Original Article

Choriocapillaris Involvement in Acute Syphilis Posterior Placoid Chorioretinitis is Responsible for Functional Impairment and Points towards an Immunologic Mechanism: A Comprehensive Clinicopathological Approach

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Abstract

Purpose: To evaluate the multimodal imaging of acute syphilitic posterior placoid chorioretinitis (ASPPC) lesions in order to elucidate their pathophysiology which seems to resemble choriocapillaritis as in primary inflammatory choriocapillaropathies such as multifocal choroiditis (MFC) and acute posterior multifocal placoid pigment epitheliopathy (APMPPE).

Methods: Charts of patients with ASPPC seen in the Centre for Ophthalmic Specialised Care, Lausanne, Switzerland, were retrieved. Fundus autofluorescence (FAF), spectral domain optical coherence tomography (SD-OCT), fluorescein angiography (FA), indocyanine green angiography (ICGA), and when available, OCT angiography were analyzed and compared to a case of MFC.

Results: One woman aged 58 and 2 men aged 50 and 31 with unilateral ASPPC were analyzed. All had positive syphilis serologies (venereal disease research laboratory [VDRL] and treponema Pallidum hemagglutination assay [TPHA]). Two were human immunodeficiency virus (HIV) positive. Mean best corrected visual acuity was 0.2 ± 0.1 at presentation and 1.0 for all patients 6 weeks later, after antibiotic treatment for neurosyphilis. All had central scotomata with a mean defect (MD) of 12.2 ± 2.6. Six weeks later, MD values were 3.9 ± 1.7. Microperimetry had a mean score of 25/560 at presentation and recovered to a mean of 444/560 6 weeks later. Multimodal imaging features consisted of FA tissue staining, ICGA hypofluorescent choriocapillaris non-perfusion, FAF hyperautofluorescence, and loss of the ellipsoid line in the diseased areas. The findings were consistent and identical in ASPPC and a case of MFC and pointed toward the involvement of the choriocapillaris.

Conclusions: Similarities seen in multimodal imaging features in ASPPC and choriocapillaritis highlight the role of the choriocapillaris in the pathophysiologic mechanism of both conditions. Inflammatory choriocapillaris non-perfusion triggered by infectious agents seems to be the common pathway through which the eye is reacting.

Keywords: Acute syphilitic posterior placoid chorioretinitis, Choriocapillaris, Fundus autofluorescence, Indocyanine green angiography, Primary inflammatory choriocapillaropathies

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Introduction

One form of posterior syphilis involvement was defined as acute syphilitic posterior placoid chorioretinitis (ASPPC) by Gass *et al.*[1] Acute syphilitic posterior placoid chorioretinitis

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is often at the origin of severe functional impairment in the form of extensive visual field deficits usually associated with a decrease of visual acuity. The clinicopathology of such an evolution deserves to be elucidated and the mechanisms investigated. If treatment is initiated rapidly, the extensive visual field deficits are entirely reversible within 2–4 weeks, indicating that no significant morphologic damage is explaining the functional morbidity, but a vasoactive inflammatory mechanism has to be hypothesized, which, in case, it is not prolonged, can be completely reversed and avoid permanent damage to the chorioretina. The structure potentially corresponding to such a mechanism is the choriocapillaris. In primary choriocapillaritis entities such as acute posterior multifocal placoid pigment epitheliopathy (APMPPE) or idiopathic multifocal choroiditis (MFC), the mechanism is inflammatory choriocapillaris non-perfusion with consequent ischemic damage to the outer retina. Loss of the ellipsoid zone produces scotomata in the involved areas. Unlike what was reported in the initial report on APMPPE by Gass,[2] the primary site of involvement is not the pigment epithelium but the choriocapillaris, as explained by Deutmann and others.[3-5] The most precise investigational modality at our disposal for the choriocapillaris is indocyanine green angiography (ICGA), the advantages being that it gives imaging access to the choroid, including the choriocapillaris, and that it yields dynamic information on the different vascular structures.[6,7] The aim of this work was to collect multimodal imaging findings in three cases of ASPPC and compare them to two episodes of MFC, one of the primary choriocapillaritis entities (primary inflammatory choriocapillaropathies). We generate the hypothesis that the clinicopathological process in ASPPC and in primary choriocapillaritis entities is identical and correspond to each other and correspond to an immunogenic process.

Methods

Charts of cases with the diagnosis of acute syphilitic posterior placoid chorioretinitis (ASPPC) seen in the uveitis clinic of the Centre for Ophthalmic Specialised Care, Lausanne, Switzerland were retrieved, and multimodal imaging appraisal was performed.

Routine clinical examination, including also visual field testing (Octopus, Haag-Streit Co., Bern Switzerland) and microperimetry (SLO/OCT, multichannel OCT, OTI, Toronto, Canada), was completed by fundus photography, fundus autofluorescence (FAF), spectral domain optical coherence tomography (SD-OCT), fluorescein angiography (FA), ICGA using a Heidelberg Retina Angiograph HRA 2 (Heidelberg Engineering Inc., Heidelberg, Germany), and when available, OCT angiography (Optovue Avanti XR, Optovue Co, Fremont, California, USA).

Imaging features of ASPPC were compared to a case of idiopathic MFC, where the clinicopathology is clearly situated at the level of the choriocapillaris.

Results

Patient demographics and characteristics

Out of the eight patients with ocular syphilis seen in our center, three (one woman aged 58 and two men aged 50 and 31) presented with typical ASPPC. Patient demographics and characteristics are shown in Table 1.

