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Pathologic Myopia

Pathologic myopia, a subset of high myopia with degenerative changes, is a leading cause of legal blindness worldwide.[1] The prevalence of myopic retinopathy ranges from 1.2% in the Blue Mountains Eye Study in Australia to 3.1% in the Beijing Eye Study in China.[2,3] High myopia, defined as -6.0 diopters or less, is projected to nearly quadruple in prevalence worldwide from 2.7% in 2000 to 9.8% in 2050.[4] As such, the prevalence of pathologic myopia is also expected to increase.

Age, increased axial length, and higher minus spherical equivalent have all been associated with increased risk for development of pathologic myopia.[5] Although genetic factors may play a role in development of high myopia, its role in myopic maculopathy is unclear.[6]

The pathogenesis of pathologic myopia is related to abnormal axial elongation and development of a posterior staphyloma. A posterior staphyloma is defined as an outpouching of all layers of the posterior globe and is pathognomonic for pathologic myopia. Posterior staphylomas are found in up to 50% of patients with high myopia and are more common in older individuals.[7,8] Development of posterior staphyloma is associated with significantly worse vision and higher likelihood of myopic maculopathy.[9]

Common manifestations of myopic maculopathy include chorioretinal atrophy, lacquer cracks, myopic choroidal neovascularization and myopic traction maculopathy.

Chorioretinal atrophy in myopic maculopathy presents as two types: diffuse and patchy (Figure 1).[10] Diffuse chorioretinal atrophy is ill-defined on fundus exam; large choroidal vessels are more visible due to thinning of most of the choroid. Patchy chorioretinal atrophy is well-defined and may develop within diffuse atrophy. On OCT, in areas of patchy chorioretinal atrophy, there is significant thinning of all choroid and RPE, and the inner retina appears to directly overlie the sclera. There is currently no treatment for chorioretinal atrophy secondary to myopic maculopathy.

Lacquer cracks appear as yellowish linear lesions within the macula and may represent linear defects in Bruch's membrane. Lacquer cracks increase in number and width with time, and new lacquer cracks may be associated with subretinal hemorrhage (Figure 2).[11] Myopic choroidal neovascular membrane are often associated with lacquer cracks.[12]

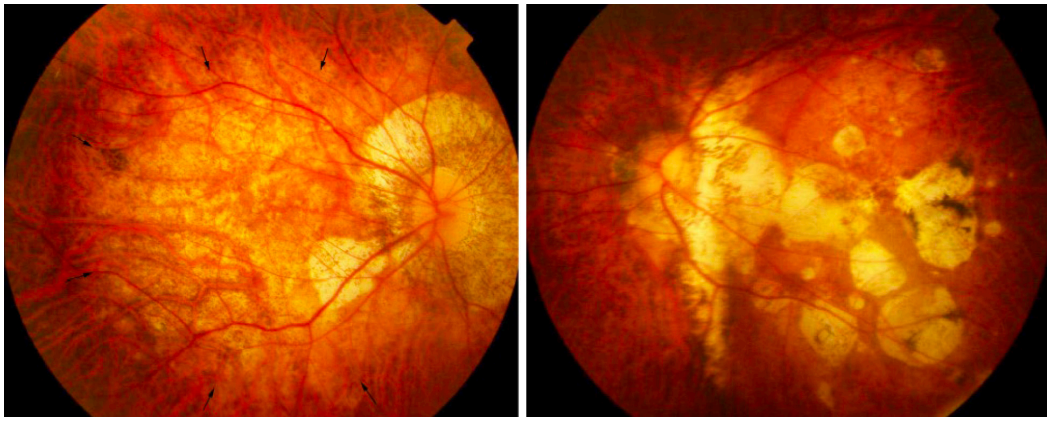

Figure 1: Diffuse chorioretinal atrophy. Right: Patchy chorioretinal atrophy. From Ohno-Matsui K, Kawasaki R, Jonas JB et al. International photographic classification and grading system for myopic maculopathy. *Am J Ophthalmol* 2015;159(5):877-83.e7.

