography (OCT). Incomplete Posterior Vitreous Detachment (PVD) increases the risk of CME development, and this may make it necessary to start treatment before cataract surgery to reduce the incidence or improve the prognosis of the condition. In this case, PVD was diagnosed in the right eye suggesting that it will be at higher risk to develop CME in case of cataract surgery when it is complicated by VL and starting CME treatment before the surgery will be highly recommended.

**Keywords:** Cataract, Cystoid, Vitreous Loss, OCT

## 1. Introduction

Posterior capsule rupture is one of the major risk factors for conversion from Phacoemulsification (Phaco) to Extracapsular Cataract Extraction (ECCE).

Capsule rupture or any cause leading to Vitreous Loss (VL) will result in tractional inflammation of the retina leading to Cystoid Macular Edema (CME) especially in the presence of Incomplete Posterior Vitreous Detachment (PVD).

Anterior Chamber Intra Ocular Lens (ACIOL) implant was done due to complication by inferior zonulolysis and VL.

He complained of reduced Best Corrected Visual Acuity (BCVA) in the operated eye from 6/9 (after 4.5 months post operation) to 6/18 (after 10.5 months post operation) within 6 months.

He has history of Diabetes mellitus (DM) and he is on medication

## 2. History and Symptoms

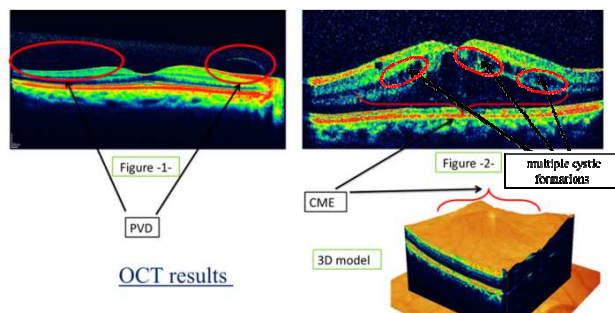
65 years old Chinese man has history of Left Eye (LE) phaco converted to ECCE, anterior vitrectomy and

## 3. Signs and Examinations

All signs and eye examination results are shown in the next table

Test	Right Eye (RE)	Left Eye (LE)
Visual Acuity (VA)	6/24 (Pin Hole (PH) 6/18)	6/18 (PH 6/18)
Slit lamp	<ul style="list-style-type: none"><li>Cataract, Moderate cortical opacities and nuclear sclerosis (NS)</li><li>No rubeosis iridis</li></ul>	<ul style="list-style-type: none"><li>Pseudophakia</li><li>ACIOL with multiple pigmented giant cells</li><li>No rubeosis iridis</li><li>Surgical peripheral iridotomy (PI) superiorly</li></ul>

Test	Right Eye (RE)	Left Eye (LE)
Goldmann Tonometry	15 mmHg	12 mmHg
Funduscopy	<ul style="list-style-type: none"> <li>Vitreous degenerative changes</li> <li>Cup to Disc Ratio (CDR) = 0.4</li> <li>No Diabetic Retinopathy (DR)</li> </ul>	<ul style="list-style-type: none"> <li>Vitreous degenerative changes</li> <li>CDR = 0.4</li> <li>No DR</li> </ul>
Optical Coherence Tomography (OCT)	Retina is normal (figure-1-) - Incomplete Posterior Vitreous Detachment (PVD)	Retina is abnormal (CME ) (figure -2-)



**Figure 1.** shows incomplete PVD in the RE around the fovea. **Figure 2.** shows accumulation of edema in the outer neurosensory retina with multiple cystic formations

### 3.1. Diagnosis

The patient was diagnosed to have LE Cystoid Macular Edema, RE cataract (showed moderate cortical opacities and NS) and RE incomplete PVD.

### 3.2. Management

Nonsteroidal anti-inflammatory drug (Ketrolac Tremetamol) one drop four times a day for LE was prescribed for 2 months ,and to come again for follow up , but patient didn't come . It was expected that the vision may not improve by much due to chronicity of CME.

## 4. Discussion

VL is a well-known complication of cataract surgery. Following VL there is an increased risk of retinal detachment, expulsive hemorrhage, CME, corneal decompensation, and secondary glaucoma.

This patient underwent ECCE, followed by partial anterior vitrectomy and ACIOL implant after VL. This VL was caused by leakage and aspiration by phaco tip due to damage of the lens posterior capsule as a result of zonulolysis (trauma may cause the zonulolysis). PI was needed to prevent pupillary block<sup>1</sup>. This procedure is a common practice in case of VL<sup>8</sup>.