All had unilateral (right) involvement. All had positive non-treponemal venereal disease research laboratory [VDRL] and treponemal treponema Pallidum hemagglutination assay [TPHA] antibodies. Two were human immunodeficiency virus (HIV) positive.

Mean best corrected visual acuity (BCVA) was 0.2 ± 0.1 at presentation and 1.0 for all patients 6 weeks later after antibiotic treatment with a regimen for neurosyphilis.

All had central scotomata with a mean defect (MD) of 12.26 ± 2.6 which had recovered almost completely 6 weeks later with a mean MD of 3.93 ± 1.7. Microperimetry collapsed to a mean of 25/560 and had recovered to 444/560 6 weeks later. Laser flare photometry was below 7.7 ph/ms in two patients, showing no significant anterior chamber involvement and measured 35.8 ph/ms in one patient with significant vitritis, also present in the latter patient.

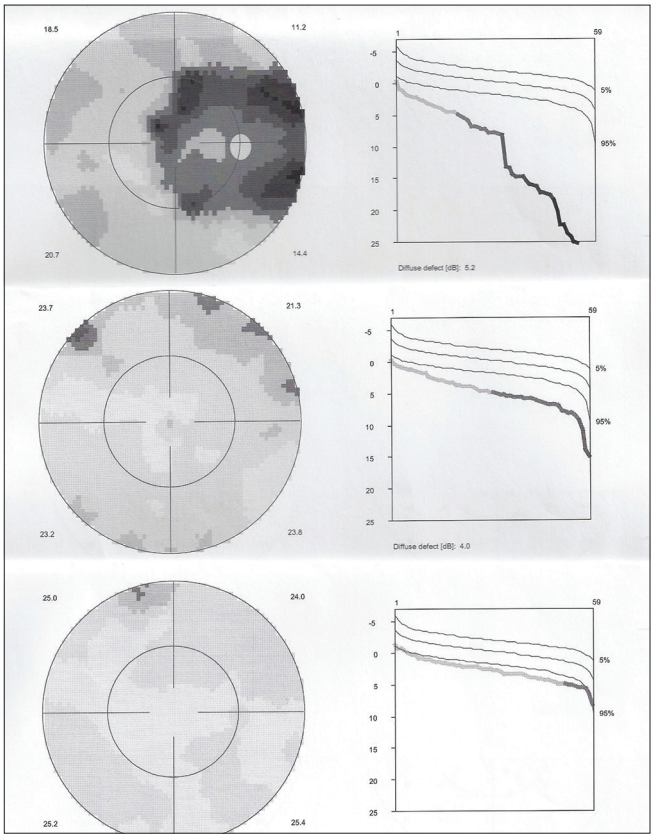

Figure 1: Octopus visual field in a case of acute syphilitic posterior placoid chorioretinitis. At presentation, there is a central scotoma (top) that almost completely recovered after 6 weeks (middle) and was completely normal 1 year later (bottom)

Table 1: Demographics and characteristics

Patients	Age	Positive syphilis serology	HIV	VAOD	VAOD (6 We)	VF-MD (OD)	VF-MD (OD) (6 We)	MP (OD)	MP (OD) (6 We)
Patient 1	58	VDRL and TPHA	Neg	0.16	1	11.6	2.7	28/560	434/560
Patient 2	50	VDRL and TPHA	Pos.	0.16	1	15.1	3.2	22/560	454/560
Patient 3	31	VDRL and TPHA	Pos	0.3	1	10.1	5.9	N/A	N/A

HIV: Human immunodeficiency virus, VAOD: Visual acuity right eye, VAOD (6 we): Visual acuity after 6 weeks, VF-MD (OD): Visual field - mean defect right eye, VF-MD (OD) (6 We): Visual field mean defect right eye after 6 weeks, MP (OD): Microperimetry right eye, MP (OD) (6 we): Microperimetry right eye after 6 weeks, N/A: Not available, VDRL: Venereal disease research laboratory, TPHA: Treponema Pallidum hemagglutination assay

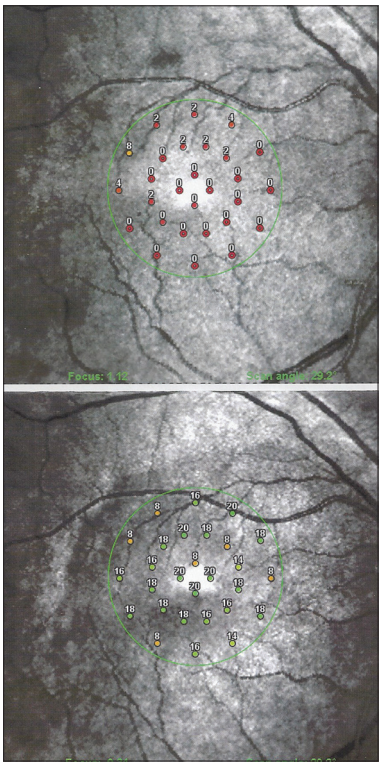

Figure 2: Microperimetry in a case of acute syphilitic posterior placoid chorioretinitis [same patient as Figure 1]. At presentation collapsed microperimetry to 28/560 (top) which almost completely recovered to 434/560 (bottom) at 6 weeks

Multimodal imaging findings in a representative patient (patient 1)

This 58-year-old female presented with a BCVA decreased to 0.16 OD, a central scotoma, and a collapsed microperimetry with a score of 28/560 [Figures 1 and 2].

