



Fibrin Membrane Pupillary-Block Glaucoma After Uneventful Cataract Surgery Treated with Intracameral Tissue Plasminogen Activator

Tülay Alpar Akçetin, Kadir Eltutar, Nurhan Dinçer, Fatma Esin Özdemir

Abstract

To evaluate the diagnostic and therapeutic methods of fibrin-dependent pupillary-block glaucoma (PBG). A 79-year-old male patient with hypertension was admitted to hospital with symptoms of pain and redness 6 days after an uneventful cataract surgery. He had acute elevation of intraocular pressure with a shallow anterior chamber. Because of severe corneal edema, the anterior chamber details could not be distinguished. After systemic and topical antiglaucoma treatment, corneal edema was reduced and a complete fibrin membrane was observed across the pupil on slit lamp examination. A complete fibrin membrane across the pupillary space, shallow anterior chamber, and deep posterior chamber was confirmed by ultrasonic biomicroscopy (UBM). The intraocular lens (IOL) was actually displaced posteriorly, with a large clear space between the IOL and iris. Despite topical and subconjunctival corticosteroid therapy as well as Nd:YAG laser membranotomy, the fibrin membrane was not resolved. Tissue plasminogen activator (tPA) (25 µg) was injected intracamerally and the fibrin membrane was completely dissolved, improving the pupillary block. Anterior segment imaging techniques, especially UBM, are a powerful diagnostic technique for differentiating the technique for the analysis of the mechanisms underlying acute angle-closure glaucoma mechanism. Fibrin-dependent PBG was successfully treated with an intracameral injection of tPA

Keywords: Fibrin membrane, pupillary block glaucoma, cataract surgery, ultrasound biomicroscopy, tissue plasminogen activator

Introduction

Fibrin membrane-dependent pupillary block glaucoma (PBG) is a rare complication seen after uneventful phacoemulsification cataract surgeries (1, 2). The clinical picture coursing with peripheral angle closure and increased intraocular pressure (IOP) due to the pupillary space being completely covered with fibrin membrane is called as fibrin-induced PBG (2). Severe corneal edemas developing because of increased IOP hinders biomicroscopic examination and makes early diagnosis difficult.

Its treatment is still controversial. The membrane can form again after Nd: YAG laser membranectomy, or an endothelial destruction can develop when laser peripheral iridotomy is performed due to corneal edema associated with IOP (1).

In this study, the treatment of the fibrin membrane through anterior segment imaging and intracameral tissue plasminogen activator (tPA) injection was presented in a patient developing PBG due to the fibrin membrane.

Case Report

A 79-year-old male patient receiving hypertension therapy was admitted to the hospital. His right visual acuity (VA) was 0.8 and left VA was 0.1. The right-left IOP was 15 mmHg. Corneas were clear in both eyes, and the depth of the anterior chamber was normal. Pseudophakia and nuclear cataract were detected in the right and left eyes, respectively. After an uneventful phacoemulsification cataract surgery, intracapsular hydrophobic acrylic intraocular lens (IOL) (Acriva-VSY) was placed. In the examination performed on the first postoperative day, the left eye VA was 0.8 and IOP was 14 mmHg. The depth of the anterior chamber was normal, and there was a mild inflammation in the anterior chamber (1+ cell reaction). Topical lomefloxacin (Okacin-Novartis) 5x1 and topical prednisolone acetate (Pred Forte 1%-Allergan) were given every hour for the first 2 days, and the dose was then switched to 5x1/day. His control examination on the third postoperative day was normal. On the sixth day, the patient was admitted to our hospital with sudden onset of pain, redness, and low-vision in the operated eye. His left VA was 0.05 and IOP was 48 mmHg. Congestion was observed in the

Clinic of Ophthalmic, Istanbul Training and Research Hospital, Istanbul, Türkiye

Address for Correspondence:
Tülay Alpar Akçetin, Istanbul Eğitim ve Araştırma Hastanesi, Göz Kliniği, İstanbul, Türkiye
Phone: +90 536 619 36 15
E-mail: talparakcetin@yahoo.com.tr

