Targeting The Vitreoretinal Interface In Diabetics

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Targeting The Vitreoretinal Interface In Diabetics

- **Targeting by brain**: to understand the nature of the vitreous changes in diabetics and its role in the pathogenesis of DR
- **Targeting by eyes**: to detect the changes by clinical examination and imaging
- **Targeting by hands**: to treat the VR interface lesions by IV injection and by surgery

TARGETING BY YOUR BRAIN

VITREOUS DIFFERENCES IN DIABETICS

In **nondiabetics**, the vitreous gel goes through aging changes consisting of:-

- Lacunae formation
- Liquefaction
- Shrinkage

In **diabetics**, the vitreous gel shows:

- Less Lacunae formation
- Less liquefaction
- More shrinkage

So, the vitreous in diabetics is more formed and more contracted = More PVD

?

VITREOUS SHRINKAGE

In diabetic eyes, even **without retinopathy:-**

- Breakdown in the blood- vitreous barrier.
- Leakage of blood constituents into the vitreous
- Leads to shrinkage of the vitreous

In diabetic eyes **with active retinopathy**,

- Retinal neovascularities also leak into the vitreous and further promote vitreous shrinkage and contraction.

VITREORETINAL ADHESIONS

In response to retinal ischemia in a diabetic eye, retinal neovessels proliferate.

These proliferations are located on the surface of the retina and insinuate between the retina and the vitreous.

When fibrocytes are laid down and proliferate, the proliferation turns from a ***neovascular*** to ***fibrovascular*** and into a ***fibrous*** proliferation.

VITREORETINAL ADHESIONS

These proliferations constitute points or areas of vitreo-retinal adhesions.

The strength of these adhesions varies depending on the nature of the adhesion and also on its chronicity

PARTIAL PVD

Partial or anomalous PVD is the result of increased vitreous shrinkage and increased VR adhesions in diabetic eyes with PDR

Initial Vitreous Findings in Patients with Diabetic Retinopathy

VITREOUS SEPARATION	None	Partial	Complete
Non-proliferative cases	71	7	22 (%)
Proliferative cases	34	64	2 (%)

Complications from PDR arise as a result of the interaction between the vitreoretinal adhesions, and the vitreous contraction in diabetic eyes.

There are mainly three complications:-

VH

TRD

Rapid neovascular growth.

PARTIAL PVD

Vitreous for the Retina Specialist

An Enemy

- That contributes to the disease process

An Ally

- That potentiates treatment by providing a reservoir for drug placement.

Successfully changing the vitreous by inducing a PVD has become an important therapeutic approach for many retinal and vitreoretinal interface diseases.

THE DVR CONCEPT

The term DR is like a two dimentional concept of the disease giving the impression that it is only an angiopathy.

The term DVR is a three dimentional one, which prompts us to think about the vascular and the mechanical aspect of the disease.

VITROUS AFFECTS THE COURSE OF DR

- **In early onset diabetes**, chronic and extensive angiopathy stimulates new vessel growth before the vitreous is totally detached.
- Partial PVD results and proliferative changes and their inherent complications of vitreous hemorrhage, tractional and rhegmatogenous retinal detachment and accelerated new vessel growth frequently follow.

VITROUS AFFECTS THE COURSE OF DR

- **In late onset diabetes**, PVD occurs before angiopathy became significant enough to stimulate new vessel growth
- Thus, nonproliferative diabetic changes generally result.

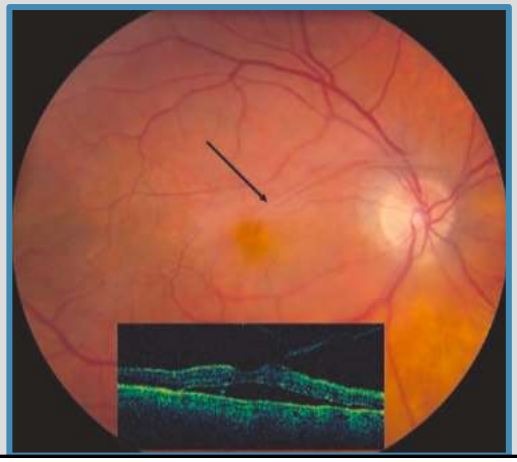
VITREOUS AFFECTS RESPONSE TO TREATENT

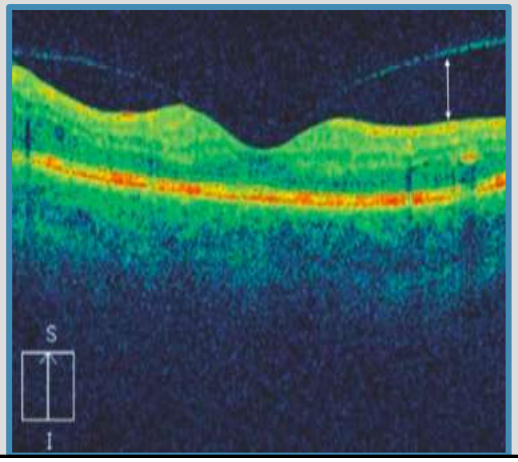
- Complete PVD is protective
- PRP is more beneficial with no anomalous PVD
- Vitreous itself act as a reservoir for the drugs after IVI