Fundus photography OD showed a vast round placoid zone in the right posterior pole [Figure 3]. The patient showed the usual imaging pattern with the FA late hyperfluorescence due to staining [Figure 4, left two frames], ICGA hypofluorescence due to choriocapillaris non-perfusion [Figure 4, right two frames], FAF hyperautofluorescence [Figure 5, top] due to loss of photoreceptor outer segments secondary to ischemia of the outer retina seen on SD-OCT, [Figure 5, bottom], and so giving access to the retinal pigment epithelium (RPE) autofluorescence without the usual filter of the retinal photopigments within the photoreceptor outer segments.

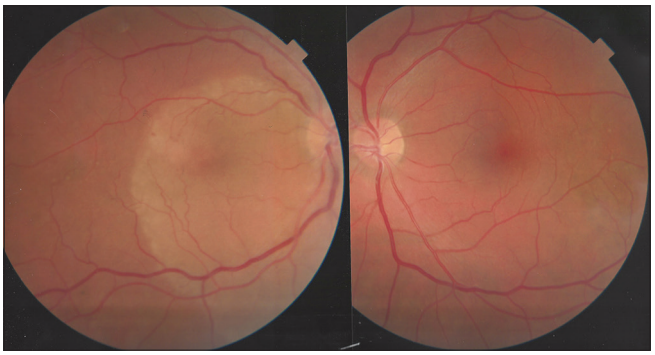

Figure 3: Fundus photography in a case of acute syphilitic posterior placoid chorioretinitis [same patient as Figure 1]. Vast round placoid yellow zone in the posterior pole

SD-OCT, at presentation showed almost complete loss of the ellipsoid line in the area corresponding to FA hyperfluorescence, ICGA hypofluorescence, and FAF hyperautofluorescence. It showed, in addition, a thickening of the inner retina indicating retinal edema [Figure 6].

Several late ICGA findings were noted, including bilateral posterior pole late ICGA hyperfluorescence on both sides [Figure 7] and bilateral peripheral hyperfluorescent pinpoints [Figure 8], indicating potential subclinical involvement of the contralateral eye.

The two other cases of ASPPC showed exactly the same imaging features such as ICGA hypofluorescent areas in the posterior pole due to choriocapillaris non-perfusion [Figures 9 and 10] and gave clear imaging information by SD-OCT on the evolution of the photoreceptor damage as well as retinal edema present in the acute stage and its subsequent evolution [Figure 11].

Angio-OCT performed 10 months after the acute episode in patient 1 showed faint residual perfusion inhomogeneity of the choriocapillaris. No Angio-OCT had been performed at presentation while the contralateral eye was normal (not shown) [Figure 12].

Comparative imaging features in a case of idiopathic multifocal choroiditis choriocapillaritis (2 episodes)

In order to show the similarity of the clinicopathologic process in ASPPC and primary choriocapillaritis entities, we report the images of two episodes of a case of MFC.

A 40-year-old female was seen for a second and third episode of idiopathic MFC. She consulted for photopsias in her left

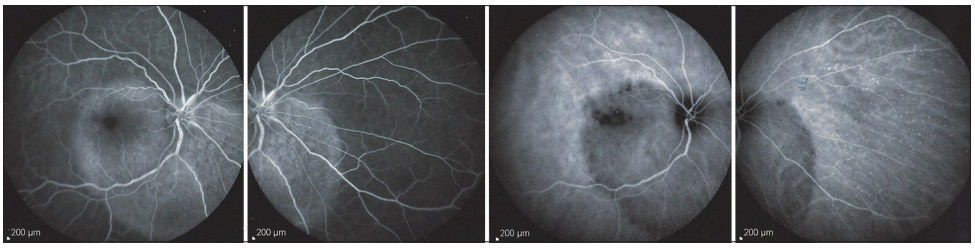

Figure 4: Acute syphilitic posterior placoid chorioretinitis. Fluorescein and indocyanine green angiography (ICGA) (same patient as preceding figures). Fluorescein angiography reveals a vast round zone of hyperfluorescence in the posterior pole (tissue staining) corresponding to ICGA choriocapillaris non-perfusion (dark hypofluorescent area) and fundus autofluorescence hyperautofluorescence [Figure 5]

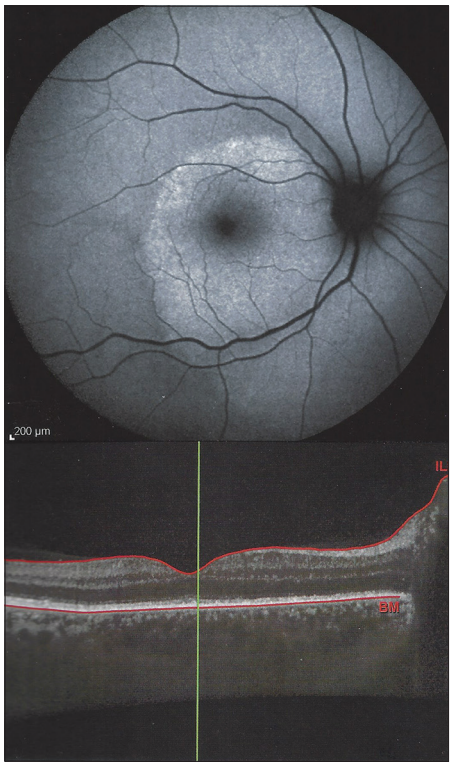

Figure 5: Fundus autofluorescence in a case of acute syphilitic posterior placoid chorioretinitis (same patient as preceding figures). Vast round placoid zone of hyperautofluorescence in the posterior pole corresponding to the fundus yellow discoloration, to the fluorescein angiography hyperfluorescent zone (tissue staining) and to indocyanine green angiography choriocapillaris non-perfusion hypofluorescent area and to the area of loss of the photoreceptor ellipsoid line shown in the bottom image

