CASE REPORT

Capsular Bag Distension Syndrome Following Phacoemulsification with Implantation of Intraocular Lens

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ABSTRAK

Sindrom distensi beg kapsul ialah salah satu komplikasi yang jarang berlaku selepas fakoemulsifikasi dengan implan kanta intraokular dimasukkan ke dalam bag kanta dalam mata. Kami menerangkan satu kes sindrom distensi beg kapsul yang berlaku pada seorang lelaki berusia pertengahan yang menjalani fakoemulsifikasi dengan 'plat haptic' implan kanta intraokular (Zeiss CT ASPHINA 509MP) ke dalam mata kanan. Penglihatan mata kanan semasa pemeriksaan susulan 1 minggu and susulan 5 minggu selepas pembedahan tidak menunjukan penambahbaikan dengan refraksi kuasa mata bertukar menjadi minus 2.5 diopter. Tekanan intraokular hanya meningkat pada 2 jam selepas pembedahan dan normal semasa pemeriksaan susulan berikutnya. Beg kapsul distensi telah disahkan dengan menggunakan IOL Master 700. IOL Master 700 menunjukkan pengasingan abnormal beg kapsul dari implan kanta intraokular. Diagnosis sindrom distensi beg kapsul yang disebabkan oleh pengekalan viskoelastik dibuat. Pengeluaran viskoelastik yang tertinggal di dalam mata kanan telah dilakukan dan sindrom distensi beg kapsul berjaya diselesaikan dengan penglihatan mata kanan kembali ke 6/6. Oleh itu, pengeluaran viskoelastik dengan sepenuhnya semasa fakoemulsifikasi dengan 'plat haptic' implan kanta intrakular adalah penting untuk mengelakkan sindrom distensi beg kapsul.

Kata kunci: asik hyaluronik, fakoemulsifikasi, kapsulotomi posterior, kelegapan kapsul, tekanan intraokular

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ABSTRACT

Capsular bag distension syndrome is a rare complication following phacoemulsification with posterior chamber intraocular lens implantation. We describe the case of a middle-age male, who developed early onset capsular bag distension syndrome of the right eye after phacoemulsification with implantation of plate haptic posterior chamber intraocular lens (Zeiss CT ASPHINA 509MP). There was persistent poor vision during follow up at 1 week and 5 weeks post-surgery with a myopic shift of 2.5 diopters sphere (DS). The intraocular pressure was only elevated at 2 hours post-operative and was normal during the subsequent follow-up. Distension of capsular bag was confirmed with the IOP Master 700 which showed abnormal separation of posterior capsule from the intraocular lens. He was diagnosed with capsular bag distension syndrome secondary to retention of viscoelastic. Right eye removal of retained viscoelastic was performed and the capsular bag distension syndrome resolved successfully with best corrected visual acuity at 6/6. Hence, complete clearance of viscoelastic during phacoemulsification with plate haptic intraocular lens is important in order to prevent capsular bag distension syndrome.

Keywords: capsule opacification, hyaluronic acid, intraocular pressure, phacoemulsification, posterior capsulotomy

INTRODUCTION

Capsular bag distension syndrome (CBDS) is an uncommon complication which occurs in less than 1% of patients undergoing phacoemulsification with implantation of posterior chamber intraocular lens (PCIOL) (Davison 1990; Kim & Shin 2008). There are a few terms which share the same meaning as CBDS, namely capsular block syndrome, capsular bag hyperdistension and capsulorhexis block syndrome. CBDS is characterised by fluid collection in between the PCIOL and posterior capsule, which pushes the PCIOL anteriorly and hyperdistension of the posterior capsule. The accumulation of fluid leads to reduction in visual acuity due to myopic shift (Haugsdal et al. 2016). CBDS can present within few weeks to years after cataract surgery (Pinarci et al. 2012; Kozeis et al. 2010). Due to the rarity of the CBDS, one needs to be aware especially when there are signs suggesting of CBDS. In this case report, we describe CBDS in a patient who underwent phacoemulsification with the implantation of plate haptic PCIOL (Zeiss CT ASPHINA 509MP).

