35 years old female patient presented with **blurring of vision of OS**.

**BCVA** was 6/18.

**IOP** was 38 mm Hg with applanation tonometer.

**Gonioscopy** showed open angle.
Examination of the conjunctiva and episclera of OS showed dilated tortuous episcleral veins (upper) while no abnormalities were present in OD (lower).
Disc examination of OS showed large glaucomatous cup.
• **Visual field** examination of OS showed dense lower arcuate scotoma with upper nasal step.
• **IOP of OD** was 18 mm Hg with normal **disc** and **field**.
• *Intracranial, orbital or systemic causes* of increased episcleral venous pressure has been excluded.

• *Medical treatment* was started with two drugs (prostaglandin and a combination of carbonic anhydrase inhibitor and beta-blocker) with a mean IOP of 22 mm Hg over a follow-up period of 6 month.
• Visual field follow up after 6 month showed deterioration in the central and upper paracentral field and BCVA dropped to 6/24.
While the **field** of OD still normal on follow up.
Because IOP can not be lowered below the *draining down* stream pressure, it was expected that episcleral evnous pressure of this eye is 22 mm Hg and IOP will not be lower than 22 mm Hg.

The decision of surgery with the possible complications has been discussed with the patient but she refused surgery and a third drug (Alpha 2 agonist) has been added with no more lowering of IOP.
Episcleral Venous Pressure

- A factor contributing to normal intraocular pressure (IOP) is episcleral venous pressure. It is not an important variable in the pathogenesis or treatment of most eyes with glaucoma because the trabecular meshwork is the major site of obstruction to aqueous outflow.

- Normal episcleral venous pressure varies according to the measurement technique and averages between 9 and 11 mm Hg. For every 1 mm Hg rise in episcleral venous pressure there is a 1 mm Hg rise in IOP.
CAUSES OF ELEVATED EPISCLERAL VENOUS PRESSURE

I. Idiopathic (The most common cause)

II. Venous obstruction
   - Thyrotropic ophthalmopathy
   - Retrobulbar tumors
   - Cavernous sinus thrombosis
   - Obstruction of superior vena cava because of aortic aneurysm, thoracic neoplasm, or surgical ligation

III. Arteriovenous communication
   - Carotid artery—cavernous sinus fistula
   - Dural shunt
   - Sturge-Weber syndrome

IV. Orbital varices
II. Venous Obstruction

- In **thyrotropic ophthalmopathy** orbital congestion associated with inflammatory cellular infiltration may cause an obstruction of ocular and orbital venous drainage resulting in elevated episcleral venous pressure and secondary glaucoma.

- **Retrobulbar tumors** and **cavernous sinus thrombosis** may also obstruct venous drainage. **Obstruction of the superior vena cava** may occur because of an aortic aneurysm or a thoracic neoplasm in the mediastinum. Even temporary surgical ligation of the superior vena cava during cardiac surgery is known to have caused bilateral glaucoma that resolved after a period of 6 weeks.
Multiple *ocular and orbital signs* may occur with venous obstruction. These include:

- **Edema** of the lids, face, and conjunctiva,
- **Engorgement and dilation of the veins** of the face, neck, conjunctiva, episclera, and the fundus.
- **Exophthalmos, optic disc edema, and glaucoma.**

All of these signs are accentuated by moving the patient from the upright to the recumbent position.
III. Arteriovenous communication

- Arterio-venous communications or fistulas allow arterial blood to flow into venous channels because of the pressure gradient between arteries and veins. As a result, intravenous pressure rises and venous blood flow is altered in rate and direction.

1. **Carotid-Cavernous fistula:**
   - If a fistula develops between the *internal carotid artery* and *the cavernous sinus*, glaucoma secondary to elevated episcleral venous pressure may occur. Such fistulas usually arise as a result of trauma.
The ocular and orbital signs include:

- Proptosis,
- Dilated episcleral veins, chemosis, an
- Audible bruit,
- Dilated retinal veins,
- Pulsation of the globe, and
- Periorbital edema.

