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Short Communication

Vascular Phenomena in Ocular Toxoplasmosis

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Vascular alterations are frequent findings in the context of ocular toxoplasmosis. They usually occur within the quadrant in which the focus of retinochoroiditis is located. Most of these phenomena do not generate occlusion of the vessels, in fact these are the least frequent cases. Ischemia resulting from occlusive lesions can cause irreversible damage to the retina even though the area involved is subsequently relocated. This translates into disorders in the visual field that are added to those generated by the tissue remodeling of the inflamed retina itself, leading to very severe visual alterations.

We present the case of a 32-year-old female patient who consulted for decreased visual acuity in her right eye. The presence of mutton-fat keratic precipitates (KPs), anterior chamber cells 1+, vitreitis 2+ and a hypopigmented focus of a papillary diameter in the lower nasal quadrant surrounded by a large hyperpigmented area whose extension wasn't able to be determined due to a severe vitreitis. An intraretinal bleeding area, exudates and vascular sheathing of approximately 4 discs diameters was observed adjacent to the lesion. She was diagnosed with recurrent ocular toxoplamosis. It was treated with trimethoprim + sulfamethoxazole 960mg every 12 hours for 6 weeks and meprednisone 1 mg/kg/day resolving the total retinochoroiditis leaving a large scar involving the nasal periphery, the lower temporal arch and a small macular sector away from the fovea. Bleeding was unchanged despite successful treatment. Therefore, after three months, a fluorescein angiography was performed and choroidal hypofluorescence was observed due to lack of filling adjacent to the chorioretinal scar area and dilated vessels over the ischemic area that filter in late recirculation. In addition, peripapillary hyperfluorescence was observed. The visual field of the affected eye showed a scotoma corresponding to the scarred and bleeding area. The ischemic zone was treated with argon laser scattered photocoagulation.

The analysis of the case presented allows us to conclude that occlusive vasculitis, although rare, can be observed in ocular toxoplasmosis. Although the ischemic zone can be reperfused, once the inflammatory picture is resolved, the damage to the photoreceptor layer is usually permanent, affecting both the final visual acuity and the visual field.

Introduction

Ocular toxoplasmosis (TO) is the most common infectious uveitis worldwide. Although its course is self-limited, it can cause serious visual sequelae due to the tissue destruction generated by the focus of retinitis and vascular ischemia which is multifactorial.

The case presented here is about a 32 years-old woman who consulted our service at the end of 2019 because of decreased visual acuity in the right eye and denied any kind of pathological background. It was unilateral granulomatous panuveitis syndrome. The visual acuity of the affected eye was less than 20/200 and the other eye was 20/20.

The IOP was 14 mmhg for both eyes. At the biomicroscopy presented mutton-fat keratic precipitates and anterior chamber cells 1+. Vitreitis 2+ and a retinitis focus in the lower nasal hypopigmented quadrant with net edges of at least one disc diameter surrounded by a hyperpigmented area whose edges were difficult to delineate because of the severe vitritis. Above the lesion, in the upper nasal quadrant area, there was an intraretinal bleeding and exudates of approximately 4 papillary diameters.

The igG value was positive (the sample needed to be repeated since we obtained a negative initial value which was reported in another publication) with negative igM, as well as HIV, vdrl, ftabs, ppd.

We diagnosed recurrent ocular toxoplasmosis and began treatment with trimethoprim + sulfamethoxazole 960mg every 12 hours for 6 weeks and meprednisone 1mg/kg/day. Visual acuity progressively improved up to 20/20 after 6 weeks. Treatment was discontinued and the area was found to be persistent with bleeding and exudates. In the RFG, choroidal hypofluorescence was observed due to the lack of fullness adjacent to the chorioretinal scar area and dilated vessels over the ischemic area that leak in late recirculation. In addition, peripapillary hyperfluorescence was observed. The visual field of the affected eye showed a scotoma corresponding to the scarred and the bleeding area. The ischemic area was treated with argon laser scattered photocoagulation due to the risk of complications such as retinal and subretinal neovascularization and vitreous hemorrhage.

As Dr. Michael W. Gaynon said in 1984 in a three cases of retinal neovascularization and TO publication "retinovascular changes in ocular toxoplasmosis can occur both early and late. The vascular phenomena in the context of this disease are very varied. In general, non-occlusive conditions are the rule, vascular occlusions are much less frequent and arterial occlusions are even rarer.

In some cases, vasculitis may be the initial finding and may mask the underlying retinitis. For this reason, we should suspect TO in young people or in immunosuppressed people in whom atypical conditions are more common.

