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# **Pseudophakic Bullous Keratopathy after Anterior Iris-Fixated Intraocular Lens Implantation**

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## **Authors' contributions**

*This work was carried out in collaboration between both authors. Author FS designed the study, collect the data and wrote the first draft of the manuscript. Author RH also managed the literature searches of the study. Both authors read and approved the final manuscript.*

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**Case Study**

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## **ABSTRACT**

**Introduction:** To report a case of pseudophakic bullous keratopathy (PBK) complication after anterior chamber (AC) iris-fixated intraocular lens (IOL) secondary implantation.

**Presentation of Case:** A 65-year-old man came with a red, painful, uncomfortable right eye. He felt a blurry vision and a severe headache. Three years ago, he had cataract surgery of the right eye. Afterward, he had IOL luxation and had an IOL exchange surgery. His right eye showed ciliary injection and visual acuity was counting finger at one meter. Examination on his cornea showed edema with deep corneal fold, stromal haze, and epithelial defect due to ruptured bullae. On his right eye, there was an iris-clip AC IOL which the position was a little bit tilted and suspected to had come in contact with the cornea. His left corneal endothelial count exceeded 2000, however, the right cornea endothelial count data could not be taken. He was diagnosed with pseudophakic bullous keratopathy (PBK) and treated with topical antibiotics and an eye lubricant.

**Discussion:** PBK is a clinical diagnosis of irreversible corneal edema and endothelial damage that occurs after cataract extraction and IOL implantation. Endothelial loss in secondary IOLs is suggested because of the mechanical injury from instruments or IOL during a surgical procedure. The important risk factors are shallow anterior chamber depth, a shorter distance between the IOL edge to the endothelium, inflammation, and design of the IOLs.

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**Conclusion:** A complete preoperative examination, careful selection of IOL types, and modification of surgical techniques could minimize the risk of endothelial damage complications.

**Keywords:** *Pseudophakic bullous keratopathy; anterior chamber intraocular lens; cataract surgery; endothelium decompensation; case report; corneal edema; endothelial damage.*

## 1. INTRODUCTION

There are numerous cataract surgery complications. Among these complications, pseudophakic bullous keratopathy (PBK) is a significant cause of morbidity and irreversible vision loss [1]. About 1-2% of people will develop PBK after cataract surgery, on average within eight months to seven years [2]. One of the known pathogenesis causing PBK is closely related to the selection of secondary IOL, particularly anterior chamber IOLs (ACIOLs) type [1].

There are several options when zonular support is insufficient to hold intraocular lens (IOL) in the capsular bag, including ACIOL, transscleral-fixated or iris-fixated posterior chamber IOL (PCIOL), 3-piece IOL in the ciliary sulcus (with or without optic capture), or a 3-piece IOL with the haptic placed in zonular weakness points [3,4]. Because of its placement, an antepupillary or anterior chamber (AC) iris-fixated lens is often categorized into the ACIOLs group. Despite their large incision, ACIOLs, especially with flexible open-looped haptics, are still a widely used technique for secondary IOL in 23% to 50% of cases [3]. Incidence of ACIOLs (closed-loop, iris-fixated, and iris-plane) progress to PBK ranges between 1-10% [1]. This article aims to report a case of pseudophakic bullous keratopathy (PBK) complication after anterior chamber (AC) iris-fixated intraocular lens (IOL) secondary implantation.

## 2. PRESENTATION OF CASE

A 65-year-old man came to an eye clinic with a chief complaint of a red, painful, uncomfortable right eye for about a week. He also felt a blurry vision and a severe headache that has disturbed his sleep ever since. The pain radiated to the right side of the face and head. He had cataract surgery of the right eye three years ago in another hospital. Two weeks after the surgery, he underwent second surgery of removing luxated IOL and implanting a new IOL. He had no complaint ever since.

On ocular examination, his right eye visual acuity was counting finger at one meter, with no improvement on pinhole. His left eye was 6/9, but the best-corrected visual acuity (BCVA) was 6/6 with his current spectacles. The right eye showed ciliary injection. On slit-lamp examination, his cornea was edema with deep corneal fold, stromal haze, and epithelial defect due to ruptured bullae. On his right eye anterior chamber, there was an iris-clip ACIOL which the position was a little bit tilted and suspected to had come in contact with the cornea. Intraocular pressure (IOP) of both eyes is was 16 mmHg with a Tonopen.

His right eye was diagnosed with pseudophakic bullous keratopathy (PBK), corneal decompensation, and corneal irritation. His left eye was diagnosed with pseudophakia. He was given a Levofloxacin drop, a Chloramphenicol 0.25% ointment, a Cendo Hyalub eye lubricant and was advised follow up next day.

