

Choroidal neo-vascularization presentation in younger age group (pre-senile)Mehak Shehzad,¹ Tariq Aziz²**Abstract**

Age-related macular degeneration (ARMD) is the most common cause of permanent visual loss in the elderly. Advancing age, as the name suggests, is a major risk factor. Vascular endothelial growth factor (VEGF) along with other factors could be responsible for the dramatic damage in the eyes. Although uncommon in pre-senile group, this disorder can also occur unrelated to age, such as pathologic myopia in which Fuch's spots can classically be seen. It can also occur following traumatic disruption of the Bruch's membrane. Herein we report a case of a 20-year-old healthy female with no known co-morbidities who presented with complaints of sudden central visual loss in her left eye over the course of a few days (one week) with no preceding history of traumatic event or predisposing factor. To investigate the cause, Optical Coherence Tomography/Fluorescein angiography (OCT/FFA) was ordered which exhibited the classical signs of choroidal neo-vascularization.

Keywords: Age related macular degeneration, Choroidal neo-vascularization, Sub-retinal hemorrhage, VEGF antagonist.

Introduction

Where so many other hereditary and environmental factors stand in threatening the visual acuity of the elderly, age-related macular degeneration (ARMD) tops the list.¹ With an 8% increased risk and incidence of this dramatic condition in the ones who have crossed seven decades of their lives,² it is now posing major ocular issues to the seniors — thus a mainstay of treatment is required to reduce its complications. Nearly 71% of the cases are suggested to be inherited.³ Complement factor H (CHF) gene might play a role in the pathogenesis of the disease.⁴ Ten pack-years of smoking along with a CFH CC genotype predisposes a person to having a 144-fold increase in developing the disease.⁵ The condition can be classified into two categories based upon the presence or absence

of exudates, namely dry ARMD (atrophic) or wet ARMD (neo-vascular/exudative). The pathogenesis underlying the process is choroidal neo-vascularization in which the blood vessels disrupt the Bruch's membrane and grow into the sub-retinal pigmented epithelial (RPE) space.⁶⁻⁸ On microscopic visualisation, dry ARMD presents as diffusely present drusen spots that are yellowish white spots on the Bruch's membrane with marked thinning of retina with RPE atrophy. Individuals with dry type of lesions can develop into wet type with an incidence of 10% to 20%. Due to less understood pathological phenomena, new choroidal vessels develop, disrupting the Bruch's membrane and penetrating the RPE. The blood may seep through the vessels into the RPE space and be appreciated as macular scars on Fluorescein angiography (FFA). Photoreceptors may ultimately die and the central vision is distorted with the peripheral vision relatively intact. In some unfortunate subjects, the haemorrhage is massive. In pre-senile group, the leading cause is high myopia. Bruch's membrane tends to thin out in high myopia which increases the risk for CMV.⁹ Vascular endothelial growth factor (VEGF) may be a triggering agent in causing the trouble where its levels tends to increase in hypoxia in the intra-ocular environment and can thus promote pathogenesis of ischaemia and related changes.

Case Report

A 20-year-old young patient presented on August 4, 2012 with a history of sudden central visual disturbances in her left eye achieving 6/18 visual acuity after correction. She had no known co-morbidities. There was no history of recent trauma to her head or eyes. She was myopic; -4.00 in both eyes. On examination, a tiny focal haemorrhage was noticed in her left eye. Findings in her right eye were unremarkable with a 6/6 visual acuity after correction. An urgent Optical Coherence Tomography/Fluorescein angiography (OCT/FFA) was suggested, which confirmed the diagnosis and depicted early signs of choroidal neo-vascularization (Figure-1). The patient consulted various other ophthalmologists, who suggested treatment with anti-VEGF injection into the vitreous of the affected eye. She was scheduled for a 3-monthly intra-vitreous anti-VEGF injections course. She received her first dose of injection bevacizumab 1.25mg/0.05ml on August 10, 2012 with

.....
¹Undergraduate Medical Student, Dow University of Health Sciences,

²Consultant Eye Surgeon, Karachi.