Non-occlusive vasculitis occurs in the form of venous and arterial sheath. Also, there may be nodular arteriolitis (keyrieleis arteritis) with deposit of yellowish plaques within the artery wall. Frosted branch angiitis is another possible finding, although not exclusive to this entity, and involves the entire vascular tree of both eyes and is therefore attributed to an immune response by the host.

The physiopathology of vasculitis is explained by different hypotheses. We know that the protozoon, once in the host, reaches the eyeball through the arterial circulation and then lodges in the internal retina, where tissue inflammation begins. One study showed that tachyocytes have a predilection for the vascular endothelium and express proteins on its surface such as ICAM-1 that allow them to pass through it and may cause vascular obstruction at that time. These intercellular adhesion molecules are also expressed on lymphocytes and monocytes and it is believed that they can collaborate by transferring the parasite through the hematoretinal barrier. The vascular endothelium of the arterioles responds to inflammation, first by generating vasoconstriction and then by generating vasodilation that promotes exudation, increased intravascular viscosity and generates a state of hypercoagulability that leads to regional tissue ischemia and, in some cases, causes vascular occlusion. Finally, the inflammatory focus can cause the blockage of the vessels which are around by a compression mechanism. Inflammation increases retinal thickness generating a mass effect on the vessel wall (it also explains why partial PVDs are developed on the focus of retinitis).

Regarding to ENVAINAMIENTO, O'connor showed that it is due to an antigen-antibody response. To reach this conclusion he injected toxoplasma gondii antigens into the vitreous cavity of rabbits causing vasculitis.

Finally, occluded vessels may or may not recur once the condition is resolved. Theodossiadis, in a prospective longitudinal study of 64 eyes, reported that 92% of the eyes presented vasculitis within the affected quadrant, the remaining 8% presented alterations within the four quadrants and only in three patients (5%) was the vasculitis of the occlusive type. In addition, they observed that vasculitis disappeared in only 14 eyes in correlation with the resolution of retinitis. Of the other 50 cases, 29 resolved retinitis within 3 months of the year on a deferred basis and the remaining 21 cases had no improvement throughout the follow-up.

The importance of these vascular phenomena lies in the fact that they cause ischemia and death of the photoreceptors. We know that the internal retina is nourished by the contribution of the retinal vessels, while the external retina does so through the choroidal vessels. Jonathan Stone et al. carried out a study on the location of mitochondria in the photoreceptors of mammals and determined that in humans they are located mainly in the terminal sectors of the axon at the level of the outer plexiform layer because there they get more oxygen. The blood supply in the macular region is determined by the superficial, intermediate and deep capillary plexuses, which run at the level of the ganglion cell layer, at the inner and

outer limits of the inner nuclear layer respectively. In the rest of the retina, the intermediate capillary plexus is absent and this is explained by the greater number of photoreceptors at the macular level, especially at the perifoveal level.

To study the damage caused by ischemia, OCT is a vitally important tool. While angiography is the choice for demonstrating vascular occlusion, OCT can more accurately reveal the area affected by ischemia than RFG even in the context of a normal angiogram.

Dr. Suquin Yu did wrote about the changes that we can observe through OCT in retinal vasculitis in acute and late stages. He divided the acute findings into three possibilities: (1) Thickening and hyperrefectivity in the layer of the internal retina attributed to ischemia in the superficial layer; (2) Hyperreflective band in the internal nuclear layer (which represents the picture known as acute paracentral middle maculopathy due to ischemia of the middle and deep vascular plexuses); (3) Diffuse thickening and hyperreflectivity in internal and middle layers of the retina representing ischemia, the latter being the pattern most frequently observed by him. In the chronic stages what he observed is a thinning of the retinal thickness due to atrophy of the different layers of the retina involved. In our experience it was difficult to obtain images of the area affected by vasculitis because initially the inflammation made it difficult for the patient to fixate and the lesion was outside the macula.

Based on the above, we believe it is necessary to stress the importance of knowing the different vascular phenomena that can be observed in Ocular Toxoplasmosis. Our clinical case is interesting, first, because it is an occlusive type of vasculitis. Secondly, because the site of the occlusion, although close, does not coincide with the quadrant affected by retinitis. So, the causes of vascular occlusions, as discussed above, are multiple. Our patient was cured, but with an extensive scar that reaches the lower temporal arcade below the papilla which translates into a large scotoma in the visual field. As a point of discussion in our work we cannot know if using classical treatment therapy would have resulted in a smaller chorioretinal scar. Bactrim is a drug that is accepted by the scientific community but is widely discussed as to its superiority over the classical scheme. Considering the sequelae described in our patient, we decided to maintain a prophylactic intake of trimethoprim/sulfamethoxazole every three days with the intention of avoiding a recurrence that would aggravate her current situation.