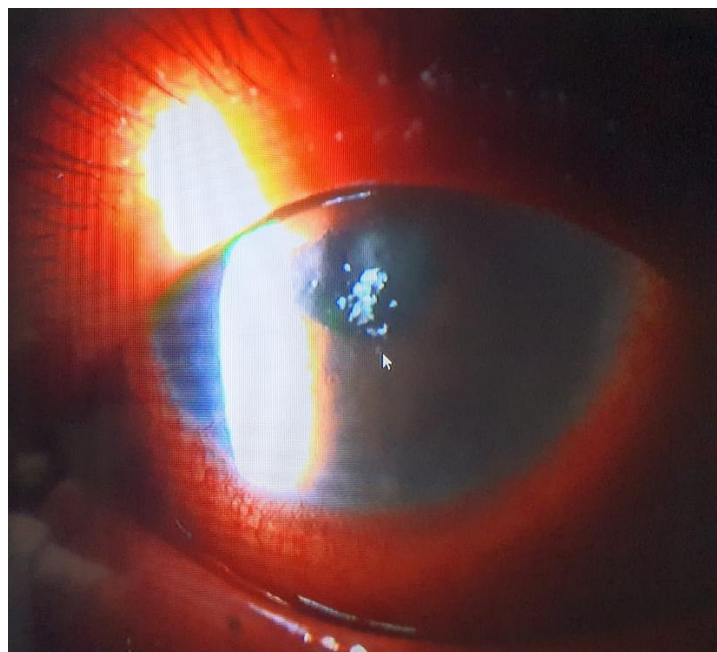
On the second day, his right eye visual acuity worsened with counting finger at 30cm with no improvement on pinhole. His anterior segment ophthalmic examination showed the same results. On the other hand, he felt much more comfortable after his medication even though he still had some pain. Specular microscopy examination of the left eye was normal. His left corneal endothelial count exceeded 2000. However, the right cornea endothelial count data could not be taken because of severe edema. He was instructed to continue the prescription and to come back for a maintenance follow-up in 2 weeks. The patient was given thorough information about his condition and prognosis of permanent visual impairment in his right eye. He was also informed about the probability of corneal transplantation and secondary IOL removal if the discomfort persisted. However, as discussed with the patient, because of a donor rarity in Indonesia and relatively expensive corneal transplantation procedure, if there was no more pain and inflammation was relatively contained by medication, the patient would undergo routine outpatient follow-up.



**Fig. 1. External view of both eyes**



**Fig. 2. Right eye: Note ciliary and conjunctival injection, hazy cornea and hyperlacrimation**



**Fig. 3. Close up view of the right eye**

### 3. DISCUSSION

The most densely innervated tissue in the human body is the cornea [5]. It is innervated by the ophthalmic nerve, the first division of the fifth cranial nerve [6]. From the outer to the inner layer, the cornea consists of epithelium, Bowman layer, stroma, Descemet membrane, and endothelium. It is avascular and received its oxygen from the tear film and nutrients from the aqueous humor [7].

The following mechanisms maintain transparency of the cornea:

1. The endothelium dual actions, first as a permeability barrier from aqueous humor with its tight junction and second as an active pump to maintain stromal dehydration state by generating negative hydrostatic pressure [7].
2. The stromal collagen regular lattice-like arrangement, even distribution, and is much smaller than light wavelength size [1].

The corneal endothelium is a non-regenerating monolayer hexagonal cell with a total cell count of approximately 3000 cells/mm<sup>2</sup> in young adults [5,7]. Cell count number is higher in the periphery, decreases with age and trauma. In normal aging, the rate of corneal endothelium cell loss is 0.6% per year [7]. After each cell loss, healing occurs through the remaining surrounding endothelium cells migration, rearrangement, and enlargement [7]. Cell count of 400-700 cells/mm<sup>2</sup> is the minimum required amount for normal function. Otherwise, aqueous influx will outweigh the remaining endothelial cells' pumping capacity, resulting in corneal edema and impaired transparency [2,5,7].

Corneal endothelial cell loss could happen pre-operatively, intra-operatively or post-operatively resulted in irreversible corneal edema [2]. Preoperatively low endothelial cell count increases the risk of PBK. Surgical trauma is an important factor, either directly from instrumentation or indirectly from excess phaco power, the toxicity of irrigating solution, or posterior capsular rupture with vitreous prolapse anteriorly. Inflammation and glaucoma are major causes that contributed to postoperative endothelial cell loss.