Received:
25.02.2013

Accepted:
30.12.2014

© Copyright 2015 by Available online at
www.istanbulmedicaljournal.org

conjunctiva. The depth of the anterior chamber was too decreased, despite the fact that it could not be evaluated with certainty due to an intense corneal edema. The patient was initiated 200 cc. Intravenous mannitol 20%, systemic acetazolamide 4x1/day, topical dorzolamide–timolol maleate 2x1 (Cosopt-Merck Sharp and Dohme), and topical tropicamide (Tropamid 0,05%-Bilim) were initiated. Because corneal edema was decreased when IOP decreased with an anti-glaucoma (25 mmHg), fibrin membrane was seen in the pupillary region (Figure 1). With the fibrin membrane covering the pupillary region, it was observed through UBM (Sonomed Planar CLI 1500) that the anterior chamber was shallow (Figure 2) and posterior chamber was deep (Figure 3). Owing to UBM imaging, the differential diagnosis of PBG was performed with capsular block glaucoma and other acute glaucoma factors.

With topical and subconjunctival corticosteroid and mydriatics therapies, the fibrin membrane was opened a little in the temporal and the anterior chamber was deepened slightly in that region, but full resolution did not happen. On the third day, membranotomy was performed from the pupillary margin with an Nd:YAG Laser. The membrane completely separated from the pupillary margin, but it occurred again the following day.

This time, an intracameral 25 µg tPA injection was given. Fibrin membranolysis developed within an hour, and the depth of the anterior chamber returned to normal (Figure 4). Systemic anti-glaucoma therapy was discontinued. With topical corticosteroid and anti-glaucoma therapy, IOP was 16 mmHg and VA was 0.5 in his control examination performed a week later.



Figure 1. The view of the fibrin membrane covering the pupillary region

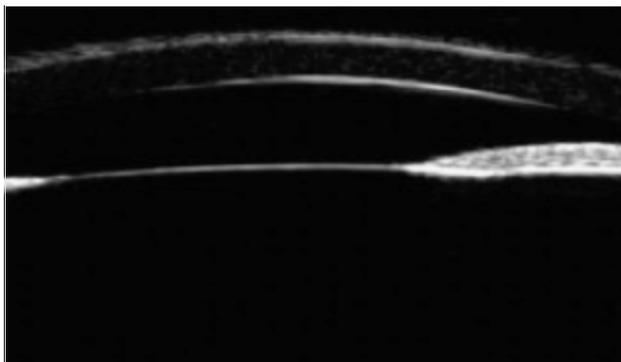


Figure 2. The fibrin membrane in the pupillary region (UBM image)

Discussion

After cataract surgery, an increased IOP and a narrowed anterior chamber can develop due to fibrin membrane-dependent pupillary block, Soemmering's ring, posterior synechia, capsular block syndrome, and malignant glaucoma (1, 2). Although fibrin membrane-dependent PBG is frequently seen after pars plana vitrectomy (PPV), there are a few studies on its development after cataract surgery (3). It is difficult to detect a fine fibrin membrane due to the intense corneal edema associated with an increased IOP, which causes the diagnosis to be delayed. UBM, with its echographic feature, allows the anterior segment to be monitored. Thus, it is possible to rapidly establish the diagnosis and to initiate the treatment early in the case of acute increased IOP.

Although anterior segment optic coherence tomography (OCT) is a useful technique for acute angle closure glaucoma, it offers limited data in the presence of a corneal haze. Moreover, because the penetration depth of anterior segment OCT is lower than that of UBM, the examination of the ciliary body, which should be evaluated for the diagnosis of malignant glaucoma, is difficult (2).

In this case, anterior segment OCT was insufficient to evaluate the localization of IOL in the posterior chamber.