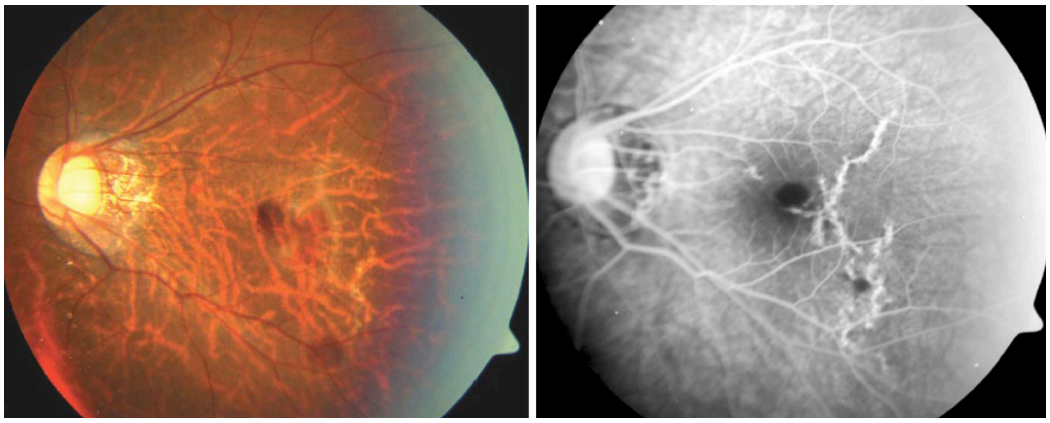

Figure 2: Lacquer cracks with associated subretinal hemorrhage on color fundus photograph (left) and fluorescein angiogram (right). From Yip LW and Au Eong KG. "Recurrent subretinal haemorrhages and progressive lacquer cracks in a high myope." *Acta Ophthalmol Scand* 2003: 81; 646-7.

Myopic choroidal neovascular membrane (CNVM) develops in up to 10% of patients with pathologic myopia.[12] Patients who have a history of myopic CNVM in one eye are at increased risk of developing CNVM in the fellow eye.[12] Presenting symptoms include metamorphopsia, central scotoma, and blurry vision. Fluorescein angiography and indocyanine green angiography show hyperfluorescence and hypercyanescence with mild leakage, which is often smaller in area compared with CNVM from other diseases such as macular degeneration (Figure 3).[13] OCT-Angiography can also be useful in identifying CNVM. Myopic CNVM is treated with intravitreal injections of anti-VEGF; bevacizumab, ranibizumab, and aflibercept have all been shown to be effective.[14,15,16] Unlike in treatment of macular degeneration, myopic CNVM is more often treated in a PRN regimen as it is more likely to regress spontaneously.[16] Regressed CNVM enter the scar phase, which is characterized by a darkly pigmented "Fuch's spot" and subsequently progress to atrophy. Although photodynamic therapy (PDT) has previously also been used to treat CNVM, patients treated with PDT show less improvement in visual acuity compared to those treated with ranibizumab; PDT remains a second-line option for patients in whom anti-VEGF is contraindicated.[17]

