INCIDENCE, CORRELATION AND NATURAL HISTORY OF EPIRETINAL MEMBRANES SURROUNDING IDIOPATHIC MACULAR HOLES

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THESES ON INCIDENCE, CORRELATION AND NATURAL HISTORY OF EPIRETINAL MEMBRANES SURROUNDING IDIOPATHIC MACULAR HOLES

Duration – 1 YEAR

AIM – to evaluate the prevalence, correlation and natural history of EPIRETINAL MEMBRANES SURROUNDING IDIOPATHIC MACULAR HOLES

PRIMARY OBJECTIVES:

1. To evaluate the prevalence of epiretinal membranes in eyes with stage 2/3/4 macular holes.

2. The correlation between epiretinal membrane and visual acuity in idiopathic macular holes

3. The correlation between size of macular hole and epiretinal membrane

Secondary objective - To assess the increase in severity of epiretinal membrane in follow up.

Study design – Prospective study, observational study

Methodology (material and methods) - All patients with idiopathic macular hole stage 2, 3, 4 who attended the OPD

Inclusion criteria - All patients with symptoms of decreased central vision and metamorphopsia and clinically proven macular hole stage 2, 3 and 4

Exclusion criteria - Patients who had traumatic macular hole and macular hole due to other causes and macular edema.
Sample size- 50 patients

Age - 30 to 80 years

Gender – both genders

- Study parameters
- Detailed history including past and present illness
- Visual acuity assessment using snellens visual acuity chart
- Anterior segment examination using slit lamp including lens and anterior vitreous.
- Fundus examination using direct and indirect ophthalmoscopic examination.
- Stereoscopic fundus examination using slit lamp biomicroscopy
- Colour fundus photography
- Optical coherence tomography.

- Data collection and methods-All cases diagnosed as idiopathic macular holes stage 2,3 and 4 will be registered and evaluated subsequently at the end of 3,6 and 12 months of follow up.

- Analysis plan- to consider the importance of factors in removal of epiretinal membrane during vitrectomy for macular hole
INTRODUCTION

Macular holes are full thickness neurosensory retinal defects surrounded by localised annular neurosensory detachment. Most of the macular holes are idiopathic and probably arising as a result of localised traction in the foveal region by the attached posterior cortical vitreous. Most patients have visual symptoms of metamorphopsia and loss of visual acuity.

Epiretinal membranes are also called primary retinal folds, secondary retinal gliosis, cellophane maculopathy, preretinal traction membranes. They are commonly benign and cause minimal symptoms but they can also be associated with significant loss of acuity and metamorphopsia. They are associated with numerous other ocular conditions and diseases. Epiretinal membranes may also be idiopathic with no associated ocular abnormality or history.

Idiopathic macular holes are associated with epiretinal membranes in majority of the cases and form a major cause of defective central vision.
REVIEW OF LITERATURE

EPIDEMIOLOGY

Macular holes usually only affect one eye, though there is a 10 per cent, one in ten, chance that the other eye will eventually be affected. They mostly occur in the age group of 60-80 years. They are twice more common in women than men.
**NATURAL HISTORY**

The natural history of macular holes is well established. Stage 1 holes usually have a 66% progression rate and stage 2 macular holes have a 74% rate of progression to full thickness macular holes. The incidence of apparent disappearance of idiopathic macular holes is low. Foveal detachment and macular break resolution seem to result from weakening of the vitreous attachment at the fovea. Reattachment of the fovea may preserve fairly good amount of visual acuity. In patients with established full thickness macular hole, the fellow eye with attached vitreous has 12% chance of developing a macular hole. The prevalence rates of PVD is 32% and 0% in the centric hole and eccentric hole groups respectively. In addition to tangential traction, an element of *oblique antero-posterior traction* may play a role in the pathology of idiopathic macular holes.
ANATOMY OF THE RETINA

Retina is the inner most coat of eyeball. It is a thin and delicate transparent membrane. This layer is where the optical image is formed by the eyes optical system. Photochemical transduction occurs so that nerve impulses created here are transmitted along the visual pathways to the brain for higher processing. It is thick at the posterior pole and peri-papillary region of about 0.56mm whereas the thickness decreases to about 0.1 mm at the ora-serrata. It is thinnest at the centre of the fovea. The outer surface of the retina is in contact with Bruchs membrane of the choroid and the inner surface is in contact with the vitreous body.

Retina extends more anteriorly on the medial aspect than laterally. Ora serrata lies closer to the limbus medially. It consists of an outer pigmented layer and an inner neurosensory layer which are derived from the neuro-ectoderm. The ORA SERRATA is the anterior wavy edge of the retina just posterior to the ciliary body.

Retina can be grossly divided into Optic disc, Macula lutea and rest of the peripheral retina.
OPTIC DISC:

It is a pale pink, circular area of about 1.5mm in diameter. All the layers of the retina terminate at the optic disc except the nerve fibres. The optic disc appears white due to the absence of vascular choroid and the presence of lamina-cribrosa and medullated nerve fibres behind it. There is complete absence of rods and cones at the optic disc and hence insensitive to light and is referred to as the BLIND SPOT. The area of sclera where the optic nerve fibres exit the eye by piercing the sclera is called the LAMINA CRIBROSA. Posterior to the optic disc, the nerve fibers are myelinated, whereas anterior to the disc they are non myelinated.

PHYSIOLOGICAL CUP:

It is a depression seen at the centre of the optic disc. The central retinal vessels emerge from the centre of this cup.

