Shallow A.C. 1 year after trabeculectomy

By

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One year ago, a 27 yrs old female presented to our glaucoma subspecialty clinic by

Left ocular discomfort and diminution of vision

Examination of the patient showed

OD		OS
6/36	**BCVA**	6/60
+10.00/-0.5@30	**Refraction**	+11.5/+0.25@65
RRR	**Pupil**	RRR
24 mmHg	**IOP**	28 mmHg
540 microns	**Pachymetry**	545 microns

Examination of the patient showed

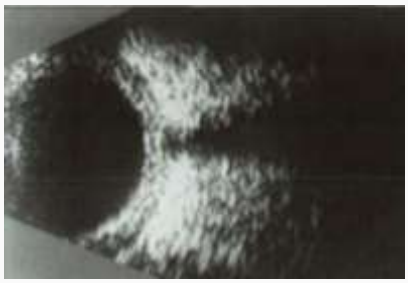
Bilateral shallow anterior chambers

Examination of the patient showed

OD		OS
C/D ratio 0.4 (Vertically elongated cup)	**Fundus**	C/D ratio 0.6 with inferior notch
Grade II in all quadrants	**Gonioscopy**	Closed angle with PAS
Nasal step	**VF**	Superior arcuate scotoma Nasal step
Border line reduction of the average RNFL thickness	**OCT ONH**	Significant reduction of the average RNFL thickness

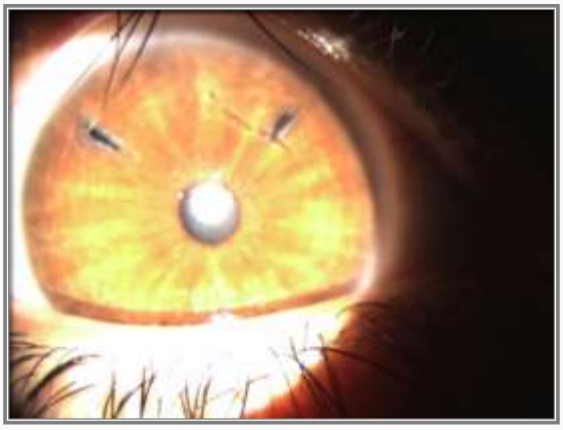
Ultrasound was done to the patient to detect the Axial length

- The AXL in the right eye was 18.5 mm
- The AXL in the left eye was 17 mm



What to do to that patient????

Antiglaucoma medications (PGs analogues+Alpha agonist were prescribed)



Also bilateral laser iridotomies were done

One week later

- The IOP in the right eye was controlled (14 mmHg)

BUT

- In the left eye the IOP was still high (26 mmHg)

Left SST with MMC

Was done

Day one post operative

IOP	8 mmHg
BCVA	6/60
AC	Well Formed A.C.
Fundus	C/D ratio 0.6 (No edema or hge)
Bleb	Diffuse posterior bleb

The patient was followed up

The IOP was controlled in both eyes over a year

After one year

The patient came presented with:

