

# Acute Posterior Multifocal Placoid Pigment Epitheliopathy After Coronary Bypass Surgery: Case Report

## Koroner Baypas Cerrahisi Sonrası Gelişen Akut Posterior Multifokal Plakoid Pigment Epiteliopati

Abuzer GÜNDÜZ,<sup>a</sup>  
Tongabay CUMURCU,<sup>a</sup>  
Selim DOĞANAY,<sup>a</sup>  
Mufide ÇAVDAR<sup>a</sup>

<sup>a</sup>Department of Ophthalmology,  
İnönü University Faculty of Medicine,  
Malatya

Geliş Tarihi/Received: 19.03.2013  
Kabul Tarihi/Accepted: 12.09.2013

*This case report was presented as a poster at 16<sup>th</sup> Afroasian Congress of Ophthalmology, İstanbul, Turkey, 13-16 June 2012.*

Yazışma Adresi/Correspondence:  
Abuzer GÜNDÜZ  
İnönü University Faculty of Medicine,  
Department of Ophthalmology, Malatya,  
TÜRKİYE/TURKEY  
abuzergunduz@hotmail.com

**ABSTRACT** This report evaluated bilateral acute posterior multifocal placoid pigment epitheliopathy (APMPPE) with unilateral papillitis. We studied a 58-year-old male patient attempt to our clinic for blurred vision 4 days after a bypass surgery. A fundus examination revealed bilateral creamy white lesions in the posterior pole and papillitis in the left eye. In fundus fluorescein angiography, lesions showed hypofluorescence in early phases and hyperfluorescence in late phases. Optical coherence tomography imaging showed impaired retinal pigment epithelial boundaries. APMPPE after coronary bypass surgery may help to find out probable etiology. In this case, we thought that direct surgical stress or surgical-induced stress factors may cause APMPPE in susceptible persons. To obtain exact results, specific immunogenetic studies are needed.

**Key Words:** Coronary artery bypass; retinal pigment epithelium

**ÖZET** Koroner baypas cerrahi sonrası gelişen bilateral akut posterior multifokal plakoid pigment epiteliopati (APMPPE) ve eşlik eden tek taraflı papilit olgusu değerlendirildi. Olgumuz 58 yaşında erkek hastada, baypas cerrahi sonrası 4. günde sol gözde bulanık görme şikayeti nedeni ile yapılan göz dibi muayenesinde bilateral arka kutupta çok sayıda krem-beyaz plakoid lezyonlar ve sol optik diskte papilit saptandı. Floresein anjiyografide plakoid lezyonlar erken fazda hipofloresan, geç fazda ise hiperfloresan olarak izlendi. Optik koherans tomografide lezyonların lokalizasyonunda retina pigment epitelinin sınırlarının bozulduğu saptandı. Etyopatogenezi tam olarak aydınlatılmamış olan APMPPE'nin koroner baypas cerrahi sonrası da saptanması, muhtemel etiyoloji açısından önemli olabilir. Bu tablonun oluşmasında direkt cerrahinin sorumlu olabileceği veya cerrahinin neden olduğu stres faktörlerinin duyarlı bireylerde tabloyu tetikleyebileceğini düşünmekteyiz. Kesin sonuçların elde edilebilmesi için spesifik immünogenetik çalışmalar gerektiği kanaatindeyiz.

**Anahtar Kelimeler:** Koroner arter baypas; retina pigment epiteli

**Türkiye Klinikleri J Case Rep 2013;21(4):186-9**

**A**cute posterior multifocal placoid pigment epitheliopathy (APMPPE), was first described by Gass in 1968, is an acquired inflammatory disorder affecting the choroidal vessels.<sup>1</sup> The disease is self-limited and is characterised by multiple yellow-white placoid subretinal lesions of the posterior pole.<sup>2</sup> The lesions are frequently bilateral, mostly affects young adults (20-40 years) and typically resolve in weeks to months.<sup>3,4</sup> The pathophysiology of APMPPE is speculative and is thought as an inflammatory vasculitis that affects the choroidal vessels. It is evaluated as focal choroidal vasculopathy.<sup>5,6</sup> Retinal pigment epithelium is affected by