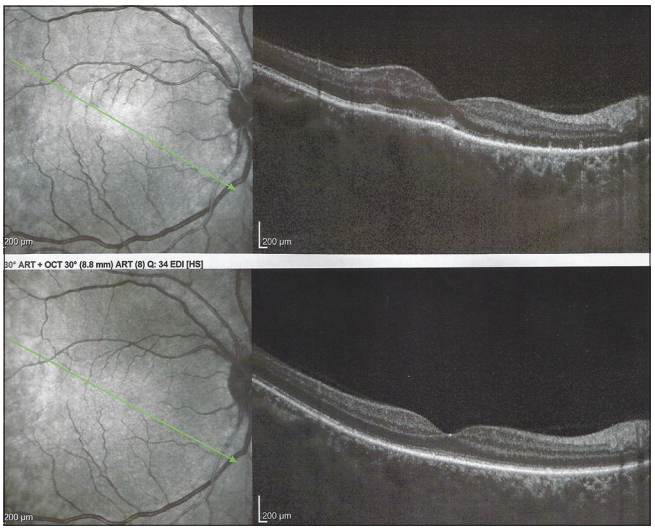

Figure 6: Acute syphilitic posterior placoid chorioretinitis. Optical coherence tomography, enhanced depth imaging (EDI) mode (same patient as preceding figures). Loss of photoreceptor ellipsoid line is clearly identified with overlying retinal edema (top). After 2 weeks, the photoreceptor ellipsoid line has partially recovered (bottom)

eye. FA showed hyperfluorescent late staining around the optic disc and along the superior temporal arcade [Figure 13a]. In the same areas, FAF showed hyperautofluorescence [Figure 13b], and ICGA showed hypofluorescence due to choriocapillaris non-perfusion [Figure 13c], corresponding to a scotoma on octopus visual field [Figure 13d]. SD-OCT showed loss of the photoreceptor ellipsoid line in the areas corresponding to FA, ICGA, and FAF lesions [Figure 13e].

One year later, the patient presented with a recurrence of MFC (3rd episode) in her left eye, showing the same characteristics including fundus foci, FA staining, ICGA hypofluorescence [Figure 14a], FAF hyperautofluorescence [Figure 14b], and a scotoma in the affected area [Figure 14c].

As in ASPPC, there was a loss of photoreceptor outer segments in the acute phase and an increased retinal thickness due to retinal edema seen on SD-OCT. After 6 months, SD-OCT showed deceased thickness, despite the restitution of the ellipsoid line, explained by the fact that the retinal edema had resolved [Figure 14d]. In contrast to ASPPC, the recovery of photoreceptor outer segments took much longer after the introduction of immunosuppressive therapy, which was introduced to avoid further recurrences.

Summary of common multimodal features in acute syphilitic posterior placoid chorioretinitis and multifocal choroiditis choriocapillaritis

As shown in Table 2, all three cases of ASPPC and two episodes of a MFC case showed the following common and constant findings: (1) a posterior pole scotomata [Figures 1, 13d, and 14c], (2) FA early perfusion delay (not shown) and late diffuse hyperfluorescence in the area of the scotoma indicating staining of dye in the retinal tissue [Figures 4, 13a and 14a], (3) ICGA

early non-perfusion (not shown) and ICGA hypofluorescence through all angiographic phases with large choroidal vessels still visible in transparency indicating choriocapillaris non-perfusion corresponding to the zone of FA hyperfluorescence [Figures 4, 13c and 14a], (4) FAF-hyperautofluorescent zones corresponding to FA and ICGA lesions in the 2 ASPPC cases where the information was available and the two episodes of MFC [Figures 5, 13b and 14b], (5) loss of the photoreceptor ellipsoid line on SD-OCT scans in the area of FA, ICGA, and FAF lesions in the 2 ASPPC cases where the information was available and the two episodes of MFC [Figures 5, 6, 11, 13e and 14d], and (6) thickened edematous retina in the acute phase, above the zone deprived of the ellipsoid line, shown on SD-OCT scans in the two ASPPC cases where the information was available and in MFC [Figures 6, 11 and 14d].

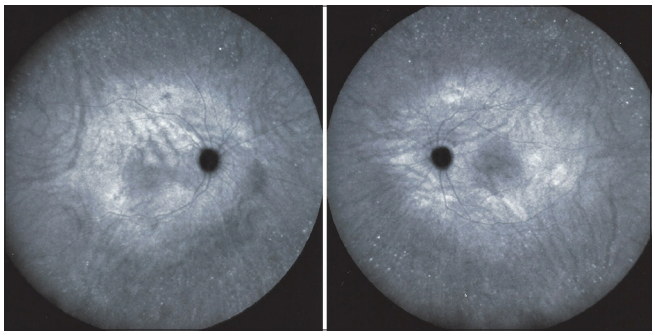

Figure 7: Acute syphilitic posterior placoid chorioretinitis. Late indocyanine green angiography (ICGA) frames in convalescent phase (same patient as preceding figures). Bilateral late ICGA posterior pole hyperfluorescent disciform area

Other findings irrelevant to construct the scenario of events were at least slight ICGA disc hyperfluorescence in all four cases, an indication of substantial inflammation as on ICGA, the disc usually remains dark. In two ASPPC cases, there were late bilateral ICGA hyperfluorescent pinpoints in the periphery [Figure 8]. In one case, there was bilateral late posterior pole disciform ICGA hyperfluorescence [Figure 7].