The low endothelial cell count leads to chronic progressive stromal and epithelial edema, the

latter results in the formation of bullae [2]. Pseudophakic bullous keratopathy (PBK) is a clinical diagnosis of irreversible corneal edema and endothelial damage that occurs after cataract extraction and IOL implantation [1]. PBK is often associated with reduced vision, ocular irritation, foreign body sensation, and epiphora [4]. Pain sensation in PBK is a result of stretching of the ophthalmic nerve or from ruptured corneal bullae which irritate the corneal nerve endings [1,2]. The ruptured bullae increases the risk of infectious keratitis. Risk factors for PBK are advanced age, preexisting Fuch's corneal dystrophy, previous intraocular surgery, shallow COA, glaucoma, previous ocular trauma, systemic conditions, and insertion of ACIOL [2].

Traditionally, with sufficient capsular integrity, IOL is placed intracapsular. In the absence of zonular support, ACIOL or PCIOL placement with various designs and implantation techniques could be the alternative solution [8]. Some older ACIOL designs have been associated with a severe or complete daily living disability because of an increased corneal decompensation post-operative complication. Iris-clip lenses with IOL placement anterior to the iris are proven to be in contact with the endothelium during eye movement [4]. Closed-loop ACIOLs produce chronic inflammation and contact with peripheral endothelial cells [4]. A review by the American Academy of Ophthalmology concluded that between 8 types of ACIOLs and PCIOLs with various designs and implantation techniques there was no difference in safety and visual outcome in an average 6-month follow-up [8]. Nevertheless, endothelial cell loss might be higher in an antepupillary (AC) than in a retropupillary iris-fixated lens [9].

Endothelial loss in secondary IOLs is proposed because of the mechanical injury from instruments or IOL during a surgical procedure. PBK in eyes with ACIOLs, the important risk factors are shallow anterior chamber depth, a shorter distance between IOL edge to the endothelium, inflammation, and design of the IOLs [1,9]. Lens positioning and stability as well as lens material and finishing, are crucial to corneal clarity maintenance [1]. The edema in ACIOLs eyes characteristically begins in a localized trauma zone which will then progress to surrounding areas [1]. As in this case, visual acuity worsening on the second day in our patient could be a sign of edema progression.

To prevent PBK in iris-fixated lens implantation, there should be complete removal of vitreous from the anterior chamber, at least 270 degrees of iris tissue with pupil size smaller than 6 mm, and an anterior chamber depth of 3 mm or greater [9]. Modification of cataract surgical techniques like generous application of viscoelastic and ultrasound sparing techniques could also lower the risk of PBK [2]. Cataract extraction without ultrasound like extracapsular cataract extraction (ECCE) can be applied to minimize intraoperative endothelial cell damage [2].

PBK initial treatment should aim to control inflammation, avoid IOP elevation, and decrease the edema with hypertonic sodium chloride drop or ointment to draw water out from the cornea [4,10]. Ruptured bullae need topical antibiotics along with bandaged contact lenses and lubricating drops [2]. The gold standard treatment for a patient with PBK is corneal transplantation which could relieve symptoms and recovers visual function [6]. Patient with PBK complication associated with ACIOL is treated by endothelial keratoplasty in combination with IOL exchange [4]. Indications for keratoplasty are decreased vision, recurrent keratitis, and pain. However, in developing countries, including Indonesia, there is a shortage of corneal donors and a long waiting list, therefore temporary symptomatic alternatives are often needed. Other temporary but symptom-improving options are corneal collagen cross-linking (CXL), amniotic membrane transplant (AMT), anterior stromal puncture (ASP), and phototherapeutic keratectomy (PTK) [2,6].

In our eye hospital, patients with endothelial cell count below 1500 cells/ mm<sup>2</sup> are made aware of the possibility of PBK and obligated to fill a specialized informed consent form. In this case, the patient had his surgery in another hospital and didn't have any specular microscopy data in the data he showed us. However, his specular microscopy on the contralateral eye was normal, excluding the suspicion of underlying corneal dystrophy as this condition is almost always bilateral [1]. Other than senility, the risk factors he had including a history of IOL subluxation and insertion of ACIOL in his second surgery which somehow has tilted. In addition, there were no data of anterior chamber depth before surgery, in which a shallow anterior chamber might be a factor causing his ACIOL to intermittently rub his cornea.

#### **4. CONCLUSION**

From this case, we learned that to prevent PBK, a complete preoperative examination including specular microscopy should be performed. If there is any indication of secondary IOL implantation, anterior chamber depth examination should also take place. Careful selection of IOL types could minimize the risk of endothelial damage complications. Modification of surgical techniques which could lower the risk of PBK should also be considered in high-risk patients. The definitive treatment for PBK is corneal transplantation, however, several temporary remedies can be given for patients in waiting.

#### **CONSENT**

All authors declare that written informed consent was obtained from the patient for publication of this case report and accompanying images.

#### **ETHICAL APPROVAL**

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

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#### **COMPETING INTERESTS**

Authors have declared that no competing interests exist.

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