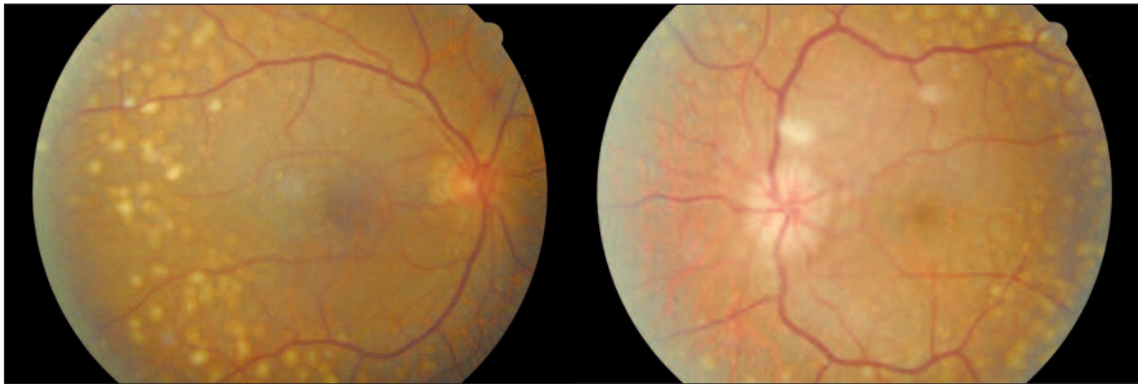
the inflammation of the choroid.<sup>3,7</sup> The similar results were demonstrated in some systemic diseases including Wegener granulomatosis, systemic necrotising vasculitis, cerebral vasculitis, and insect bites.<sup>7-9</sup> Scleritis, serious retinal detachment, retinal vasculitis, papillitis, and Harada disease are the ocular disorders associated with APMPE.<sup>10-13</sup>

The present study investigated an APMPE case that occurred after coronary bypass surgery. As far as we know, no APMPE case after cardiac surgery or any other surgery has been reported before. Our case is the first report in the literature.

## CASE REPORT

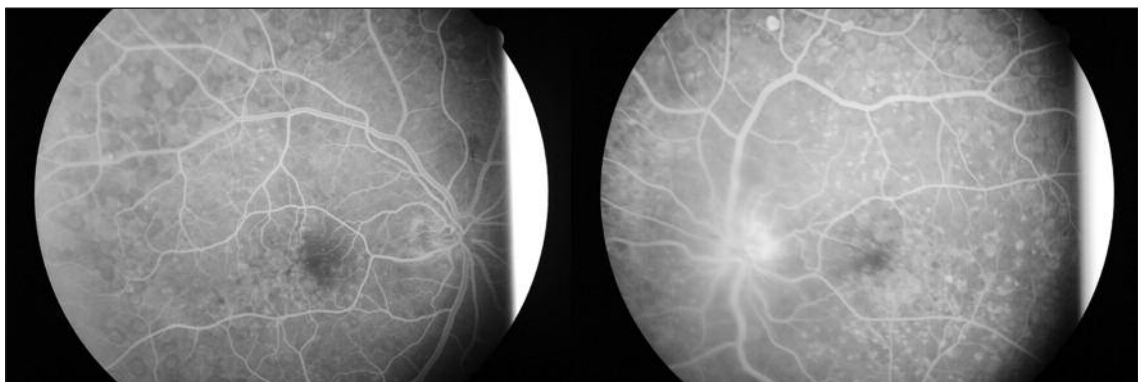
A 58-year-old man underwent bypass surgery for three vessels due to subacute inferior myocardial infarction. Four days after the surgery, the patient consulted to our clinic due to vision loss. There was