Integrative and comprehensive scenario for primary and secondary choriocapillaritis based on multimodal imaging features [Figures 15 and 16]

The first event to occur is inflammatory choriocapillaris

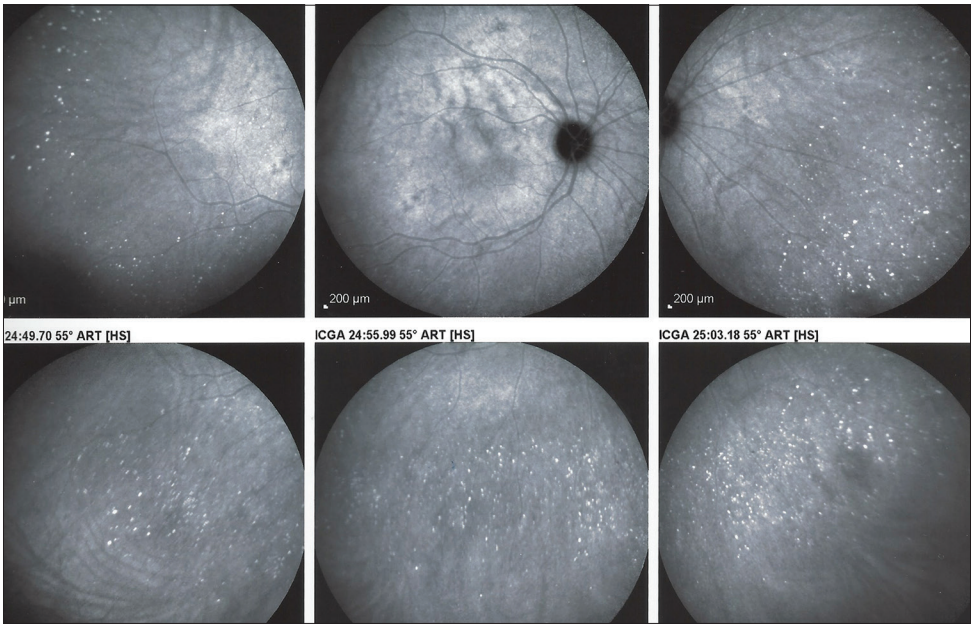

Figure 8: Acute syphilitic posterior placoid chorioretinitis. Convalescent indocyanine green angiography 1 year after the acute phase. Numerous hyperfluorescent pinpoints in the periphery (also present in the contralateral eye [not shown])

Table 2: Imaging summary

Patients	Central scotoma	FA late hyperfl/ tissue staining	Hot disc	ICGA disc hyperfl.	FAF hyperfl	OCT loss of ellipsoid line	OCT acute phase retinal edema	Other
Patient 1	Yes	Yes	Yes	Yes	Yes	Yes	Yes	ICGA peripheral pinpoints ODS
Patient 2	Yes	Yes	Yes.	Yes	Yes	Yes	Yes	ICGA peripheral pinpoints ODS
Patient 3	Yes	Yes	Yes	Yes	N/A	N/A	N/A	N/A
MFC case	Yes	Yes	Yes	±	Yes	Yes	Yes	No pinpoints

MFC: Idiopathic multifocal choroiditis, FA late hyperfl/tissue staining: Fluorescein angiography late hyperfluorescence/retinal tissue staining, ICGA: Indocyanine green angiography, Hyperfl: Hyperfluorescence, FAF: Fundus autofluorescence, OCT: Optical coherence tomography, N/A: Not available, ODS: Right and left eye

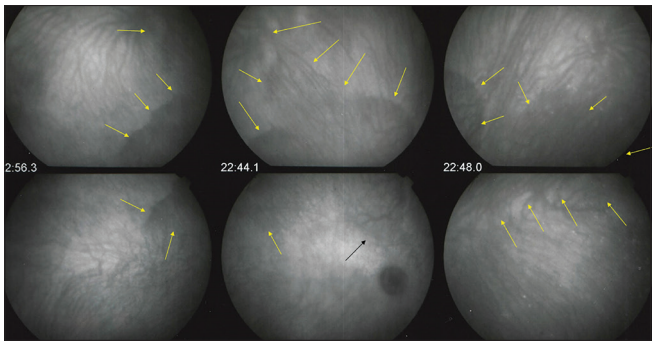

Figure 9: Acute syphilitic posterior placoid chorioretinitis. Indocyanine green angiography (ICGA) (patient 2). Extended zone of ICGA hypofluorescence indicating choriocapillaris non-perfusion