Lost AC mainly centrally

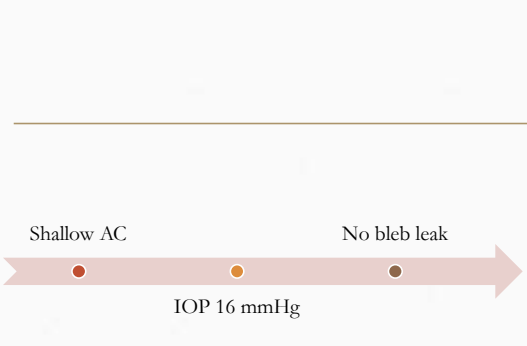
Vascularized pupillary membrane

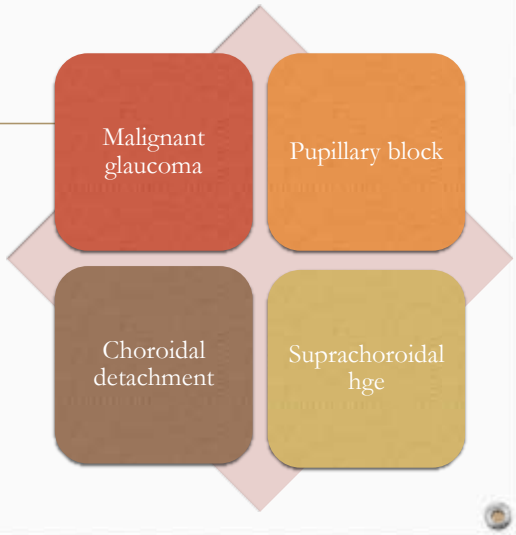
IOP

16 mmHg

The bleb

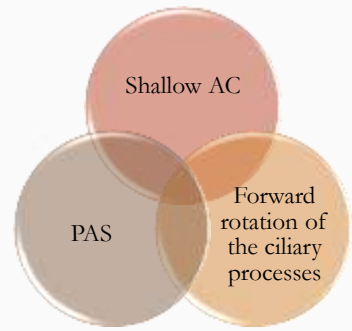
Localized posterior bleb (Negative Siedle test)

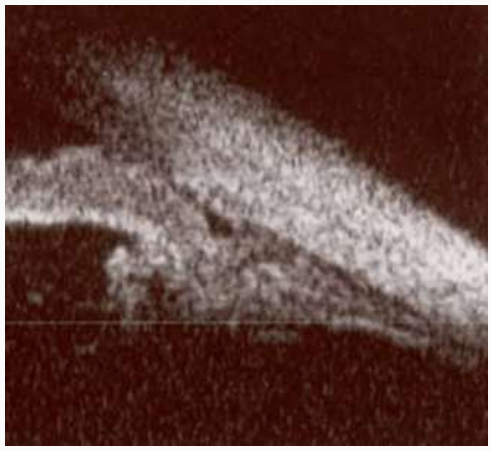




	Malignant Glaucoma	Choroidal Separation	Pupillary Block	Suprachoroidal Hemorrhage
Anterior chamber	Flat or shallow	Flat or shallow	Flat or shallow	Flat or shallow
Intraocular pressure	Normal or elevated	Subnormal	Normal	Normal or elevated
Fundus appearance	Normal; no choroidal Elevation	Large, smooth, light brown choroidal elevations	Normal; no choroidal elevation	Dark brown or dark red choroidal elevations
Patent iridectomy present	Yes	Yes	No	Yes

UBM





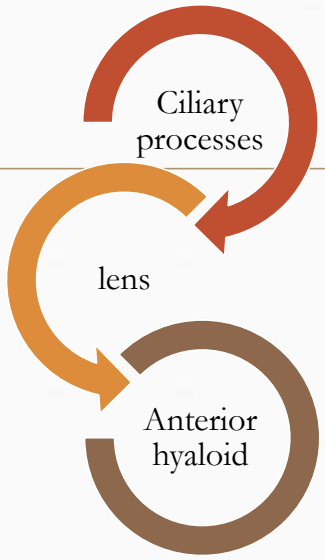
Malignant glaucoma

Malignant glaucoma

- The term was given by von Graefe in 1869 to describe an aggressive form of postoperative glaucoma that was resistant to treatment
- Reported to occur in 0.4–6% cases of incisional surgery for angle-closure glaucoma
- Onset: Variable, ranging from the immediate postoperative period to many years after surgery

Pathogenesis

- Abnormal anatomical relationship between

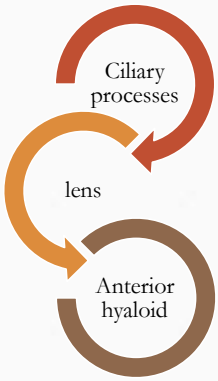


Risk factors

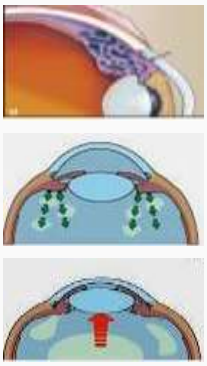
- Axial hyperopia
- Old age
- High IOP
- Post operative hypotony
- Hypertension