no vision loss in his previous history. In his ophthalmologic examination: Eye movements were free in every direction in both eyes, and lids are normal. His left eye had reactive afferent pupillary defect. His right eye vision was 1.0 (-0.5 D), and his left eye vision was at the hand-movement level (-1.25 D). In biomicroscopic examination anterior segment structures were normal. The results of the fundus examination showed multiple yellowish lesions above the retina, some of which spread to the whole retina. Majority of the lesions were located at the equatorial region. Left optic disc margins were blurred and papilla were edematous (Figure 1). Fundus fluorescein angiography (FFA) and optical coherence tomography (OCT) were performed. The FFA results showed hypofluorescence lesions in early phases (Figure 2), and hyperfluorescence in late phases (Figure 3). The FFA image of the left optic disc showed leakage in the early



**FIGURE 1:** In the first diagnosis of the patient, bilateral diffuse placoid lesions, left optic disc edema, and peripapillary exudates were seen.

(See color figure at <http://www.turkiyeklinikleri.com/journal/turkiye-klinikleri-journal-of-case-reports/1300-0284/tr-index.html>)



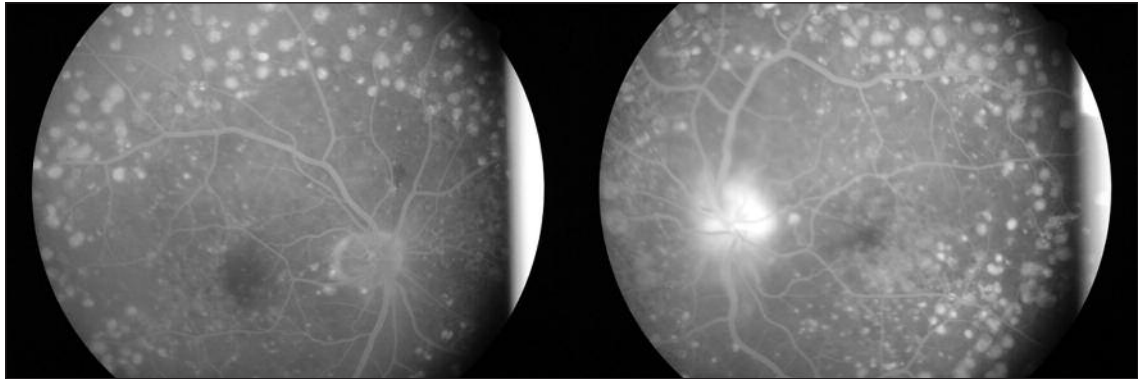
**FIGURE 2:** Placoid lesions were hypofluorescent in early phases of the FFA, and the left optic disc was dyed.

phases and stains in the late phases. In OCT images, placoid lesions were seen at the subretinal space, and lesions originated from the choroid (Figure 4). According to these findings, the patient was diagnosed with APMPE. Blood samples were taken from the patient to evaluate blood count, erythrocyte sedimentation rate, liver function, C-reactive protein, blood urea nitrogen, creatinine, rhomatoid factor, venereal disease markers, antinuclear anti-core (ANA), dsDNA, P-ANCA (myeloperoxidase-ANCA), Borellia Ig G and Ig M, HIV, toxoplasmosis Ig M and Ig G, and anticardiolipin anticores. Cranial tomography and chest x-ray were also performed. All test results were normal. With the test results, bypass surgery was considered, but no treatment was given to the patient. Ophthalmological examination 2 months after the diagnosis showed that the patient's vision was 1.0 (-0.5 D) on the right eye and 0.7 (-1.25 D) on the left eye. The anterior segment was normal bilaterally. The fun-

