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Case report
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Posterior placoid chorioretinitis: a disguise of ocular syphilis

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Abstract

Background: To report a case of ocular syphilis presenting as acute syphilitic posterior placoid chorioretinitis.
Case presentation: A 38-year-old male presented with reduced vision in the left eye for 2 weeks. Best-corrected Snellen visual acuity was 6/60 in the affected eye and 6/9 contralaterally. Fundoscopy revealed mild vitritis, hyperaemic optic disc, a yellowish placoid lesion at the macula, and choroiditis. Optical coherence tomography of the macula showed disruption at the outer retinal layers and hyperreflective lesions in the choriocapillaris. Fundus fluorescein angiography showed a “hot” disc and patches of vasculitis. The diagnosis was delayed by spontaneous clinical resolution and false negative non-treponemal test due to the prozone phenomenon. Due to high clinical suspicion, the test was repeated with a diluted serum sample that turned out to be positive and was treated with intravenous benzylpenicillin.
Conclusion: A placoid lesion at the macula should raise high suspicion of ocular syphilis.

Keywords: chorioretinitis, education, placoid lesion, syphilis

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Korioretinitis plakoid posterior: penyamaran penyakit sifilis okular

Abstrak

Latar belakang: Untuk melaporkan satu kes sifilis okular yang dikenali sebagai korioretinitis plakoid posterior sifilis akut.
Pembentangan kes: Seorang lelaki berusia 38 tahun hadir dengan penglihatan yang berkurangan pada mata kiri selama dua minggu. Ketajaman penglihatan Snellen yang paling baik diperbetulkan ialah 6/60 pada mata yang terjejas dan 6/9 secara kontralateral. Fundoskopi mendedahkan vitritis ringan, cakera optic yang hiperemik, luka plakoid kuning pada makula dan koroiditis. Tomografi koheren optik pada makula menunjukkan gangguan pada lapisan luar retina dan luka hiper-reflektif dalam koriokapilaris. Fundus fluorescein angiography menunjukkan cakera 'panas' dan tompok vaskulitis. Diagnosis telah tertangguh disebabkan oleh resolusi klinikal spontan dan ujian bukan-treponemal yang negatif palsu kerana 'fenomena prozon'. Disebabkan syak wasangka klinikal yang tinggi, ujian diulangi dengan sampel serum yang dicairkan dan keputusan ternyata positif dan kes dirawat dengan benzylpenicillin intravena.
Kesimpulan: Luka plakoid di makula sepatutnya menimbulkan syak wasangka yang tinggi terhadap sifilis ocular.

Kata kekunci: korioretinitis, pendidikan, luka plakoid, sifilis

Introduction

Syphilis is a sexually-transmitted disease caused by the spirochete *Treponema pallidum*.[1,2] It is re-emerging in Malaysia with a reported incidence rate per 100,000 population of 10.63 in 2020, doubling from 5.7 in 2012.[3] Ocular involvement is reported to range from 1% to 8% and usually occurs during the secondary stage.[1,2] It is known as the "great mimicker" owing to its ability to manifest in a wide variety of clinical presentations. Posterior segment involvement is more common, of which chorioretinitis is the commonest.[4] Here, we present a specific pattern of ocular syphilis known as acute syphilitic posterior placoid chorioretinitis (ASPPC) and discuss the possible explanations for its pathophysiology.

Case presentation

A 38-year-old Malay male presented with painless central blurring of vision in the left eye associated with floaters for 2 weeks. Upon systemic review, he developed a scrotal ulcer that preceded the current presentation. He stated having unprotected sexual intercourse with multiple partners in the past. There was no skin rash and his past ocular and medical history was unremarkable.