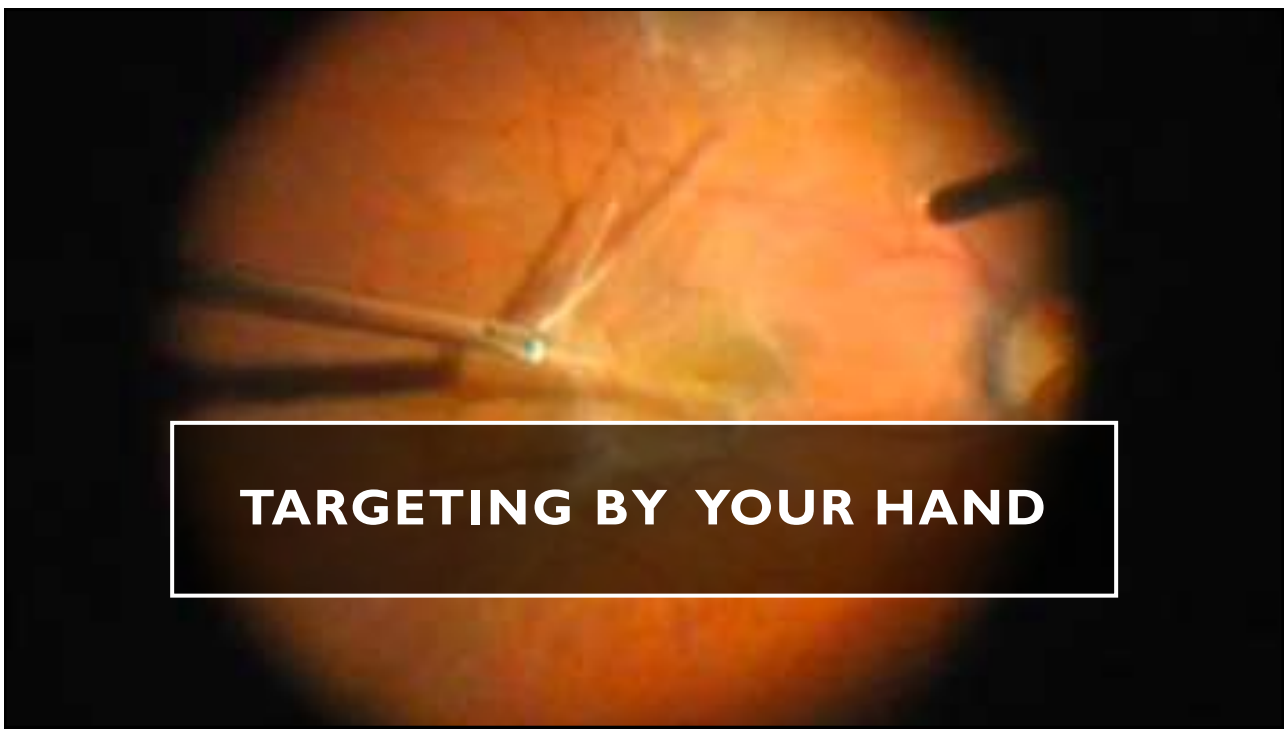
CLINICAL EXAMINATION

- Clinically, the vitreous could be examined using a +78 D lens and a slit lamp using a thin strong slit beam.
- The 78 D lens facilitates detection of the vitreous cortical movement on eye excursions.
- It is also important for the observer to become dark adapted before vitreous examination (30 sec).
- Thirdly, ensuring that the patient's pupil is widely dilated would also facilitate the examination.