MACULA LUTEA:

It is otherwise called the yellow spot. It is a relatively darker area of about 5.5mm in diameter situated at the posterior pole just temporal to the optic disc.
FOVEA CENTRALIS:

Most sensitive portion of the retina is the fovea centralis. It is about 1.85 mm in diameter and 0.25 mm thick. It corresponds to about 5 degrees of the visual field. Structurally, there are no rods; cones are larger and tightly packed as against the other areas which are thin.

FOVEOLA:

It is about 0.35 mm in diameter. It forms the floor of the fovea and is situated about 2 DD away from the temporal edge of optic disc and 1 mm below the horizontal meridian. Umbo is a central tiny depression at the centre of the foveola that corresponds to the foveolar reflex. There are no blood vessels overlying the fovea and no rod cells in the floor of fovea. The closeness of packing of the photoreceptors and therefore the angle each subtends ultimately limit the visual acuity obtained by the retina.

FOVEAL AVASCULAR ZONE is about 350 to 500 microns in diameter. PARAFOVEAL ZONE is about 0.5 mm in diameter and the PERIFOVEAL ZONE is about 1.5 mm in diameter.
PIGMENTED LAYER OF THE RETINA

RPE consists of a single layer of cells that extends from the margin of the optic nerve head to ora serrata anteriorly. These cells are tall and narrow in the posterior pole and they become flattened near the ora serrata. These cells are hexagonal in cross section. The basal end of each cell is infolded and rests on a basement membrane. The apical ends of cells show numerous microvilli measuring 5-7 microns long. These microvilli are embedded in glycosaminoglycans which may act as an adhesive binding the pigment layer to the neural layer. The adjacent cell membranes are bound together in the basal region by zonula adherens and in the apical region by zonula occludens. These tight junctions are very important in maintaining the isolation of the retina from the systemic circulation.

FUNCTIONS:

1. Absorption of light
2. Turnover of outer segments of photoreceptors
3. Vitamin A metabolism
4. Antireflection mechanism
5. Blood retinal barrier - the tight junctions that completely encircle the cells in the RPE forms a barrier
that limits the flow of ions and prevents diffusion of large molecules from the choroid capillaries to the photoreceptors of neural retina

NEURAL RETINA:

Embryologically derived from the inner layer of the optic cup, it mainly consists of photoreceptors, bipolar cells and ganglion cells. Other important cells include horizontal cells and amacrine cells.

THE RODS AND CONES interdigitate with the pigment epithelium and are referred to as the outer segments. They are responsible for photopic vision (cones) and scotopic vision (rods).

THE BIPOLAR CELLS:

There are three types of bipolar cells namely rod bipolar cells, flat or diffuse bipolar cells and midget bipolar cells. They synapse with the ganglion and amacrine cells.

GANGLION CELLS:

They resemble cells seen in the nervous ganglia. They are the second order neurons in the visual pathway. They have non-myelinated axons that converge at the exit of optic nerve head.
The other cells include **horizontal cells** and **amacrine cells**.

Histologically, the retina consists of **ten layers**.

The pigment epithelium

1. The rods and cones
2. The external limiting membrane
3. The outer nuclear layer
4. The outer plexiform layer
5. The inner nuclear layer
6. The inner plexiform layer
7. The ganglion cells
8. The nerve fiber layer
9. The internal limiting membrane
ANATOMY OF THE MACULA

Fovea is the seat of central vision. In this area, there are no rods, cones are larger, in abundance and tightly packed and the other areas of retina are thin.

The foveola largely consists of cones and their nuclei covered by a thin internal limiting membrane. All other retinal layers are very thin at the foveolar region. In the foveolar region, the cone axons are obliquely arranged to reach the margin of the fovea called the Henle’s layer.
OPTICAL COHERENCE TOMOGRAPHY (OCT)

OCT is a non invasive non contact imaging system based on interferometric optical tomographic evaluation of ocular tissues. It produces micro resolution retinal images in vivo. It is analogous to B scan except that it uses light instead of sound. Image resolutions to the order of 1-15 microns have been achieved both in situ and real time. There is an increased advantage of increased resolution and speed of acquisition.

It is based on the principle of low coherence interferometry.
The techniques used are

1. **Time domain OCT**: The path length of the reference arm is translated longitudinally in time. The time delay of the reflected or back scattered light from microstructures within the tissue is used for evaluation and analysis.

2. **Frequency domain OCT**: The broad band interference is acquired using spectrally spaced detectors. The depth scan is obtained immediately using a fourier transform from the acquired spectra without the movement of the reference arm.

The use of optical coherence tomography in diagnosing posterior segment disorders is one of the most mature applications of OCT. OCT has contributed to a better understanding of the pathogenesis of macular holes, vitreomacular traction and has provided a quantitative method of accurately detecting changes in the retinal thickness due to diabetes, epiretinal membrane and cystoid macular edema.

The posterior boundary of the neurosensory retina is represented by a highly reflective layer of retinal pigment epithelium and choriocapillaris. The outer segments of photoreceptors are represented by a dark area of minimal reflectivity just anterior to the highly reflective
band of RPE and chorio capillaris. The inner margin of the retina, the
nerve fibre layer is represented by a highly reflective red band due to
bright back scatter against the contrast of non reflective vitreous. The
intervening layers are represented by alternating areas of moderate to low
reflectivity.

Longitudinal surveillance of patients with macular disease requires
six radial scans centered at the fovea. Evaluation of the entire macular
region is possible using the retinal thickness map. Retinal thickness is
computed for 600 macular locations and then plotted on a false colour
topographic map.

