CLINICAL CASES
CLINICAL CASE PRESENTATION
Y. Barkana

A 70 year-old generally healthy woman presented with discomfort and decreased vision in her only good-seeing right eye. She had had extracapsular cataract extraction with PCIOL 12 years previously. In this right eye visual acuity was 20/150, IOP was 34 mmHg. The external eye was quiet, cornea dystrophic but without edema. The anterior chamber was uniformly shallow. There was a sector iridectomy superiorly. Angle details could not be visualized even with forceful corneal compression with the gonioscope. There was no vitreous or choroidal pathology. The disc had 0.7 cupping. Initial treatment with pilocarpine resulted in elevation of IOP and was immediately discontinued. Subsequent IOP-lowering medications were only mildly effective. Addition of cycloplegia resulted in chamber deepening from 2.2 mm to 2.5 mm and IOP decrease to 19 mmHg. UBM and US did not reveal any significant pathology other than Elsching pearls in the capsular bag. Attempt at discontinuing cycloplegia resulted in immediate reversal of treatment effect and so it was continued. After a week IOP elevated again, and with the diagnosis of aqueous misdirection the patient was treated with pars-plana vitrectomy, hyaloidectomy and zonulectomy. During a follow-up of 4 months untreated IOP has been 10 mmHg, the chamber deep and the angle open. This case represents a most unique example of "malignant glaucoma", not after recent surgery. The differential diagnosis according to the Ritch mechanistic classification of angle-closure is nicely demonstrated, with the necessary conclusion that indeed a posterior-segment source was responsible for the angle closure and chamber shallowing. The diagnosis had to be firmly established since the consequent treatment was unusual surgery in an only seeing eye. I am aware of only one report in the literature of a similar case.
EVALUATION OF OPTIC NERVE HEAD CUPPING IN THE PRESENCE OF
MYELINATED NERVE FIBRES-A REAL CHALLENGE?
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68 year old female, a nurse by occupation presented for a glaucoma workup. She had an
iridencleisis surgery in her left eye way back in 1972 when she had developed corneal edema and
persistently high intraocular pressure. On examination the best corrected visual acuity was 6/6 in
the right eye and 6/60 in the left eye. The intraocular pressure by applanation tonometry was 15 mm
Hg and 19 mmHg in the right and left eyes respectively. The anterior segment of the right eye was
normal. The central corneal thickness was 530 microns. A +90 D examination revealed a cupping of
0.5 in the right eye. The rims were healthy nasally and temporally. Due to the presence of
myelinated nerve fibres at the optic disc, the health of the superior and inferior neuroretinal rims
could not be commented upon. (Figure 1). Gonioscopy showed open angles with no peripheral
anterior synechiae. The Humphrey Central 24-2 SITA standard visual field analysis was normal.
(Figure 2). Optical coherence Tomography showed increased retinal nerve fibre thickness in all
quadrants. (Figure 3). The patient was using Timolol 0.5% in the left eye. The cornea had
epitheliopathy with features of dry eye. The central corneal thickness was 602 microns. There was a
flat bleb which had pigmentation which probably incorporated the pillars of iris (iridencleisis).
(Figure 4). The chamber was normal with a cataractous lens. In addition, the inferior iris revealed
multiple pigmented spots suggestive of iris naevus syndrome. The view of the left fundus was hazy
and a cupping of 0.9 with very thin neuroretinal rims was noted. Due to the hazy cornea, details of
gonioscopy could not be made out. Similar visual field analysis of the left eye showed superior
arcuate defects. (Figure 5). Retinal nerve fibre analysis by Optical coherence Tomography could not
be done due to the corneal changes.
SUPERIOR OPHTHALMIC VEIN THROMBOSIS MASQUERADING AS ACUTE ANGLE CLOSURE ATTACK
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A 45 year old post menopausal lady of South Asian descent presented with congested left eye of 6 months duration along with diminished vision in the same eye for past 4 months. Her previous treatment records revealed a baseline IOP of 42 mmHg OS and 16 mmHg OD and that she had been managed as a case of acute angle closure glaucoma and was prescribed Brimonidine and Timolol in the OS and had undergone a YAG laser iridotomy. Due to non remittance of her symptoms the treating ophthalmologist then started her on oral steroids assuming it to be a thyroid ophthalmopathy with which she was non compliant. The patient was obese and hypertensive controlled on medications. She did not give a history of heat intolerance, weight loss or mood disturbances. There was no past history of trauma.

Examination: Her best corrected vision at presentation was 6/6 in OD and 6/24 in OS. Left eye had relative afferent pupillary defect and a proptosis of 3 mm with limitation of movement in end gazes. The right eye was apparently normal. Left eye had dilated episcleral vessels in a corkscrew configuration (Fig. 1a & 1b). Fundus examination showed dilated and tortuous veins in the left eye but there were no hemorrhages or choroidal folds. The cup: disc ratio was 0.3: 1 in the right eye and 0.5 in the LE with mild temporal pallor. The vessels in the LE showed tortuosity but there was no disc swelling in the left eye (Fig. 2). Gonioscopy showed occludable angles. The exophthalmos was non-pulsatile and no bruit could be heard in the forehead or periorbital region.

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Investigations: Her Thyroid function tests (T3, T4 and TSH values) were normal and an MRI showed bulky extraocular muscles with relative sparing of the tendons. The superior opthalmic vein on the left side showed subtle dilatation. A provisional diagnosis of a carotico cavernous fistula was made. An magnetic resonance angiography was then performed to look for any abnormal arteriovenous communication or fistula. However, no such anomalous arteriovenous communication was picked up on either of these imaging modalities. In the absence of a carotid cavernous fistula we considered treating her on lines of a Thyroid ophthalmopathy and patient was advised oral Prednisolone of 1 mg/kg dose and anti glaucoma medication. The patient was discharged.

