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Chronic Central Serous Chorioretinopathy Sequelae in Autofluorescence: Case Report and Literature Review

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Abstract

Central serous chorioretinopathy is the fourth most common nonsurgical retinopathy associated with subretinal fluid (SRF), whose chronic presence can ultimately damage the retinal pigment epithelium (RPE), although in some cases underlying multifocal choroidal vascular dysfunction can directly affect the RPE without the presence of SRF.

In our case report, we present a patient with decreased visual acuity and photopsia with a history of corticosteroid treatment, whose complementary studies, such as OCT and autofluorescence (FAF), led us to conclude that we were dealing with chronic central serous chorioretinopathy with minimal residual accumulation of SRF, with RPE damage.

In our review, we found the importance of autofluorescence (FAF) images that can help estimate the duration of the SRF episode and the damage induced by it, in addition to helping determine the appropriate treatment strategy.

Keywords: Central Serous Chorioretinopathy, Chronic, OCT, Autofluorescence (FAF)

Introduction

Central serous chorioretinopathy (CSC) is a chorioretinal disease that causes idiopathic serous retinal detachment, which is associated with one or more areas of choroidal leakage through a defect in the outer retinal pigment epithelium (RPE) blood-retinal barrier.

Most patients are men who have reduced and/or distorted vision, generally associated with a decreased quality of life. The age of onset of CSC can be as early as 7 years and as late as 83 years, with a peak at 40-50 years.

CSC is common and is considered the fourth most common non-surgical retinopathy associated with subretinal fluid leakage, after wet neovascular age-related macular degeneration, diabetic macular edema, and retinal vein occlusion. Although subretinal fluid (SRF) may resolve spontaneously, many patients have significant clinical sequelae, such as retinal and/or pigment epithelial atrophy, and may develop subretinal neovascularization [1].

The chronic presence of subretinal fluid may ultimately damage the RPE, although in some cases underlying multifocal choroidal vascular dysfunction may directly affect the RPE without the presence of SRF [2, 3].

The pathophysiology is thought to be related to increased hydrostatic pressure in the choroid and choriocapillaris hyperpermeability leading to pigment epithelial detachments (PEDs) and RPE monolayer defects, allowing fluid to leak beneath the neuroretina. This differs from neovascularization, where pigment epithelial detachments occur due to leakage of newly formed vessels [4].Regarding complementary studies, autofluorescence (FAF) images can help estimate the duration of the CSC episode and the damage induced by it, in addition to helping determine the appropriate treatment strategy [5, 6].

Finally, the combination of OCT, FAF, ICGA and A-OCT can be used to detect subretinal neovascularization, which can sometimes be difficult to confirm.

In our case, the combination of OCT and FAF with the clinical correlation of history allowed us to infer that we were dealing with chronic CSC.

Clinical Case

A 66-year-old patient with no previous personal or ophthalmological history came to the consultation, reporting photopsies of 2 months' duration without referring to any other associated symptoms.

Autorefractometry showed +4.50 sf -1.25 x 99 cyl in the RE and +4.25 sf -1.00 x 80 cyl in the LE, with a BCVA 20/200 RE and 20/40 in the LE.

Biomicroscopy showed no particularities, as well as her IOP which was 12 mmHg both eyes. The fundus examination was performed with a 90 D magnifying glass, and then with an BIO and a 20 D magnifying glass, in which diffuse hypopigmentation areas were found that emerge from both optic nerves superiorly and inferiorly, limited to the middle periphery of both eyes, together with pigment redistribution. In addition, alteration of the macular brightness of both eyes was observed, and the rest of the ophthalmoscopic examination was unremarkable.

In light of this finding, the patient was re-interrogated, who told us that she was unaware of her bilateral decrease in visual acuity, and as the only positive data, she reported that, prior to the myodesopsias, she received intramuscular injections of corticosteroids for non-rheumatological low back pain.