Traditionally the diagnosis of macular holes is done using contact
lens slit lamp bio microscopy. There are a number of lesions like partial
or lamellar thickness macular holes, Pseudo holes, macular cysts that may
be difficult to distinguish from full thickness macular holes. All the above
mentioned disorders lack the characteristic full thickness defect with fluid
cuff and flask shaped appearance. OCT aids in identification and staging
of macular holes according to the GASS classification.
HISTORY OF MACULAR HOLES

*Knapp and Noyes* were the first to describe macular holes in the late 1800s.

*Gass* first described a series of changes in the formation of idiopathic macular holes. Epiretinal membrane was first described by *Iwanoff* in 1865.

TIMELINE IN THE EVOLUTION OF MACULAR HOLE CONCEPTS

1. 1869 - **Knapp** – First case description of macular hole (traumatic)
2. 1871 – **Noyes** – First detailed clinical description of macular hole (traumatic)
3. 1900 – **Kuhnt** – Atraumatic theories of cystic retinal degeneration leading to macular hole.
4. 1901 – **Fuchs** and 1907 – **Coats** – Early histopathologic changes in macular hole including cystic changes.
5. 1912 – **Zeeman** – Histopathologic recognition of premacular vitreous condensation.
6. 1924 – **Lister** – Vitreous forces and traction bands may cause premacular holes.
7. 1967 – REESE ET AL – Vitreous separation critical to macular hole formation.


10. 1986 – MORGAN AND SCHATZ – Involutional macular thinning is a premacular hole condition.


ETIOLOGY OF MACULAR HOLE

I. PRIMARY:

Idiopathic (70-80 %)

- Occurs due to aging unrelated to any ocular or any other antecedent events.

II. SECONDARY:

1. Trauma
2. Hypertensive retinopathy
3. Associated with Cystoid macular oedema -
   (inflammation, retinal vascular disease, macular pucker)
4. Secondary to Retinal detachment
5. Extreme myopia
6. Post LASER therapy
7. Lightning strike
PATHOPHYSIOLOGY OF MACULAR HOLE

**Vitreo - macular traction** from posterior vitreous surface is the main event in the creation of macular hole. Antero-posterior trans-vitreal traction by vitreous fibres extending to the vitreous base may lead to pathologic changes that lead to full thickness macular hole. **Gass and Johnson** had proposed a theory whereby shrinkage of adjacent cortical vitreous and subsequent tangential vitreous traction first causes a circumscribed foveolar dehiscence followed by retinal dehiscence. Then subsequently vitreofoveal separation occurs and finally complete posterior vitreous detachment with enlargement of the macular hole.

**Guyer and Green** later proposed three theories for the formation of idiopathic senile macular hole.

1. Fluid movements and counter currents
2. Cellular remodelling of the cortical vitreous
3. Contraction of a cellular membrane on the inner surface of the tapered cortical vitreous.

Proliferation of fibrous astrocytes and muellers cells occurs with the formation of macular hole. This reparative tissue was previously interpreted as operculum. Full thickness macular holes have previously
been documented to arise in eyes with complete PVD. So pathogenesis other than tangential traction is likely in the formation and progression of senile macular holes.

**Hydrodynamic model** of macular hole states that a macular hole is formed or maintained by fluid flow caused by the macular retinal pigment epithelial pump.

In reality the aetiology and pathophysiology of macular hole may be multifactorial. Recognising the primary event probably is less important than considering factors like vitreo-macular traction, foveolar dehiscence etc. which can change the management and decision making.
PATHOGENESIS OF MACULAR HOLE

NORMAL MACULA

STAGE 1 - A FOVEAL DETACHMENT

STAGE 1 - B FOVEAL DETACHMENT

STAGE 2 EARLY HOLE, ECCENTRIC

STAGE 2 EARLY HOLE, CENTRAL

STAGE 3 HOLE WITH OPERCULUM

STAGE 3 HOLE WITHOUT OPERCULUM

STAGE 4 HOLE WITH POSTERIOR VITREOUS SEPARATION
A. TRAUMATIC THEORY

Traumatic theory was put forth by Knapp in 1869, when he published the first case description of a macular hole in a patient with ocular trauma and an initial diagnosis of a macular hemorrhage. Most other early observers attributed macular holes to ocular trauma.

Noyes published the first accurate case report and a detailed ophthalmoscopic description of macular hole, which was secondary to blunt trauma. He noted the difference in depth of focus from the retinal surface to the base of the lesion and probably was the first to recognize that the hallmark of the lesion was a full-thickness defect in retinal tissue within the center of the macula.

In 1900 Ogilvie compiled the first case series in holes at the macula, and proposed terminology including macular hole, as well as floor and edge of the macular hole. Many of the first reported cases of macular holes were in young patients, and trauma was estimated to account for as many as 50%.

The majority of traumatic macular holes occur in men, whereas it is now known that the majority of age related macular holes occur in women.
B. CYSTOID DEGENERATION THEORY

Full-thickness and lamellar macular holes were first described by Fuchs (1901) and Coats (1907).

Coats noted cystic intra-retinal changes adjacent to the macular hole and concluded that these changes could be caused by trauma as well as other mechanisms. In some cases of trauma in which there was not immediate macular hole formation, trauma was believed to cause reactive vasoconstriction followed by vasodilation, thus leading to cystic degeneration of the central macula.

Blunt ocular trauma could cause immediate macular hole formation from mechanical energy created by vitreous fluid waves and contrecoup macular necrosis or macular laceration. Indirect ocular trauma had also been reported to cause macular hole formation.