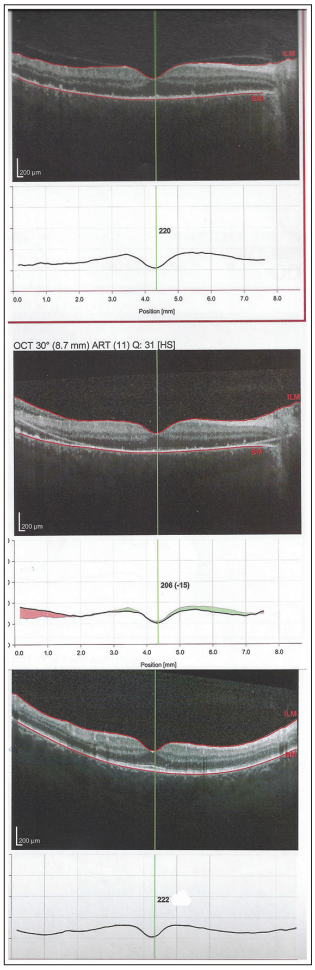

Figure 11: Acute syphilitic posterior placoid chorioretinitis. spectral domain optical coherence tomography scans showing evolution in time of ellipsoid line and retinal thickness (case 2). The sequential optical coherence tomographies (OCTs) show (1) Increased retinal thickness/edema despite loss of ellipsoid zone at presentation (top OCT scan). (2) After 1 week of antibiotic treatment, retinal thickness decreased by 15 microns due to resolution of retinal edema, while ellipsoid line is still missing (middle OCT scan). (3) Lower OCT scan performed 5 weeks later shows re-increased retinal thickness without retinal edema, explained by the reconstitution of the ellipsoid line (lower OCT scan)

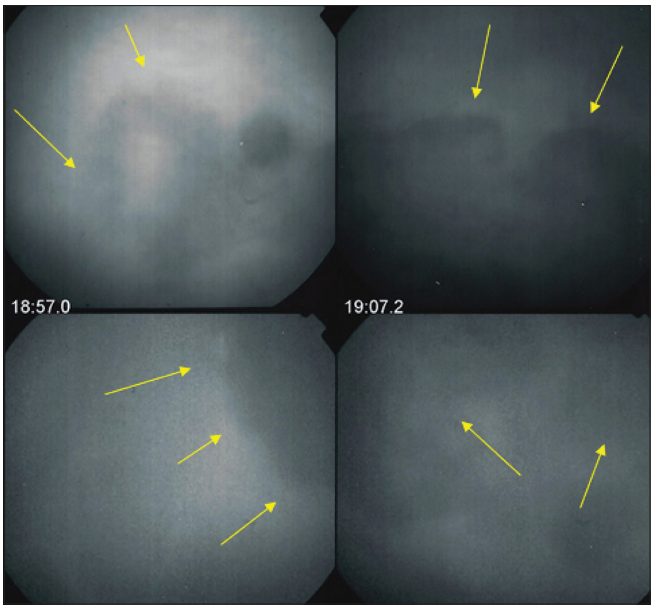

Figure 10: Acute syphilitic posterior placoid chorioretinitis. Indocyanine green angiography (ICGA) (patient 3). Extended zone of ICGA hypofluorescence indicating choriocapillaris non-perfusion

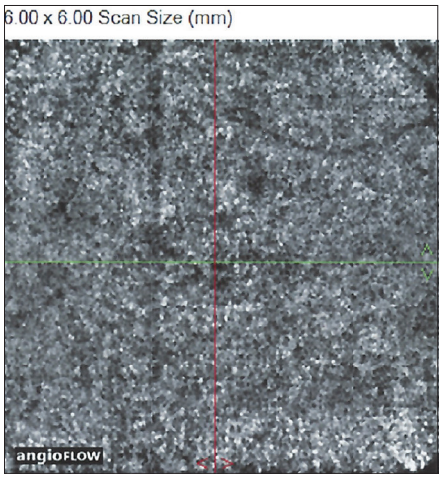

Figure 12: Angio-optical coherence tomography in acute syphilitic posterior placoid chorioretinitis (ASPPC). Scan taken 10 months after acute episode of ASPPC showing residual inhomogeneity of the choriocapillaris circulation

non-perfusion triggered by an infectious agent, an unknown potential virus in primary inflammatory choriocapillaropathies or bacteria (here T. pallidum), or other known agents in secondary inflammatory choriocapillaropathies, as described in a previous report.[8] This causes ischemia of the outer retina and the disruption/loss of the photoreceptor ellipsoid line. In reaction to outer ischemia, inner retinal vessels develop a compensatory permeability increase, causing the FA intraretinal hyperfluorescent staining and sometimes pooling (not shown in this report). The dilatation of inner retinal vessels is well illustrated in Figure 16. This causes retinal edema, well shown in Figures 6, 11, and 14d.

