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Acute Syphilitic Posterior Placoid Chorioretinitis: A Narrative Literature Review

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A B S T R A C T

Acute syphilitic posterior placoid chorioretinitis (ASPPC) is defined by the presence of a placoid, yellowish, often solitary lesion, typically involving the areas of the temporal vascular arcades, the juxtapapillary retina, and/or the macular region, with the lesions being present at the level of the outer retina and retinal pigment epithelium (RPE). The visual acuity may range from no light perception to 20/20. There may be associated anterior chamber inflammation and vitritis. Therapy should include antibiotics for neurosyphilis. The role of corticosteroids is controversial. HIV coinfection should always be tested for. With appropriate therapy, the prognosis seems to be good.

1. Introduction

In 1988, Souza et al.[1] described three young patients with "unilateral central chorioretinitis", associated with severe vision loss. The patients were seropositive for syphilis and responded dramatically to penicillin treatment.[1] The term acute syphilitic posterior placoid chorioretinitis (ASPPC) was first described by Gass et al. in 1990.[2-4] These studies reported on six patients with evidence of secondary syphilis who presented with vision loss due to placoid lesions at the level of the pigment epithelium. The lesions were yellowish, large, and located in the juxtapapillary and macular regions.[2] Since then, ASPPC has been associated with ocular syphilis, with or without HIV coinfection.

Acute syphilitic posterior placoid chorioretinitis (ASPPC)

ASPPC is a rare ocular manifestation of syphilis. It has initially been associated with immune suppression (such as HIV+ status) but is now reported in both immunocompetent and immunocompromised patients. Clinically, ASPPC is defined by the presence of a placoid, yellowish, often solitary lesion, typically involving the areas of the temporal vascular arcades, the juxtapapillary retina, and/or the macular region, with the lesions being present at the level of the outer

retina and retinal pigment epithelium (RPE), as seen in Figure 1. It can be unilateral or bilateral with a wide visual acuity (VA) range from 20/20 to no light perception.[4] Different types of visual field defects have also been described. Burkholder et al.[5] described central and peripheral scotomata in their cases. Optical coherence tomography (OCT) may show thickening of the subretinal (subfoveal) choriocapillaris complex, choroidal punctate hyperreflectivity, nodular RPE thickening, or irregularity, disruption and loss of the ellipsoid zone, transient localized subretinal fluid, and punctate, hyperreflective, intraretinal inflammatory infiltrates (Figure 2).[3-5] It has been suggested that the hyperreflective lesions at the RPE, as seen on OCT, may be due to circulating spirochaetes entering from the choroidal circulation. Reaching the outer retina, they may also cause subretinal edema[4,6]. Christakopoulos C et al.[6] reported a case of ASPPC with typical placoid circular yellowish bright margins. On OCT, there were disruptions of the ellipsoid zone and external limiting membrane and multiple hyper-reflective vertical finger-like hyperdensities extending from the outer to the inner retina called the "pitchfork sign." The other eye had serous macular detachment and linear deformity attached to the RPE.[6]

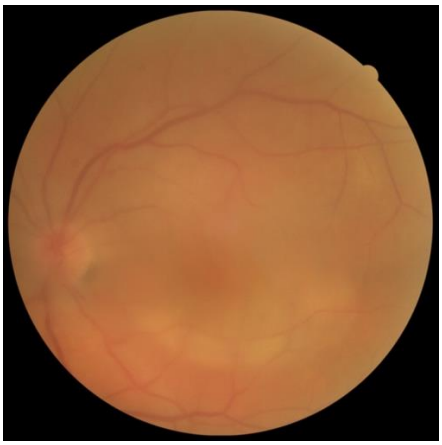

Figure 1. Fundus photograph of the left eye in a patient with ASPPC: vitritis; hyperemia of the optic disc; deep, subretinal, yellow placoid lesions in the posterior pole. (Courtesy of Gueorgui Markov).

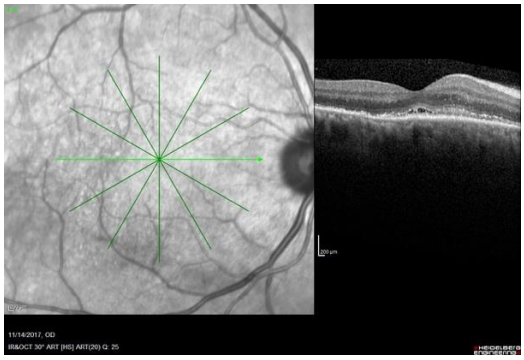

Figure 2. OCT of the macula in a patient with ASPPC: hyperreflective vitreous body opacities (vitritis), neurosensory retinal thickening nasally, subfoveal RPE detachment, alterations on the level of the RPE and photoreceptors, thickened choroid with hyperreflective dots. (Courtesy of Gueorgui Markov).

