

Macular Telangiectasias Associated With a New Choroid Vessel, in One Case

Chourouk Aabdi, Hajar Boutahar, Siham Chariba, Asmae Maadane Rachid Sekhsoukh and CHU Mohammed VI Oujda

ARTICLE INFO

Article history:

Received: 22 February 2020;

Received in revised form:

6 July 2020;

Accepted: 17 July 2020;

Keywords

Macular Telangiectasia,
 Microanevrismal Dilations.

ABSTRACT

Macular telangiectasias are characterized by the presence of microanevrismal dilations around the fovea. This appellation brings together two very different entities, type 1 and type 2. We are talking in this work about Macular telangiectasia type 2. We report the case of a 48-year-old patient followed for macular telangiectasia 2, complicated by neovessels treated by anti VEGF

© 2020 Elixir All rights reserved.

Introduction

Macular telangiectasias are defined as unilateral or bilateral alterations of the juxtafoveolar capillaries, dilated and abnormally permeable. It is a rare condition of which the epidemiology is poorly understood. Several classifications have been given to this pathology, the most used remains that proposed in 2006 by Yannuzzi and which defines two groups of macular telangiectasias. Group 1 represents unilateral, congenital, exudative macular telangiectasias often complicated by cystoid macular edema. Group 2 macular telangiectasias are occult, acquired, bilateral telangiectasias. They are not associated with cystoid macular edema, but rather with retinal atrophy and can be complicated by neovascularization. In this work we will talk about macular telangiectasias2

The objective of this work is to recall the clinical characteristics of macular telangiectasia2, the contribution of imaging in this pathology and in its complications and the therapeutic means that can be used.

Observation

We report the case of a 48-year-old patient, followed for 5 years for macular telangiectasia2 with on clinical examination a corrected visual acuity calculated at 10/10 P2 for both eyes and at the bottom of the eye, we found, bilaterally, dilations of the macular capillaries The rest of the ophthalmological examination was normal, angiography confirmed the diagnosis by ascending a poorly limited macular hyperfluorescence from early times with diffusion in late times (figure1).

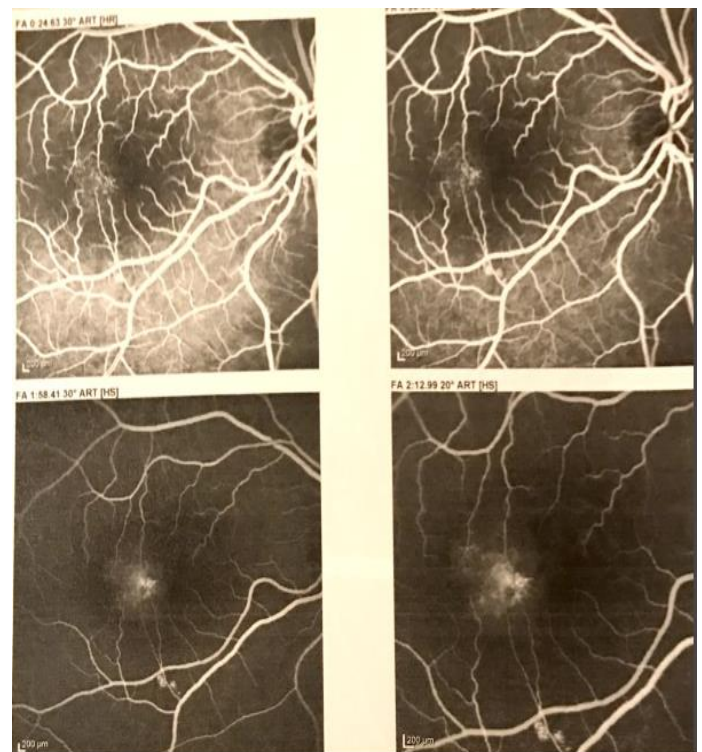


Figure 1. angiography showing a poorly limited macular hyperfluorescence, from early times with diffusion in late times.

The evolution was marked by the installation of metamorphopsies in the left eye with acuity estimated at 7/10 with correction. The oct angiography had helped to detect a neovascular complications (figure2).

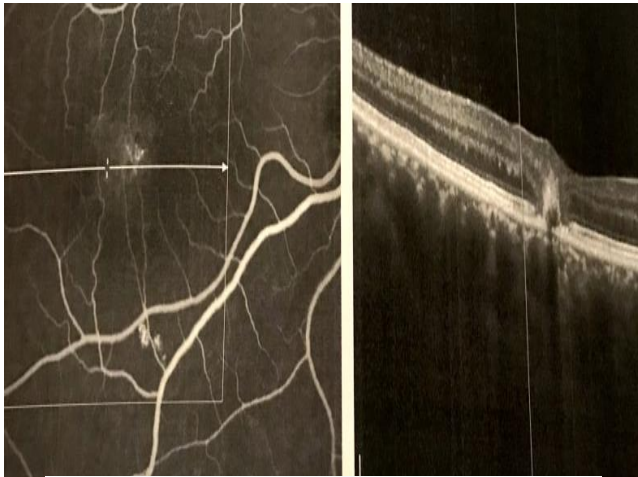


Figure 2. oct angiography showing a neovascular complication.

Treatment with intravitreal injections anti VEGF was started, currently at 6 weeks of treatment visual acuity increased by one line and macular thickness decreased

Discussion

The etiology of these telangiectasias is complex, multiple and still poorly understood.