Recognizing that cystoid degeneration was not only due to posttraumatic macular sequelae, Kuhnt concluded that macular holes were caused by cystoid degeneration in the macula, not necessarily related to trauma.

Aaberg in 1970 found that only 9% of eyes with a macular hole were associated with trauma, compared with an earlier report of 50%. He described macular hole and cystoid oedema in association with a variety
of conditions, including severe hypertension, central retinal artery occlusion, retinal venous occlusive disease, Coats’ disease, syphilis, solar maculopathy, arc welding maculopathy, electrocution, and vitreous traction

C. VASCULAR THEORY

Although trauma was once believed to be the primary or sole cause of macular holes, it was probably the histopathologic and clinical observations of cystoid degeneration in the surrounding tissue of macular holes that led to considerations of atraumatic causes and, in particular, the vascular theory of pathogenesis. Coats and Kuhnt together believed that aging-related changes of the retinal vasculature led to cystoid degeneration and subsequent macular hole formation

D. VITREOUS THEORY

Avila and Jalkh, noted vitreoretinal traction arising from the vitreous base and concluded that persistent vitreous-to-macula adhesions were important in the pathogenesis of macular holes. Other proponents of a vitreous theory emphasized that the process of vitreous separation from the macula was the critical event in the pathogenesis of a macular hole. It is reported that macular holes can develop despite having a pre-existing complete posterior vitreous separation.
E. INVOLUTIONAL MACULAR THINNING

Morgan and Schatz proposed a mechanism that they described as involutional macular thinning, incorporating vitreous, vascular, and cystic degeneration theories. The macular lesion was described as “thin, mildly atrophic fovea that has lost its normal architecture and appearance.” The foveal lesion was a subtle abnormal depression with variably associated retinal cystic changes or a surrounding yellow ring.
CLASSIFICATION OF MACULAR HOLES

STAGE – 1 MACULAR HOLES:

(Pre-macular holes, macular cysts, Involutional macular thinning)

With a unilateral stage 1 macular hole, the patient typically is asymptomatic with both eyes open. For this reason and because they are evanescent lesions, stage 1 macular holes are not observed commonly and their diagnosis can be difficult. When symptoms are present, they consist of painless metamorphopsia or decreased vision or both.

In a stage 1 macular hole, no true neural retinal defect is present. The photoreceptor layer is believed to be intact, and no vitreo-foveal separation has occurred. Oblique vitreous traction on the fovea is
believed to be the inciting event and can typically be observed on OCT. Stage 1 holes are further divided into Stage 1a and Stage 1b, based on clinical appearance. In a stage 1a macular hole, a small central yellow spot is seen on ophthalmoscopy (Foveolar detachment). The fovea may be thickened along with a loss of the normal foveal contour. In a stage 1b macular hole, a yellow ring is visible in the foveal area (foveal detachment).

Foveal Pseudocyst

OCT studies conclude that a stage 1a macular hole actually represents a cystic change within the fovea, rather than a true photoreceptor detachment from the retinal pigment epithelium (foveal pseudo-cyst).
In a stage 1b macular hole, the cyst-like space is accompanied by a foveal detachment that coalesces to a point just short of actual dehiscence. Stage 1 holes spontaneously resolve in about 50% of eyes with no visual sequelae. The worse the initial visual acuity, the less likely is spontaneous resolution.
STAGE - 2: MACULAR HOLES:

When peri-foveal vitreous cortex shrinks, a stage 1 hole advances to a stage 2 hole. Stage 2 holes have a small (100–300 µm), full-thickness neural retinal defect, either centrally or eccentrically. The defect can be round, oval, crescentic, or horseshoe shaped. The visual acuity typically is diminished and a pseudo-operculum, which represents condensed vitreous, may overlie the hole. It is believed that once a stage 2 hole occurs, it nearly always progresses to stage 3, with little hope for spontaneous visual improvement. The visual acuity with a stage 2 hole varies between 20/50 (6/15) and 20/400 (6/120).
STAGE - 3 : MACULAR HOLES:

Stage 3 macular hole is the end result of continued vitreo-foveal traction on a stage 2 hole. At stage 3, the hole is developed fully and has the classic appearance of an idiopathic macular hole. This consists of a round, 350–600 µm full-thickness neural retinal defect with smooth edges, and a small, surrounding, doughnut-shaped rim of sub-retinal fluid. This fluid rarely progresses to cause a widespread retinal detachment. Yellow deposits can be seen in the base of the defect, and peri-foveal cystic retinal changes are present. With time, retinal pigment epithelial alterations may develop at the leading edge of the sub-retinal fluid cuff. The visual acuity typically is 20/200-20/800 (6/60-6/240); however, visual acuity as good as 20/40 (6/12) may be seen with a stage 3 hole, but rarely. Vitreo-foveal separation still has not occurred.
STAGE - 4: MACULAR HOLE:

A stage 4 macular hole has all the features of a stage 3 hole, but with complete posterior separation of the vitreous from the fovea.
STAGES OF MACULAR HOLE

Normal macula

Stage I

Stage II

Stage III

Stage IV
I. CLINICAL SYMPTOMS

Patients usually experience visual symptoms like metamorphopsia, loss of central vision and a central scotoma. The visual acuity of the patient varies with the size, duration and location and stage of the macular hole.

In patients with impending macular holes, central visual acuity may be reduced to the range of 20/25 to 20/50. In fully developed macular holes, central visual acuity may be reduced to the range of 20/80 to 20/200. The central visual acuity may be correlated to the size of neurosensory detachment surrounding the macular hole. Both the hole size and neurosensory detachment size correlates with the duration of symptoms.