Secondary glaucoma occurs in 30% to 41% of these cases.
Another type of arteriovenous communication that elevates episcleral venous pressure and causes secondary glaucoma is a dural shunt. Arterial blood flows through small meningeal branches of the carotid arteries to enter the venous circulation near the cavernous sinus.

The ocular and orbital signs of a dural shunt are typically milder than those associated with carotid artery-cavernous sinus fistulas. Most patients will have spontaneous improvement in their clinical signs (including normalization of 10P) and surgical treatment of the shunt should be avoided if possible.
3. Sturge-Weber syndrome

- The **Sturge-Weber syndrome** refers to the association of a usually unilateral cutaneous facial hemangioma (port-wine stain or nevus flammeus), ipsilateral meningeal hemangioma, and ocular hemangioma.

- The **ocular findings** include conjunctival and episcleral hemangiomas, heterochromia (with the darker iris on the affected side), glaucoma, tortuosity of the retinal vessels, and choroidal hemangioma.

- Thirty percent of patients with Sturge-Weber syndrome develop glaucoma. If the cutaneous hemangioma involves the skin of the upper eyelid, ipsilateral glaucoma is almost invariably present. Glaucoma is usually present at birth but may become apparent later in childhood, adolescence, or adult life.
Another cause of elevated episcleral venous pressure and secondary glaucoma is orbital varices.

This lesion is characterized by intermittent exophthalmos associated with stooping over or the Valsalva maneuver.
I. Idiopathic Elevated Episcleral Venous Pressure (IEEVP)

- In this group which is the most common there is no recognized cause outside the eye for elevation of venous pressure and thus termed idiopathic cases but the vessels on the globe are dilated and tortuous large and measurement shows the pressure in these vessels to be elevated, to more than 30 mm Hg in some cases.

- Diagnostic evaluation should include a complete ophthalmic examination for a glaucoma suspect; radiological imaging, such as an MRI, and non-invasive vascular imaging to exclude a cerebrovascular disorder should also be performed.
• The diagnosis of IEEVP is one of exclusion after intraorbital and intracranial pathology has been excluded. The diagnosis is based on the clinical findings of dilated tortuous episcleral veins, elevated IOP causing characteristic optic nerve and/or visual field changes typical of glaucoma, and an open angle on gonioscopy. Blood in the Schlemm canal is a general sign of elevated EVP but is not present in all cases of IEEVP.

• Because IOP can not be lowered below the draining down stream pressure, in these conditions the IOP remains elevated after initiation of medical therapy.
• If surgery is inevitable, IOP should be lowered as much as possible with glaucoma medical therapy. The following precautions should be taken in glaucoma surgery:

➢ Prophylactic *posterior sclerotomies*,
➢ *Smaller* incision, excision trabeculotomies and
➢ Anterior chamber *viscoelastics* should be used at the time of filtering surgery in such patients.
• It is assumed that the intravascular pressure in the choroid and ciliary body in such eyes is elevated. When the eye is opened, the IOP drops to the level of atmospheric pressure. This promotes an extravasation of fluid from choroidal vessels.

• With a preexisting increase in the venous pressure of the choroid, the extravasation of fluid is more massive and rapid and a non-rhegmatogenous serous retinal detachment may also occur.
The technique of **V-shaped unsutured sclerotomy** was as follows:

1. **Radial conjunctival incisions** 10 mm in length were made in the inferotemporal and inferonasal quadrants.

2. The location of the triangular sclerotomy was first outlined on sclera with monopolar underwater diathermy to provide hemostasis when the sclera is incised in the next step.

3. Each side of the triangle is 4 mm long and its apex is located 4 to 5 mm behind the surgical or posterior limbus.
4. A No. 15 Bard – Parker blade was used to make full thickness scleral incisions along two sides of each triangle.

5. The base is left attached. The blade is then used to dissect sclera from underlying choroids.
6. A cylodialysis spatula inserted in the suprachoroidal space on both sides to drain the suprachoroidal fluid.
7. Conjunctiva is closed with 10/0 mattress sutures over the triangular sclerotomy which were left open without closure.
THANK YOU