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Choroidal Neovascularization Complicating Adult-Onset Foveomacular Vitelliform Dystrophy: A Multimodal Imaging Case Report

Fiqhi Aissam[1*], M. Boukssim[1], H. Moumene[1], Mouzari Yassine[1]

[1]Department of Ophthalmology, Mohammed V Military Teaching Hospital, Mohammed V University, Faculty of Medicine, Rabat, Morocco

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***Corresponding author:** Fiqhi Aissam
Department of Ophthalmology, Mohammed V Military Teaching Hospital, Mohammed V University, Faculty of Medicine, Rabat, Morocco

Abstract | **Case Report**

We report a case of choroidal neovascularization (CNV) complicating adult-onset foveomacular vitelliform dystrophy (AOFVD) in a 62-year-old patient. Multimodal imaging, including spectral-domain optical coherence tomography (SD-OCT), fluorescein angiography (FA), and optical coherence tomography angiography (OCT-A), confirmed the presence of a neovascular vascular plexus within the outer retina and choriocapillaris layer. Anti-vascular endothelial growth factor (anti-VEGF) therapy resulted in anatomical and functional stabilization.

Keywords: Adult-onset foveomacular vitelliform dystrophy (AOFVD), Choroidal neovascularization (CNV), Optical coherence tomography angiography (OCT-A), Anti-VEGF therapy, Multimodal imaging, Pattern dystrophy.

INTRODUCTION

Adult-onset foveomacular vitelliform dystrophy (AOFVD) is a retinal pattern dystrophy first described by Gass in 1974 [1]. It is characterized by the accumulation of yellowish subretinal material in the macular region and typically presents in the fifth to seventh decades of life. Although the course is often slowly progressive, significant visual loss may occur due to macular atrophy or choroidal neovascularization (CNV) [2,3].

CASE PRESENTATION

A 62-year-old male presented to our ophthalmology department with a progressive decrease in central vision in his right eye over a four-month period. He described metamorphopsia and central distortion without ocular pain, photophobia, or floaters. There was no history of trauma, intraocular surgery, systemic vascular disease, diabetes mellitus, hypertension, autoimmune disease, or known hereditary retinal disorders. The patient was not on any chronic medication. Family history was negative for macular dystrophies.

Best-corrected visual acuity (BCVA) was 2/20 in the right eye and 20/20 in the left eye. Refraction did not significantly improve visual acuity in the affected eye. Intraocular pressure measured by Goldmann applanation tonometry was 15 mmHg in both eyes. Pupillary examination revealed equal, round, and reactive pupils with no relative afferent pupillary defect. Ocular motility is preserved in all nine directions

Slit-lamp biomicroscopy showed a clear cornea, deep and quiet anterior chamber without cells or flare, normal iris architecture, and a clear crystalline lens without significant cataract. There were no signs of anterior or posterior segment inflammation.

Dilated fundus examination of the right eye revealed a well-circumscribed, yellowish, subfoveal elevated lesion consistent with vitelliform material accumulation. The lesion was associated with subtle retinal pigment epithelium (RPE) irregularities and focal hyperpigmentation. No extensive soft drusen typical of neovascular age-related macular degeneration were observed. There was no vitreous hemorrhage. The left eye examination was unremarkable.

Spectral-domain optical coherence tomography (SD-OCT) demonstrated hyperreflective subretinal material located between the neurosensory retina and the RPE, with disruption of the ellipsoid zone and irregular RPE elevation. Minimal subretinal fluid was present.

OCT angiography (OCT-A) analysis revealed a distinct high-flow neovascular vascular plexus. On the outer retinal slab, an irregular, tangled vascular network was observed. At the level of the choriocapillaris, a well-defined neovascular complex was identified, surrounded by areas of relative flow void, consistent with type 1 choroidal neovascularization.

Fluorescein angiography showed early hyperfluorescence corresponding to the lesion with progressive late leakage, confirming active choroidal neovascularization.