UBM is a strong diagnostic assistant in acute angle-closure glaucoma (4). With the detection of separation between the posterior chamber IOL and iris, a differential diagnosis of PBG can be made with malignant glaucoma, capsular block syndrome, and intra-vitreous gas expansion. The diagnosis is supported when the 360°

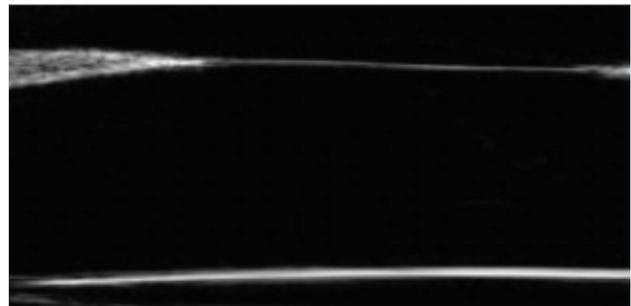


Figure 3. The deep posterior chamber and localization of IOL at the back in the UBM image

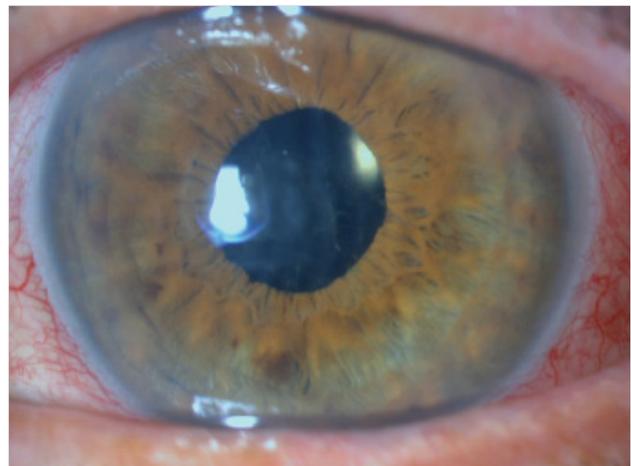


Figure 4. The view after the intracameral tPA injection

peripheral iridocorneal contact, the transparent space between the posterior chamber IOL and iris, and distant localization of IOL from the iris and fibrin membrane are detected through UBM (2).

Khor et al. (2) examined fibrin-dependent PBG after phacoemulsification in their study including four cases, and they defined the role of OCT and UBM in differentiating fibrin block glaucoma from other postoperative acute glaucoma reasons. In this particular situation, the aqueous accumulates in the posterior chamber as the fibrin membrane completely covers the pupilla. It was reported that 3 of these 4 patients had diabetes mellitus (DM) (2). On the other hand, previous reports suggest that the development of fibrin reaction after cataract surgery in patients with DM is associated with an impaired blood–aqueous barrier (5).

The incidence of the occurrence of fibrin after cataract surgery is between 2% and 8% in the general population (2). In ocular events such as uveitis and pseudoexfoliation syndrome, this rate increases in systemic diseases such as HT and DM (1, 2, 5, 6). In a large series, the development of fibrin after extracapsular cataract extraction was observed to be more frequent in patients with DM, and the development of fibrin-dependent PBG in a patient was reported (5).

Increased IOP is considered as a result of fibrin formation, and it is not very common. In a series including six cases, IOP was found to be high only in two of the patients with fibrin membrane (7).

In patients having small pupilla, iris manipulation or trauma during surgery causes an impaired blood–aqueous barrier, inflammation, and fibrin formation. Moreover, small pupilla can be more easily closed with the fibrin membrane. Therefore, patients with small pupilla should be evaluated with regard to mydriatic agents (2).

The treatment of fibrin-dependent PBG has not been determined with certainty to date (1). Cases developing after PPV are often treated with laser peripheral iridotomy (8) or intraocular tPA (9). In a case series, fibrin PBG developing after cataract surgery was successfully treated with Nd:YAG laser peripheral iridotomy (1). Nd:YAG laser fibrin membranotomy was applied to two patients, but the membrane developed again in one of them. In our case, after Nd:YAG laser fibrin membranotomy was performed, the membrane developed again on the following day.