Pathogenesis

Several theories are postulated but there is an agreement about



- Anterior rotation of the ciliary body
- Increased contact of the ciliary process with the lens equator
- Posterior flow of the aqueous humor
- Increase in the vitreous pressure
- Forward movement of iris lens diaphragm
- Shallow AC

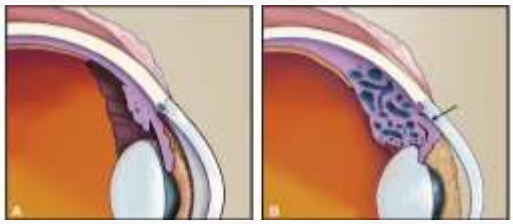


Anterior rotation of the ciliary body

Pathogenesis

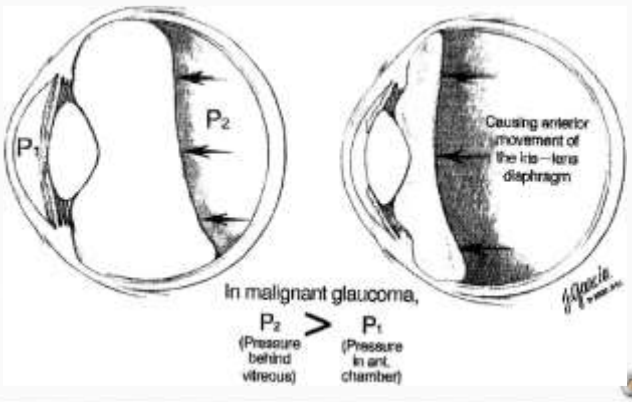
Why there is anterior rotation of the ciliary body?????

- Over-filtration postoperatively leads to shallowing of the anterior chamber and anterior rotation of the ciliary body as a result of posterior pressure
- Spasm of the ciliary muscle, leading to laxity of zonules and increased contact between ciliary processes and equator
- Inflammation and congestion of the ciliary body leading to supraciliary effusion and forward rotation of the ciliary body



Pathogenesis

- A) **Shaffer and Hoskins** suggested that posterior diversion of aqueous flow causes accumulation of aqueous behind a posterior vitreous detachment with secondary forward movement of the iris-lens diaphragm

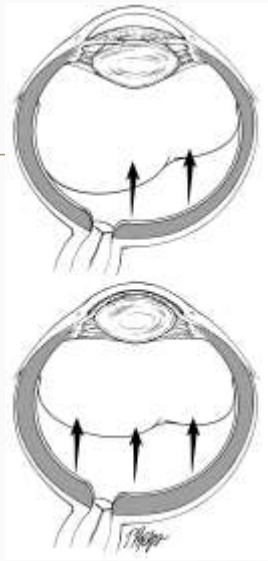


Pathogenesis

B) **Chandler**

Proposed that laxity of lens zonules coupled with pressure from the vitreous leads to forward lens movement.

The higher the pressure in the posterior segment, the more firmly the lens is held forward