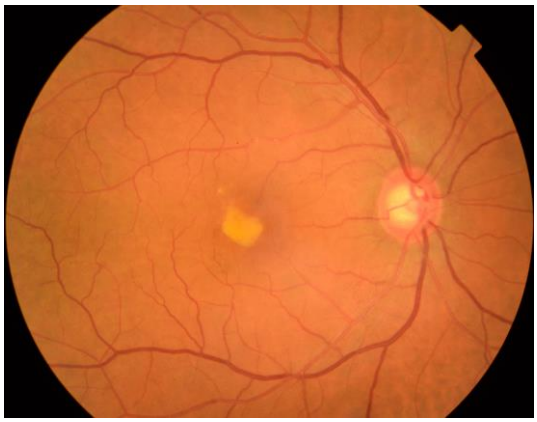

Figure 1: Color fundus photograph of the right eye demonstrating a well-demarcated subfoveal yellow vitelliform lesion with mild surrounding retinal pigment epithelium (RPE) alterations. The optic disc and retinal vasculature appear normal without extensive drusen, supporting the diagnosis of adult-onset foveomacular vitelliform dystrophy

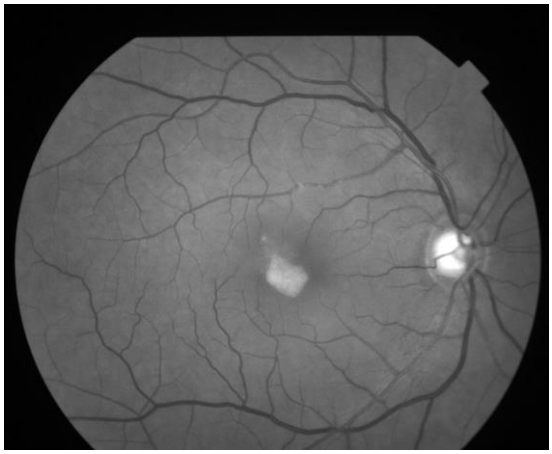

Figure 2: Fundus autofluorescence imaging showing intense hyperautofluorescence corresponding to the vitelliform material, reflecting lipofuscin accumulation within the subretinal space. The surrounding retina demonstrates relatively preserved autofluorescence

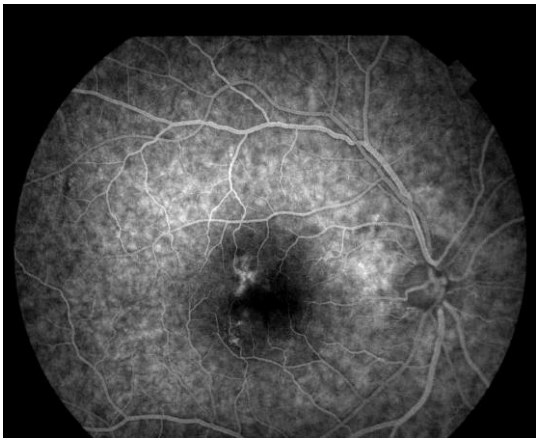

Figure 3: Fluorescein angiography (late phase) Marked dye leakage and pooling are evident within the subfoveal region, confirming the presence of active choroidal neovascularization complicating adult-onset foveomacular vitelliform dystrophy

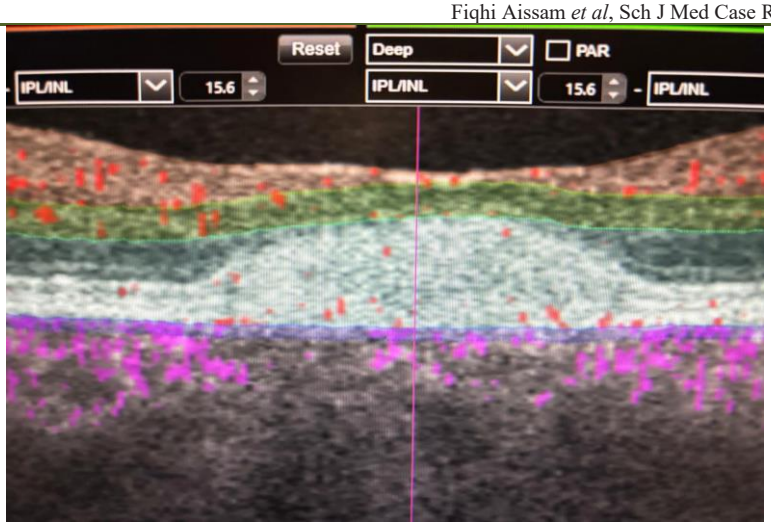

Figure 4: OCT angiography at the level of the choriocapillaris demonstrating a distinct neovascular vascular complex surrounded by areas of flow void