Fluorescein angiography (FA) discloses early hypofluorescence (due to choriocapillaris non-perfusion) with late hyperfluorescent due to late staining[3], as seen in Figure 3. Autofluorescence reveals diffuse areas of mottled hypo-and hyperautofluorescence (due to RPE and photoreceptor loss and dysfunction).[3] All patients with ASPPC should be tested for coinfection with HIV and for neurosyphilis. Eandi et al.[7] reported on 16 syphilis (+) patients with active ASPPC, 9 of whom (53,6%) were HIV (+), with a mean CD4-cell count of 250 cells/μL. Zamani et al.,[8] reported a case of corticosteroid-induced modulation of ASPPC in syphilis (+) patients. The patient had syphilitic uveitis, but developed placoid macular lesions when he started taking oral prednisolone. The ASPPC resolved after discontinuing the corticosteroid therapy. ASPPC can also be accompanied by central nervous system involvement and mucocutaneous lesions.

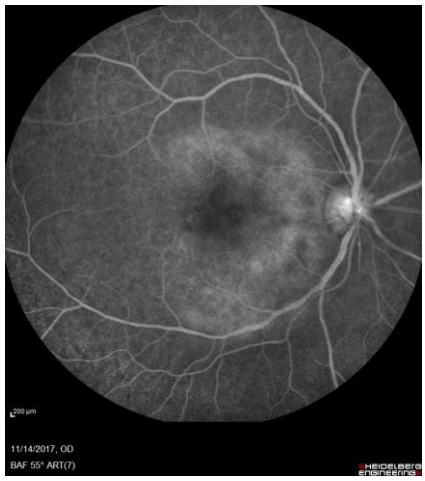

Figure 3. FA of OD of a patient with ASPPC: “hot disc”, placoid hyperfluorescence between the temporal arcades, areas of focal periphlebitis. (Courtesy of Gueorgui Markov).

The natural course of the disease is still unclear since most patients with ASPPC receive antimicrobial therapy. Some of the reported cases of ASPPC showed spontaneous resolution.[4,9] Franco et al.,[9] reported a case of severe unilateral ASPPC. The patient was HIV (-) and syphilis (+). The initial visual acuity of the affected eye was hand movements (HM). When observed again, VA of the affected eye had spontaneously recovered to 20/20. Herbort et al.,[3] reported on three cases of unilateral ASPPC, two of whom had HIV coinfection. After 6 weeks of antimicrobial treatment, all the affected eyes had visual acuity of 20/20[3]. Burkholder et al.[5] described 3 patients with unilateral ASPPC. The first patient, who was HIV (-), had an initial VA of the affected eye of 20/50 and a central scotoma.[5] He received IV penicillin G 24 million units/qd[5]. After a 3-month period, the visual acuity of the affected eye was 20/20. The second patient had peripheral scotoma, an initial VA of 20/63 of the affected eye, and 1+ cells in the anterior chamber. He received 2 g IV ceftriaxone qd for 14 days and VA improved to 20/25, with a resolution of inflammation in the same eye. The third patient, who was HIV+, had initial VA in the affected eye of 20/60 and cells in the anterior chamber. He received IV penicillin G and, in the follow-up period of three months, had improved visual acuity to 20/25 with a resolution of inflammation.[5] Christakopoulos et al.,[6] reported a patient with bilateral ASPPC. The VA of one eye was HM and of the other - 20/20. He had mild vitritis in both eyes and 1+ anterior chamber cells. The patient received IV penicillin G, 6 million IU t.i.d., and

methylprednisolone at a starting dose of 80 mg/qd and subsequently 40 mg/qd for 1 week, followed by IV ceftriaxone 2 g for 10 days. During the follow-up period, the patient had improved, reaching a VA of 20/20 for both eyes in the seventh month.[6]

2. Conclusion

ASPPC has characteristic lesions seen on ophthalmoscopy, OCT, FA, and FAF. With appropriate antimicrobial treatment, ASPPC seems to have a good prognosis, but the natural course of this condition remains unclear. All patients with ASPPC should be tested for HIV and neurosyphilis.

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