Pathogenesis

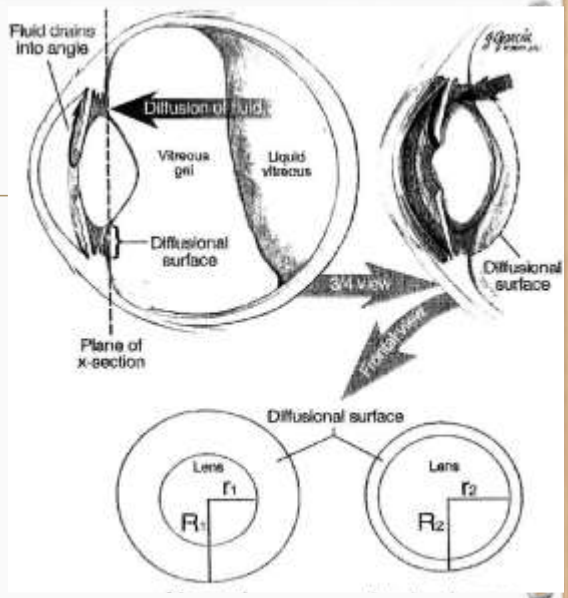
C) **Choroidal expansion :**

Is the precipitating event which increases vitreous pressure

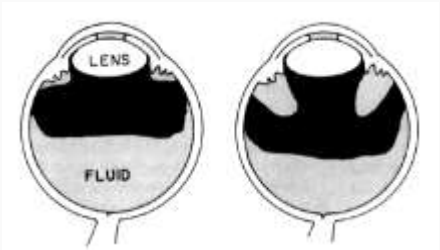
With a 20% increase in choroidal thickness, there is 100 μL in volume displacement—equal to the volume of the anterior chamber in eye with angle closue. If there is no anterior wound, the pressure–volume relationship of the human eye suggests that IOP would rise to 60 mm Hg with a similar expansion.

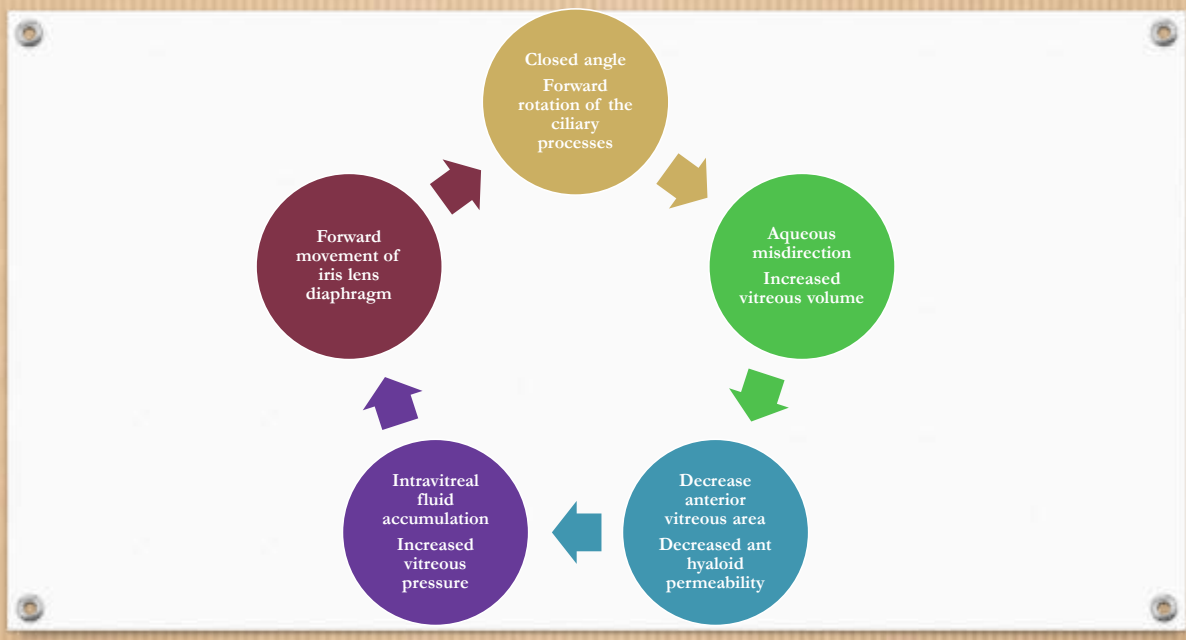
D) Decreased vitreous permeability

- The fluid that must traverse the vitreous body from posterior to anterior can only exit the vitreous gel from an anterior surface that is limited by the vitreous base peripherally and the vitreous–lens contact zone centrally.
- If this doughnut shaped diffusional surface area is smaller (As in shallow AC), the less the permeability of the vitreous and the more the fluid accumulation.