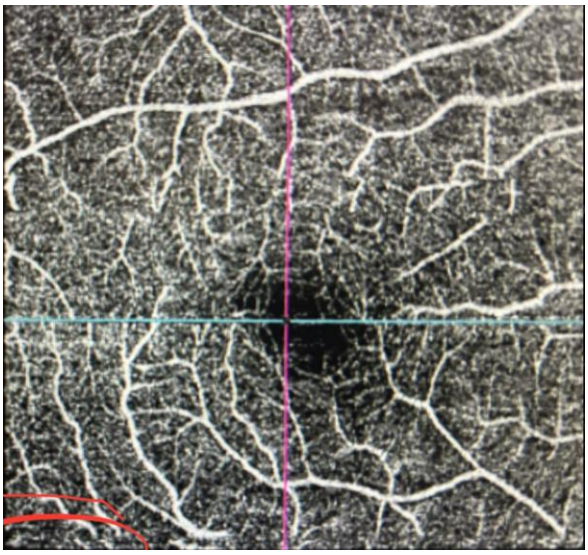

Figure 5: OCT Angiography: Superficial Capillary Plexus

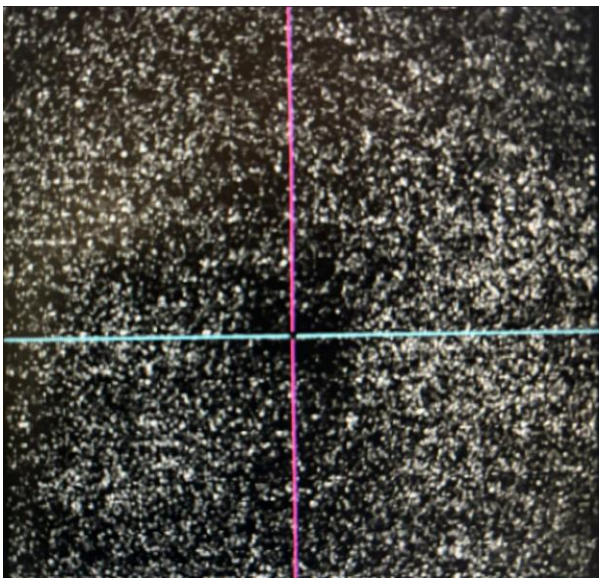

Figure 6: OCT Angiography: Outer Retina Slab

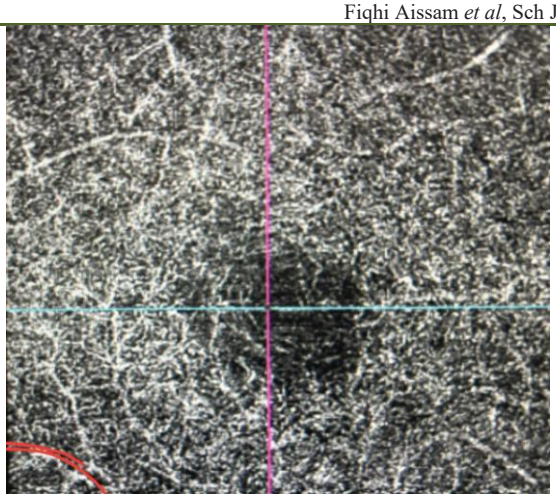

Figure 7: OCT Angiography: Deep Capillary Plexus

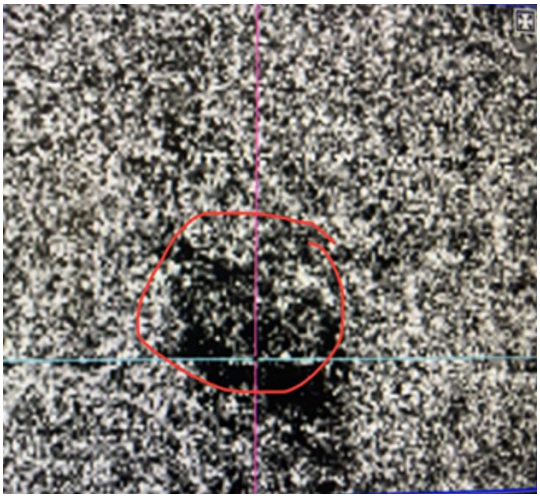

Figure 8: OCT Angiography: Choriocapillary plexus: Showing Subfoveal Vitelliform Material with Associated Type 1 Choroidal Neovascularization in Adult-Onset Foveomacular Vitelliform Dystrophy

DISCUSSION

AOFVD is generally considered a relatively indolent macular dystrophy; however, its clinical course may be complicated by structural degeneration and secondary choroidal neovascularization. Several studies have emphasized that although many patients maintain relatively preserved visual acuity for years, progression to atrophy or CNV significantly alters prognosis [2,3]. Renner *et al.,* reported that visual decline in AOFVD is typically mild unless complicated by neovascularization or geographic atrophy [3]. Similarly, Francis *et al.,* demonstrated that substantial loss of central vision correlates strongly with the development of CNV [4].