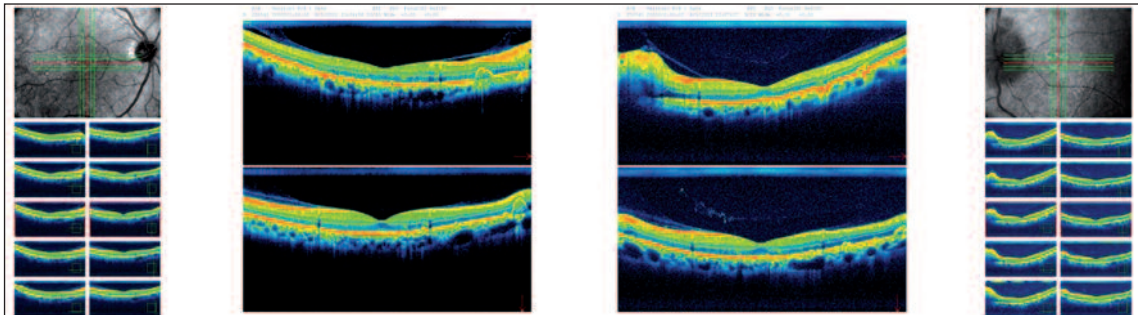
dus examination also revealed pale placoid lesions, left optic disc edema was resolved and became pale (Figure 5). Patient's informed consent was taken for all interventions and presentations.

## DISCUSSION

The etiopathogenesis of the APMPE is not known. Different theories were suggested for etiopathogenesis. The first of these theories was presented by Gass in 1968.<sup>1</sup> According to Gass, a viral agent causes acute cellular response against the choroid, and retinal pigment epithelium was responsible for the disease. In another study, Van Buskirk suggested late vasculopathy as an etiologic factor.<sup>14</sup> Vedantham and Ramasamy explained that retinal vascular endothelium and choroidal vascular structures play a role in the pathogenesis of the disease.<sup>7</sup> Another study pointed out that the underlying mechanism is believed to be an obstructive vasculi-

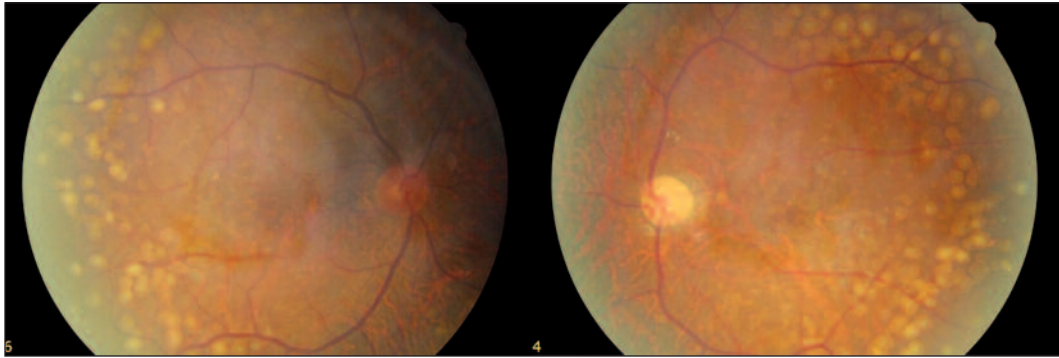


**FIGURE 3:** Placoid lesions were hyperfluorescent bilaterally in late phases of the FFA, and the left optic disc was dyed more.



**FIGURE 4:** RPE borders distorted by placoid lesions in optical coherence tomography.

(See color figure at <http://www.turkiyeklinikleri.com/journal/turkiye-klinikleri-journal-of-case-reports/1300-0284/tr-index.html>)



**FIGURE 5:** 2 months after the diagnosis: Placoid lesions were blurred, and the optic disc edema was lower than the first diagnosis time.

(See color figure at <http://www.turkiyeklinikleri.com/journal/turkiye-klinikleri-journal-of-case-reports/1300-0284/tr-index.html>)

tis caused by sensitive T lymphocytes and causing nonperfusion of the terminal choroidal lobules in the posterior pole of the eye.<sup>15</sup> This study showed late type hypersensitivity associated with sarcoidosis, pulmonary tuberculosis, and schistosomiasis. In our case, the physical examination and laboratory tests did not reveal any disorder that may cause APMPE. Surgery-induced acute stress probably caused APMPE, but we could not explain its mechanism. Acute retinal pigment epitheliopathy, birdshot retinopathy, punctate internal choroidopathy, serpiginous choroiditis, multifocal choroiditis and geographic choroidopathy should be considered in differential diagnosis of the disease.

