Retinal Complications of High Myopia

Dr. Timothy YY Lai

MBBS, MMedSc, MRCSEd, FCOphthHK, FHKAM(Ophthalmology)

Associate Professor, Department of Ophthalmology & Visual Sciences, The Chinese University of Hong Kong

Dr. Timothy YY Lai

Introduction

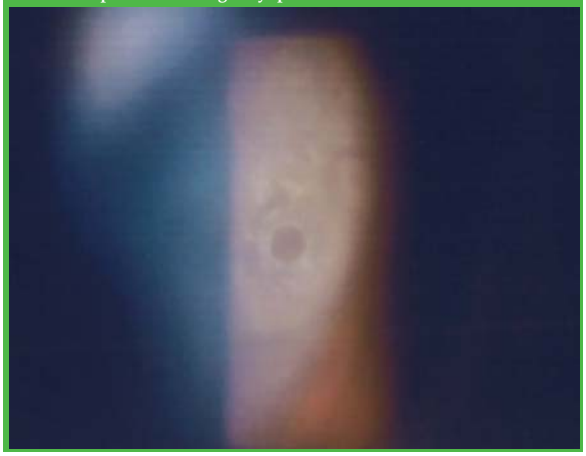
High myopia or pathological myopia is associated with globe elongation and a refractive error of at least 6 diopters (D) and/or axial length of greater than 25.5 mm.[1-3] The prevalence of high myopia varies considerably in different ethnic groups and has been estimated to be around 10% in Asian populations.[1,2] Excessive axial elongation of the globe in high myopia can cause mechanical stretching and thinning of the choroid and retinal pigment epithelium layers, resulting in various retinal degenerative changes.[4] It is well known that individuals with high myopia have increased risks of retinal complications such as peripheral retinal degenerations, retinal tears, retinal detachment, posterior staphyloma, chorioretinal atrophy, retinal pigment epithelial atrophy, lacquer cracks, choroidal neovascularisation (CNV) and macular haemorrhage.[4-6] In a cross-sectional community-based epidemiological study in Hong Kong, 56.1% and 11.3% of subjects with high myopia were found to have one or more peripheral retinal degenerative lesion or posterior pole lesion respectively.[7] Some of these retinal lesions may be associated with severe irreversible visual loss and therefore it is important for clinicians to be aware of the retinal pathologies in high myopia. This review aims to provide an overview on some of the important retinal complications associated with high myopia.

Peripheral retinal degenerations and rhegmatogenous retinal detachment

Epidemiological studies have demonstrated increased prevalence of peripheral retinal degenerations in association with high myopia and increased axial length.[4-13] Among the different types of peripheral retinal degenerations in high myopia, lattice degeneration is the most important peripheral retinal degeneration which can predispose to rhegmatogenous retinal detachment (RRD).[14] This is because retinal tears can develop at the posterior and lateral margins of the lattice degeneration caused by strong vitreoretinal adhesions following posterior vitreous detachment. Symptoms of posterior vitreous detachment and retinal break formation include sudden or gradual increase in the number of floaters and/or flashes. In patients with RRD, they may also develop symptoms of curtain-like progressive visual field loss and blurring of vision. Dilated fundus examination should be carried out in patients with these symptoms as soon as possible to detect for the development of retinal break or retinal detachment.

Laser photocoagulation is used for the treatment of eyes which have developed retinal hole or break. This can be performed in the majority of patients under topical anaesthesia as an out-patient procedure. Several rows of laser are applied onto the retina to surround the retinal defect in order to seal off the retinal break (Fig. 1). Since around 30% of eyes with acute RRD have been found to have lattice degeneration, prophylactic laser treatment can also be performed in patients with peripheral retinal degenerations,[15] especially those with a history of retinal detachment in the fellow eye.

Figure 1. Retinal hole surrounded by fresh laser photocoagulation marks in a patient with high myopia



In eyes with retinal detachment, laser photocoagulation alone is insufficient to treat the condition and vitreoretinal surgery is required. Surgical modalities for RRD include pneumatic retinopexy, scleral buckling surgery with cryopexy, and pars plana vitrectomy with intravitreal tamponade such as gas or silicon oil. The goal of the surgery is to identify and seal off all retinal breaks. For patients in whom the macula is still attached, they will generally have favourable visual outcome postoperatively. However, for patients in which the central of the macula i.e. the fovea is detached, the visual prognosis of the patient is more variable and some patients might develop irreversible visual loss despite successful retinal detachment surgery. Therefore, prompt ophthalmic consultation is advised for early detection of retinal detachment in order to prevent irreversible visual loss.

Myopic foveoschisis and macular hole

Due to excessive axial elongation of the globe in high myopia, patients can develop posterior bulging or ectasia

of the globe known as posterior staphyloma. Recent advancement in retinal imaging technology using optical coherence tomography (OCT) has demonstrated that highly myopic patients with posterior staphyloma are predisposed to develop macular pathologies such as myopic foveoschisis and macular hole. Myopic foveoschisis is the splitting of the retinal layers in the macula and can result in metamorphopsia and blurring of vision.[16] Macular surgery may be performed in myopic foveoschisis to prevent further deterioration of vision.[17,18] In more advanced stage, myopic macular hole can develop which may be associated with retinal detachment and patients will suffer from severe visual loss with reduced visual acuity. Various surgical procedures have been performed for macular hole with or without retinal detachment and they include pars plana vitrectomy with gas or silicone oil tamponade, macular buckling, and scleral shortening surgeries.[19-21] However, despite these interventions, reopening of the macular hole and retinal redetachment may still develop and some patients will require multiple surgeries to achieve attachment due to the loss of chorioretinal tissue and retinal pigment epithelial atrophy.