CASE REPORT

A 50-year-old male underwent an uneventful right eye phacoemulsification with implantation of plate-haptic PCIOL. Preoperation, his best corrected visual acuity (BCVA) in the right eye was 6/24.

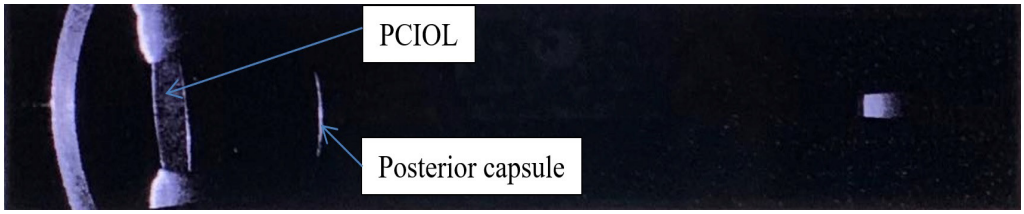

Figure 1: Intraocular lens appears to be shifted forward with a potential space between the posterior capsule and the intraocular lens.

Examination of the right eye revealed nucleus sclerosis of grade 1 with posterior subcapsular cataract grade 2. Otherwise, the rest of the examination was unremarkable. Intraoperative, ZEISS Z-HYALIN viscoelastic was used and an in the bag foldable single piece plate haptic ZEISS CT ASPHINA 509MP lens was implanted. The surgery was uneventful. Intraocular pressure at 2 hours post-operative was 30 mmHg secondary to retained viscoelastic in the anterior chamber. He was treated with a 3-day course of oral acetazolamide 250 mg twice daily dose for total of 3 days along with routine post-operative topical steroid and antibiotic every two hourly.

At 1-week postoperative visit, the right eye unaided visual acuity was 6/60 and pinhole visual acuity was 6/36. The anterior segment examination under slit lamp was unremarkable except for occasional microscopic cells seen in the anterior chamber. The PCIOL was stable in the capsular bag and intraocular pressure was 16 mmHg. Fundus examination was unremarkable too. Thus, the frequency of steroid and antibiotic eye drops was reduced to 4 hourly. Due to unexplained poor vision postoperative, patient was given an earlier appointment in 2 weeks.

At 3-week postoperative visit, the patient unaided visual acuity remained 6/60, with pinhole visual acuity of 6/24. Ocular examination findings were unremarkable with resolved anterior chamber inflammation. Hence, the topical steroid and antibiotic were tapered off.

At 5-week postoperative visit, the unaided visual acuity dropped to 3/60 and 6/36 with pinhole. Manifest refraction in the right eye was -2.50 DS. On slit lamp examination, the anterior chamber of the right eye was deep but appeared to be shallower compared to the left eye. The PCIOL appeared to be displaced anteriorly and the posterior capsule was seen convex towards the vitreous. Distension of capsular bag was confirmed with the Carl Zeiss IOL Master 700 which showed PCIOL appeared to be shifted forward with a potential space seen in between the posterior capsule and the PCIOL (Figure 1). Diagnosis of right eye capsular bag distension syndrome secondary to retention of viscoelastic was made.

The patient underwent removal of retained viscoelastic of the right eye 6 weeks, postoperatively. Intraoperatively, retained viscoelastic was visible posterior to the lens. The lens was displaced anteriorly just behind the iris with posterior

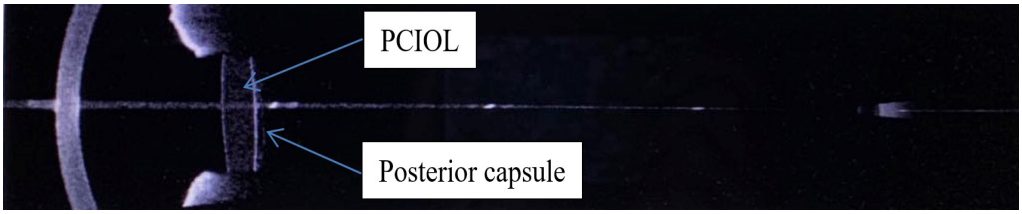

Figure 2: Intraocular lens is located in normal pseudophakic anatomy without any potential space in between the intraocular lens and posterior capsule. Anterior chamber is deeper compare to Figure 1.