OCT







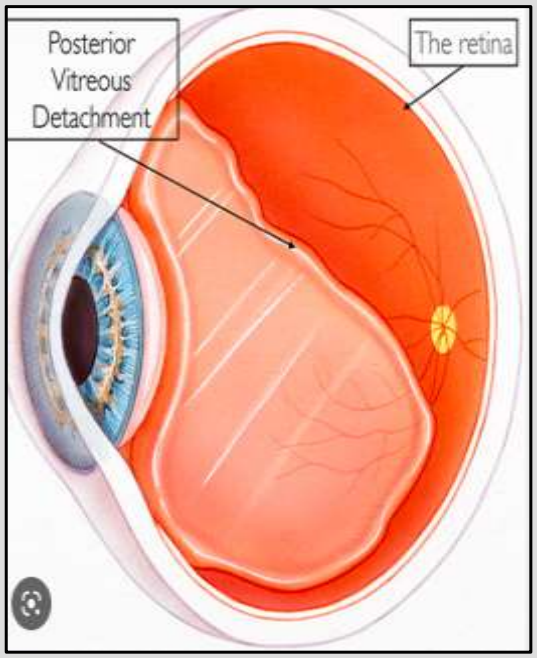
TREATMENT

- Creating a complete PVD to relieve an anomalous PVD is the aim.
- This can occur in 3 situations:-

1- Unintended PVD after IV injections or after PRP

2- Pharmacological vitreolysis

3- Surgery



UNINTENDED PVD

After PRP:-

-- PVD occurs in 2/3 (66%) of cases with no PH of VH

-- PVD occurs in 1/3 (33%) of cases with PH of VH

After IVI:-

-- PVD occurs in 1/4 (25%) of cases after 3 IVI

PHARMACOLOGICAL VITREOLYSIS

- It involves the use of drugs that liquefy the vitreous and weaken the adhesion between the vitreous and the ILM
- IV hyaluronidase and collagenase, Chondroitinase, dispase, plasmin, and tissue plasminogen activator (tPA) have been tested in animals and sporadically in humans
- Successful induction of PVDs has been achieved after intravitreal injections of Ocriplasmin (about 29%)