Correspondence: Mehak Shehzad. Email: idealequations@live.com

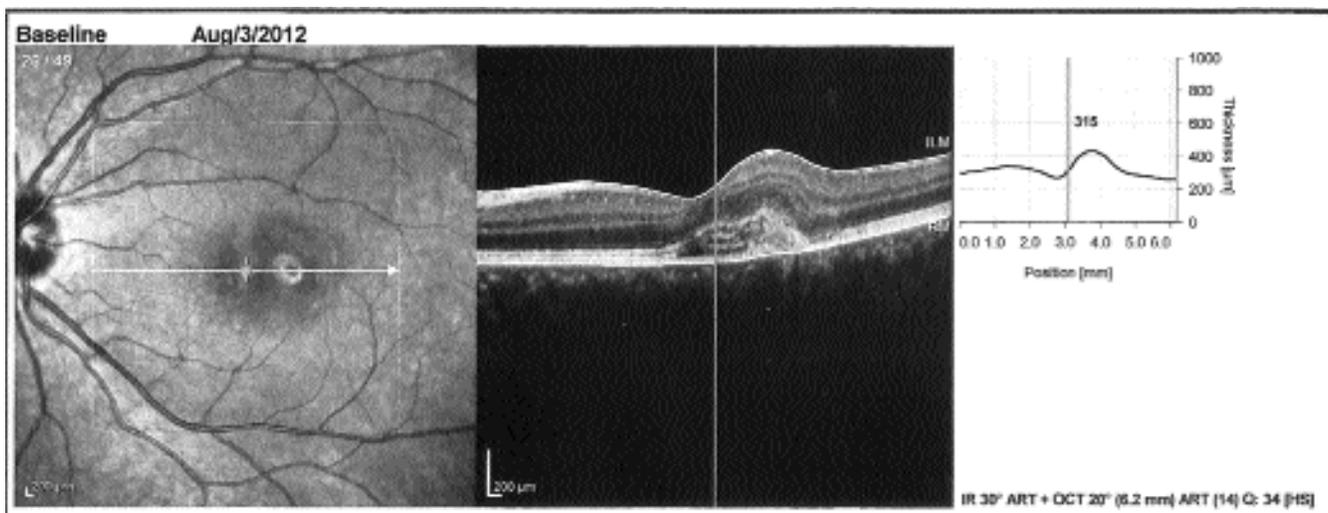


Figure-1: Pre-intervention.

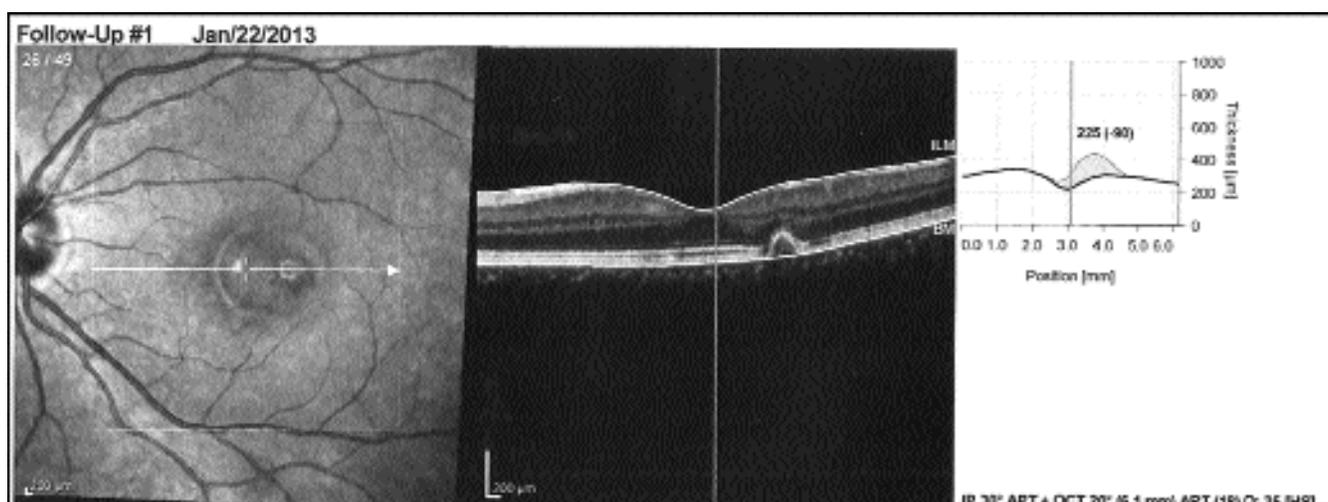


Figure-2: Post-intervention.

dramatic improvement in her visual acuity and correction to 6/9 after one month. The second dose was given on September 14, 2012 which restored the vision to 6/6. The final dose of ranibizumab was given on October 19, 2012. OCT was repeated in January 2013 (Figure-2) after completion of the treatment which showed no further deterioration of vision. She had regular follow-ups every 3 months. Her vision was found to be 6/6 with no distortion. She has been advised to follow-up every six months. In the meanwhile, she was directed to evaluate her central vision on the Amsler Grid.