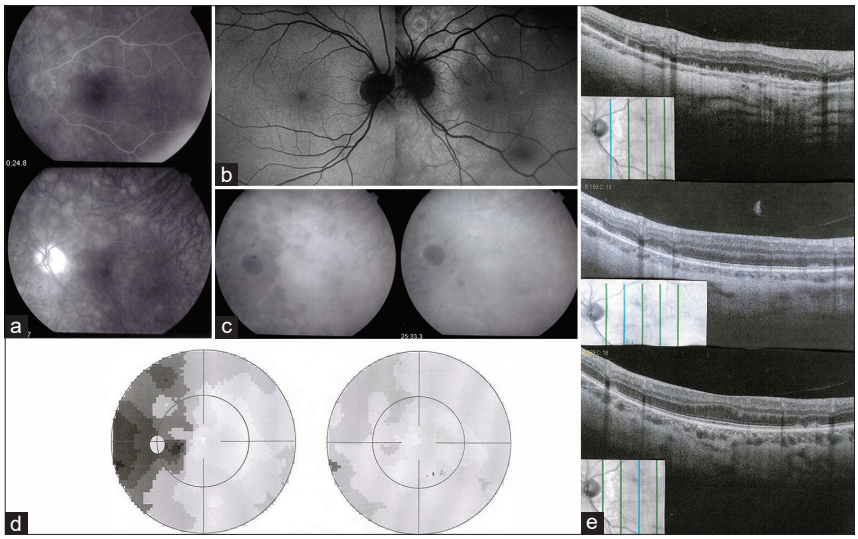

Figure 13: (a) Idiopathic multifocal choroiditis (MFC). Fluorescein angiography (FA) (left eye). Hot disc and vast zone of progressive hyperfluorescence around disc and along superior temporal arcade corresponding to fundus autofluorescence (FAF) hyperautofluorescence [Figure 12b] and indocyanine green angiography (ICGA) choriocapillaris non-perfusion [Figure 12c]. (b) Idiopathic MFC. FAF. Hyperautofluorescence in the left eye (right picture) in the same area as fluorescein hyperfluorescence [Figure 13a] and as ICGA hypofluorescence/choriocapillaris non-perfusion [Figure 13c]. In comparison, the right eye has a normal FAF (left picture). (c) Idiopathic MFC. ICGA. (left eye) ICGA hypofluorescence due to choriocapillaris non-perfusion (left picture) in the same area as fluorescein hyperfluorescence [Figure 12a] and as hyperautofluorescence [Figure 12b] The right picture shows repermeabilisation of choriocapillaris 1 month after initiation of systemic corticosteroid therapy. (d) Idiopathic MFC. Octopus visual field. (left eye) Scotoma around the optic disc extending towards the superior temporal arcade and corresponding to the area of FA hyperfluorescence, FAF hyperautofluorescence ICGA hypofluorescence. Visual field almost entirely recovered, 1 month after introduction of systemic corticosteroids. (e) Idiopathic MFC. Spectral domain optical coherence tomography (left eye). Diffuse loss of the photoreceptor layer in the scan going through FA hyperfluorescent, FAF hyperautofluorescent and ICGA hypofluorescent area (top scan). The middle and bottom scans are going through areas at the limits of the affected area with some disruption of the photoreceptor layer

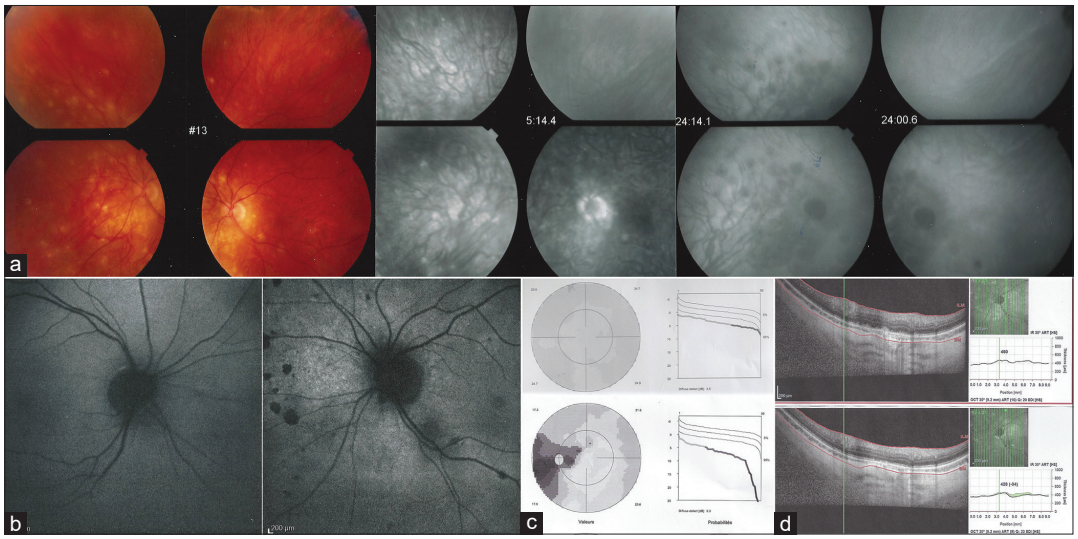

Figure 14: (a) Idiopathic multifocal choroiditis (MFC) recurrence, left eye. Fundus, fluorescein angiography (FA) and indocyanine green angiography (ICGA). Characteristic MFC in fundus picture (left); peripapillary and nasal staining on FA; ICGA hypofluorescence due to choriocapillaris non-perfusion in the same area as FA staining. (b) Idiopathic MFC recurrence, left eye. Fundus autofluorescence. Characteristic peripapillary and nasal hyperautofluorescence in the same area as FA and ICGA lesions. (c) Idiopathic MFC recurrence, left eye. Octopus visual field. Visual field had recovered since the previous MFC episode (top) [Figure 13d] but a new peripapillary scotoma was noted, due to a new recurrence (3rd episode) of MFC (bottom). (d) Idiopathic MFC 2nd recurrence, left eye. Spectral domain optical coherence tomography. Severe disruption of ellipsoid line and retinal edema during acute phase of MFC recurrence (top). Bottom scan taken 6 months later shows decreased retinal thickness (−34 μ) despite reconstitution of photoreceptor outer segments, explained by the resolution of the retinal edema