In the literature, APMPE was explained with unilateral or bilateral papillitis.<sup>3</sup> Reports claim that choroidal vasculitis in the peripapillary area may cause papillitis in immunogenetically sensitive patients. In our case, there was papillitis in the left eye, but we could not explain its mechanism.

As a result, it is thought that APMPE is a vasculitis triggered by different agents in susceptible patients. In our case, we thought that coronary bypass surgery or acute stress factors caused by surgery were the responsible agents for APMPE. However, we need specific immunogenetic research studies to explain its pathophysiology.

## REFERENCES

- Gass JD. Acute posterior multifocal placoid pigment epitheliopathy. *Arch Ophthalmol* 1968;80(2):177-85.
- Alpay A, Sađık HM, Uđurbař SH. Acute posterior multifocal placoid pigment epitheliopathy: Association with granulomatous anterior uveitis: Case report. *Turkiye Klinikleri J Med Sci* 2012;32(2):532-6.
- Abu El-Asrar AM, Aljazairy AH. Acute posterior multifocal placoid pigment epitheliopathy with retinal vasculitis and papillitis. *Eye (Lond)* 2002;16(5):642-4.
- Jones NP. Acute posterior multifocal placoid pigment epitheliopathy. *Br J Ophthalmol* 1995;79(4):384-9.
- Deutman AF, Oosterhuis JA, Boen-Tan TN, Aan de Kerk AL. Acute posterior multifocal placoid pigment epitheliopathy. Pigment epitheliopathy of choriocapillaris? *Br J Ophthalmol* 1972;56(12):863-74.
- Spaide RF, Yannuzzi LA, Slakter J. Choroidal vasculitis in acute posterior multifocal placoid pigment epitheliopathy. *Br J Ophthalmol* 1991;75(11):685-7.
- Vedantham V, Ramasamy K. Atypical manifestation of acute posterior multifocal placoid pigment epitheliopathy. *Indian J Ophthalmol* 2006;54(1):49-52.
- Hsu CT, Harlan JB, Goldberg MF, Dunn JP. Acute posterior multifocal placoid pigment epitheliopathy associated with a systemic necrotizing vasculitis. *Retina* 2003;23(1):64-8.
- O'Halloran HS, Berger JR, Lee WB, Robertson DM, Giovannini JA, Krohel GB, et al. Acute multifocal placoid pigment epitheliopathy and central nervous system involvement: nine new cases and a review of the literature. *Ophthalmology* 2001;108(5):861-8.
- Matsuo T, Horikoshi T, Nagai C. Acute posterior multifocal placoid pigment epitheliopathy and scleritis in a patient with pANCA-positive systemic vasculitis. *Am J Ophthalmol* 2002;133(4):566-8.
- Kirkham TH, Fytche TJ, Sanders MD. Placoid pigment epitheliopathy with retinal vasculitis and papillitis. *Br J Ophthalmol* 1972;56(12):875-80.
- Savino PJ, Weinberg RJ, Yassin JG, Pilkerton AR. Diverse manifestations of acute posterior multifocal placoid pigment epitheliopathy. *Am J Ophthalmol* 1974;77(5):659-62.
- Wright BE, Bird AC, Hamilton AM. Placoid pigment epitheliopathy and Harada's disease. *Br J Ophthalmol* 1978;62(9):609-21.
- Van Buskirk EM, Lessell S, Friedman E. Pigmentary epitheliopathy and erythema nodosum. *Arch Ophthalmol* 1971;85(3):369-72.
- Park D, Schatz H, McDonald HR, Johnson RN. Acute multifocal posterior placoid pigment epitheliopathy: a theory of pathogenesis. *Retina* 1995;15(4):351-2.