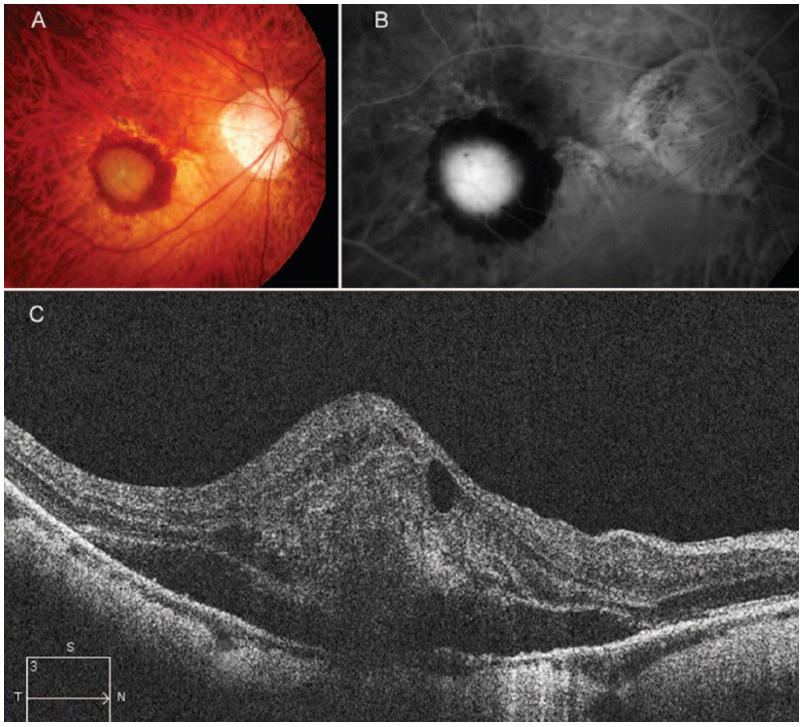

Figure 3: Myopic choroidal neovascular membrane on fundus photo (upper left), fluorescein angiogram (upper right) and OCT (bottom). From Wong TY, Ohno-Matsui K, Leveziel et al. Myopic choroidal neovascularization: current concepts and update on clinical management. *Br J Ophthalmol 2015;99(3):289-96.*

In addition to atrophic and neovascular myopic maculopathy, patients with pathologic myopia can also develop vision loss from myopic traction. Myopic traction maculopathy (MTM), including maculoschisis, lamellar macular hole, and foveal retinal detachment, can occur in up to 9% to 34.4% of patients with pathologic myopia (Figure 4).[18] Timing of surgical intervention for MTM can be controversial. Although pars plana vitrectomy with or without internal limiting membrane peeling and gas tamponade has been reported to improve MTM, these surgeries are more likely to be complicated by full thickness macular hole.19 Recent studies have reported successful resolution of MTM with fewer complications with fovea-sparing internal limiting membrane peeling.[19]

High myopia has implications for the peripheral retina in addition to the macula. Patients with high myopia are at 5-6 times higher risk for retinal detachment compared to patients with low myopia.[20] Lattice degeneration is more common in highly myopic eyes compared to the general population and may be more likely to be associated with retinal detachment.[21] All patients with high myopia and peripheral pathology or new floaters and photopsias should undergo a detailed examination of the peripheral retina with scleral depression.

Special consideration must be given to children with high myopia. In a study of children under 10 years old presenting with high myopia, only 8% had simple myopia without other associated ocular or systemic conditions.[21] Among the most common associations were extreme prematurity (10%), Stickler syndrome (8%), and Marfan syndrome (5%), which are associated with increased risk for retinal detachment at a young age.[22] Young, highly myopic patients may warrant referral for retina examination, especially if there is a family history of retinal detachment at an early age.

Research into early prevention of myopia with dilute atropine has shown promising results. In the Atropine for the Treatment of Myopia 2 (ATOM 2) trial, low dose 0.01% atropine was shown to be most effective in preventing overall myopia progression and axial elongation at 5 years after initiating therapy, even compared with higher atropine doses.[23] Importantly, 0.01% atropine was well tolerated with minimal pupil dilation and loss of accommodation.

Pathologic myopia is a bilateral, progressive condition that can affect working age adults. With the projected increase in prevalence of high myopia, pathologic myopia will likely have an even greater societal and economic impact in the future. Fortunately, treatments for vision threatening complications of pathologic myopia, such as CNVM and traction maculopathy, can lessen the burden of this disease. New research into early prevention of myopia may curb the upcoming pandemic.

