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Clover Leaf Vision: Persistent Placoid Maculopathy

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Case History

A 51-year-old woman, presented with bilateral decreased central vision over a 3- week period. She states that there is a smudge in her central vision, "the shape of a cloverleaf" (Figure 1).

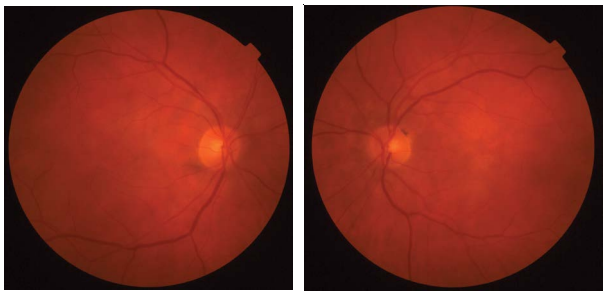

Figure 1: Cloverleaf Vision.

She has an unremarkable past medical history with the exception of a viral illness one-month prior.

At presentation, best-corrected vision was 20/70 in the right eye and 20/50 in the left eye. Dilated fundus examination demonstrated a well demarcated whitish macular plaque in a jigsaw pattern, sparing the parapapillary region, of both eyes. There were no signs of anterior chamber cell or vitritis. Additionally, there was no choroidal neovascularization or subretinal hemorrhage.

Fluorescein angiography demonstrated early blockage and slight late hyperfluorescence corresponding to the macular plaque (Figure 2A,2B). Indocyanine green demonstrated early and late blockage, involving the macula [Figure 2C,2D]. Spectral domain optical coherence tomography displayed discontinuity of the ellipsoid zone and the inner digitation zone [Figure 2E,2F].

Diagnosis

Persistent Placoid Maculopathy (PPM) is characterized by

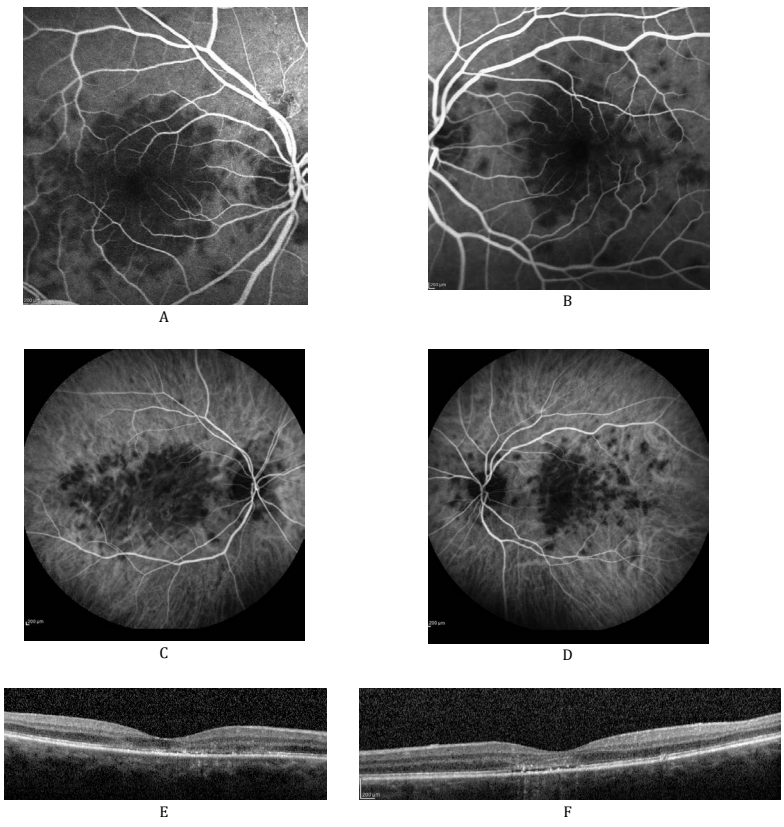

Figure 2: A, B: Right (A) and Left (B) eye, Fluorescein Angiography demonstrating early blocking defect of macula, **C,D:** Indocyanine Green depicting blockage of the choroid in the early and late phases of the study, **E:** OCT Right eye with diffuse disruption of the ellipsoid and inner digitation zone, **F:** OCT Left eye with subfoveal disruption of the ellipsoid and inner digitation zone.

whitish plaque lesions involving the macula and sparing the peripapillary region. PPM was first categorized by Golchet et al, [1] when they described six patients who had clinical features of both acute posterior multifocal placoid pigment epitheliopathy (APMPPE) and macular serpiginous choroidopathy.

Visual loss occur secondary to loss of outer retina photoreceptors and complications from choroidal neovascular membranes [2]. Clinically, patients with PPM can appear similar to macular serpiginous choroiditis, acute posterior multifocal placoid pigment epithelipathy and syphilitic posterior placoid chorioretinitis [3]. It is important to differentiate between these entities because treatment and prognosis can vary. Most patients with APMPPE do not require treatment and the condition subsides. Conversely, many patients with PPM can benefit from treatment, often in the form of high-dose corticosteroids (0.75–1 mg/kg/day) or immunosuppressant agents like mycophenolate (cellcept) [2,3].

Persistent placoid maculopathy has an inflammatory etiology involving the inner choroid and has subsequent changes involving the retinal pigment epithelium and the outer retina [4]. Indocyanine green (ICG) highlights the inner choroid pathology, displaying hypoperfusion as an early blocking defect on ICG. Ocular Coherence Tomography will often show disruption of the ellipsoid and innerdigitation zone, ultimately preventing proper photoreceptor function [4].

Most patients with persistent placoid maculopathy develop choroidal neovascularization (CNV), resulting in central vision loss [5,6]. Golchet et al. [1] noted 9 of the 12 eyes with PPM developed CNV and poor vision.

Fortunately, as described by Eadie et al. [7] Bevacizumab has been used successfully in regression of CNV secondary to PPM.

Take Home Points

- Distinguish PPM from other similar clinical entities by first ruling out ocular syphilis, then differentiating between APMPPE and PPM. PPM may benefit from early high dose steroidtreatment.
- Counsel your patient and remain vigilant of the long-term complication of choroidal neovascularization (CNV). Complications from CNV are prevalent in PPM but may be amenable to anti-VEGF treatment.

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