On follow-up: 4 weeks later her vision in the left eye was reduced to 6/60. The proptosis had increased to 4 mm. There was severe conjunctival chemosis, an increase in congestion and severe limitation of eye movements (Fig. 3). The patient had developed a Cushingoid facies. Her IOP on maximal topical medication and Acetazolamide 500mg T.DS was 50mmHg. The patient was admitted and over the next 4 days her vision dropped to hand movement close to face. A repeat MRI was performed, but no cavernous sinus lesion was detected (Fig. 4). The patient then underwent an urgent digital subtraction angiography (DSA) that showed a small left side indirect low flow cavernous fistula that was partially thrombosed arising from the internal carotid artery along with mildly dilated superior ophthalmic vein with anterograde flow and a retrograde flow into the dural plexus (Fig. 5).
Figure 1

FIG 1a

FIG 1b

Figure 2
A 72-year-old female was referred to the phaco service to determine the etiology of episodes of blurry vision in left eye. She has recently been diagnosed by few ophthalmologists for recurrent hyphemas in her left eye but they did not detect an etiology for her condition.

**Her past ocular history:** Pseudophakic (ECCE and PMMA PCIOLs implantation) in both eyes 18 years ago (1994); Posterior capsulotomy by Laser Yag in left eye 17 years ago (1995); Trabeculectomy in left eye because of high IOP in left eye 14 years ago (1997). Vitreous hemorrhage diagnosed and treated with medications by an ophthalmologist in February, 2009.

**Her past medical history and family history:** unremarkable.

**Office Examination:** when she presented to phaco service.

Her BSCVA was 6/6 OD and 6/18 OS. Her IOP with applanation tonometry measured 18 mmHg OD and 28 mmHg OS. Pupil diameter was 1.5 mm OD and 3.5 mm after dilated (Fig. SLE OD1, OD2). Pupil diameter was 2.5 mm OS and fixed (Fig. SLE OS1). We noted PCIOLs in sulcus both eyes and an open posterior capsule OS. We did not see the bleb of previous trabeculectomy OS. (Fig.SLE OS 6). In her left eye, hyphema was present with 2 + cell in the anterior chamber and vitreous (Fig. SLE OS2). With transillumination, we also noted the iris defect at 2.30 o’clock with an IOLs haptic nearly exposed (Fig. SLE OS 4, 5). At the site of iridectomy at 12 o’clock in her left eye, we saw a part of IOLs optic (Fig. SLE OS3). We also observed greater cupping of the optic nerve in her left versus her right eye (Fig. FO). We did not note any blood in the vitreous. On gonioscopy, the total closed angle 360° with PAS in her left eye (Fig. Gonioscopy). On UBM, we suspected the IOLs haptic pressing between ciliary body and iris in her left eye (Video clip of UBM).

**Diagnosis:** Uveitis, glaucoma, hyphema (UGH) syndrome in the patient’s left eye.

**How would we proceed?**

1/ **How to manage the closed-angle glaucoma on the patient?** Perform surgery? If so, what type?
2/ **With her left vision 6/18, could we remove and replace the PCIOLs in the patient’s left eye?** If so, what type of IOLs we should use?
Nasal

OD

OD1
Superior

Temporal
A 5 year-old girl suffered from acute IOP elevation in the right eye following a pupil dilatation for routine retinal examination. She was a premature baby with a gestation age of 27 weeks and a body weight of 980 grams. She developed ROP, stage IV, and received scleral buckle in both eyes during infancy. Visual acuity (VA) was 20/200 in the right eye and light sense positive in the left eye. After the acute attack occurred, she had the typical ocular presentations of acute angle closure glaucoma, including edematous cornea, shallow anterior chamber, fixed-dilated pupil, and glaucomatous flecks of the lens, etc. (Fig. 1) Ultrasound biomicroscopy (UBM) showed that the angle in the right eye was closed 360 degrees circumferentially. The IOP remained over 50 mmHg although oral diamox and maximal tolerated topical antiglaucoma medications were treated. When the IOP control can not be achieved by the initial medical treatment following an acute attack of pupillary block, we need to advocate a surgical procedure to lower the IOP. A traditional procedure, including (a) laser iridotomy and (b) surgical peripheral iridectomy under sedation, could be an option. However, sometimes pupillary block can not be easily broken with this traditional procedure. If the peripheral anterior synechiae remains and the IOP remains high, additional surgical procedure is still needed. At this point, since visual prognosis was greatly demanded for this one-eye case, we had to assess all possible surgical options, including filtering procedure, glaucoma drainage, and diode cilioablation. As the patient was encircled with a scleral buckle, we also had to consider all possible technical obstacles which might be encountered during the surgical procedure. Weighing the advantages and disadvantages for each surgical option, a filtering surgery of trabeculectomy with mitomycin C was finally decided upon. With the advances in neonatology, the survival rate of premature infants with lower birth weight and lower gestation age has increased and the incidence of ROP is thereafter on the rise. Acute attack of angle closure glaucoma is likely to occur following the pupil dilatation in patients of ROP. Management of acute angle closure glaucoma in pediatric ROP is not as easy as management in adult patients.

Photo following acute attack: edematous cornea, shallow anterior chamber, fixed-dilated pupil, and glaucomatous flecks of the lens.
ABSTRACT WITHDRAWN
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CLINICAL CASE PRESENTATION

Introduction: Pigmentary glaucoma (PG) is a well-recognized form of secondary glaucoma caused in part by iris pigment liberation resulting in outflow obstruction. Primary PG is commonly treated with topical medications or laser trabeculoplasty. Secondary PG is becoming increasingly common in developing nations with the growing utilization of IOLs. There is minimal awareness of the relationship between IOL placement and secondary PG in the less developed world.

Summary: We report on both the development of late onset secondary PG from poor IOL placement and its surgical management.

History: A 63 year old male patient presented with history of eye strain and blurred vision in his right eye. He had manual small incision cataract surgery with IOL implantation in both eyes, over 2 years ago. There was no prior history of glaucoma.

Examination: His Best corrected visual acuity was 6/9 in RE with -3.5 DS and -3.0 D Cylinder at 180° and 6/6 with -1.75 D Cylinder at 90° in LE. The right eye’s anterior segment examination demonstrated a temporal scleral tunnel, localized shallow anterior chamber superiorly for 2 clock hours, and superior anterior iris displacement. After dilatation we found an anteriorly displaced superior haptic and decentered single piece PMMA IOL. The superotemporal haptic was in the sulcus and the inferior haptic in the bag. The left eye’s PCIOL was in the bag. IOPs were 36 mm Hg RE and 18 mm Hg LE. Gonioscopy revealed extremely heavy trabecular pigmentation for 360° with 2 hours of localized peripheral anterior synechiae superiorly in RE and minimal pigmentation with a pseudophakic depth chamber angle in the left. Fundus examination of the RE found a 0.7 cup with an inferior notch and NFL thinning and a 0.5 centrally placed healthy disk cup LE. Visual fields with Humphrey’s field analyser (HFA 24-2) showed early superior arcuate scotoma in RE. AS-OCT showed localized elevation of iris superiorly in RE.