For group 2, the pathophysiology seems different and more complex. There are two hypotheses:

- or an inaugural anomaly of the macular capillaries which are hyperpermeable and cause macular exudation;
- or an initial process of degeneration of Müller cells that support architecture and retinal homeostasis. In this case, the anomaly of the capillaries would be secondary and later[1].

In the non-proliferative phase, neurodegeneration is the predominant event, which results in a decrease in the function of photoreceptors, a loss of the integrity of the internal retina, vascular abnormalities, leakage of fluid and progressive loss of vision slow later. If the retinal damage is severe enough to initiate hypoxic pathways leading to the release of the angiogenesis mediator and neovascularization, patients enter the proliferative phase[2].

Macular telangiectasia also affects both sexes and is very likely to be genetically determined[3], indicating that it should be present from the earliest stages of retinal development. The signs and symptoms of macular telangiectasia can be silent, especially at the beginning of the course of the disease, and patients may not be diagnosed during a routine examination. The first symptoms have the highest incidence in the 5th and 6th decades. Patients usually present with reading difficulties, metamorphopsia and paracentral scotomas. The loss of vision is slowly progressive and the best corrected visual acuity is preserved until late in the disease.

The differential diagnoses of this pathology are essentially diabetic, radiation maculopathy, post-OVCR, diabetic macular edema

In this group, telangiectasias are bilateral, barely visible at the back of the eye and little exudative and can be complicated by initially intraretinal neovascularization more or less associated with a retinochoroid anastomosis.

The diagnosis of macular telangiectasia 2 is becoming easier with the progress of new imagery and the increase in resolution. The images of OCT opposite allow us today to visualize the telangiectasias.

Regarding the treatment of these anomalies, apart from neovascular complications, there is, to date, no established

protocol. The literature reports the use of Argon laser photocoagulation, PDT, steroid IVT, anti-VEGF or combination therapy. The results are very controversial[4,5].

Anti-VEGF drugs seem to be useful for treating the OMC secondary to group 1 IMDs as well as the neovascular complications of group 2 IMDs. In fact, in our work, the injections in this case have improved. Most studies of anti-VEGF therapy for the proliferative phase of MacTel have reported anatomical and functional improvement [6,7,8]. A single retrospective study by Roller et al. reported borderline results [9].

Conclusion

Macular telangiectasias remain rare pathology, still imperfectly understood. Imaging, OCT, angiography, autofluorescence exams help better identify these macular abnormalities. Many uncertainties remain as to the therapeutic course to adopt. We found in this work an anatomical and functional efficiency of intravitreal anti-VEGF injections in case of neovascular complication

Reference

1. Yannuzzi L.A., Bardal A.M.C., Bailey Freund K., Chen K.J., Eandi C.M., Blodi B. Idiopathic maculat telangiectasia Arch Ophthalmol 2006 ; 124 : 450-460
2. Shen W, Fruttiger M, Zhu L, Chung SH, Barnett NL, Kirk JK et al. L'ablation conditionnelle des cellules de Müller provoque des pathologies neuronales et vasculaires indépendantes dans un nouveau modèle transgénique. J Neurosci. 2012; 32 (45): 15715–15727
3. Klein R, Blodi BA, Meuer SM, Myers CE, Chew EY, Klein BEK. Prévalence de la télangiectasie maculaire de type 2 dans l'étude sur l'œil de la digue de castor. Suis J Ophthalmol. 2010; 150 (1): 55–62.e52.
4. Kotoula M.G., Chatzoulis D.Z., Karabatsas C.H., et al. Resolution of macular edema in idiopathic juxtafoveal telangiectasis using PDT Ophthalmic Surg Lasers Imaging 2009 ; 40 : 65-67
5. Arevalo J.F., Sanchez J.G., Garcia R.A., et al. Indocyanine-green-mediated photothrombosis (IMP) with intravitreal triamcinolone acetonide for macular edema secondary to group 2A idiopathic parafoveal telangiectasis without choroidal neovascularization: a pilot study Graefes Arch Clin Exp Ophthalmol 2007 ; 245 : 1673-1680
6. Dave V, Chhablani J, Narayanan R. Différentes modalités de traitement pour la néovascularisation choroïdienne dans deux yeux d'un patient avec une télangiectasie parafovéale bilatérale de type 2A. Indian J Ophthalmol. 2013; 61 (7): 353–355
7. Baz O, Yilmaz I, Alagoz C, Demircan A, Perente I, Ozkaya A, et al. Efficacité du bevacizumab intravitréen dans le traitement de la télangiectasie juxtafovéale idiopathique proliférative de type 2. Turk J Ophthalmol. 2017; 47 (3): 144-148
8. Narayanan R, Chhablani J, Sinha M, Dave V, Tyagi M, Pappuru RR, et al. Efficacité de la thérapie anti-vasculaire par facteur de croissance endothélial dans la néovascularisation sous-rétinienne secondaire à une télangiectasie maculaire de type 2. Rétine (Philadelphia, PA) 2012; 32 (10): 2001-2005.
9. Roller AB, Folk JC, Patel NM, Boldt HC, Russell SR, Abramoff MD, et al. Bévacizumab intravitréen pour le traitement de la télangiectasie maculaire idiopathique proliférative et non proliférative de type 2. Retina (Philadelphia, Pennsylvanie) 2011; 31 (9): 1848–1855.