OCT Findings in High Myopia

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Pathological High Myopia

- Axial length exceeding 26.50 mm.
- Refractive error more than -6 diopters.
- Characteristic pathological changes: tessellated fundus, lacquer cracks, patchy or diffuse atrophy, CNV, posterior staphyloma, straightened and stretched vessels, temporal peripapillary atrophic crescent, hemorrhages and tilting of the optic disc.
Prevalence

- Pathological myopia is a major cause of irreversible vision loss and is the fourth to ninth most frequent cause of blindness.
- The prevalence is high in Asian populations and lower in African groups, with most countries having a prevalence of about 1-4%.

Curtin in 1977 classified posterior staphyloma into 10 types
Avila et al. in 1984 developed a classification of myopic maculopathy

- **M0**, normal-appearing posterior pole;
- **M1**, choroidal pallor and tessellation (the choroidal vessels can be seen through the retina owing to reduced RPE pigmentation);
- **M2**, choroidal pallor and tessellation with posterior staphyloma;
- **M3**, choroidal pallor and tessellation with posterior staphyloma and lacquer cracks;
- **M4**, choroidal pallor and tessellation with lacquer cracks, posterior staphyloma, and focal areas of deep choroidal atrophy;
- **M5**, large geographic areas of deep chorioretinal atrophy and ‘bare’ sclera.

Because of the characteristic features of the retina in degenerative myopia (i.e., tigroid fundus, thin retina, areas of choriocapillaris atrophy, retinal pigment epithelium hypopigmentation and/or hyperpigmentation, posterior staphyloma, etc), the early stages of traction maculopathy can be easily underestimated by biomicroscopy, angiography, or ultrasonography, and consequently, its presence can remain undiagnosed.

OCT greatly facilitates the study of the posterior vitreoretinal anatomy in eyes with high myopia and allows the detection of subtle macular changes that are otherwise undetectable.
**OCT**

- Dehiscence of retinal layers-retinoschisis.
- Paravascular inner retinal cysts and lamellar holes.
- Peripapillary intrachoroidal cavitation.
- ERM.
- Tractional internal limiting membrane detachment.
- Macular holes (lamellar and full thickness).
- Dome-shaped macula.
- Posterior neurosensory detachment.
- Choroidal neovascular membranes.

In 1999, Takano and Kishi identified foveal retinoschisis and retinal detachment without retinal holes in severely myopic eyes by OCT. In 2004, Panozzo and Mercanti first used the term 'myopic traction maculopathy' MTM to refer to these pathologies of the posterior pole in high myopia, such as macular retinoschisis, shallow retinal detachment without retinal holes, lamellar macular holes, and macular holes with or without retinal detachment.
In 2007, Panozzo suggested that the term 'retinoschisis' used in the literature for high myopic maculopathy was not appropriate as the term schisis suggested a complete separation between the retinal layers that resulted in the irreversible and total loss of retinal function. This retinal damage is not a schisis but rather is a retinal swelling.

Smiddy in 2009 supported this notion and suggested that myopic maculoschisis and vitreoschisis should all fall under the family of MTM.
Myopic foveoschisis may remain stable for many years without affecting vision (Gaucher et al., 2007), however it can result in metamorphopsia and blurring of vision (Timothy, 2007), vision worsening increases when there are associated premacular structures such as epiretinal membranes or a partially detached vitreous cortex (Johnny et al., 2010).

Genetic Factors of Pathological Myopia

- High myopia most commonly appears as a complex disease caused by a combination of genetic and environmental factors working together.
- However, it sometimes presents as one of the features in a wide variety of genetic disorders. Genes responsible for these syndromic genetic disorders with myopia as a consistent clinical finding have been identified: collagen 2A1 and 11A1 for Stickler syndromes type 1 and 2, lysyl-protocollagen hydroxylase for type 4 Ehler-Danlos syndrome, and fibrillin for Marfan syndrome.

Each of these genes is expressed in the sclera.
The pathogenesis of MTM

1) Thickening and stiffening of the internal limiting membrane (ILM), which can prevent the retina from adapting to high myopia posterior scleral staphyloma that produce centripetal traction and tangential traction to the retina.

2) choroidal atrophy, which can lead to the shortage in blood supply in the retina that reduces adhesion between the retinal layers.

3) the retinal pigment epithelium and choroid atrophy, thus reducing the adhesion between the retinal pigment epithelium and neuroretina.