We therefore requested a macular OCT, which showed alterations in the outer layers of the retina in the right eye with hyperreactivity of the choroid due to the window effect, without thickening of the thickness of the same, with a subfoveal hyperreflective point with a slight accumulation of subretinal fluid, which could correspond to a leak point, while in the left eye no foveal retinal changes were observed, although the presence of atrophy of the outer layers of the retina with a window effect can be seen in both eyes at the upper, nasal and lower levels with respect to the papilla.

These findings, in turn, correspond to a requested autofluorescence in which we can see that these areas of atrophy are observed to be hypoautofluorescent, surrounded by a halo of hyperautofluorescence.

These lesions found in the autofluorescence, made us initially reconsider the suspected diagnosis, since an important differential diagnosis that presents the same pattern of hypoautofluorescent plaques with a hyperautofluorescent halo is serpiginous choroidopathy and its main variant, serpiginous-like choroidopathy, associated with mycobacterium tuberculosis, with a negative PPD and a normal chest X-ray that allowed us to rule it out, in addition to not seeing choroidal thickening, in which pathology it is present.

Therefore, due to the images and the history of having received corticosteroid injections, we resumed the initial diagnosis, finding ourselves facing a bilateral chronic serous central choroidopathy, with macular involvement of the right eye.

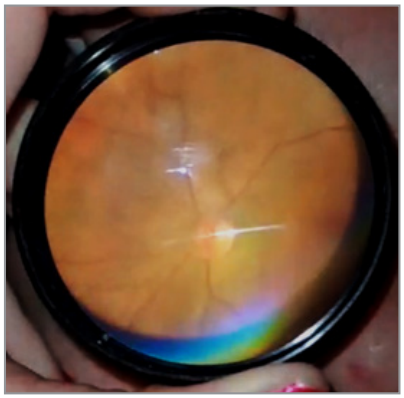


Figure 1: RE Faint peripapillary hypopigmentation is evident towards the upper and lower nasal area.

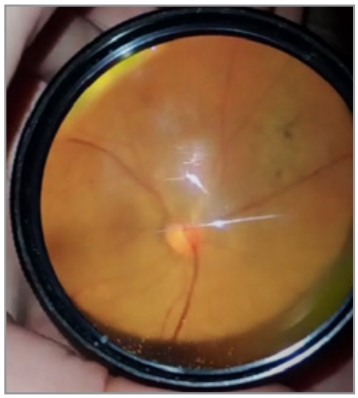


Figure 2: LE The same mild peripapillary hypopigmentation with pigment redistribution is observed.

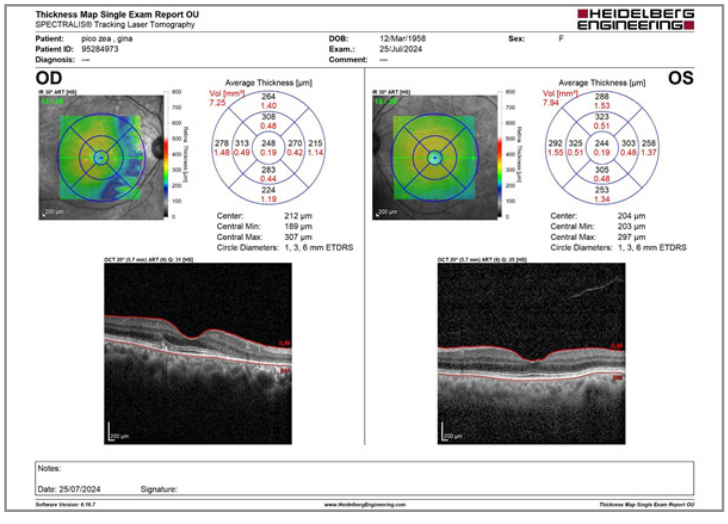


Figure 3: Macular OCT: RE Atrophy of the outer layers of the retina, slight accumulation of subretinal fluid with hyperreflective focus. LE Preservation of retinal layer architecture and foveal thickness.