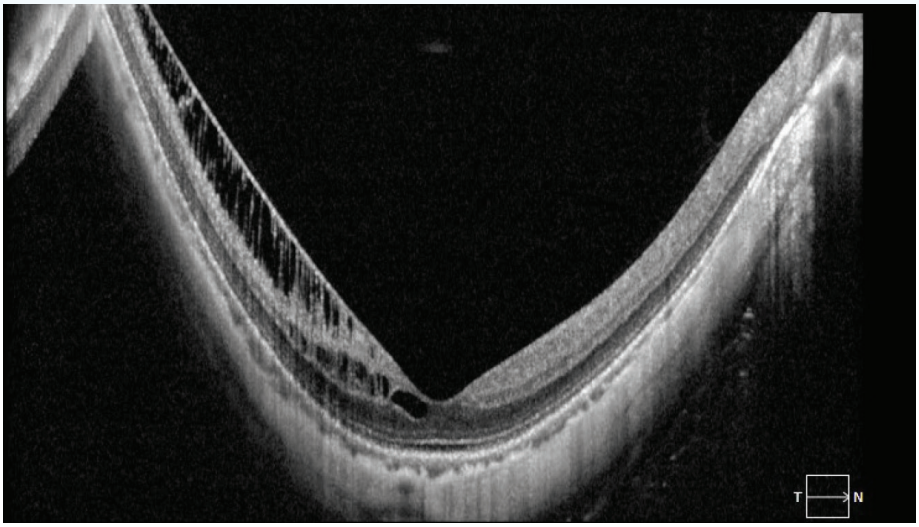

Figure 4: Myopic maculoschisis

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At the forefront of clinical research

NJRetina continuously conducts clinical trials at key locations. Our clinical research coordinators will be happy to discuss the inclusion/exclusion criteria or any other aspect of these studies with you or your patients. If you have any questions, please feel free to contact:

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Enrolling Studies:

Wet AMD

Edison

Opthea Coast: A Phase 3, Multicenter, Double-masked, Randomized Study to Evaluate the Efficacy and Safety of Intravitreal OPT-302 in Combination with Aflibercept, Compared with Aflibercept Alone, in Participants with Neovascular Age-related Macular Degeneration (nAMD)

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Opthea Shore: A Phase 3, Multicenter, Double-masked, Randomized Study to Evaluate the Efficacy and Safety of Intravitreal OPT-302 in Combination with Ranibizumab, Compared with Ranibizumab Alone, in Participants with Neovascular Age-related Macular Degeneration (nAMD)

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Elevatum: A Phase IIIB/IV, Multicenter, Open-Label, Single-Arm Study to investigate Faricimab treatment in response to treatment-naïve, underrepresented patients with Diabetic Macular Edema

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Luna: A Multi-Center, Randomized, Double-Masked Phase 2 Study to Assess Safety and Efficacy of ADVM-022 (AAV.7m8-aflibercept) in Anti-VEGF Treatment Experienced Patients with Neovascular (Wet) Age[1]related Macular Degeneration

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Eye Point: A Phase 2, Multicenter, Prospective, Randomized, Double-Masked, Parallel Study of EYP-1901, a Tyrosine Kinase Inhibitor (TKI), Compared to Aflibercept in Subjects with Wet AMD

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Upcoming Studies:

GA / Dry AMD

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Alexion: A Phase 2, Double-Masked, Placebo-Controlled, Dose Range Finding Study of Danicopan (ALXN2040) in Patients with Geographic Atrophy (GA) Secondary to Age-Related Macular Degeneration (AMD)

Janssen: Phase 2/3, Randomized, Double-masked, Multicenter, Dose-ranging, Sham[1]Controlled Clinical Trial to Evaluate Intravitreal JNJ-81201887 (AAVCAGsCD59) Compared to Sham Procedure for the Treatment of Geographic Atrophy (GA) Secondary to Age-related Macular Degeneration

RVO

Teaneck, Toms River, Edison

Bayer Study: A Randomized, Double-Masked, Active-Controlled, Phase 3 Study of the Efficacy and Safety of Aflibercept 8 mg With Macula Edema due to Retinal Vein Occlusion

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