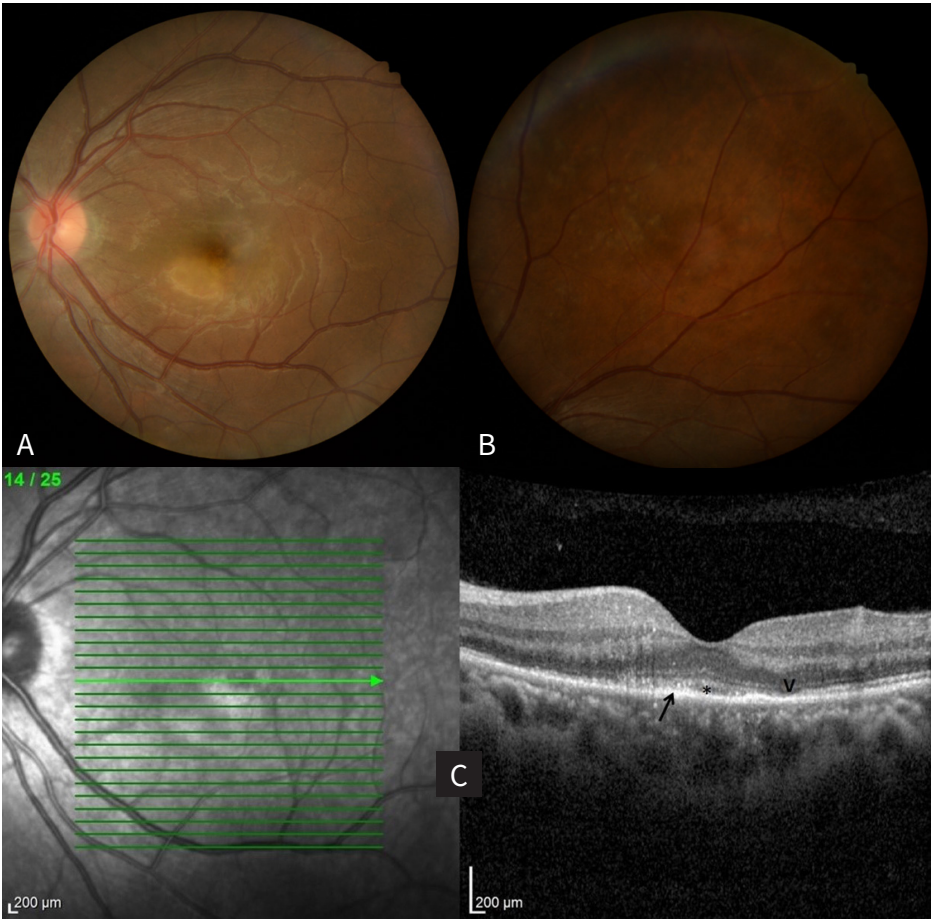

Fig. 1. (a) Fundus of the left eye showing a hyperaemic disc with a placoid lesion at the macula. *(b)* Multiple choroiditis lesions seen in the superior retina. *(c)* OCT of the left macula showing nodular thickening and hyperreflectivity of the RPE layer *(arrow)*, disruption of the photoreceptor inner segment-outer segment junction *(arrowhead)*, minimal subretinal fluid *(asterisk)*, and hyperreflective dots at the choriocapillaris-RPE-photoreceptor complex *(orange arrow)*.