Figure 4: RE Image of atrophic lesion of external layers with hyperreflectivity of choroid (window effect)

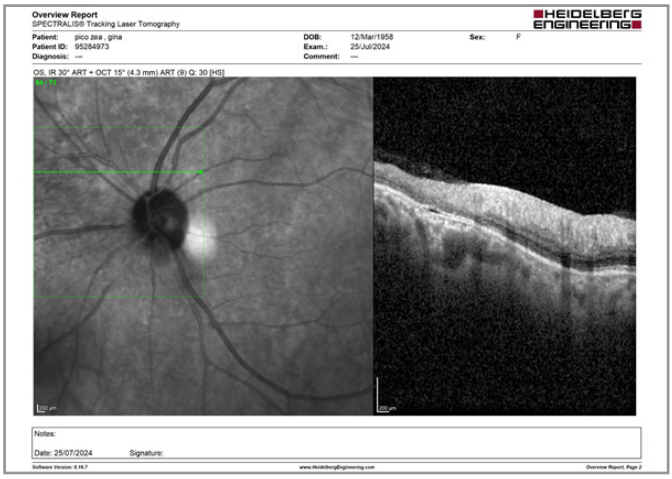


Figure 5: LE atrophy of the outer layers of the retina with window effect and minimal accumulation of subretinal fluid (SRF).

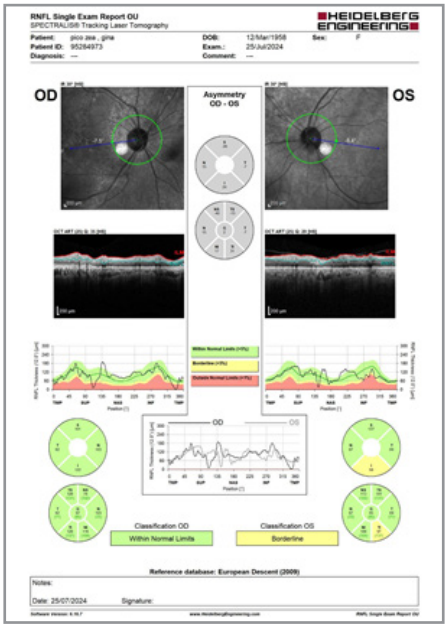
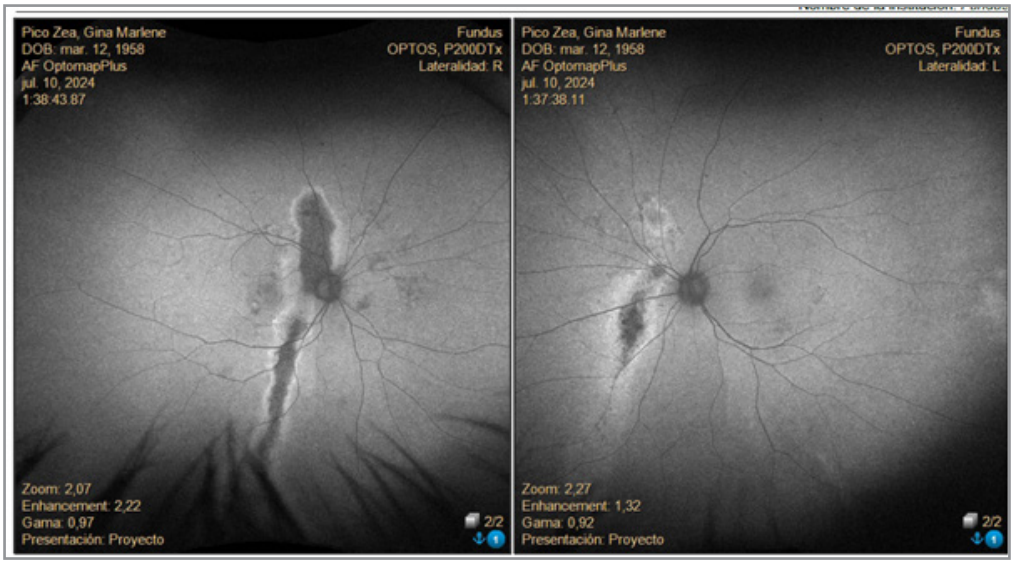


Figure 6: OCT RNFL, fiber layer preserved in both eyes, LE segmentation alteration.