Management: We made a diagnosis of secondary PG due to IOL positioning. We performed an IOL exchange to prevent further pigment liberation, decrease the amount of synechial closure, and reduce the myopic shift. Two months later, his BCVA was 6/6 RE with -1.0 D Cyl at 140° and his IOP was 15 mm Hg without therapy. Postoperative AS-OCT showed flattening of iris elevation.
BILATERAL AND SIMULTANEOUS ANGLE CLOSURE GLAUCOMA FOLLOWING ANAESTHESIA
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62 year old female presented with complaints of defective vision both eyes for 10 days. She gave history of undergoing hysterectomy 10 days earlier under spinal anesthesia elsewhere. She was treated with the post op room with antiglaucoma drops & topical steroids. On examination her visual acuity were 4/60 in right eye & 2/60 in left eye. Ocular examination showed corneal oedema & dilated pupils in both eyes. Intra ocular pressures recorded were 28 mmHG in right eye & 30 mm Hg in left eye. Laser iridotomies were tried in both eyes, but was not successful. Trabeculectomy was done in both eyes, in a gap of 4 days. Intra ocular pressures were 10 mmHg in Right eye & 8 mm Hg in left eye after filtering surgeries. Fundus examination were normal in both eyes. She later developed cataract following filtering surgeries in both eyes, for which Phacoemulsification with Intraocular lens implantation was done.
Mrs. M.H., a 60 year-old Caucasian, was referred for advanced chronic angle closure glaucoma of the RE diagnosed a year previously. Two trabeculectomies had already been done. The LE had undergone no laser or incisional surgery. Her visual acuity was RE: +3.00 (-1.25 × 17°) 2/10 Add +3.00 P 10 and the LE +2.75 (-1.00 × 70°) 8/10 Add +3.00 P2. IOP was RE 18 mmHg on no medical treatment and LE 17 mmHg on a prostaglandin analog QHS and dorzolamide/timolol combination BID. CCT was RE 605 μ LE 592 μ. The RE had a relative afferent pupillary defect, a patent surgical iridectomy, and an intact filtering bleb. Gonioscopy of the RE showed PAS for over 270° completely covering the trabecular meshwork. Indentation gonioscopy of the LE showed significant relative pupillary block in all quadrants with the nasal angle completely closed by apposition. The nasal trabecular meshwork was lightly pigmented. The temporal angle, however, was open to the ciliary body band with no indentation necessary to easily observe the angle. There was heavily pigmented tissue resembling dense iris processes or uveal meshwork covering the angle structures. Question to the audience: This pigmented tissue is: normal iris processes? PAS inserted more posteriorly? On ocular fundus examination the RE had advanced glaucomatous optic atrophy, and the LE showed no cupping at all. The visual field RE had a centro-temporal island: the LE was entirely normal. Question to the audience: In light of the unusual gonioscopy findings LE, what is the next examination to be performed? Anterior segment OCT? UBM? Something else? The UBM LE showed a closed nasal angle both in the dark and with illumination of the pupil. The temporal angle remained open at all times. The ciliary body was thicker on the temporal side with a ciliary body lesion measuring 6.35 x 1.05 mm. In summary, this patient has a pigmented ciliary body lesion (nevus versus melanoma) keeping the angle open by centripetal displacement of the iris root. Because of the small size and relative thinness of the lesion, the presumptive diagnosis is a nevus. The LE underwent a small laser PI nasally, and is also being followed at an ocular oncology center. (A case report is being prepared for publication). Iconography: Videogonioscopy of temporal and nasal angle LE. Video UBMs (in dark and light) of temporal and nasal angle LE. 24-2 visual fields.
A 16 year old boy presented in May 2010, with a history of pain, redness & diminished vision in the Left eye since the past few months. H/o injury to the LE with a fire cracker at the age of 1 year. H/o Cataract surgery done with a posterior chamber intra ocular implant done at the age of 13 years. A posterior capsular opacification developed & a Yag Capsulotomy was done 2 years later. Glaucoma was diagnosed in April 2008 & a trabeculectomy was done in Oct 2008. O/E : RE was WNL BCVA; 6/9 & N6. IOP was 20 mm Hg Fundus was normal so was the Perimetry. LE showed BCVA to be 6/18, N36, IOP was was 36 mm Hg. There was a flat bleb superiorly with a pseudophakia & an open PC. There were pigments on the anterior surface of the IOLI. Pupil was RTL. Fundus showed a normal sized disc , with a c/d of 0.85 & thin NRR all round. The perimetry showed a generalised depression with a paracentral defect. A diagnosis of failed trabeculectomy was made & a RETRAB was done in MAY 2010. The immediate post op period was uneventful, with vision improving to6/12 , N10. & IOP's at 14 mmHg. At 3 months the IOP started rising, & the bleb seemed to be failing, with episcleral fibrosis setting in. S/C Inj 5 FU was given in addition to topical Beta blockers.4 such inj were given on alternate days. 10 days later a nice diffuse superior bleb was seen with an IOP of 5 mm Hg & Vision at 6/12, N10. Routine post-op tapering steroids were continued. 6 weeks later the bleb was getting encysted with an IOP of 42 mmHg. A bleb needling was done, the bleb was formed & IOP came down to 4 mmHg. Patient's vision started dropping, BCVA 6/60 as patient developed choroidals with a maculopathy. Steroids were stepped up, Choroids are settling, but no change in vision. IOP's rising. With every intervention the IOP's lower, how long to keep the steroids on? What is the cause of episcleral fibrosis setting in so quickly < 1 month How do you proceed?
A 30 year old Male patient was referred to us. He had been a k/c/o glaucoma since 13 years of age, with poor vision in the Left eye. Was using G. Timolol 0.5% bd and G. Latonoprost HS. in OU. Poor compliance O/E:OD showed a BCVA 6/9, IOP : 14 mm Hg N6 Quiet AC, Pupils reacting well to light & a fundus showing a normal sized disc with a c/d of 0.9 with a thin & pale NRR. OS: showed a BCVA: PL PR inaccurate, IOP of 36 mm Hg, Mid dilated fixed pupil, ciliary staphyloma seen superiorly & inferiorly & a fundus shows a glaucomatous optic neuropathy with a total pale cup. Perimetry: OD showed superior para- central + superior arcuate defect, OD macular program also showed superior arcuate defect. OS Perimetry could not be done due to poor vision. As patient was non compliant with meds on follow up a trabeculectomy was suggested. In the interim period patient was lost to follow up & he came to us 6 weeks later with an IOP of 32 mmHg in the Right Eye. Trabeculectomy with MMC was done. The immediate post up was uneventful, with IOP in OD staying at 6 mm Hg with a diffuse bleb. Later, the RE was doing fine, the non seeing eye was a problem. What next? He is on 2 drugs. Will you add more medications? Cyclodestructive procedures? Trabeculectomy? Advised: LE Ahmed Valve as he had a large ciliary staphyloma in the LE & was known to be non compliant. OS Ahmed valve insertion done post-op VA OS PL+ PR inaccurate. IOP : 03 mmHg. OS Conjunctival bleb +AC Slightly shallow, Hyphaema + Pupil 4.0 mm dilated. Fundus: C:D::0.95. Bipolar notching. Early Post op was uneventful 2 months later: IOP OD 05 mmHg OS 28 mmHg OS – tube not seen in the Anterior chamber. Impression – tube withdrawn from the AC and blocked. How to proceed? We decided to wait & watch, & started Alpha agonist e d in the LE. In subsequent follow-up the patient developed a conjunctival hole over the implant with Siedel’s positivity. He was advised conjunctival suturing which he deferred initially. Conjunctival suturing was eventually done. Later, on a routine follow-up, the patient was found to have Extrusion of the Ahmed valve plate in the left eye. IOP (GAT) OD 04 mmHg OS 02 mmHg. The valve had extruded out completely with shallow-flat anterior chamber. NOW WHAT? Pressure bandage was applied and removed after 4-5 hours after which the anterior chamber was relatively well formed OS removal of the Ahmed valve and conjunctival suturing was done. Post-op recovery was uneventful with normal to low IOP and Siedel’s negative. When he last presented to us VA with PG OD 6/12p; OS PL+ IOP: 7 & 24 mmHg, OD . Trabeculectomy bleb+, OS - Ciliary+Intercalary staphyloma with temporal conjunctival scarring. Fundus OD C:D::0.95 with no inferior rim, OS Glaucomatous optic atrophy. Patient was asked to continue G. Combination Therapy OS and follow-up in 2 months. All pictures are well documented. Do we need to send all the pictures right away?
I have a small eye & a hole in my eye & I can see less