Nd:YAG laser iridotomy is a valid treatment method for fibrin-dependent PBG. However, if there is an intense corneal edema and iridocorneal contact, its implementation is difficult, and there is also the risk of endothelial damage (1).

In a randomized prospective study, it was reported that a single dose of 10 µg tPA injection decreased the incidence of fibrin formation and its amount without any side effect in patients developing fibrin reaction in the anterior chamber after cataract surgery (10).

In cases where it is difficult to evaluate the anterior chamber, tPA injection can be safely applied to the anterior chamber after a carefully performed ultrasound examination (1).

Conclusion

In patients developing a narrow anterior chamber after cataract surgery, the differential diagnosis of fibrin membrane PBG should be carefully made. UBM is an important diagnostic tool in cases with an intense corneal edema. In our case, fibrin membrane-dependent pupillary block was successfully treated with an intracameral tPA injection.

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - T.A.A.; Design - T.A.A., N.D.; Supervision - N.D., K.E.; Funding - T.A.A.; Materials - T.A.A.; Data Collection and/or Processing - T.A.A., F.E.Ö.; Analysis and/or Interpretation - T.A.A., N.D.; Literature Review - T.A.A., K.E.; Writer - T.A.A.; Critical Review - N.D., K.E.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

References

1. Yoshino H, Seki M, Ueda J, Yoshino T, Fukuchi T, Abe H. Fibrin membrane pupillary-block glaucoma after uneventful cataract surgery treated with intracameral tissue plasminogen activator: a case report. *BMC Ophthalmol* 2012; 12: 3. [\[CrossRef\]](#)
2. Khor WB, Perera S, Jap A, Ho CL, Hoh ST. Anterior segment imaging in the management of postoperative fibrin pupillary-block glaucoma. *J Cataract Refract Surg* 2009; 35: 1307-12. [\[CrossRef\]](#)
3. Jaffe GJ, Lewis H, Han DP, Williams GA, Abrams GW. Treatment of post-vitrectomy fibrin pupillary block with tissue plasminogen activator. *Am J Ophthalmol* 1989; 108: 170-5. [\[CrossRef\]](#)
4. Mansouri K, Sommerhalder J, Shaarawy T. Prospective comparison of ultrasound biomicroscopy and anterior segment optical coherence tomography for evaluation of anterior chamber dimensions in European eyes with primary angle closure. *Eye* 2010; 24: 233-9. [\[CrossRef\]](#)
5. Krupsky S, Zalish M, Oliver M, Pollack A. Anterior segment complications in diabetic patients following extracapsular cataract extraction and posterior chamber intraocular lens implantation. *Ophthalmic Surg* 1991; 22: 526-30.
6. Ozveren F, Eltutar K. Therapeutic application of tissue plasminogen activator for fibrin reaction after cataract surgery. *J Cataract Refract Surg* 2004; 30: 1727-31. [\[CrossRef\]](#)
7. Norris JW, Chirls IA, Santry JG, Norris JW III. Severe fibrinous reaction after cataract and intraocular lens implantation surgery requiring neodymium: YAG laser therapy. *J Cataract Refract Surg* 1990; 16: 637-9. [\[CrossRef\]](#)
8. Lewis H, Han D, Williams GA. Management of fibrin pupillary-block glaucoma after pars plana vitrectomy with intravitreal gas injection. *Am J Ophthalmol* 1987; 103: 180-2. [\[CrossRef\]](#)
9. Jaffe GJ, Lewis H, Han DP, Williams GA, Abrams GW. Treatment of post-vitrectomy fibrin pupillary block with tissue plasminogen activator. *Am J Ophthalmol* 1989; 108: 170-5. [\[CrossRef\]](#)
10. Heiligenhaus A, Steinmetz B, Lapuente R, Krallmann P, Althaus C, Steinkamp WK, et al. Recombinant tissue plasminogen activator in cases with fibrin formation after cataract surgery: a prospective randomized multicentre study. *Br J Ophthalmol* 1998; 82: 810-5. [\[CrossRef\]](#)