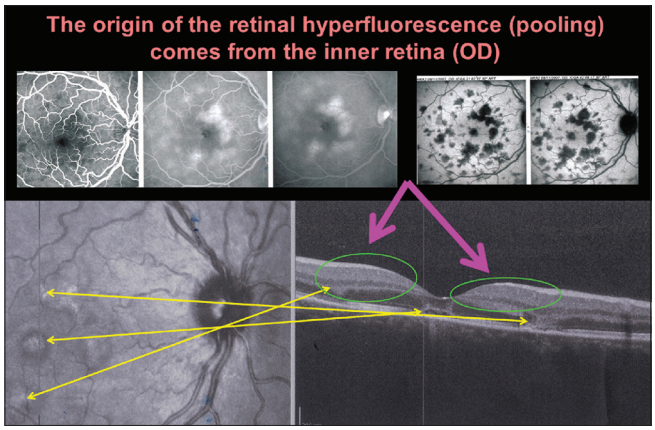

Figure 15: Scenario of clinicopathologic sequence in choriocapillaritis (in choriocapillaritis case of acute posterior multifocal placoid pigment epitheliopathy). Multiple zones of choriocapillaris non-perfusion on indocyanine green angiography in intermediate and late angiographic phase (top right two images). The choriocapillaris non-perfusion can also be seen on the early fluorescein frame (top far left). Because the outer retina is ischemic, there is increased permeability of the inner retinal vessels at the origin of exudation causing staining and even pooling of dye and retinal edema in the areas of choriocapillaris non-perfusion (top two middle frames). The two lower images show dilated vessels on the optical coherence tomography (right) in relation with lesion sites (left). Inner retinal vessel dilatation and permeability is better shown in Figure 16

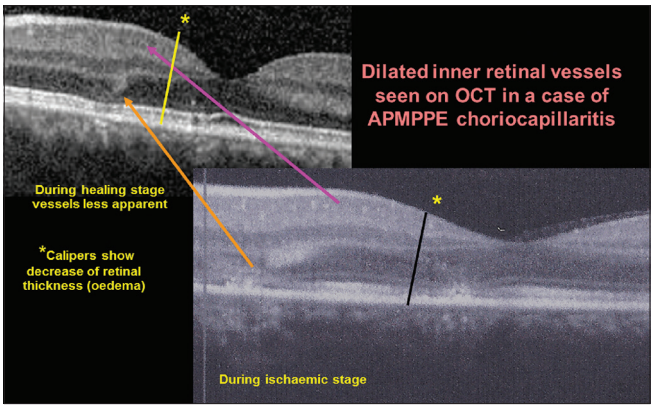

Figure 16: Scenario of clinicopathological sequence in choriocapillaritis case of acute posterior multifocal placoid pigment epitheliopathy (APMPPE). In the active stage of APMPPE (lower optical coherence tomography [OCT] scan) there is retinal edema and inner vessels are more well-recognized because they are dilated and have increased permeability in the areas of photoreceptor ellipsoid line disruption. In the convalescent phase (upper OCT scan) thickness of the retina has diminished and inner retinal vessels are less well visible

Discussion

We report the multimodal imaging features of a series of three patients with ASPPC. Findings were consistent in all three cases and corresponded to the first description by Gass as far as (1) fundus aspect, (2) FA description with early hypofluorescence and late staining, (3) as well as rapidity of resolution after antibiotic treatment are concerned.[1] The constellation of FA, FAF, ICGA, OCT, and OCT angiography findings corresponded and pointed towards a major initial inflammatory involvement of the choriocapillaris in the lesion process. We are thus in line with an earlier report stressing the role of the choriocapillaris in ASPPC as well as with a very recent report indicating choriocapillaris flow reduction.[9,10] Our findings, however, did not support the thesis of the RPE being the origin of inflammatory events in ASPPC that was put forward in a past report.[11] Clinically our findings were in line with the results reported by Eandi *et al*.[12] and confirmed some of the angiographic findings by Mora *et al*.[13]

To support our hypothesis of the predominant choriocapillaris involvement in ASPPC, we compared the multimodal findings in our three patients to two episodes of a case of idiopathic MFC where the crucial role of the choriocapillaris in the development of the disease has been commonly accepted, as in other primary inflammatory choriocapillaropathies (primary choriocapillaritis entities), such as APMPPE.[14-19] As can be seen in Table 2, the imaging features, FA, FAF, ICGA, and OCT, do absolutely correspond in both conditions. While the sequence of events after initial choriocapillaris damage has been clearly exposed in our study and in others, the pathophysiology leading towards inflammatory choriocapillaris non-perfusion in primary choriocapillaritis entities is unknown. In about half of these conditions, symptoms of a viral flu-like infection are preceding the eye involvement, pointing towards a virus or other agent possibly triggering an immune reaction at the level of the choriocapillaris that in most cases responds to steroidal and/or non-steroidal immunosuppression.[20,21] Similarly, for ASPPC, while the bacterial infection is clearly at the origin of the inflammatory choriocapillaris non-perfusion, the reason why it develops in a certain percentage of ocular syphilis cases is unexplained. A recent publication showed that an erroneous prednisone treatment in a case of ASPPC resolved the placoid lesions while the vasculitis and papillitis persisted. Furthermore, the placoid lesions recurred when the prednisone dosage was lowered. Once specific antibiotic therapy was given, the placoid lesions, vitreous haze, vasculitis, and papillitis resolved.[22] This points toward an immunopathologic explanation of the particular clinical presentation of ASPPC. The choriocapillaris involvement and its consequences with its stereotyped imaging features in both situations might be a common pathway the choriocapillaris takes to react to an infection involving a potential unknown virus in primary inflammatory choriocapillaropathies and the known agent *Treponema Pallidum* in the secondary choriocapillaritis ASPPC.

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Conflicts of interest

There are no conflicts of interest.

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