Neurologic Complications of Acute Posterior Multifocal Placoid Pigment Epitheliopathy: A Case Series of 4 Patients

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Abstract

Acute posterior multifocal placoid pigment epitheliopathy (APMPPE) is a self-limited idiopathic inflammatory ophthalmologic condition with characteristic fundoscopic and fluorescein angiography findings. It is typically characterized by a flu-like prodrome followed by monocular or binocular vision loss. Often, prognosis is excellent with complete or near-complete recovery of vision. Rarely, however, APMPPE is associated with neurologic complications, including meningitis, cerebral vasculitis, and stroke. Treatment in patients with central nervous system (CNS) involvement involves steroids and ultimately other immunosuppressive therapy, as there can be significant resulting morbidity and mortality otherwise. Evidence or guidelines regarding duration of treatment are lacking. We present 4 patients diagnosed with APMPPE who demonstrate the spectrum of neurologic sequelae associated with APMPPE. The first 2 cases highlight cerebrospinal fluid lymphocytic pleocytosis as an indicator of active CNS inflammation and the potential utility of serial lumbar punctures (LPs) to guide treatment duration. Cases 3 and 4 demonstrate the neurovascular complications seen in CNS vasculitis. Case 4 also highlights the potential use of magnetic resonance vessel wall imaging (VWI) as a noninvasive means for disease surveillance and treatment guidance. This case series emphasizes the importance of recognition by neurologists of APMPPE as an entity associated with strokes and cerebral vasculitis in order to provide appropriate and timely treatment. Active CNS inflammation warrants continued aggressive immunosuppressant treatment, and we propose that serial LPs and/or magnetic resonance VWIs may be effective tools to guide disease surveillance and subsequent treatment duration.

Keywords

headache, cerebral vascular disease, meningitis, vasculitis, uveitis

Introduction

Acute posterior multifocal placoid pigment epitheliopathy (APMPPE) is a self-limited idiopathic inflammatory ophthalmologic condition first described by Gass in 1968 that is characterized by multiple cream-colored lesions in the posterior pole of the retina which evolve into areas of circumscribed pigmentary changes.¹ Fundus fluorescein angiography (FA) characteristically reveals early hypofluorescence and late hyperfluorescence of these lesions. One-third of patients present after a flu-like viral prodrome and most have painless mild visual loss in 1 or both eyes.² Rarely, APMPPE is associated with neurologic complications, including cerebral vasculitis and stroke. We present 4 patients diagnosed with APMPPE who experienced neurological complications. Each case demonstrates the spectrum of neurologic sequelae associated with APMPPE. We report the potential utility of serial lumbar

puncture (LP) and magnetic resonance vessel wall imaging (VWI) as a means for disease surveillance and treatment guidance. The risk of neurologic complications associated with APMPPE is underrecognized and can be associated with significant morbidity and mortality. Patients presented in cases 1,

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2, and 4 each gave verbal consent to have their cases included in the article. Patient 3 succumbed to complications of the condition being presented and consent could not be obtained.

Case Reports

Case 1: Meningitis

A 53-year-old man presented with a 3-week history of headache and bilateral central blurred vision associated with photopsias. Review of systems was notable for 3 days of chills, sweats, and myalgia. Visual acuity was 20/20 OD and 20/50 OS. Neuroimaging (Table 1) was unremarkable. A dilated fundoscopic examination revealed multiple cream-colored placoid lesions involving the central macular region of the retina without retinal vasculitis or vitritis in both eyes (Figure 1). Fluorescein angiography revealed early hypofluorescence and late hyperfluorescence of the placoid lesions. Serial LPs were notable initially for elevated opening pressures and 5 days later development of lymphocytic pleocytosis (Table 1). Treatment consisted of high-dose intravenous methylprednisolone followed by a 3-month course of prednisone and transition to mycophenolate mofetil. He experienced resolution of headaches and recovery of visual acuity to 20/20 OD and 20/40 OS, after 3 weeks. Repeat LP 7 months later showed resolution of the pleocytosis. Mycophenolate mofetil was tapered over several weeks, and the patient has remained free of neurologic symptoms at 7 years from initial presentation. He developed subfoveal choroidal neovascularization as a sequelae of having APMPE in both eyes some years later after having moved from the area (Table 1).