- 11 yrs old male patient presented with C/O decreased vision with present pair of glasses
- No H/O trauma
- S/A: (RE) -2.50 DS /-2.0DC x 60 6/24,N8
  (LE) +10.0DS/+1.0DC x 35 FC 1 ½ metres,N36

- IOP: (RE) 32 mmHg, (LE) 33 mmHg
- On Examination : Hypertelorism + built Small for age.

(LE)-Microcornea .Corneal diameter Vertical & horizontal : 9 mm.
AC-irregular, Gonioscopy showed closed angles, Atypical iris coloboma
- Anterior subcapsular cataractous lens
- FUNDUS : normal sized disc .CDR 0.5,HNRR, no chorioretinal coloboma seen
( LE) : Cornea-normal , vertical & horizontal diameter : 12mm , Iris Typical iris coloboma,
Gonioscopy-pigmented TM visualised temporally & inferiorly ,
Lens-Aphakia(absorbed lens)
Fundus- normal sized disc, CDR 0.5,HNRR, no choroidal coloboma seen

Investigations :
- Perimetry done in the RE shows normal VF & perimetry cannot be done in the LE due to poor VA.
- OCT done in BE shows RNFL to be WNL.

How do you proceed?
Systemic examination does not yield anything positive.
Dentition normal, hearing normal ,no skeletal deformities, no cardiac involvement, no umbilical abnormality, no renal problem.
The poor vision in the LE is probably due to Amblyopia.
Poor vision in the RE ??? only cataract?? Cause of cataract??
IOP??? Treat medically ???
- Started medical treatment for the IOP, responded patially, IOP’s came down to 24 mm hg with combination therapy, but patient is very uncompliant.
- Cataract in the RE , to be operated??
- 50% chances of this child developing Glaucoma in the future??
Laser ruled out as the angle is closed.
Impression?? Iridogoniodygenesis?? Heredity?? Genetic work up ??
A seven year old boy presented to us with gradual progressive deterioration of vision for last two years. On examination his best corrected visual acuity was finger counting at 1/2 meter in right eye and finger counting at 1 m in left eye from aphakic area. He was on therapy with dorzolamide and timolol 0.5% in both eyes. Applanation intraocular pressure was 44 mm of Hg in RE and 42 mm of Hg in LE. His horizontal corneal diameters were 11.5 mm right eye (RE) and 11.5 mm left eye (LE), with pachymetry of 650 µm in both eyes. His lens was cataractous and superiorly subluxated in both eyes. Fundus evaluation revealed a vertical cup to disc diameter ratio was 0.9:1 RE and 0.8:1 LE, with foveal hypoplasia in both eyes. He was diagnosed as both eyes Aniridia with subluxated lens with glaucoma. The case will be put to the panel for surgical options. We performed both eye pars plana lensectomy with AC maintainer with implantation of Ahmed glaucoma valve with pars plana clip.
SWAN SYNDROME - POST TRABECULECTOMY
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Background: Focal vascularization from an ingrowth of episcleral vessels at the cataract wound site, resulting in recurrent intraocular bleeding, has been termed Swan syndrome\(^1\). It has been reported following intracapsular cataract extraction, extracapsular cataract extraction (including clear corneal incisions), iridocyclectomy, and glaucoma filtering procedures.\(^1,2,3\)

Case Report: A 32 yr old one eyed Asian Indian male, with history of high myopia, presented with uncontrolled Primary open angle Glaucoma, Left eye. (Feb 2007).

Past history: His Right eye had been enucleated in his late teens after phthisis bulbi following unsuccessful retinal detachment surgery. He also had a past history of prophylactic cryo to retinal break in the Left eye in 2000. He also had a uneventful Phacoemulsification with IOL (hydrophobic acrylic) implantation OS for a posterior subcapsular cataract in 2006. He was a known steroid responder.