concavity of the posterior capsule. The posterior capsule appeared to be more mobile with wrinkling of the posterior capsule after removing the retained viscoelastic. The patient was followed-up at 1-week post viscoelastic-washout in the clinic. BCVA improved to 6/6 with resolution of myopic shift. Carl Zeiss IOL Master 700 was used to demonstrate the PCIOL was located in normal pseudophakic anatomy. It also showed that the posterior capsule was located behind the PCIOL without any potential space separating the two structures (Figure 2).

DISCUSSION

CBDS can be classified into 3 categories according to its time of onset. The three classifications are intraoperative, early postoperative and late postoperative. Intraoperative CBDS is characterised by hyperdistension of the posterior capsule which is secondary to high irrigation pressures during hydrodissection. There is a higher risk of capsule rupture in this type of CBDS. Early postoperative CBDS is caused by incomplete removal of viscoelastic posterior to the intraocular lens. The signs of early postoperative CBDS are anterior displacement of the PCIOL, anterior chamber shallowing, intraocular pressure (IOP) elevation and myopic shift. This can occur in the first few weeks following cataract operation. Late postoperative CBDS can occur months or years after an uncomplicated cataract extraction. It is only noticeable when there is reduced visual acuity which is recognised by the patient as it does not exhibit any other symptoms (Patel et al. 2018). Intraocular pressure is usually normal in late onset CBDS which is different from the intraoperative and early-onset CBDS where intraocular pressure is usually high (Koh et al. 2016).

There are a few postulations on how CBDS can occur. One of it is due to the failure of intracapsular fluid to escape from the capsular bag. This is explained by tight adhesion between the anteriorly displaced IOL to anterior capsule which trap the intracapsular fluid. Besides that, it may also occur due to the nature of sodium hyaluronate, (a component in the viscoelastic) which imbibes fluid into the capsular bag to re-establish the osmotic balance (Dhaliwal et al. 2011; Sugiura et al. 2000). Other than the two postulations mentioned above, residual cortical cells secondary to incomplete removal during cataract surgery is thought to be the cause of late-onset CBDS. Proliferation of these residual cortical

Table 1: Reported cases of CBDS following cataract surgery

Author	Ashbala Khattak	Ashbala Khattak	Kozeis et al.	Dilraj et al.	Kah Joon et al.
Year	2017	2017	2010	2013	2019
Age/Gender	35/male	35/male	67/female	54/male	50/male
Intraocular lens	AT LISA tri toric 939MP	AT LISA tri toric 939MP	Acrysof IQ	-	CT Asphina 509MP
Type of haptic	Plate haptic	Plate haptic	Modified C-loop haptic	-	Plate haptic
Onset time	1 week	1 week	1 week	7 years	1 week
Uncorrected visual acuity	20/50	20/60	0.4	20/40	6/60
Postoperative refraction	-2.50 sphere	-2.0 sphere	-2.0 sphere	-1.50 sphere	-2.50 sphere
Treatment	Nd:YAG posterior capsulotomy	Nd:YAG posterior capsulotomy	Nd:YAG posterior capsulotomy	Nd:YAG posterior capsulotomy	Anterior chamber washout
Uncorrected visual acuity after treatment	20/25	20/30	0.9	20/25	6/6
Refraction after treatment	+0.50 sphere	-0.50 sphere	-0.75 sphere	-0.25 sphere	plano

cells will lead to production of alpha-crystalline proteins and ultimately forming a turbid fluid appearance in between the intraocular lens and the posterior capsule (Bao et al. 2008).