Significant visual improvement following spontaneous closure of macular holes is very rare.
II. CLINICAL SIGNS:

1. Watzke Allen test:

With the use of a thin slit beam during bio-microscopy, an absolute scotoma can appear to the patient as a break in the beam when it is centered over large holes (Watzke allen sign). If centered over smaller holes, or over the surrounding neuro-sensory retinal detachment in larger holes, only narrowing or distortion of the beam is experienced by the patients.

2. Laser aiming beam test:

For detecting the absolute scotoma associated with smaller defects, 50 microns argon laser aiming beam is directed at the macular hole. Disappearance of the aiming beam is noted in patients with true full thickness macular hole, whereas in patients with epi-retinal membrane or pseudo-macular hole, the laser aiming beam does not disappear.

3. Macular micro perimetry:

Here patient is tested in a similar fashion as a static or kinetic perimetry. The absolute and relative scotomas are mapped directly on to the retinal surface. This technique has demonstrated that visual loss in
eyes with macular holes is due to absence of retinal function and reduction in function in the surrounding areas.

4. Flourescein angiography:

To rule out masquerading lesions as seen in cystoid macular oedema or exudative maculopathy, flourescein angiography may be helpful.
5. Optical coherence tomography in macular holes:

In **stage 1 macular holes** – peri-foveal separation of posterior hyaloid with focal vitreous attachment to the fovea. There can be presence of foveal detachment or an intraretinal space called **PSEUDOCYST**. There is no full thickness defect in the retina.

**Stage 2 macular holes** – spontaneous partial vitreo-foveal separation. Small full thickness retinal defect or rupture of the roof of the pseudocyst.

**Stage 3 macular holes** – full thickness hole with or without an overlying operculum. The size of the hole is less than 400 microns in diameter.
Stage 4 macular holes - full thickness hole with a complete posterior vitreous separation, the size of the hole being more than 400 microns in diameter.

PROGNOSIS:

Optical coherence tomography can also be used for the prognostication of macular hole.

Macular hole index:

The macular hole index is the ratio of the hole height to the basal diameter of the hole. The higher the macular hole index, the better the visual results. If the diameter of the macular hole is more it is a bad sign.

Diameter hole index and traction hole index and the minimum diameter of the macular hole are also used to prognosticate the results after macular hole. A minimum diameter of less than 311 microns and a traction hole index of more than 1.41 have been associated with a better prognosis.
**Hole form factor**

OCT can be used accurately in predicting the anatomical and functional closure for vitrectomy.

*Puliafito* was the pioneer in calculating the hole form factor. He considered the ratio between the overlying tissue dimensions and the hole base diameter to be of greater influence on the anatomical success rate than the basal diameter alone. 80% of patients with HFF greater than 0.9 had anatomical success. Anatomical success rate was less than 25% in patients with HFF less than 0.5. The association between HFF and anatomic success rate was found to be statistically significant.
EPIRETINAL MEMBRANES

(Macular pucker or cellophane maculopathy or preretinal vitreous membrane or epiretinal astrocytic membrane or surface wrinkling maculopathy)

Epiretinal membrane with pseudo hole

SYMPTOMS:

Decreased vision and metamorphopsia

SIGNS:

- *Retinal distortion and retinal oedema*

  The severity of symptoms is related to the area of macular involvement, degree of retinal surface irregularity, the thickness of the membrane and the presence or absence of macular oedema.
• **Presence of retinal striae, retinal vascular tortuosity, straightening, foveal ectopia**

There may be associated inner retinal haemorrhages, microaneurysms, retinal telangetasia and retinal oedema.

• **Full thickness macular hole**

ERM is present in about 30% of eyes with a true full thickness macular hole. ERM usually forms after the hole develops.

• **Fluorescein angiography shows the degree of retinal vascular tortuosity and the presence of vascular leakage.**

It also helps to rule out other causes like neo-vascular membrane, retinal vascular disease and cystoid macular oedema following cataract surgery.

• **OCT shows degree of retinal distortion and vitreous traction** and quantitatively the degree of macular thickening. It also helps in post operative monitoring of return of normal retinal architecture.
GRADES OF EPIRETINAL MEMBRANE:

Grade 1: Presence of a cellophane membrane

Wrinkling of inner retina

No edge or ERM elevation

Grade 2: The edge of ERM is elevated

Full thickness retinal distortion

Less than half of the ERM is opaque

Grade 3: Thick membrane with more than half of ERM being opaque

Marked distortion of the retinal architecture

Obstruction of the underlying retinal architecture
Differential Diagnosis

1. Cystoid Macular Edema:

In case of cystoid macular oedema, rupture of a large cyst results in macular hole formation. But there is significant leakage in fluorescein angiography.

![Image of fluorescein angiography]

2. EpiRetinal Membrane with Pseudo Hole:

A central opening in the epiretinal membrane may mimic a retinal defect also called pseudo macular hole but there is no surrounding neurosensory detachment. The membrane may have a fibrotic appearance with distortion of perifoveal capillaries that may leak in angiography.
3. EXUDATIVE MACULOPATHY:

Exudative maculopathy such as central serous retinopathy and age related macular degeneration may mimic a macular hole. Fluorescein angiography and OCT can be used to differentiate this condition from a full thickness macular hole.
4. LAMELLAR MACULAR HOLE:

Some form of aborted macular hole, OCT will demonstrate the absence of full thickness defect, possibly with vitreofoveal separation and a pseudo operculum or epiretinal membrane.