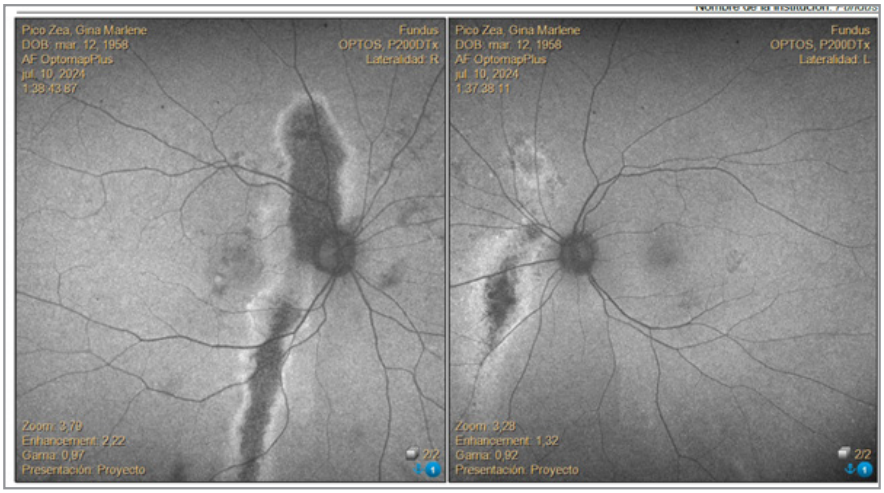


Figure 7: Autofluorescence evidence in compromised areas in the OCT, plaques of hypoautofluorescence accompanied by halos of hyperautofluorescence.

Discussion

Although in the case of our patient, due to her clinical history of previous corticosteroid treatment, associated with OCT images corresponding to areas of RPE atrophy, we were able to infer the presence of chronic central serous chorioretinopathy, autofluorescence not only provided us with data on the affected area of the RPE, but also the halos of hyperautofluorescence are indicating dysfunction of the RPE.

It is known that FAF is related to changes in lipofuscin within the retinal pigment epithelium, with alterations in the outer retina and the subretinal space [7].

In general, the chronicity of CSC has been determined by the subjective memory of the patient, since there was no defined objective method to estimate it. In the case of our patient, who was unaware of the onset of her pathology, AF allowed us to demonstrate the degree of damage to the RPE and presume its chronicity.

In the retrospective study by Lee, W. et. al, the correlation of subjective symptoms and changes evidenced in autofluorescence was analyzed, classifying them into acute, chronic and sequelae according to what was found [5]. In acute CCS, AF showed staining patterns with various levels of fluorescence signal in the area of subretinal detachment (SRD) [25]. In patients with chronic CSC, some showed discrete points with increased FAF intensity corresponding to subretinal precipitates, this was not observed in acute CSC, and some showed an intense area of hyper-FAF just inferiorly adjacent to the SRD [25]. In those patients with sequelae of CSC, the so-called "descending tracts" are found, these tracts showed various FAF patterns corresponding to the state of the RPE. Descending tracts with increased FAF without window defects in FAF were observed, with an area of an intact RPE with a damaged outer photoreceptor layer in OCT. On the other hand, in other cases, descending tracts with decreased FAF have been found, which corresponded to areas with window defects in AF and areas of atrophy of both the RPE and the outer photoreceptor layer in OCT. Those with CSC sequelae revealed focal absolute hypo-FAF and heterogeneous FAF patterns that mixed hyper- and hypointensity. The area of focal absolute hypo-FAF corresponds to the area with atrophic thinning of the RPE in OCT. This pattern was only observed in eyes with CSC sequelae.