4) Incomplete posterior vitreous detachment or adhesion between the remaining vitreous and the retina, which can produce centripetal traction and tangential traction to the retina.

5) One subretinal factor is the formation of posterior scleral staphyloma from the extension of the axis of the eye, which produces centrifugal traction on the retina.
The pathogenesis of MTM

6) Panozzo and Mercanti reported that women constituted 87.5% of MTM patients, suggesting that MTM may be linked to hormonal differences. Others have shown that high myopia is a polygenic inherited disease. Seven pairs of related genes have been identified, with six located on autosomal chromosomes and one on the X chromosome (Xq28).

Evolution and Progression

Macular retinoschisis and lamellar macular holes occur in the early stages of MTM, while high myopic macular holes and sensory detachment occur in the advanced stages of MTM. Myopic macular retinoschisis is often stable for many years without progression, but it can progress to retinal detachment or macular hole. Development of a foveal retinal detachment in myopic macular retinoschisis impairs vision more severely. RD is an intermediate-stage change in development of FTMH in some MRS cases.
OUR STUDY

The study involved 100 eyes of 100 patients with high myopia complaining of recent diminution of vision. Inclusion criteria were a minimum spherical equivalent (SE) of -6.0 diopters (D) and recent complaint of diminution of vision. OCT examination was done to detect macular abnormalities.

RESULTS

There were 40 males and 60 females and 60% of the studied eyes were the right eyes. The mean age of the patients was 52.9±12.9 (range, 33 to 70) years. Mean SE in these eyes was -14.5±5.8 (range, -8.75 to -22.75) D.

All eyes had one or more chorioretinal features typical of degenerative myopia (tigroid fundus, stretched vascular arcades, peripapillary atrophy, chorioretinal atrophy and lacquer cracks).
RESULTS

- ERM was present in 65 eyes.
- Vitreomacular traction (Anteroposterior traction) was detected in 10 eyes.
- Lamellar macular hole was present in 10 eyes.
- Full thickness macular hole was present in 4 eyes.
- Macular retinoschisis was present in 33 eyes.
- Posterior neurosensory detachment was present in 2 eyes.
- ILM detachment was present in 7 eyes.
- Myopic CNV was present in 15 eyes.
- Atrophy of retinal layers was detected in 10 eyes.

Normal
ERM-Foveal sensory detachment

Foveal schisis
Chorioretinal atrophy

Traction Myopic Maculopathy
Differentiation of ILM detachment from epiretinal membrane is based on the presence of columns that bridge the membrane to the retinal surface. It seems that tractional myopic maculopathy described by Panozzo in 2004 is a variation of longstanding tractional ILM detachment with loss of column bridges due to extreme traction and retinal atrophy. SD-OCT image showed multiple columnar structures within the retinoschisis space and also in the split spaces beneath the ILM detachment.
ILM detachment in the superior and/or inferior peripheral macula is a sign of persistent strong traction that can cause a foveal retinal detachment, which is likely transmitted to the outer retina through the dense columnar structures in the retinoschisis at each retinal level.

Foveal schisis with Lamellar Macular Hole
Lamellar Macular Hole
Full Thickness Macular Hole

Natural evolution from macular retinoschisis to full-thickness macular hole in highly myopic eyes

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dome-shaped macula

first described by Gaucher et al as an unexpected finding in myopic staphyloma and was characterized as an inward convexity of the macula that occurred in highly myopic eyes within the concavity of a posterior staphyloma.

Imamura et al suggested that the dome-shaped macula is the result of relatively localized variations in the thickness of the sclera beneath the macula.
It is interesting to note that, although extrafoveal retinal schisis was present, foveoschisis was absent. The bulge in eyes with a dome-shaped macula may act as a macular buckle-like mechanism.
Choroidal neovascularization (CNV) secondary to pathological myopia is the leading cause of vision impairment in patients younger than 50 years.

They are located in the subretinal space, as opposed to the sub-RPE space in AMD. Subretinal or sub-RPE fluid, hemorrhage or exudates are uncommon.
Myopic CNV

Eyes in the scar or atrophic stage have a higher risk of developing a macular hole.
Conclusion

In summary, in this large case series, we showed that retinal abnormalities in the macular area can be a frequent finding in eyes with degenerative myopia. Epiretinal traction probably plays a major role in this condition. Optical coherence tomography can greatly contribute in the examination of eyes with high myopia.