The pathophysiology of CNV in AOFVD remains incompletely understood. Chronic dysfunction of the retinal pigment epithelium appears to play a central role. Histopathologic studies have shown disruption of photoreceptor outer segments and RPE alterations with accumulation of subretinal material [5]. Over time, persistent metabolic stress may compromise Bruch's membrane integrity, facilitating angiogenic signaling and neovascular ingrowth from the choroid. Boon *et al.,* suggested that chronic RPE dysfunction in pattern dystrophies generates a pro-angiogenic microenvironment similar to that observed in early age-related macular degeneration (AMD) [6].

Differentiating CNV secondary to AOFVD from neovascular AMD is clinically crucial. Although the age overlap can be misleading, several imaging features assist in differentiation. AOFVD typically lacks extensive soft drusen and demonstrates characteristic vitelliform material with hyperautofluorescence on fundus autofluorescence imaging [2,7]. In contrast, AMD is often associated with widespread drusen and pigment epithelial detachments. However, once CNV develops, imaging patterns may overlap, making advanced imaging modalities essential.

Fluorescein angiography has historically been the cornerstone for CNV detection. In AOFVD, FA may show staining of vitelliform material without leakage in earlier stages [8]. However, progressive late leakage strongly suggests neovascular activity. Battaglia Parodi *et al.,* described ICGA findings demonstrating hypocyanescence corresponding to vitelliform lesions

but highlighted the difficulty of identifying occult CNV using dye-based angiography alone [9].

The advent of OCT angiography has dramatically improved the ability to detect CNV in retinal dystrophies. OCT-A allows noninvasive visualization of flow within abnormal vascular networks. Querques *et al.,* reported that OCT-A could identify subclinical neovascular membranes not evident on FA [10]. In our case, OCT-A demonstrated a distinct high-flow neovascular vascular plexus within the outer retina and choriocapillaris slab. The vascular complex exhibited irregular branching morphology and a tangled configuration, consistent with type 1 neovascularization. Surrounding flow void areas may reflect localized choriocapillaris hypoperfusion or shadowing effects.

Recent literature supports the utility of OCT-A in distinguishing between avascular vitelliform deposits and true neovascular complexes. Freund *et al.,* emphasized that the presence of a well-defined vascular network on OCT-A confirms CNV even when structural OCT findings are ambiguous [11]. Moreover, Mimoun *et al.,* reported that CNV complicating AOFVD often presents as type 1 lesions located beneath the RPE, similar to our findings [12].

Therapeutically, anti-VEGF therapy remains the standard of care for CNV secondary to AOFVD. Multiple reports have demonstrated favorable anatomical and functional outcomes following intravitreal injections. Mimoun *et al.,* described significant reduction in exudation and stabilization of vision after anti-VEGF treatment in AOFVD-related CNV [12]. Similarly, Freund *et al.,* observed regression of neovascular flow signals on OCT-A following therapy [11]. Our patient exhibited decreased flow density within the neovascular complex and resolution of intraretinal fluid after a loading phase of three-monthly injections, consistent with these findings.

Long-term prognosis depends on early detection and timely intervention. Delayed treatment may lead to photoreceptor loss, fibrotic scarring, and irreversible visual decline. Therefore, close monitoring with multimodal imaging is essential in patients with AOFVD, particularly when new symptoms arise. OCT-A has become a valuable surveillance tool for detecting early neovascular changes before irreversible structural damage occurs.

In summary, CNV represents a critical turning point in the natural history of AOFVD. The integration of structural OCT and OCT-A enable precise diagnosis and guides therapeutic decision-making. Our findings reinforce the growing body of evidence supporting OCT-A as an indispensable modality in the management of macular dystrophies complicated by neovascularization.

CONCLUSION

Choroidal neovascularization significantly worsens the prognosis of AOFVD. Multimodal imaging, particularly OCT angiography, is crucial for accurate diagnosis. Early anti-VEGF therapy can stabilize vision and prevent irreversible retinal damage.

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Consent: Informed consent was obtained from patients included in this study.

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