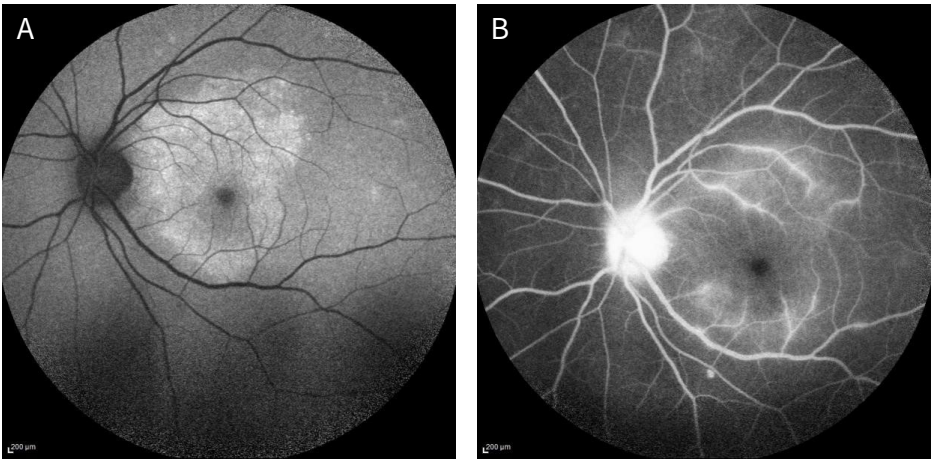

Fig. 2. (a) FA showed an area of hyperautofluorescence larger than the placoid lesion seen clinically. *(b)* FFA showed a "hot" optic disc as well as vasculitis at the perifoveal region and peripheral retina.

His best-corrected Snellen visual acuity (BCVA) was 6/60 in the left eye and 6/9 in the right eye. Relative afferent pupillary defect was negative. Anterior segment examination was unremarkable and intraocular pressure was 16 mmHg bilaterally. Fundus examination of the left eye revealed mild vitritis, hyperaemic optic disc, and a yellowish placoid lesion measuring around one disc diameter located inferonasal to the fovea (Fig. 1a). There was also a cluster of choroiditis lesions in the superior retina (Fig. 1b). Fundus examination of the right eye was unremarkable. Optical coherence tomography (OCT) of the left macula showed disruption at the ellipsoid zone (Fig. 1c), while the right eye showed no abnormalities. He was tested for syphilis and tuberculosis, and treated with topical steroid in the left eye.

Upon review 5 days later, the left eye BCVA had improved to 6/30 and the placoid lesion had mostly resolved on fundoscopy. Fundus autofluorescence (FA) showed that the placoid lesion and choroiditis lesions at the superior retina was more extensive than was clinically visible (Fig. 2a). Fundus fluorescein angiography (FFA) showed a "hot" optic disc and vasculitis at the perifoveal region and peripheral retina (Fig. 2b).

Laboratory investigations indicated normal full blood count as well as kidney and liver profile but raised erythrocyte sedimentation rate of 25 mm/hr. Chest X-ray was unremarkable and tuberculin skin test was negative (5 mm). The first serum sample for rapid plasma (RPR) test was negative, but due to high clinical suspicion, a second diluted serum sample was requested and turned out to be of high titre (1:512). This was further confirmed with a positive *Treponema pallidum* hemagglutination test. HIV testing was negative. Unfortunately, the patient

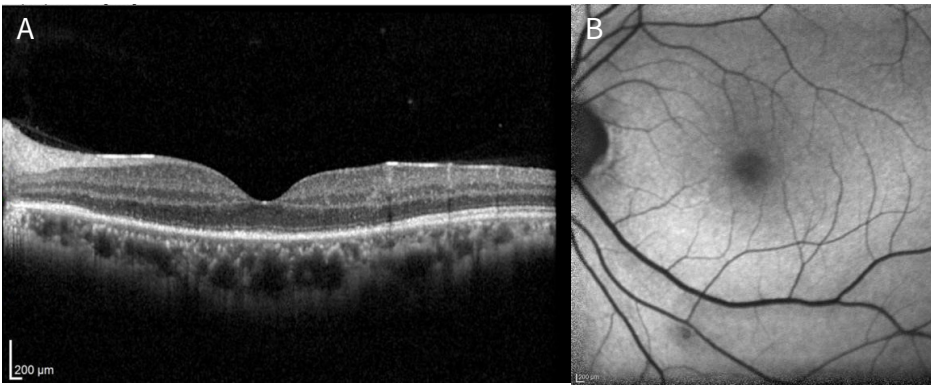

Fig. 3. (a) OCT and *(b)* FA of the macula showing resolution of previous changes.

defaulted subsequent follow-up and only returned after three weeks of initial presentation. He was then referred to the infectious disease physician and admitted for intravenous benzylpenicillin 4 MU every four hours for two weeks. By the time the antibiotic was initiated, the left BCVA had recovered to 6/6, and OCT and FA of the macula showed resolution of previous changes (Fig. 3). It is noteworthy to mention that no systemic steroid was given during the interval. The patient defaulted follow-up again after antibiotic treatment and thus a reduction in RPR titre could not be elicited.