Case 2: Meningitis

A 32-year-old woman with Crohn disease (off immunosuppression) presented for evaluation of a 2-week positional headache in the setting of subjective fevers, sweats, and chills. She developed bilateral blurred vision for 1 week and then acute left face and hand numbness 1 day prior to presentation to an outside hospital where an LP showed an elevated opening pressure. After transfer, ophthalmological examination was notable for visual acuity of 20/40 OD and 20/50 pinholing to 20/25 OS, and fundus examination and FA supported the diagnosis of APMPE (Figure 1). Neurological examination was notable for decreased pinprick sensation on the left face and hemibody. Magnetic resonance imaging (MRI) of the brain showed T2-weighted hyperintensities in the left basal ganglia and subinsular region without associated restricted diffusion, thought to be unrelated to her active symptoms. Other neuroimaging studies were considered unremarkable for clinical presentation (Table 1). Repeat LP 2 days later showed elevated opening pressure and new lymphocytic pleocytosis. Treatment consisted of high-dose intravenous methylprednisolone for 5 days followed by high-dose prednisone. The patient experienced resolution of her headache, numbness, and blurred vision after 3 months. Repeat LP at 7 months

showed resolution of pleocytosis. She was weaned off prednisone and remained free of neurologic symptoms until 36 months after initial presentation when she developed head pain consistent with migraine-type headache, successfully treated with amitriptyline.

Case 3: Central Nervous System Vasculitis

A 64-year-old man with hypertension, tobacco use, and chronic obstructive pulmonary disease underwent ophthalmologic evaluation for 4 days of binocular blurry vision, kaleidoscopic visual hallucinations, and right-sided head, ear, jaw, and neck pain with scalp tenderness. Visual acuity was 20/30 pinholing to 20/20 OD and 20/40 pinholing to 20/20 OS. Fundoscopic examination demonstrated evidence of retinal edema bilaterally. Fluorescein angiography showed findings consistent with APMPE. He was started on 80 mg daily prednisone. By week 3, visual acuity improved (20/30 pinholing to 20/25 OD, 20/20 OS) and headaches had resolved. Prednisone was tapered to 60 mg daily. Within 1 week he presented to the emergency department with acute-onset left hemiparesis and dysarthria. Magnetic resonance imaging revealed bihemispheric multifocal infarcts (Figure 2). Conventional angiography revealed arterial beading of the posterior cerebral artery, superior cerebellar artery (SCA), and middle cerebral artery (MCA), supportive of vasculitis (Figure 2). Despite treatment with high-dose intravenous methylprednisolone, the patient deteriorated, developed acute respiratory failure from a presumed aspiration event, and was intubated. Lumbar puncture was not performed. His course was further complicated by seizures and serotonin syndrome. He was started on cyclophosphamide. Repeat MRI 3 weeks after his initial presentation revealed new extensive multifocal infarctions (Figure 2). The patient was transitioned to comfort care and terminally extubated 33 days later.

Case 4: Persistent Central Nervous System Vasculitis

A 55-year-old man with hypertension, hyperlipidemia, alcohol-related liver cirrhosis, and chronic kidney disease presented with progressive painless, bilateral central vision loss. Three weeks prior, he experienced 5 days of subjective fevers, chills, myalgia, frontal headache, and blurred vision (OS > OD). His constitutional symptoms resolved and he was referred to ophthalmology for visual loss. Ophthalmological examination revealed several hypopigmented retinal lesions. Following his dilated examination, he experienced complete left eye visual loss and was referred for emergent neurological evaluation that revealed right ptosis and bilateral central scotoma. A serological evaluation revealed elevated antinuclear antibodies without elevations in F-actin (autoimmune hepatitis) or antimitochondrial antibodies. Lumbar puncture was notable for increased opening pressure and lymphocytic pleocytosis. Magnetic resonance imaging revealed restricted diffusion in the distribution of the right SCA (Figure 2).