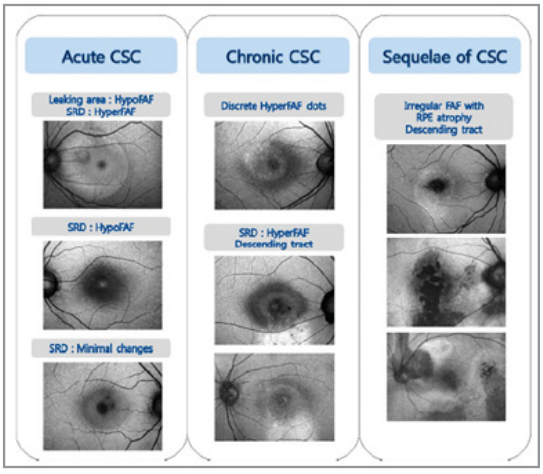


Figure 8: FAF signs according to CSC evolution from Lee, W., Lee, JH. & Lee, B. Fundus autofluorescence imaging patterns in central serous chorioretinopathy according to chronicity. Eye 30, 1336–1342 (2016).

Han J. et al, classified FAF patterns into blocked FAF, mottled FAF, hyper FAF, hyper/hypo FAF or the descending tract in eyes with CSC, according to the chronicity of the pathology [17].

Spider Richard F., et al., in their article mentions that focal hyperpigmentation may indicate not only alterations in the retinal pigment epithelium (RPE), but also of the outer segments of the photoreceptors, in response to chronic exposure of subretinal fluid [6].

In general, it is believed that autofluorescence arises from lipofuscin in the RPE, as mentioned by Ozmert, E, et al., who explains that it could be due to a greater metabolic activity of the retinal pigment epithelium, leading to a more intense accumulation of lipofuscin [7-10].

However, autofluorescence would seem to arise also from the posterior surface of the retina. An important component of RPE lipofuscin is N-retinylidene-N-retinylethanolamine which serves as a useful marker for assessing lipofuscin production [11]. Its precursors, such as dihydro-N-retinylidene-N-retinylphosphatidylethanolamine, N-retinylidene-N-retinylphosphatidylethanolamine and N-retinylidene-N-retinylrhodopsin, all autofluorescent, are formed in the outer segments prior to phagocytosis by the RPE and help to demonstrate the concept that autofluorescence does not necessarily have to arise from the RPE [13, 18]. Phagocytosis of the outer segments would cause autofluorescent material to accumulate in the subretinal space [19, 20, 21]. In rhegmatogenous retinal detachments, the outer segments are dragged from the subretinal space by the flow induced by eye movements and convection currents [14, 15]. In CSC, there is a non-escapeable fluid space for the outer segments detached from the photoreceptors. The accumulation of the outer segments, mixed with possible lipoproteins from the serous fluid that leaks into the subretinal space, can lead to the accumulation of autofluorescent material in these patients, as was observed in the case of our patient in the form of a halo around the hypoautofluorescent image (Image 7) [16].

Atrophy of the RPE and a decrease in the amount of material on the external surface of the retina are observed, suggesting that the atrophy of both occurs in parallel.

In the retrospective study by Zola, M. et al., the earliest change in patients with chronic CSC is diffuse hyperFAF and occurs approximately 4 months after the first episode. In their 3-year follow-up, they demonstrated that eyes with punctate hyperFAF appear more likely to slowly change their pattern, either to diffuse hyperFAF or hypoFAF patterns. In addition, eyes with granular hypoFAF patterns changed to confluent hypoFAF patterns after an average of 2 years. They report that diffuse homogeneous hyperFAF usually indicates the presence of subretinal fluid or reactivation, while punctate hyperFAF spots represent chronicity.

In addition, confluent hypoFAF was shown to be a poor visual prognostic factor [22-24, 26]. In the case of our patient, her right eye presents a confluent hypoFAF pattern with a halo of hyperFAF that is consistent with BCVA of 20/200.

Conclusion

Based on the above, we can conclude that our patient's case, due to the findings found in both the OCT and the autofluorescence image, corresponds to a bilateral chronic central serous chorioretinopathy with a pattern of confluent hypofluorescence with peripheral hyperfluorescence, which is consistent with the atrophy of the RPE and external segments of photoreceptors observed in the OCT, which tells us about a sequelae phase of months of evolution, which correlates with her poor BCVA.

According to our review of the subject, autofluorescence allows us, according to the different patterns found, to be able to orient ourselves in the evolutionary pattern of the disease, and thus be able to guide the therapeutic strategy.

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