Systemic history: He had no systemic illnesses.

Meds: Tab Acetazolamide 250 mg BD, Timolol 0.5% bd, Latanoprost 0.005% od, Brimonidine 0.15% bd

On examination- OS (Feb 2007)--
- BCVA- 6/9, n6.
- A/S- Pseudophakic, One posterior synechia to ant capsule
- Pupil irregular , briskly reactive
- Gonio – Open Angle, IOP- 38 mmhg (with MMT)
- Disc – Myopic-0.4 c:d, peripheral retinal laser scars, lattice degeneration.
- Visual fields- Generalised depression of retinal sensitivity

- March 2007. He underwent uneventful Trabeculectomy with Mitomycin C with Releasable sutures
- Post trab IOPs were well controlled 10-12 mmhg, without medications, sutures cut at 2 months after surgery.
- Aug, Sep 07
  - Two episodes of mild Anterior Uveitis which responded to topical steroids.
  - Uveitis Workup- Normal.
  - DD- intermittent low grade inflammation-
    - Anterior non granulomatous uveitis of unknown etiology.
    - ? Mild low grade Endophthalmitis s/p Trab.
    - AC tap- positive for P Acnes Genome
  - One episode of Ant uveitis in Feb and May 2008, resolved with Dexamethasone e/d.
  - Repeat episodes in March 2009, and June 2009
- August 2009
  Patient presented with c/o blurred vision OS since morning .No h/o antecedent trauma.
  O/E-A/S- Small Hyphema, which resolved uneventfully in a few days,
  BCVA, A/S, disc, retina and IOP were maintained

Gonioscopy: Showed a pinkish blush on the internal ostium ? neovascularisation and the vessels were seen to actively bleed with pressure from the Goniolens. Rest of the angle was open with moderate pigmentation. A diagnosis of SWAN syndrome was made. Vascularisation of the internal ostium was probably causing repeated small hyphemas, which would probably set up a reactionary inflammation, which were the episodes of repeated mild ant uveitis seen all along. Management: Diode laser ablation , using High magnification Gonio lens, slit lamp delivery.- Sep 2009. However we noted one recurrance of microhyphema episode in Dec 2009, and one more recurrence in May 2010. The new vessels were seen to have recurred on the internal ostium. The patient was given Intravitreal Bevacizumab 2.5 mg in 0.1 ml, with Sub conj injection at Trab site. The patient has had no recurrances till date(Feb 2011), BCVA, IOP, Retinal status stable.
SWAN syndrome is rare, but needs to be kept in mind as a DD for recurrent inflammation with hyphema after anterior segment surgeries, with a scleral approach. Anti VEGF drugs can be effectively used in the management of this condition.

Fig 1: Anterior segment OS

Fig 2: A/S photo with pupil dilated OS

Fig 3: Gonio photo showing internal ostium with NV

Fig 4: Active bleeding into AC during Gonioscopy
Fig 5: Recurrence of NV on ostium after laser

Fig 6: Resolution of the new vessels after Bevacizumab
CASE PRESENTATION
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Case Description: A 31 years old female with history of rheumatic heart disease and Mitral valve replacement with a mechanical prosthetic valve on anticoagulants for life. History of wearing contact lenses, repeated use of steroid drops, increased IOP that decreased by the use of anti-glaucoma medications. Best corrected visual acuity 6/18 in both eyes, IOP 16 mmHg bilaterally on medications, normal anterior segment and bilateral pale optic disc with bilateral contracted fields. Electrophysiological testing showed rod/con dystrophy consistent with the diagnosis of Retinitis Pigmentosa. The patient is poorly compliant to medical treatment and follow-up visits.

**Diagnosis:** Steroid induced glaucoma in a case of retinitis pigmentosa on anticoagulant therapy for Mitral valve replacement

**Management Problems:**
1. What prognosis is expected in this case?
2. If Medical treatment only were decided, what would be the safest drugs to be used?
3. If surgery is decided which type is recommended to be safest for the retinal condition?
4. If surgery is recommended should anti-coagulant therapy “warfarine” be stopped and bridged with Heparin in the peri-operative period?
5. If surgery is recommended should the patient receive prophylactic antibiotics for prevention of bacterial endocarditis?
Background: To report our experience in managing a challenging case of trauma related glaucoma whereby multiple surgeries performed resulted in successful IOP control.

Method: Interventional case report.

Results: A 25-year old Malay man alleged a fire cracker injury to his right eye one month prior to presentation was referred for uncontrolled intraocular pressure (IOP). He sustained right corneal abrasion, traumatic mydriasis, subluxated lens and secondary glaucoma. Right intracapsular cataract extraction (ICCE) and anterior vitrectomy were performed by referring ophthalmologist. Ocular examination revealed right aphakia with best corrected vision of 6/30 and IOP of 39 mmHg. There was presence of minimal vitreous strands in the anterior chamber at 9 to 10 o’clock position and cup to disc ratio of 0.7. He underwent Right Baerveldt implantation and the silicone tube was placed at 12 o’clock position. At Day 1 postoperative day, vitreous was noted to be attached to the tip of the silicone tube and the IOP was 14 mmHg. Unfortunately within few days the IOP started to climb up again, ranging from 22 to 63 mmHg. Antiglaucoma medications were stepped up and at Day 9 post Barveldt implantation, right Trans Pars plana vitrectomy was performed to clear the vitreous. Initially the IOP was low but elevated again to 55 mmHg at Day 4 post vitrectomy. At Day 21 post Baerveldt implantation, the silicone tube stent was removed and subsequently the IOP normalized without antiglaucoma treatment. No hypotony noted. Three months postoperatively the best corrected visual acuity was 6/18.

Conclusion: Multiple surgeries in trauma cases are inevitable. Baerveldt implantation in aphakic eyes can be complicated with presence of vitreous in anterior chamber. Adequate vitrectomy is warranted to prevent vitreous incarceration in glaucoma implant tubes.
DIFFERENT STROKES FOR DIFFERENT FOLKS
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Case: A 38 year old lady with hypermetropia, in the first trimester of pregnancy presented with a painful, red left eye. Acute primary angle closure (PAC) was diagnosed in the left eye. No response was obtained with supine posture, firm pressure with goniolens and pilocarpine drops following punctal occlusion. Diffuse corneal edema precluded the use of argon laser iridoplasty. She underwent controlled anterior chamber paracentesis (ACP) on the slit lamp with aseptic precautions. The IOP reduced from 48mm Hg to 16mmHg and YAG iridotomy was possible within 3 hours with clearing of corneal edema. The patient remains under review following an otherwise uneventful pregnancy with a healthy baby born at full term.