Table 1. Clinical Data of 4 Cases With Acute Multifocal Placoid Pigment Epitheliopathy and Central Nervous System Findings.

Case	1	2	3	4
Age/sex	55/M	32/F	64/M	55/M
Ophthalmologic symptoms	Bilateral blurred central vision, photopsias	Bilateral blurred vision	Bilateral blurred vision, kaleidoscopic visual hallucinations	Bilateral central vision loss
Neurologic symptoms	"Vice-like" headache that worsened with recumbence	Positional "pressure-like" headache worse with recumbence; left face and arm numbness	Dysarthria, left-sided hemiparesis	Bifrontal headache, right ptosis
Laboratory evaluation	Anticardiolipin, anti-RNP, anti-Smith, anti-lupus AC, anti-Ro, anti-La, ANA, ANCA, dsDNA Ab, ACE, syphilis/RPR negative; CRP 27, ESR 39	ANA, SPEP, serum ACE, anti-RNP, anti-Smith, anti-Ro, anti-La, ANA, dsDNA Ab, ACE, syphilis/RPR negative; ANCA 40:1; CRP 74, ESR 88	Lupus anticoagulant, anticardiolipin, β 2 glycoprotein, anti-RNP, anti-Smith, anti-Ro, anti-La, ANA, ANCA, dsDNA Ab, ACE, syphilis/RPR negative	F-actin, antimitochondrial antibodies negative; ANA elevated
CSF findings (time from neurologic symptom onset)	LP1 (day 0): OP 270 LP2 (day 5): OP 200 68 WBC/hpf (91% lymphocytic) Protein 66 mg/dL LP3 (7 months): OP 240	LP1 (day 0): OP 550 LP2 (day 2): OP 130 65 WBC/hpf (69% lymphocytic) Protein 58 mg/dL LP3: OP 240	NA	LP1 (day 20): Increased OP: 21 WBC/hpf (87% lymphocytic) Protein 40 mg/dL
Imaging	MRI, MRV, CA: unremarkable	MRI: FLAIR $\pm$ DWI—lesions in the left basal ganglia and left subinsular regions. MRV/MRV/CTA head and neck: unremarkable CA: mild (nonspecific) luminal irregularities of the proximal A2 and callosomarginal arteries bilaterally	MRI 1: FLAIR $\pm$ DWI+ infarcts of bilateral thalami, right central pons, and scattered throughout the bilateral frontal, right parietal, and bilateral insular cortex CTA head: narrowing of basilar artery and posterior cerebral arteries bilaterally with dilatation of collateral vessels CA: vasculitic changes along bilateral PCAs, SCAs, temporal MCAs. Mild stenosis of right vertebral artery origin	MRI 1: FLAIR+/DWI+ right cerebellar infarcts MRA: left ACA aneurysm enhancement CA: Vasculopathic irregularities in the right PVA and SCA. Left frontopolar artery origin aneurysm MRI 2 (3 weeks after initial MRI): FLAIR-/DWI+ bilateral basal ganglia and internal capsule strokes; unchanged circumferential wall enhancement of the bilateral MCAs at the level of the lentículostriate and anterior choroidal vessels compatible with vasculitis. Previously seen enhancement of the ACA not appreciated
Treatment	High-dose IV methylprednisolone, 3 months oral prednisone taper, transition to mycophenolate mofetil tapered after 7 months	High-dose IV methylprednisolone for 5 days followed by oral prednisone, tapered after 7 months	Oral prednisone prior to neurological symptoms; during hospitalization, high-dose IV methylprednisolone with taper and concomitant initiation of cyclophosphamide	High-dose oral prednisone with 3-month taper, followed by high-dose methylprednisolone 5 months later due to recurrent stroke with outpatient taper and transition to cyclophosphamide
Outcome	Complete resolution of headache. Stable recurrence of APMPE with choroidal neovascularization requiring repeated treatment with mycophenolate mofetil and intravitreal anti-VEGF therapy (bevacizumab and aflibercept). Last vision examination: 20/30–1	Resolution of headache, numbness, and blurred vision within 3 months of steroids. Later developed migraine-type headaches	Death (comfort care) 33 days following hospital admission	Permanent visual loss. Recurrent mild headaches. Resolution of aphasia, dysarthria, facial droop