5. TRAUMATIC MACULAR HOLE:

In cases of trauma, macular hole may be associated with subhyaloid hemorrhage and blood in vitreous cavity.
TREATMENT MODALITIES

INDICATIONS FOR TREATMENT:

1. Stage 3 and 4 macular holes

2. Definite symptomatic full thickness macular holes with visual acuity in the range of 20/40 to 20/60

3. Epiretinal membrane causing trampoline traction, foveal ectopia, retinal vascular leakage and macular oedema

4. Epiretinal membranes causing diplopia or debilitating metamorphopsia or severe visual decline

GENERAL SURGICAL PRINCIPLES

- Surgery performed using three port pars plana vitrectomy, including a separate continuous infusion cannula, endoilluminator, suction cannula and other instruments.

- After removal of the central vitreous, the posterior cortical vitreous is identified and separated from the retinal surface.
• When the suction is applied to the silicone tipped cannula, the orifice becomes occluded by the vitreous as the cannula is close to the retinal surface called “fish strike sign”

• If posterior vitreous detachment is not present, create one by using active suction close to the optic disc in a posteroanterior direction

• Vitrectomy has to be completed. Thorough vitrectomy can be verified using intravitreal triamcinolone.

• Epiretinal membrane and ILM PEELING can be done at this stage

• This procedure is done using a small gauge retinal pick or a barbed myringotomy blade.

• The ILM is removed in a circular fashion called maculorhexis

• Staining of the ILM and epiretinal membrane can be done using dyes like ICG and brilliant blue in an air/fluid filled eye for easy identification.
Staining of ILM

- ICG stains only the acellular ILM but *does not stain the epiretinal membrane*. So preferential staining can be done. But this dye is toxic to the retina. Hence not preferred

- *Trypan blue and triamcinolone acetonide* are alternatives to ICG.

Pre and post operative OCT images of macular hole
SURGICAL ADJUNCTIVES

- **Intravitreal transforming growth factor**

  It was reported to have dose dependent efficacy and higher anatomic success rate in eyes undergoing macular hole surgery.

- **Autologous blood serum**

  Blood serum and concentrated platelets are said to have higher anatomic success rate in eyes with idiopathic macular hole.

- **Adjunctive laser photocoagulation**

  Laser photocoagulation directed only to the RPE in the base of macular hole along with fluid gas exchange has been suggested as a treatment to persistent macular hole following vitrectomy.

- **Pharmacologic PVD:**

  **OCRIPLASMIN** is a newer drug that has been used to create pharmacologic PVD. It has been suggested as a treatment for macular hole.
• **Tamponade and post op positioning:**

Intravitreal gas tamponade and post op face down positioning are necessary. Previously non expansile concentrations of SF6 were used but required post op face down positioning. In order to reduce the dependence of surgical outcomes on prone face positioning, silicone oil is used.

**Complications of surgery**

1. Iatrogenic retinal breaks
2. Intraoperative light toxicity
3. Post operative exudative detachments
4. Subretinal fibrosis
5. Epiretinal membrane formation
AIM OF THE STUDY

• To evaluate the incidence, prevalence and correlation of epiretinal membranes surrounding idiopathic macular holes (stage 2/3/4)

• To find out the correlation between epiretinal membrane and visual acuity in idiopathic macular holes

• To find the correlation between size of the macular holes and epiretinal membrane.

• To assess the increase in severity of epiretinal membranes during follow up.
INCLUSION CRITERIA

• All patients with symptoms of decreased central vision and metamorphopsia.

• All patients presenting with complaints of central scotoma and distortion of images.

• All patients with clinically proven idiopathic macular hole.

• All patients with OCT proven idiopathic macular hole.

EXCLUSION CRITERIA

• All patients who have previous history of trauma and an identified aetiology of macular hole to be trauma.

• Other coexisting causes for macular hole like cystoid macular edema and lamellar macular hole and pseudohole.
MATERIALS AND METHODS

The study was conducted at the Regional institute of ophthalmology and Govt ophthalmic hospital, Chennai between Sep.2012 - Oct 2013.

Retrospective study design was followed.

50 eyes of 50 patients were included in the study. The patients underwent a detailed history including their past and present illness, relevant ocular history including any surgery, trauma, usage of any topical medications for any ocular complaints etc.

Uncorrected visual acuity was assessed using Snellens visual acuity chart and best corrected visual acuity was assessed using Autorefractometer. Anterior segment including the anterior vitreous face was examined using slit lamp examination techniques. Fundus examination was performed using direct and indirect ophthalmoscopes. Slit lamp Biomicroscopy using 90 D lenses was used to get a
stereoscopic view of the fundus. The findings were recorded using colour fundus photography.