Discussion: There is limited experience in management of glaucoma in pregnancy with lack of safety data on glaucoma medications especially systemic therapy. PAC has been described during pregnancy in only two patients, both during labour, induced by the associated sympathetic overdrive and a possible effect of labour inducing drug (Ritodrine, a selective β2 adrenergic agonist). To our knowledge, this is the first reported case of PAC in early pregnancy. Evidence based systematic approach to PAC is discussed especially in a young patient.

Message/Lesson: The case is an example of good practice that led to risk of glaucoma medication to the fetus being eliminated with a rapid resolution of the PAC episode. It reminds us of the role of alternative management strategies for PAC like controlled ACP in selected cases where systemic and/ topical therapies are unsuitable or inadequate.
IRIDOCORNEAL ENDOTHELIAL SYNDROM (ICE)
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A 41-year-old woman was presented in October 2008 to our clinic with elevated IOL. The patient’s past medical history included disseminated sclerosis with intermittent immunosuppressive corticosteroid therapy from 1999. She was examined by the ophthalmologists for repeated headache. On examination, the patient’s BCVA was 6/24 OP and 6/6 OS. Her IOP measured 28mmHg OD and 15mmHg OS with Goldmann applanation tonometry. Central corneal thickness was 545 um OD and 550 um OS. The slit-lamp examination of the right eye revealed abnormalities of the anterior chambre angle, and iris. Gonioscopy showed peripheral anterior synechiae which extend beyond Schwalbe’s line and small degrees of corectopia directed toward the quadrant with the most prominent area of peripheral anterior synchia. The right optic nerve had advanced glaucomatous damage with a loss of the rim and pallor. The left eye examination was normal. The patient’s examination suggested a diagnosis of Iridocorneal Endothelial Syndrom (ICE) with glaucoma and the patient initially was treated by combined therapy ICA and betablockers - Cosopt. On July 2009 - 8months later, her IOP elevated on 47 mmHg and the trabeculetomy (TE) was performed, 2months after surgery the needling was performed for the scarring of the bleb. We continued local treatment with Cosopt. On January 2010 the IOP increased on 30mmHg and the surgery was neccessary to repeat, TE with Mitomycin C was done. Despite undergoing surgeries on the right eye the long –term prognosis of IOP was poor. On July 2010 Ahmed glaucoma valve was done. We did not observe any complications during the procedure but the late failure after the shunt re-operation occur. What would be the next step?
CLINICAL CASE PRESENTATION
A. Manassakorn
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A 41-year-old woman presented with a history of left eye pain and blurred vision for 2 days. Present illness: Three years ago, she was diagnosed with cataract, primary open angle glaucoma and mild non-proliferative diabetic retinopathy in both eyes. Her visual acuity was 20/200 and Fc 3 ft. She was currently treated with 0.5% timolol to BE bid and switch to brimonidine/timolol fixed combination.

Six months ago, after the fasting blood sugar was controlled, she underwent phacoemulsification with IOL, RE. After that, proliferative diabetic retinopathy of the right eye was found. Panretinal photocoagulation of the right eye was performed.

Five months ago, she underwent phacoemulsification with IOL, LE. Two months later, she presented with terrible eye pain, LE with nausea and vomiting. Left eye examination revealed iridocorneal touch, LE with IOP of 55 mmHg. She was treated with anterior chamber paracentesis and 100% Glycerin stat, acetazolamide 1 tab oral q 6 hr, brimonidine/timolol fixed combination, BE bid, brinzolamide, LE bid, 1% Prednisolone acetate, LE q 1 hr, and moxifloxacin, LE qid. After the IOP decreased to 14 mmHg, peripheral iridotomy, LE at 2 o’clock was performed. At that time her visual acuity was 20/70 and 20/100 for right and left eyes with brimonidine/timolol fixed combination, BE bid, and brinzolamide, LE bid.

Two months ago, she presented at the emergency department with left eye pain. Her visual acuity was Fc 1 ft. and IOP of 60 mmHg. The PI was not patent with shallow peripheral anterior chamber and corneal microcystic edema. After IOP was controlled, the second PI was done at 10 o’clock. The IOP decreased to 14 mmHg, LE. She still treated with brimonidine/timolol fixed combination, BE bid, Brinzolamide, LE bid.

2 days ago, she started to have left eye pain again. Visual acuity was Fc 2 ft. with IOP of 70 mmHg. Left eye examinations revealed corneal microcystic edema with ciliary injection, small 1 patent PI and 1 occluded PI with iris bombe and rubeosis iridis around PI.

Note: This history and physical examination will be presented with anterior segment photographs and ultrasound biomicroscope printout.
ABSTRACT WITHDRAWN
CASE REPORT: AN UNUSUAL CAUSE OF POSTOPERATIVE ATHALAMIA
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A 56 years old male with uncontrolled advanced chronic angle closure glaucoma underwent uneventful phacotrabeculectomy with implantation of hydrophobic single piece lens in the lens capsule (Acri.Smart 46LC, Zeiss). At the end of the procedure a small amount of viscoelastic solution (Viscoat®) was left in the anterior chamber in order to prevent postoperative athalamia. A week later, VA was counting fingers and the examination showed: shallow anterior chamber, IOP of 4 mmHg, negative seidel test and no choroidal detachment. Refraction was -3.50 sph -0.50 cil 150. Carefully examination under complete pupillary dilatation showed an optically clear liquid between the posterior surface of the IOL and the posterior capsular membrane, the capsular bag was distended: iris and IOL moved forward while posterior capsular membrane backwards. Almost immediately after posterior capsulotomy with Nd:YAG laser was performed, the athalamia was completely solved.