Lacquer cracks

Lacquer cracks are formed by spontaneous ruptures in the Bruch's membrane and small haemorrhages may develop within the lacquer cracks. Lacquer cracks predispose patients with high myopia to have sudden visual loss as macular CNV may develop in close proximity to the lacquer cracks. Small ingrowth of fibrovascular tissue may also give rise to small elevated pigmented circular lesions and are known as Fuchs' spots.[22]

Choroidal neovascularisation in high myopia

Among various lesions associated with high myopia, macular CNV is one of the most vision threatening complications(Fig. 2).[23] It develops in around 5 to 10% of eyes with high myopia and is the commonest cause of CNV in young individuals and accounts for around 60% of CNV in young patients aged 50 years or younger.[24-26] The incidence of myopic CNV in patients with pre-existing myopic CNV in the fellow eye is even higher, as more than 30% of patients will develop CNV in the second eye within eight years after the first eye.[26]

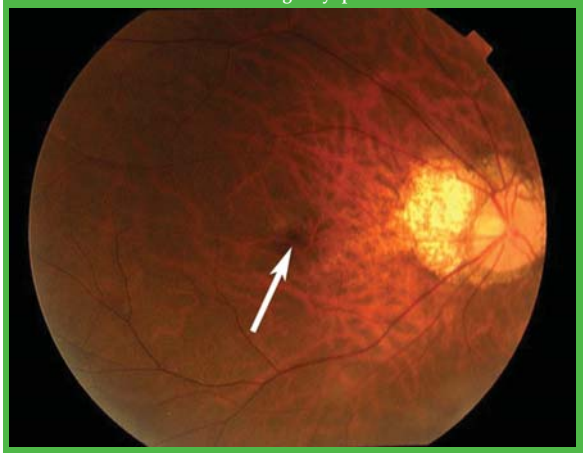
Patients with new onset myopic CNV may develop metamorphopsia, central or paracentral scotoma and reduction in visual acuity. On clinical examination, myopic CNV appears as a flat, small, greyish subretinal membrane beneath or in close proximity to the fovea. Fluorescein angiography (FA) is used to document fluorescein leakage in the CNV and to assess the location of the CNV for treatment planning.

The natural history of myopic CNV is generally poor and a large proportion of patients may have visual acuity of 20/200 or less after five years.[27,28] Poor prognostic factors for patients with myopic CNV include age of greater than 40 years, larger CNV, and worse initial visual acuity.[29,30] Based on studies on the natural history of myopic CNV, active interventions should be considered to avoid gradual visual deterioration. This is particularly important for patients with poor prognostic factors like older age of onset, larger CNV and worse visual acuity at initial presentation.

Direct thermal laser photocoagulation of myopic CNV has been attempted for treatment but this will lead to considerable visual loss due to expansion of the laser scar in the long term and therefore thermal laser treatment is no longer performed for myopic CNV. Other treatment modalities such as submacular surgery and macular translocation surgery for myopic CNV have also been performed with some success but the procedures are technically demanding and are potentially associated with high CNV recurrence rate.[31,32] The most commonly used method in the treatment of myopic CNV currently is photodynamic therapy (PDT) with verteporfin. It is a two-steps procedure involving infusion and activation of a photosensitising drug. The selectivity and efficacy of PDT on the abnormal CNV are caused by differential clearance of the photosensitising drug within the blood stream and preferential binding to low-density lipoprotein receptors on CNV endothelial cells.[33] Studies have shown that PDT with verteporfin can result in stabilisation of vision following treatment.[34-36] However, only around 20-30% of patients will have improvement in vision after PDT with verteporfin. Combined PDT with intravitreal triamcinolone acetonide has also been attempted to further improve the outcome of PDT for myopic CNV but no significant difference was observed compared with eyes which had PDT monotherapy.[37]

More recently, the use of angiogenesis therapy with anti-vascular endothelial growth factor (VEGF) agents like intravitreal bevacizumab has demonstrated encouraging results in the treatment of myopic CNV as patients had visual gain after treatment.[38,39] In a recent study by Chan et al,[38] three monthly injections of bevacizumab resulted in a mean improvement of 2.6 lines at 6 months with 68% of patients having visual improvement of two or more lines. With the increasing availability of other anti-VEGF agents like ranibizumab, targeted angiogenesis therapy will play an increasing role in the management of myopic CNV and may become the treatment of choice for myopic CNV in the near future.

Figure 2. Macular haemorrhage (white arrow) associated with choroidal neovascularisation in high myopia



Conclusions

Individuals with high myopia are subject to various retinal pathologies including peripheral retinal degenerations, retinal detachment, and posterior pole chorioretinal lesions. Since these retinal pathologies might be associated with serious sight-threatening complications, patients with high myopia should be educated about the symptoms of retinal complications such as retinal detachment, macular hole, and myopic CNV. Patients should be advised to seek medical care promptly should such symptoms arise. Prompt referral to ophthalmologists will be useful in preventing severe visual loss as effective surgical and medical treatments are available for these retinal complications especially in the early stages.

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