Discussion

In 1988, de Souza and colleagues reported 3 cases of unusual central chorioretinitis in young adults with active syphilis.[1] Two years later, Gass *et al.* coined the term ASPPC to describe large, placoid, and yellowish subretinal lesions in the macula found in 6 cases of secondary syphilis.[4] As more cases had been reported the following years, it was found to involve both eyes in almost half the cases and occurred in patients regardless of their HIV status.[1,4] Presenting visual acuity varies greatly from 20/20 to no light perception.[1,2] It is usually associated with mild to moderate vitritis in up to 88% of cases and to anterior chamber inflammation to a lesser extent in nearly one-third of cases.[1,2]

The natural course and pathogenesis of ASPPC has not been well established. In most of previously reported cases, antibiotic treatment led to rapid resolution of the lesion.[4] But recently there have been reports of spontaneous resolution of ASPPC even prior to antibiotic treatment, as reflected in our case.[5,6] Noteworthy are also reports of disease progression in untreated as well as steroid-treated cases. At least 3 cases of ASPPC developing after intravitreal and systemic steroids have been reported to date.[4] Progression of untreated ASPPC has also

been reported: 1 case which progressed to panuveitis in 6 weeks and another which initially resolved but only to develop into posterior uveitis 9 months later.[7] Adding to the complexity are also reported cases of non-progression of ASPPC even under systemic steroid therapy.[8]

Various theories have been put forward to explain the pathogenesis of this disease. As initial cases showed high prevalence of HIV-coinfection, an altered immune response to syphilis by HIV was thought to be the cause.[4] However, this was later disputed as there were no consistent differences in the clinical manifestation of patients with and without HIV.[1] A more plausible explanation would be that disseminated spirochetes may enter choroidal circulation and gain access to the outer retina, causing a local immune response much similar to the mucocutaneous rash seen during secondary syphilis.[1,2] This was evidenced by multimodal imaging, where OCT showed hyperreflective dots in the choroid, nodular retinal pigment epithelium (RPE) thickening, and disruption of overlying photoreceptors in the inner segment-outer segment junction.[9] Increased autofluorescence seen within the lesions is consistent with accumulation of lipofuscin or photoreceptor outer segment remnants in the RPE.[2] Indocyanine green angiography showed persistent hypocyanescence corresponding to the areas at FA likely due to either choriocapillaris hypoperfusion or masking effect from overlying affected RPE.[2]

The spontaneous resolution of ASPPC likely points to a successful immune response in controlling the infection locally, with possible contribution from the immune-privileged status of the eye.[10] Alternatively, it can also be explained by disease latency, a pathognomonic feature of syphilis.[4] Some authors suggest that ASPPC will run its natural clinical course of onset-aggravation-resolution regardless of treatment with systemic steroids.[10] Prompt resolution with antibiotic treatment reflects early clearance of disseminated spirochetes.

Our case presents multiple diagnostic challenges that characterises syphilis. Differential diagnoses included acute posterior multifocal placoid pigment epitheliopathy and infective retinitis, which require different treatments. The diagnosis was also delayed by the prozone phenomenon seen during early infection. This, in combination with spontaneous resolution of ASPPC, may cause a missed diagnosis.

Conclusion

The presence of a placoid macular lesion should raise high suspicion of ASPPC. Although it can resolve spontaneously, timely diagnosis and antibiotic treatment is crucial in preventing the progression of neurosyphilis.

Declarations

Informed consent for publication
The patient provided informed consent for the use of the clinical images and data contained in this case report.

Competing interests
None to declare.

Funding
None to declare.

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