Discussion: Capsular Bag Distension Syndrome (CBSD) is an uncommon and rarely recognized cause of postoperative athalamia after combined surgery in glaucoma. This syndrome occurs after remaining viscoelastic solution or cortex is trapped between the IOL and the posterior lens capsule. The rhexis diameter must be smaller than the optic diameter of the lens. Remaining viscoelastic or cortex in the lens capsule makes an osmotic gradient across the posterior lens capsule that would cause liquid accumulation in the capsular bag. As shallow anterior chamber and hipotony are very common features after glaucoma surgery, this syndrome is frequently misdiagnosed. In many cases, CBDS have been described even years after the phacoemulsification. Carefully examination is mandatory, especially when it is done without pupil dilatation and remaining viscoelastic or cortex is suspected to be inside the lens capsule. Nd:YAG laser posterior capsulotomy is a simple procedure that solves almost immediately this syndrome. The fluid material inside the lens capsule is released through the posterior capsulotomy to the vitreous cavity while the iris and the lens move back to its correct position.
A 14 old girl child presented to the out patient department of Dr Rajendra Prasad Centre For Ophthalmic Sciences, All India Institute of Medical Sciences, New Delhi with the chief complaints of progressive enlargement of the both eyeballs noticed since birth by her parents. The enlargement of the eyeballs was associated with poor vision, watering and photophobia. The parents did not seek treatment for the child at an earlier age due to lack of finances. Her visual acuity was perception of light in both eyes with accurate projection of rays in all four quadrants. The general physical examination and the systemic examination did not reveal any abnormality. Corneal diameters measured approximately 23 mm x 22 mm OD and 24 mm x 23 mm on horizontal and vertical axis. The cornea revealed stromal haze with haab striae and superficial vascularisation in all quadrants with an enlarged limbus. Gonioscopy was not possible but the angle was visible directly with a torch light examination revealing an open angle and anterior insertion of the iris. The pupils were fixed dilated and not reacting to light. Tonopen reading of both eyes measured 42 mm Hg OD and 46 mmHg OS. The fundus examination on indirect ophthalmoscopy revealed severe chorioretinal degeneration with near total glaucomatous atrophy in both eyes. The axial length was 38.15 mm OD and 38.12 mm OS. The child was started on oral acetazolamide, a fixed dose timolol and brimonidine topical combination and latanoprost. The surgical management of this case is very challenging and we want the experts to discuss the management options for such cases.
CLINICAL CASE
A. Oliveira

A male patient, 35 years old, Brazilian, Caucasian, with glaucoma history in his family, had all kind of glaucoma exams within normal limits, including normal intra-ocular pressure. After 3 months, he came back to the office and had everything changed. His cup/disc ratio was 0.8 on both eyes and a trabeculectomy was performed.
CLINICAL CASE
A. Oliveira

GFO, female, 30 years old, Brazilian, Caucasian, enter the office and told that she was in pain for about one week on both eyes after started a treatment for uveitis. She had intra-ocular hypertension (42 mmHg), conjunctival hyperaemia, corneal edema, anterior chamber cells, mydriasis, visual acuity reduced.

She stopped all medicines in use. The doctor treated her hypertension and she came back in 2 days: without pain, better visual acuity and intra-ocular pressure, her fundus exam without alteration (retina and optical nerve). An iridectomy by laser was done on both eyes but after a few days were not working. After a week another iridectomy was done but still not worked. The patient returned to the office and told the physician that she has pain and blurry vision on the right eye in the afternoon. Another medicine was prescribed.

at the last visit, she was with good intra-ocular pressure and vision but refer dry eye sensation.
A 28-year-old woman, with a history of left corneal perforation from a contact lens-related pseudomonas keratitis, was treated elsewhere with antibiotics and a conjunctival flap. She presented three months after the initial episode, with hand movements vision, a conjunctival flap covering three quarters of the cornea superiorly, stromal oedema inferiorly, new vessels and cataract. The eye was quiet. It was not possible to applanate the cornea, but intraocular pressure (IOP) by digital measurement was substantially elevated. The fundus and optic nerve could not be visualized; ultrasound confirmed an attached retina. There was synechial closure of the superior half of the angle.

The IOP was uncontrolled despite maximal therapy, necessitating an Ahmed valve implant in the superotemporal quadrant. As the patient was phakic, the tube was placed in the anterior chamber (AC). This preceded a penetrating keratoplasty following which her visual acuity (VA) improved to 0.3 – 0.4 and IOP was relatively controlled (23mmHg with 3 agents). Her cataract worsened, VA decreasing to 0.1, and was removed. The IOP again reached unacceptable levels. Numerous bleb needlings were carried out with injections of 5-FU, and the tube patency confirmed through the AC. The elevated IOP persisted until a secondary implant was added, this time in the superonasal quadrant with a ciliary sulcus tube.

Two years later, the IOP is 16mmHg on two medications and VA is 0.8. The patient is followed up every 4 – 6 months with stereoscopic disc photos, visual fields and endothelial cell counts.
TRIPLE SURGERY COMBINING PARS PLANA VITRECTOMY, AHMED VALVE IMPLANTATION AND BOSTON TYPE 1 KERATOPROSTHESIS TO TREAT COMPLICATED CONGENITAL GLAUCOMA

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A 35-year-old male arrived at our institute complaining of intense discomfort and loss of vision in his only, right eye. He had been diagnosed with bilateral congenital glaucoma within a week of birth and had had a left enucleation aged 10 for a painful, blind eye with uncontrolled hypertension, despite multiple surgical procedures. In his remaining eye, goniotomy, trabeculectomy, cataract surgery without IOL implantation and 3 penetrating keratoplasties had been performed elsewhere. Vision 2 years previously had been hand movements according to his medical notes. He was using Combigan and Lumigan drops.

On examination, we recorded light perception without projection. There was bullous epithelial and diffuse stromal corneal oedema, aphakia with cortical and capsular remnants but no evidence of vitreous in the anterior chamber (Visante anterior OCT proved inconclusive due to media opacity). Goldmann tonometry gave a reading of 22mmHg, against a pachymetry of 820 microns. The iridocorneal angle had multiple, diffuse, broad peripheral anterior synechiae, and fundoscopy showed a flat retina, with almost total glaucomatous neuropathy.

A provisional Eckardt keratoprosthesis was performed, combined with a 23 gauge pars plana vitrectomy and Ahmed valve implantation (tube in the posterior segment), and a definitive Boston type 1 keratoprosthesis (video).