What ever the mechanism, there is aqueous misdirection resulting into trapped fluid posterior to the vitreous and may be inside the vitreous gel as well





How to manage a case of malignant glaucoma?

Medical therapy
(Reported to be curative in 50% of the cases)

Cycloplegics:

- Mydriatics (atropine and phenylephrine) should be given immediately in order to tighten the lens zonules and pull the anteriorly displaced lens backwards

Reduction of IOP:

- Oral acetazolamide
- Topical beta-blockers and alpha agonists are used to reduce aqueous production.

Medical therapy

Decrease vitreous volume

- Osmotic agents (mannitol or glycerol) are used to reduce vitreous volume, deepen the anterior chamber, and possibly increase vitreous permeability

Anti-inflammatory

- Topical steroids can help to reduce inflammation

Laser therapy

Aims to restore a normal aqueous flow pattern by establishing a direct communication between the vitreous cavity and anterior chamber.

Disruption of Anterior Hyaloid Face

An intact hyaloid face is an important pathogenic factor in malignant glaucoma and in pseudophakic or aphakic patients,

Nd:YAG laser capsulotomy with disruption of the anterior hyaloid face is often effective

Surgical treatment

- Core vitrectomy
- Phacoemulsification and IOL implantation

And this is what was done to our patient

Core vitrectomy Entry of AC Reformation of the AC

Anterior and posterior synechiolysis



Phacoemulsification and implantation of PC IOL

Postoperatively the patient was on:

- Topical steroids and antibiotics
- Topical atropine
- Topical combined timolol and brimonidine(Combigan)

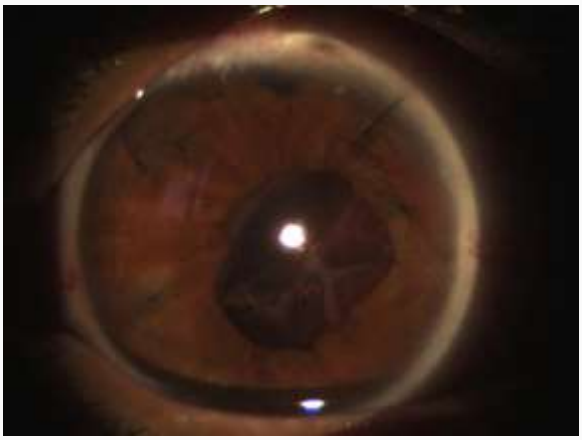
- The medications were withdrawn gradually over 5 weeks.

Now after 2 months

Deep anterior chamber

IOP: 12 mmHg (No medications)

BCVA 6/60

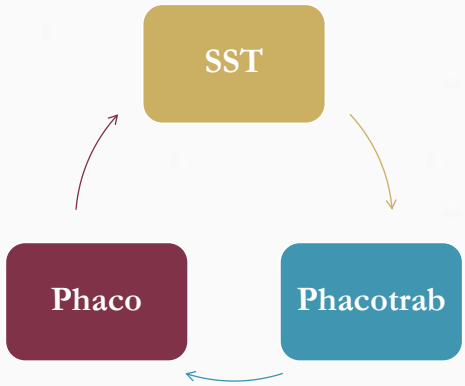


What about the other eye??

The IOP is 14 on a single medication

Refraction is: +10.00/-0.5@30

What to be done?



Phacoemulsification

Prophylactic measures:

- Preoperative atropine eye drops
- Phacoemulsification and IOL implantation
- Core vitrectomy
- Posterior capsulotomy by vitrectomy probe
- Postoperative atropine eye drops.

Take home message

- Consider lens extraction in the hyperopic eyes
- Consider core vitrectomy in the hyperopic eyes
- Malignant glaucoma can affect the eye years after the surgery
- Malignant glaucoma can be presented with a normal IOP
- Malignant glaucoma occurs only with an intact anterior hyaloid

Take home message

Always keep an eye on the glaucoma patient and never give your back to glaucoma