PERSISTENT PUPILLARY MEMBRANE- AN OVERVIEW

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PRESENTATION OF CASE

A 43-year-old working female came to Eye OPD of our hospital with complain of diminished vision in both eyes since childhood, especially at night. She had no relevant past history and also there was no systemic illness.

Persistent Pupillary Membrane (PPM) is the most frequently encountered ocular congenital anomaly in general ophthalmological practice.[1] Waardenburg stated that remnants of the embryonic vasculature are found in 80% dark eyes and 35% of light eyes.[2] PPM is due to the continued existence of part of the anterior vascular sheath of the lens, a foetal structure which normally disappears shortly before birth. If it persists, may appear as delicate cobweb like strands or threads stretched across the pupil or may be anchored down to the lens capsule and instead into the iris collarettes.

Here, we report a case of bilateral persistent pupillary membrane, which was successfully treated by Nd-YAG laser.[3]

OCULAR EXAMINATION

On examination, her uncorrected visual acuity by Snellen's chart in both eyes was 6/36, which could not be improved through refraction. She was having near visual acuity of N-10. Auto-refractometer reading was +7.50 D Sph in right eye and + 4.50 D Sph/ +2.0 D Cyl X 40^0 in left eye. On slit lamp examination, lid and conjunctiva was unremarkable, cornea was clear, anterior chamber was deep and quiet in both eyes. Both the pupils were round, but pupillary area was covered with dense pupillary membrane. Small clumps of iris pigments were present on anterior surface of lens capsule and spider-like remnants of pupillary membrane were present in both eyes. These membranes were attached to the collarettes. Anterior chamber angles were open in all quadrants in both eyes. Posterior chamber was normal.

On attempting pupillary dilatation with tropicamide-phenylephrine combination eye drop, only partial pupillary dilation could be achieved. On the basis of all the clinical findings, the diagnosis of persistent pupillary membrane in both eyes was established.

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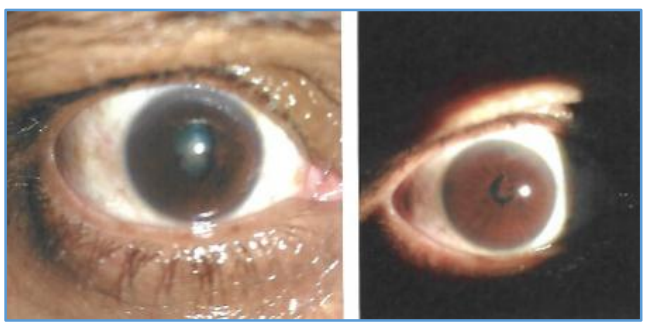

Figure 1. Showing persistent Pupillary Membrane

DIFFERENTIAL DIAGNOSIS

1. Post-inflammatory synechiae.
2. Congenital idiopathic microcoria.
3. Accessory iris membrane.
4. Axenfeld-Rieger syndrome.
5. Iridocorneal endothelial syndrome.

CLINICAL DIAGNOSIS

Persistent Pupillary Membrane (PPM) represents incomplete regression of the tunica vasculosa lentis, which normally involutes by 6th month of gestation. When this process is incomplete, strands of connective tissue may attach to the iris collarettes. Usually, minimal connective tissue remnants do not affect vision, although if symptomatic mydriatic agent may occasionally relieve a partially obstructed aperture.

Large membranes may disrupt the visual axis resulting in either visual symptoms or amblyogenic opacities requiring surgical excision or laser lysis.[4]

Duke-Elder has Classified PPM in Several Variants[5]

- That are attached solely to the iris.
- Iridolenticular adhesions.
- Sub-variants are pigmented dendritic iris stromal melanocytes, singly or in clumps, are situated aberrantly or to the anterior lens capsule. These pigments strands of anterior lens capsule are called "Chicken tracks."
- That are attached to the cornea, typically occurs in the Axenfeld-Rieger Syndrome.
  Although most cases of PPMs imperforated sheets of iris stroma may occlude the pupil, such lesions have been photo disrupted successfully with Nd-YAG laser.

MANAGEMENT

Mydriatics, laser treatment or surgery is used to clear the visual axis and tries to improve visual development. Surgical intervention is reserved for large dense PPMs.

In this case, patient was advised for Nd-YAG Laser therapy after instillation of Nepafenac eye drops 3 times a day and tropicamide-phenylephrine combination eye drops 3 times daily in both eyes for 1 week. Nd-YAG laser has been used to cut the PPMs in two settings at 1 week apart. Settings

were on Q switch, single pulse having power 6-8 MJ. Number of shots used to disrupt the PPMs were varied from 4 - 5 bursts in each eye applied.

In patients with thin sparse membrane, Neodymium: YAG laser membrane lysis may be performed.[6] Presence of blood vessels in these PPMs can lead to hyphema due to photo disruption of membrane. This procedure can also have risk of cataract formation and pigment disruption. Pigment disruption and bleeding occur in this procedure, which were managed by topical corticosteroid, NSAID, anti-glaucoma and mydriatics. 2 weeks after the Nd-YAG laser therapy, refractive status of both eyes were UCVA 6/18 in right eye and 6/24 in left eye.

BCVA in both eyes with +2.0 D Sph in right eye and +1.5/+0.75 D Cy x 70 D in left eye. Near vision was N-12 with +2.50 D Sph.

Embryological Discussion and Pathogenesis

During the early development of the eye, the crystalline lens is nourished by the tunica-vasculosa lentis. The posterior part of which is derived from branches of hyaloid artery called the Vasa hyaloidea propria. Although, its anterior part interconnects with the vasa hyaloidea propria, the pupillary membrane is derived from vascular arcades that bud from the annular vessels that skirts the rim of the developing neuroectodermal optic cup.

At the height of its development during the 5th month of gestation, the vascular arcades of pupillary membrane nearly reach the centre of the pupil. These blood vessels begin to undergo remodelling and regression in a process involving phagocytosis by macrophage at around 5 months gestation. There might be dysfunction of macrophage invasion and phagocytosis that lead to key role in pathogenesis of PPM.[7]

Involution and atrophy of the pupillary membrane and associated anterior iris stroma commences during the 6th month of gestation and proceeds peripherally, forming the thinner pupillary zone and collarette of the adult iris.

Pupillary membrane fully developed by 9th week of gestation and disappeared by 34th week of gestation.[8]

There is some more recent evidence that intrauterine stress, particularly from chronic maternal hypertension, may accelerate the disappearance of these membranes.[9]

These membranes are often observed by neonatologist as they are frequently seen in premature babies.[10] They can be used to identify the gestational age of new-borns (between 27 and 34 weeks). PPMs should be distinguished from the congenital idiopathic microcoria, which result from underdevelopment of dilator pupillae.[11] During the 1st year of life, PPMs undergo atrophy and require no treatment. If membrane persisting after 1 year are less likely to regress spontaneously, increases the risk of deprivation amblyopia.[12] A 1.5 mm pupillary aperture is necessary for adequate retinal stimulation and visual cortex development.

DISCUSSION OF MANAGEMENT

PPM which are large enough to interrupt the visual axis are uncommon. If they are left untreated, may cause stimulus deprivation amblyopia.

This case illustrates that adequate pupillary opening in PPMs lead to normal visual development. There is no evidence of amblyopia due to adequate opening in pupillary membrane in both eyes. So, Nd-YAG laser membrane lysis is beneficial in this case. Minimal bleeding is observed during the procedure.

FINAL DIAGNOSIS

Persistent Pupillary Membrane

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