There are a few reported cases describing CBDS following cataract surgery in the literature. Table 1 summarises these published cases of CBDS following cataract surgery, including this case (Khattak 2017; Dilraj & Robert 2013; Galvin et al. 2018; Kozeis et al. 2010). From Table 1, most of the patients presented as early onset postoperative CBDS. Their presentations were poor visual acuity with myopic shift which were seen in all cases. The main treatment for early onset postoperative CBDS is Nd: YAG posterior capsulotomy. Outcome of the YAG laser was resolution of the myopic shift and improved in the visual acuity. On the other hand, late-onset CBDS cases were reported by Galvin et al. in 2018. Both the patients showed milky substance seen in between the intraocular lens and posterior capsule. They were treated with pars plana vitrectomy with posterior capsulotomy. Visual acuity of both patients improved following pars plana vitrectomy and posterior capsulotomy. Resolution of myopic shift was seen in one of the late-onset CBDS. As mentioned before, late-onset CBDS is due to proliferation of the incomplete removal of cortical cells. Thus, posterior capsulotomy alone will impose risk of exposing any protein into the vitreous cavity which can lead to intraocular inflammation. Hence, pars plana vitrectomy with posterior capsulotomy was used to achieve the aim of complete removal of milky fluid

and retained cortical material (Galvin et al. 2018).

This case report describes an early-onset CBDS in a patient that underwent uncomplicated cataract surgery with a single-piece plate haptic ZEISS CT Asphina 509MP PCIOL. Carbonic anhydrase inhibitor medication was given in attempt to resolve the CBDS but failed. Removal of the retained viscoelastic successfully resolved the capsular block in the eye.

Studies have shown that four-haptic PCIOLs, compared to C-loop PCIOLs, are at greater risk of developing CBDS after cataract extraction (Kim & Shin 2008). However, Khattak (2017) reported a case of bilateral CBDS in a patient that underwent cataract extraction with a single-piece plate haptic design PCIOL (AT LISA tri toric 939MP). The lens implanted in our patient was CT Asphina 509MP which is also a plate haptic design PCIOL.

Diagnosis of CBDS is usually clinical which can be made at the slit lamp examination. Visualisation of turbid and opaque fluid in between the PCIOL and posterior capsule is the diagnostic sign of CBDS. However, in our patient, there was no turbid fluid seen behind the PCIOL. This leads to difficulty in diagnosis of CBDS. Clinical suspicion of CBDS can be made when there is significant myopic refractive error post-surgery along with an anteriorly displaced IOL and presence of a significant space between the PCIOL and the posterior capsule. These signs were seen in our patient. CBDS could be diagnosed earlier if awareness of CBDS is high among cataract surgeon. Anterior segment optical coherence tomography (OCT) and anterior ultrasound biomicroscopy (UBM) are proven to be able to identify CBDS. (Das 2010) However, due to operator dependent factor, UBM findings vary with different operators. In this case, the Carl Zeiss IOL Master 700 was useful in identifying the position of PCIOL and the posterior capsule in order to diagnose CBDS.

The outcome of CBDS cases treated conservatively without any intervention is not favourable. Only 2 (15%) out of 13 subjects had spontaneous resolution of CBDS without treatment (Durak et al. 2001). Posterior capsulotomy or peripheral anterior capsulotomy with Nd:YAG laser is proven to be effective treatment for CBDS (Pinsard et al. 2011). However, this was not performed in our patient due to fine posterior capsule outline and concern of inducing lens-pitting caused by poor focusing of posterior capsule during the procedure of YAG capsulotomy. Instead, removal of retained viscoelastic was done. YAG capsulotomy in a patient who has had phacoemulsification done 5 weeks ago can potentially worsen the inflammation. Dr. Vlasenko is also in the opinion that although YAG capsulotomy is the choice of treatment, surgical aspiration is recommended in cases with inflammatory symptoms or signs (Binder 2018). The result of the surgically removal of retained viscoelastic in this case was resolution of the myopic shift and normal vision which is comparable to the treatment with YAG capsulotomy.

In primate aqueous humors, the half-life of sodium hyaluronate is approximately 2-7 days depending

on the viscosity (Schubert et al. 1981). Clinical observations in humans have supported this result. However, we could still see viscoelastic trapped in between the intraocular lens and the posterior capsule during the operation. This could be due to the tight adhesion between the anteriorly displaced intraocular lens with the anterior capsule, which trapped the viscoelastic inside the capsular bag. Thus, removal of viscoelastic from the eye via physiological aqueous drainage system is impaired.