**Optical coherence tomography** was then performed in all patients with the above findings. These patients were followed up and reviewed at three months, six months and one year and the same tests were repeated and observations recorded. Results were analysed using standard statistical methods and conclusions derived.
OBSERVATIONS AND ANALYSIS

The study was done among 50 patients with idiopathic macular hole (n=50)

30 patients included in this study were phakic and 20 were pseudophakic.
GENDER DISTRIBUTION

In our study, 27 females and 23 males had idiopathic macular hole.
## GRADING OF MACULAR HOLES

<table>
<thead>
<tr>
<th>MACULAR HOLE</th>
<th>GRADE 2</th>
<th>GRADE 3</th>
<th>GRADE 4</th>
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<tbody>
<tr>
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<td>23</td>
<td>8</td>
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</tbody>
</table>

Number of patients with stage 2 macular holes -- 19

Number of patients with stage 3 macular holes -- 23

Number of patients with stage 4 macular holes -- 8
GRADING OF MACULAR HOLES

- stage 2: 16%
- stage 3: 38%
- stage 4: 46%
ANALYSIS OF VARIOUS GRADES OF MACULAR HOLE

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<td>3/60 NIP to 4/60 NIP</td>
<td>1/60 NIP to 2/60 NIP</td>
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ANALYSIS OF GROUP 1 (STAGE 2 MACULAR HOLES)

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<td>79</td>
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<tr>
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</tbody>
</table>

- Number of patients with stage 2 macular holes – 19 (based on OCT and clinical findings)

- Visual acuity of patients in this group ranging from **3/60 to 6/36** snellens visual acuity

- Mean visual acuity in this group was between **5/60 to 6/60 NIP**

- During **follow up the mean visual acuity is 5/60 NIP**

- The presence of epiretinal membranes was noted in **4 patients** with stage 2 macular holes.
• Percentage of stage – 2 macular holes with ERM
• Amongst those patients who had epiretinal membrane 2 were pseudophakic and 2 were phakic.
## ANALYSIS OF GROUP 2 (STAGE 3 MACULAR HOLES)

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<td>43</td>
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</table>
• Number of patients with stage 3 macular holes – 23

• Visual acuity at presentation in this group ranged from 1/60 to 5/60 NIP

• The mean visual acuity in this group was between 3/60 to 4/60 NIP

• The mean visual acuity during follow up in this group at the end of one year is 4/60 NIP

• The presence of EPIRETINAL MEMBRANES is noted in 13 patients
• Percentage of Stage – 3 macular holes with ERM.
• Amongst those who had EPIRETINAL MEMBRANES, 7 were pseudophakic and 6 were phakic patients

• During follow up, 9 patients with stage 3 macular holes showed increase in severity of EPIRETINAL MEMBRANES.
ANALYSIS OF GROUP 3 (FULL THICKNESS MACULAR HOLES)

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</table>

- The number of patients with stage 4 macular holes were – 8
- The visual acuity at presentation in this group is between 2/60 and 1/60 NIP
- The mean visual acuity during follow up in this group is 1/60 NIP
- The presence of EPIRETINAL MEMBRANES is noted in 6 patients with stage 4 macular holes, amounting to about 75% of patients with stage 4 macular holes are associated with epiretinal membranes.
• Percentage of Stage – 4 macular holes with ERM.
All the 6 patients in this ERM group were found to be pseudophakics in our study.
Among the 20 pseudophakics, 14 patients (70%) had epiretinal membrane.
Among the 30 phakics, 13 patients (43 %) had epiretinal membrane
## VISUAL ACUITY VS ERM

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<th>≤ 1/60</th>
<th>2/60</th>
<th>3/60</th>
<th>4/60</th>
<th>5/60</th>
<th>6/60</th>
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<td>7</td>
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<tr>
<td>ERM (n in %)</td>
<td>85</td>
<td>66</td>
<td>72</td>
<td>66</td>
<td>16</td>
<td>50</td>
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</tbody>
</table>
Number of patients in each group according to visual acuity and the incidence of epiretinal membrane.
INCREASE IN SEVERITY OF EPIRETINAL MEMBRANES DURING FOLLOW UP

- 16 patients showed an increase in severity of epiretinal membranes during follow up out of the 27.
- % of patients who showed an increase in severity of epiretinal membranes during follow up = 60%
RESULTS

• In this study, out of the 50 eyes of 50 patients, 30 were phakics and 20 were pseudophakics.

• In our study, there was a slight female preponderance noted - 27 females as against 23 males with a male to female ratio of 1:1.7.

• 38% of patients had stage 2 macular holes. 46% of patients had stage 3 macular holes and 16% of patients had stage 4 macular holes.

• Stage 4 holes had a poorer visual acuity compared to stage 2 and stage 3 macular holes.

• Epiretinal membranes were found associated with all three stages of macular holes. As the stage of macular hole increases, the incidence of ERM also increases. 26% of stage 2 macular holes, 70% of stage 3 and 75% of stage 4 macular holes were found to have epiretinal membranes.

• 60% of patients showed an increase in severity of epiretinal membranes during follow up.

• The mean visual acuity in patients with stage 2 macular holes was ...... with a range of 3/60 to 6/36.
• The mean visual acuity in the stage 3 macular holes was ....... with a range of 1/60 to 5/60 NIP.
• The mean visual acuity in stage 4 macular holes was .... with a range 2/60 NIP and less.
• During follow up, 9 eyes out of 17 showed a drop in visual acuity in stage 2 macular holes (53%)
• 7 eyes out of 23 showed a drop in visual acuity in stage 3 macular holes (30%)
• 2 eyes out of eight eyes showed a drop in visual acuity further in stage 4 macular holes.
DISCUSSION

Idiopathic macular holes are common in the older age group. They usually present with defective central vision and metamorphopsia. It constitutes a fraction of ocular morbidity in the elderly.

Macular holes can occur in younger age group, though the causes may be different like trauma, cystoid macular oedema or even a pseudohole secondary to vasculitis or retinal degenerations.

Macular holes are significant because they cause gross loss of central vision. The prevalence is more in females as shown in our study. It is also more among pseudophakics than phakics. Visual acuity is measured with snellens chart in our study and the visual acuity is very less in stage 4 than stage 2 macular hole.