There are many signs of VL during the operation such as, sudden deepening or shallowing of the AC and momentary pupillary dilatation, the nucleus falls away and cannot be approached by the phaco tip, vitreous aspirated into the phaco tip often manifests with a marked slowing of lens material aspiration and the torn capsule or vitreous gel may be directly visible<sup>1</sup>.

In this case, the VA dropped significantly within 6 months and OCT imaging shows that CME in the left eye has occurred. It has been found that the incidence of CME

was higher in cases of VL<sup>2</sup> and the most common cause of vision loss after cataract surgery is the development of CME<sup>3</sup>.

Vitreous traction plays a key role in the pathogenesis of aphakic CME (absence of anterior support of the vitreous)<sup>3</sup>. It is related to the disruption of the blood–retinal barrier, blood–aqueous barrier and the inflammation induced by prostaglandins or other inflammatory mediators<sup>4</sup>. VL and IOL presence in AC (posterior chamber is empty - absence of anterior support of the vitreous) cause continuous tractional force to the macular area (the site of firm attachment between vitreous and retina) which will lead to chronic CME .The Non-steroidal anti-inflammatory (NSAID) drug (Ketrolac Tremetamol) given to control this inflammation process may not help much because of chronicity.

It has been reported that incomplete PVD is related to CME development. What is shown in the RE (figure-1-)is incomplete PVD and this may put the patient at higher risk to develop CME<sup>9</sup> in future cataract operation. Thus it will be important to start the NSAID drugs preoperatively<sup>4</sup>.

One of the signs seen on the ACIOL in LE is foreign body giant cells; these cells are a typical part of the reaction of the inner eye to these implants. These giant cells are spread to an extremely large size, they may exhibit up to a hundred nuclei, and they often contain phagocytised pigment in their protoplasm. This reaction is limited and clinically quite harmless<sup>5</sup>. The most common cells found were iris pigment epithelial cells and the thickest membranes were probably formed by iris stromal cells<sup>6</sup>.

The progression of DR after ECCE is well documented<sup>7</sup>. A recent study demonstrated a progression of DR in both eyes when only one has had ECCE and systemic risk factors such as poor metabolic control may play a larger role in its progression<sup>7</sup>. However, this patient did not show DR suggesting a good sugar control. Careful and regular monitoring is important.

## 5. Conclusion

VL followed by posterior capsule damage is a complication that leads to conversion from phaco to ECCE and ACIOL implant. This will cause CME mainly by the continuous vitreous traction (chronic) with poor vision prognosis even with treatment by NSAID. Incomplete PVD shown in the RE will make it necessary to start NSAID preoperatively to prevent or decrease the incidence of CME.

In all diabetic patients who underwent cataract operation, giving them more attention and regular follow up is very

important in order to detect and manage the early or accelerated signs of DR after the surgery.

## Acknowledgment

I would like to thank the staff of Selayang Hospital for their help and support.

---

## References

- [1] Kanski, J. 2011. Clinical Ophthalmology: A Systematic Approach, 7th Edition, 288.
- [2] Preston, H.B., Rajiv, M.R. 2002. Visual outcomes after vitreous loss during cataract surgery performed by residents. *J Cataract Refract Surg* 28:847–852.
- [3] Sebag, J., Balazs, E.A. 1984 Pathogenesis of cystoid macular edema: An anatomic consideration of vitreoretinal adhesions. *Surv Ophthalmol* 28: 493-8.
- [4] Yavas, G. F., Ozturk, F., Kusbeci, T. 2007. Preoperative topical indomethacin to prevent pseudophakic cystoid macular edema. *J Cataract Refract Surg* 33:804–807.
- [5] Wolter, J.R. Foreign body giant cells on intraocular lens implants. *Graefe's Archive for Clinical and Experimental Ophthalmology* 219:103-111.
- [6] Puck, A., Tso, M. O. M., Yue, B. 1985. Cellular deposits on intraocular lenses. *Acta Ophthalmologica* 63: 54–60.
- [7] Aroca, P. R., Ballart, J. F., Garcia, M. A., et al. 2006. Nonproliferative diabetic retinopathy and macular edema progression after phacoemulsification: Prospective study. *J Cataract Refract Surg* 32:1438–1444.
- [8] Pearson, P.A., Owen, D.G., Maliszewski, M., Smith, T.J. 1989. Anterior chamber lens implantation after vitreous loss. *Br J Ophthalmol* 73:596-599.
- [9] Roldan, M., Serrano, J.M. 1989. Macular edema and vitreous detachment. *Ann Ophthalmol* 21(4):141-8.