Discussion

Pathologic myopia is the second leading cause of

choroidal neo-vascularization in ARMD and the first cause in patients younger than 50 years of age.¹⁰ The process of neo-vascularization originates from choroidal blood vessels and causes insult to the Bruch's membrane, often invading the RPE area. The RPE usually becomes incompetent, causing retinal detachment in severe cases, as a consequence of accumulation of serum and/or blood. The patient classically complains of metamorphosis, that is, crooked vision because the underlying pathology causes the retinal surface to bulge and become irregular. FFA together with OCT is used to confirm the diagnosis and see the extent of the lesion. Visual changes may not come into the patient's notice at an early age and if the sub-retinal haemorrhage remains masked for a long time,

scarring may occur. This threatens the vision further, and can result in death of the photoreceptors leading to drastic consequences and poor prognosis. Currently the mainstay of treatment is injection of VEGF antagonist into the vitreous of the affected eye.^{10,11} The treatment has no reported shortcomings/side-effects and is very well tolerated by the patients of any age group.¹²

Conclusion

Prompt treatment and management of ARMD and choroidal neo-vascularization in any age group is an important aspect in saving the patient's vision and ensuring a better life. VEGF antagonist remains the mainstay of treatment for this debilitating eye condition due to its efficacy and safety.

References

1. Barouch FC, Miller JW. Anti-vascular endothelial growth factor strategies for the treatment of choroidal neovascularization from age-related macular degeneration. *Int Ophthalmol Clin* 2004; 44: 23-32.
2. Klein R, Klein BE, Knudtson MD, Meuer SM, Swift M, Gangnon RE. Fifteen-year cumulative incidence of age-related macular degeneration: the Breaver Dam Eye Study. *Ophthalmology* 2007; 114: 253-62.
3. Scholl HPN, Fleckenstein M, Issa PC, Keilhauer C, Holz FG, Weber BHF. An update on genetics of age-related macular degeneration. *Mol Vis* 2007; 13: 196-205.
4. Maller J, George S, Purcell S, Fagerness J, Altshuler D, Daly MJ, et al. Common variation in three genes, including non coding variant in CFH, strongly influences risk of age related macular degeneration. *Nat Genet* 2006; 38: 1055-9.
5. DeAngelis MM, Ji F, Kim IK, Adams S, Capone A Jr, Ott J, et al. Cigarette smoking, CFH, APOE, ELOVL4, and risk of neovascular age-related macular degeneration. *Arch Ophthalmol* 2007; 125: 49-54.
6. Green WR, Enger C. Age-related macular degeneration histopathologic studies. The 1992 Lorenz E. Zimmerman Lecture. *Ophthalmology* 1993; 100: 1519-35.
7. D'Amore PA. Mechanisms of retinal and choroidal neovascularization. *Invest Ophthalmol Vis Sci* 1994; 35: 3974-9.
8. Green WR, McDonnell PJ, Yeo JH. Pathologic features of senile macular degeneration. *Ophthalmology* 1985; 92: 615-27.
9. Green WR, Wilson DJ. Choroidal neovascularization. *Ophthalmology* 1986; 93: 1169-76.
10. Cohen SY. Anti-VEGF drugs as the 2009 first-line therapy for choroidal neovascularization in pathologic myopia. *Retina* 2009; 29: 1062-6. doi: 10.1097/IAE.0b013e3181b1bb1a.
11. Jager RD, Mieler WF, Miller JW. Age-related macular degeneration. *N Engl J Med* 2008; 358: 2606-17.
12. Spaide RF, Laud K, Fine HF, Klancnik JM Jr, Meyerle CB, Yannuzzi LA, et al. Intravitreal bevacizumab treatment of choroidal neovascularization secondary to age-related macular degeneration. *Retina* 2006; 26: 383-90.