CONCLUSION

CBDS is a very rare condition following cataract surgery. As we discussed earlier, early CBDS is usually secondary to retained viscoelastic following cataract surgery. Hence, complete clearance of viscoelastic during phacoemulsification with plate haptic intraocular lens is important in order to prevent CBDS.

REFERENCES

Bao, Y.Z., Pei, X.T., Li, M.W. 2008. Late postoperative capsular block syndrome versus liquefied after-cataract. *J Cataract Refract Surg* **34**(10): 1799-1802.

Binder, S.P. 2018. Capsular bag distension syndrome: classification and management strategies. https://www.eyeworld.org/capsular-bag-distension-syndrome-classification-and-management-strategies [12 June 2019].

Das, K. 2010. Delayed capsular bag distension syndrome. *Oman J Ophthalmol* **3**(3): 155-6.

Davison, J.A. 1990. Capsular bag distension after endophacoemulsification and posterior chamber intraocular lens implantation. *J Cataract Refract Surg* **16**(1): 99-108.

Dhaliwal, D.K., Farhi, P., Eller, A.W., Kowalski, R.P. 2011. Late capsular block syndrome associated with propionibacterium acnes. *Arch Ophthalmol* **129**(2): 246-7.

Dilraj, S.G., Robert, S.F. 2013. The curious case of progressive myopia. https://www.aao.org/eyenet/article/morning-rounds-curious-case-of-progressive-myopia [12 June 2019].

Durak, I., Ozbek, Z., Ferliel, S.T., Oner, F.H., Söylev, M. 2001. Early postoperative capsular block syndrome. *J Cataract Refract Surg* **27**(4): 555-9.

Galvin, J.C., Berdoukas, P., Fung, A.T. 2018. Two cases of very late-onset capsular bag distension syndrome. *Am J Ophthalmol Case Rep* **10**: 268-70.

Haugsdal, J., Killoran, E., Oetting, T.A. 2016. Capsular block syndrome: an unusual presentation. http://EyeRounds.org/cases/238-Capsular-Block-Syndrome.htm [11 June 2019].

Khattak, A. 2017. Bilateral early capsular block syndrome following implantation of the new trifocal toric lens. *Oman J Ophthalmol* **10**(3): 238-40.

Kim, H.K., Shin, J.P. 2008. Capsular block syndrome after cataract surgery: clinical analysis and classification. *J Cataract Refract Surg* **34**(3): 357-63.

Koh, J.S., Song, Y.B., Wee, W.R., Han. Y.K. 2016. Recurrent late-onset fibrotic capsular block syndrome after neodymium-yttrium-aluminum-garnet laser anterior capsulotomy: a case report. *BMC Ophthalmol* **16**(1): 86.

Kozeis, N., Gatzioufas, Z., Schirra, F., Loew, U., Seitz, B. 2010. Focal 6-O'-Clock Nd: Yag-capsulotomy for the treatment of early onset incomplete capsular block syndrome. *Hippokratia* **14**(4): 277-8.

Patel, A.S., Mammo, D.A., Delmonte, D.W. 2018. Capsular bag distension syndrome. https://eyewiki.aao.org/Capsular_bag_distension_syndrome [11 June 2019].

Pinarci, E.Y., Bayar, S.A., Sizmaz, S., Canan, H., Yilmaz, G. 2012. Late capsular block syndrome presenting with posterior capsule opacification. *J Cataract Refract Surg* **38**(4): 672-6.

Pinsard, L., Rougier, M.B., Colin, J. 2011. Neodymium: yag laser treatment of late capsular block syndrome. *J Cataract Refract Surg* **37**(11): 2079-80.

Schubert, H., Denlinger, J.L., Galzs, E.A. 1981. Na-hyaluronate injected into the anterior chamber of the owl monkey: effect on iop and range of disappearance. *ARVO* **9**(Abstr): 118.

Sugiura, T., Miyauchi, S., Eguchi, S., Obata, H., Nanba, H., Fujino, Y., Masuda, K., Akura, J. 2000. Analysis of liquid accumulated in the distended capsular bag in early postoperative capsular block syndrome. *J Cataract Refract Surg* **26**(3): 420-5.

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