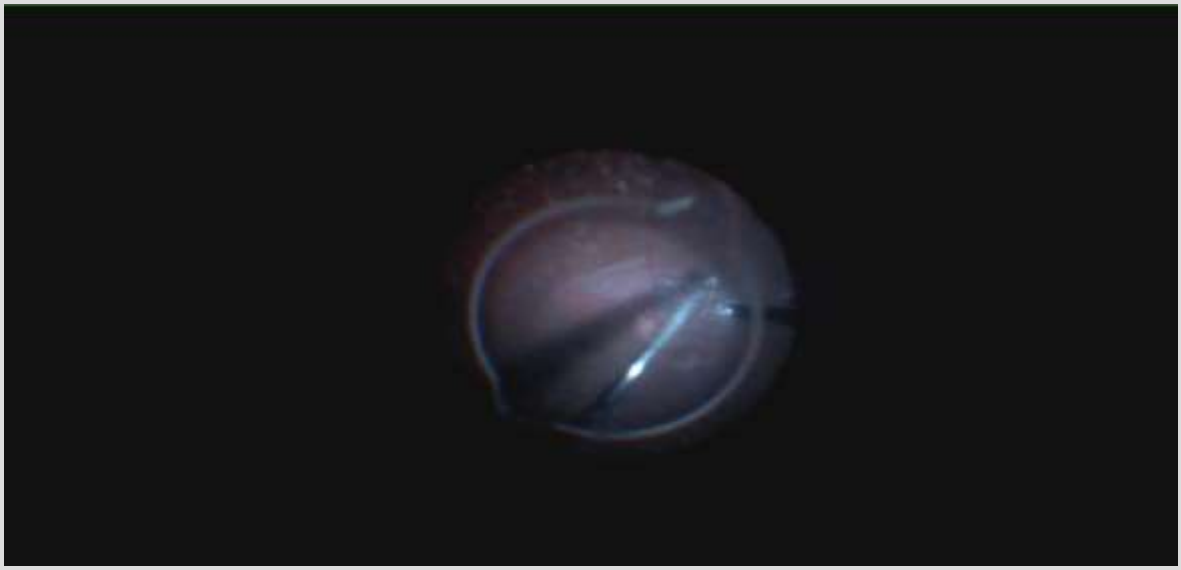
SURGERY

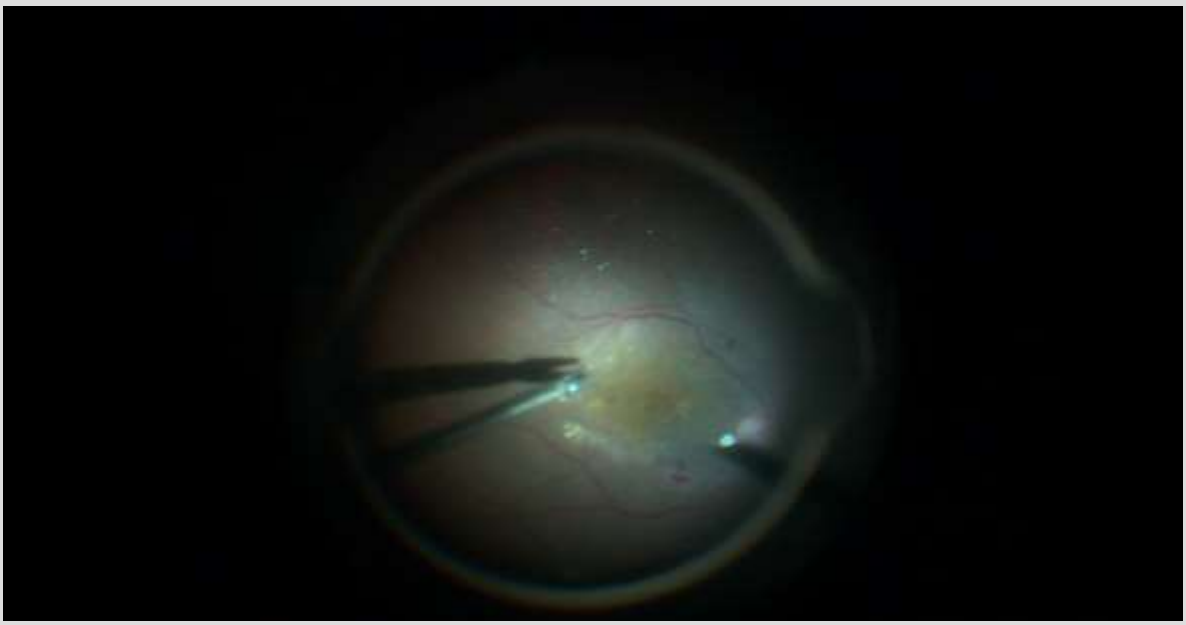
- **Can be done by**

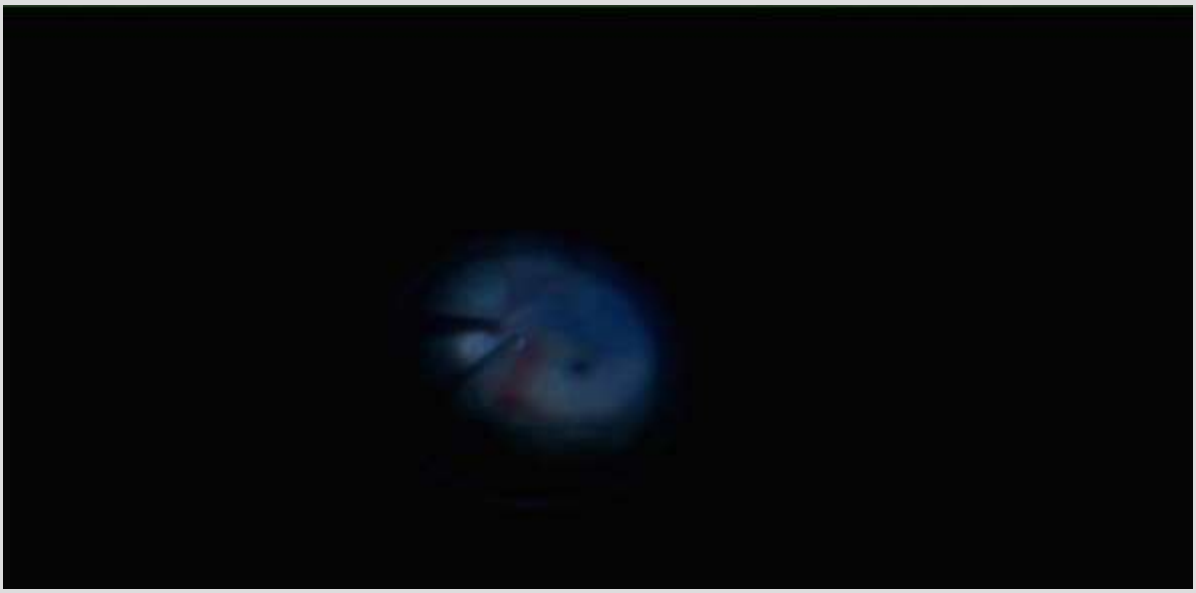
1- PPV with removal of posterior hyaloid

2- PPV with removal of posterior hyaloid and ERM

3- PPV with removal of posterior hyaloid and ILM peeling







DON'T FORGET

- It is a diabetic **VITREO**retinopathy

THANK YOU