6 months after his triple surgery, vision is HM and he tolerates a therapeutic contact lens well. The keratoprosthesis shows no sign of breakdown or endophthalmitis (on long-term steroids, vancomycin and a fluoroquinolone antibiotic), and the tube appears well-positioned and patent. The retina remains flat, and stereoscopic viewing suggests a stable optic nerve. Digitally-measured IOP is between 10 and 20 mmHg, on no topical treatment, and the patient no longer feels any discomfort. Follow-up is 3-monthly.
PARRY ROMBERG SYNDROME: PROGRESSIVE HEMIFACIAL ATROPHY ASSOCIATED WITH SCLERAL MELT: MANAGEMENT OVER 10 YEARS
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We present a unique case & share extremely challenging clinical management issues over 10 years. A 60 year old man presented with irritation of the right eye a sclera melt causing limbal bleb and hypotony. He was diagnosed to have progressive hemifacial atrophy (Parry Romberg Syndrome) for which he underwent facial reconstructive plastic surgery. A detailed clinical investigation was done to rule out other causes of sclera melt. He was observed for 8 years for signs of hypotony maculopathy. He developed reduced vision and hypotony maculopathy after 8 years. We performed bleb repair with donor sclera graft. He developed extremely high intraocular pressure rise postoperatively requiring inpatient treatment with intravenous acetazolamide and mannitol. He had to carry out cyclodiode ablation of ciliary body to control IOP after 72 hours. This resulted in hypotony, cataract and reduced vision.
Parry Romberg syndrome is a rare disorder of unknown etiology. Scleral melt should be recognised as an association of this syndrome. Opinion of international expert will be highly valuable on how would they have approached this case and on specific issue of how to surgically close these sclera melt.
A 56-year-old man was clinically admitted at the glaucoma department complaining of low visual acuity and mild discomfort on the left eye. He was first admitted at the ER 1 year earlier complaining of blurred vision, redness and photophobia on the right eye. He was diagnosed and treated as ocular toxoplasmosis and treated accordingly. Later he developed cataract and vitreous opacities. 3 month prior to his admission he was submitted to phacoemulsification and posterior vitrectomy in his right eye. Best-corrected visual acuity (BCVA) was hand movements on the right eye and 20/20 on the left eye. Anterior segment examination of the right eye revealed: anterior bowing of the peripheral iris, and a complete pupillary block due to synechia. IOP OD: 28 mmHg. He was then submitted to laser iridectomy and topical medication. After the laser, biomicroscopy showed a flatten iris. IOP 22 mmHg. After 15 days IOP was 32 mmHg and he was still complaining. Gonioscopy showed a closed angle 360 degrees. After a long discussion we programmed a goniosynechialysis. The final IOP was 12 mmHg without topical medication.
RECURRENT OF MALIGNANT GLAUCOMA POST VITRECTOMY SURGERY
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Background: Malignant glaucoma continues to present a difficult clinical challenge to the ophthalmologist as regards to pathogenic mechanism, treatment and predictors for resolution.

Methods: A 44 year lady, a known case of angle closure glaucoma presented status post failed repeat trabeculectomy with decreased vision in the left eye. On examination, her IOP was 12 and 32 mm Hg in the right and left eye, respectively with shallow axial and peripheral anterior chamber and patent peripheral iridotomy, absent osteum and pseudophakia in the left eye. A hyaloidotomy was done through the surgical PI site following which she underwent uneventful Ahmed glaucoma valve implantation for uncontrolled IOP. After initial control of IOP for 1 month, she developed recurrent shallowing of the anterior chamber with raised IOP(Figure 1). She underwent Pars plana vitrectomy with inferior surgical peripheral PI after which her IOP still remained high, though the anterior chamber formed well. On examination, a thin inflammatory membrane was visible through the PI which was broken by hyaloidotomy performed through the PI site (Figure 2a and 2b).

Results: The final IOP in the left eye on brimonidine 0.2% and timolol maleate 0.5% remained stable at 18mm Hg with well formed anterior chamber till last follow up of 2.5 years.

Conclusions: While diagnosis and treatment of malignant glaucoma is still controversial, a high index of suspicion is required for chronic angle closure patient with increased IOP and patent peripheral iridotomy. There may be a recurrence of the condition, which needs to be detected and managed appropriately.

Figure 1: Slit lamp photograph of left eye 1 month post AGV
Figure 2a and 2b: Slit lamp photos of left eye post hyaloidotomy with formed central and peripheral anterior chamber.
ABSTRACT WITHDRAWN
Case Description
A 7 years old boy came to our Hospital 10 months ago with a chief complaint of blurred vision, redness and itching in both eyes. The redness and itching was complaining since 3 years ago and he used the topical steroid regularly without opthalmologist control in 1 last year. Since 3 months ago he felt blurred vision in both eyes. There is no history of other allergies.
On eye examination, we revealed the visual acuity was counting finger at 1 meter in both eyes. The eye position was orthophoria with no limitation of the eye movement on both eyes. The intraocular pressures (non contact tonometry) were 45.8 and 42.4 mmHg in the right and left eyes respectively. In slit lamp examination, we found mild papillary reaction in superior tarsal conjunctiva, limbal papillary reaction and bulbar conjunctival injection in both eyes. In cornea, punctate epithelial keratitis was found in both eyes with Shield’s ulcer in the right eye. In posterior segment examination we revealed c/d ratio 0.8-0.9 in both eyes.

The therapy were:
- Fluorometholone ed
- Lodoxamine 0.1% ed
- Artificial tears ed
- Timolol maleat ed
- Acetazolamide eye drops and oral

And the patient underwent filtering surgery (trabeculectomy + MMC) in both eyes.

Two months after surgery the intraocular pressure in the left eye raised and the intraocular pressure in left eye could not control with 2 hypotensive medication until now (6 months).

Present eye examination
Visual acuity: counting finger at one meter in both eyes
Intraocular pressures (non contact tonometry): 8 mmHg in the right eye and 46.7 mmHg in the left eye.

In anterior segment examination, we revealed active vernal keratoconjunctivitis in both eyes and posterior subcapsular cataract in the left eye.

The plan of patient assessment for the left eye:
1. Trabeculectomy + MMC + lens extraction + intraocular lens OR
2. Trabeculectomy + MMC without lens extraction OR
3. Trabeculectomy with glaucoma drainage implant