Optical coherence tomography is a very important tool in assessing the stage of macular hole, presence and absence of epiretinal membranes associated, hole size and also prognosticating post surgery. It also plays a key role in assessing whether the macular hole surgery is successful or not.

Epiretinal membranes are commonly associated with macular holes. The incidence of epiretinal membranes increases with the stage of
macular hole. The severity of these epiretinal membranes also increases during follow up in a significant number of patients eyes. Though commonly associated with macular holes, a significant correlation has not yet been elucidated between epiretinal membranes and idiopathic macular holes.

In our study, we have attempted to find out the associations of epiretinal membranes and various stages of macular holes. Epiretinal membranes are a cause of tangential traction that plays a role in the pathophysiology of macular holes. We established that epiretinal membranes are associated with all stages of macular holes and majority show an increase in severity of macular holes during follow up. The incidence of epiretinal membranes is also more among pseudophakics than phakics who were enrolled in this study.

Stanley p azen, University of Atlanta, Florida in his journal publication in 2000 states that no significant correlation exists between epiretinal membranes and idiopathic macular hole with respect to visual acuity.

With greater advancements in instrumentation and surgical techniques, larger leaps have been made in macular hole surgery and good results are obtained.
CONCLUSION

Idiopathic macular holes are common in the elderly. It is an important cause of loss of central vision. This study shows that epiretinal membranes are commonly associated with full thickness macular holes. The prevalence of epiretinal membrane increases with the severity and size of macular hole. With the advent of OCT, and finer instrumentation greater advances have been made in macular hole surgery. Though the prevalence of epiretinal membrane is common in idiopathic macular hole, there is no significant correlation between epiretinal membranes in macular hole and surgery. These factors may be considered during vitrectomy for macular hole.
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<th>BCVA</th>
<th>ERM</th>
<th>FOLLOW UP(3M,6M,1YR)</th>
<th>V/A FOLLOW UP</th>
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<p>| 25  | MEERA    | 64/F   | 4     | PSEUDOPHAKIC | 1/60   | 1/60     | ++       | ++       | _        | 1/60     |
| 26  | RAJAM    | 69/F   | 4     | PSEUDOPHAKIC | 2/60   | 2/60     | +        | ++       | _        | 1/60     |
| 27  | KUPPU    | 72/F   | 4     | PHAKIC     | 2/60     | 2/60     | _        | _        | _        | 2/60     |
| 28  | SARASU   | 70/F   | 3     | PHAKIC     | 3/60     | 3/60     | +        | ++       | _        | 3/60     |
| 29  | SUNDARI  | 63/F   | 4     | PSEUDOPHAKIC | 1/60   | 1/60     | +        | ++       | _        | 1/60     |
| 30  | SAKUNTA  | 64/F   | 3     | PHAKIC     | 2/60     | 2/60     | _        | _        | _        | 2/60     |
| 31  | SEKAR    | 59/M   | 2     | PHAKIC     | 6/60     | 6/60     | _        | _        | _        | 5/60     |
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<td>2/60 WITH + 1DS 3/60</td>
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“PREVALENCE, CORRELATION AND NATURAL HISTORY OF EPIRETINAL MEMBRANES SURROUNDING IDIOPATHIC MACULAR HOLES”
GUIDE

PROF. DR. V. REVATHY
head of the department of UVEA AND RETINA SERVICES

CO GUIDES

PROF. DR. B. CHANDRASEKARAN
professor of ophthalmology,
RIO GOH
CO GUIDE

DR.G. BALAJI,
assistant professor,
RIO GOH
SYNOPSIS

- **TITLE** – “prevalence, correlation and estimates of epiretinal membranes surrounding idiopathic macular holes“
- **Principal investigator** – DR. M. ROSHNI
- **GUIDE** – PROF. DR. V. REVATHI, head of the department UVEA AND RETINA SERVICES
- **STUDY CENTER** – regional institute of ophthalmology, egmore, chennai
Duration – 1 YEAR

AIM – to evaluate the prevalence, correlation and natural history of EPIRETINAL MEMBRANES SURROUNDING IDIOPATHIC MACULAR HOLES

PRIMARY OBJECTIVES- 1. to evaluate the prevalence of epiretinal membranes in eyes with stage 2/3/4 macular holes.
2. the correlation between epiretinal membrane and visual acuity in idiopathic macular holes
3. the correlation between size of macular hole and epiretinal membrane
Secondary objective - To assess the increase in severity of epiretinal membrane in follow up.

Study design - Prospective study, observational study

Methodology{material and methods } - All patients with idiopathic macular hole stage 2, 3, 4 who attended the OPD

Inclusion criteria - All patients with symptoms of decreased central vision and metamorphopsia and clinically proven macular hole stage 2, 3 and 4

Exclusion criteria - Patients who had traumatic macular hole and macular hole due to other causes and macular edema.
Sample size- 50 patients
Age - 30 to 80 years
Gender – both genders

- **Study parameters**
  - Detailed history including past and present illness
  - Visual acuity assessment using Snellens visual acuity chart
  - Anterior segment examination using slit lamp including lens and anterior vitreous.
  - Fundus examination using direct and indirect ophthalmoscopic examination.
  - Stereoscopic fundus examination using slit lamp biomicroscopy
  - Colour fundus photography
  - Optical coherence tomography.
Data collection and methods-All cases diagnosed as idiopathic macular holes stage 2,3 and 4 will be registered and evaluated subsequently at the end of 3,6 and 12 months of follow up.

Analysis plan- to consider the importance of factors in removal of epiretinal membrane during vitrectomy for macular hole