Abbreviations: ACA, anterior cerebral artery; ACE, angiotensin converting enzyme; ANA, anti-nuclear antibody; ANCA, antineutrophil cytoplasmic antibodies; APMPE, acute posterior multifocal placoid pigment epitheliopathy; CA, conventional angiography; CRP, C-reactive protein; CSF, cerebrospinal fluid; CTA, computed tomography angiography; dsDNA, double-stranded DNA; F, female; ESR, erythrocyte sedimentation rate; FLAIR, fluid attenuated inversion recovery; LP, lumbar puncture; M, male; lupus AC, lupus anticoagulant; MCA, middle cerebral arteries; MRA, magnetic resonance angiogram; MRI, magnetic resonance imaging; MRV, magnetic resonance venogram; OP, opening pressure (mm H₂O); PCA, posterior cerebral artery; PVA, proximal vertebral artery; RNP, anti-ribonuclear antibody; RPR, rapid plasma reagin; SCA, superior cerebellar artery; SPEP, serum protein electrophoresis; WBC, white blood cell.

Figure 1. Ophthalmologic findings for case 1 (A-C) and case 2 (D-F). A, Fundus photograph of the left eye showing multiple coalescing creamy white lesions at the level of the retinal pigmented epithelium (RPE). B, Fluorescein angiography of the left eye in early arteriovenous phase showing hypofluorescence. C, Fluorescein angiography of the left eye in late arteriovenous phase showing hyperfluorescence. D, Fundus photograph of the right eye showing multiple coalescing creamy white lesions at the level of the RPE. E, Fluorescein angiography of the right eye in early arteriovenous phase showing hypofluorescence. F, Fluorescein angiography of the right eye in late arteriovenous phase showing hyperfluorescence.

Conventional angiography was notable for multifocal stenoses involving the right SCA and right posterior cerebral arteries as well as a 2.5×3.0 mm aneurysm in the second division of the left anterior cerebral artery (ACA; Figure 2). Vessel wall imaging was remarkable for perivascular enhancement of the left ACA (Figure 2). He was started on high-dose prednisone with a prolonged taper. His vision stabilized with an acuity of 20/400 OS with improvement to 20/300 OU. His presentation was consistent with APMPE with cerebral vasculitis.

Approximately 5 months later, while tapering steroids, he represented with sudden-onset aphasia, dysarthria, and right facial weakness. Magnetic resonance imaging confirmed the presence of an acute ischemic infarct in the distribution of the left MCA. Repeat VWI showed resolution of the ACA perivascular enhancement but persistent enhancement of the left MCA (Figure 2) suggestive of a second vasculitic infarct. Repeat LP was not performed. He was treated with high-dose intravenous methylprednisolone, a prednisone taper, and transitioned to intravenous biweekly cyclophosphamide for 6 weeks. Three-month follow-up showed resolution of dysarthria and facial paresis but persistent central scotomas.

Discussion

Acute posterior multifocal placoid pigment epitheliopathy rarely involves the central nervous system (CNS), with about 60 cases reported in the scientific literature to our knowledge, including our 4.³ Typically, when APMPE remains isolated to ophthalmologic symptoms, it has a monophasic course with complete or near-complete recovery. However, CNS involvement with APMPE is associated with severe morbidity and mortality typically related to cerebral vasculitis, meningitis, meningoencephalitis, stroke, cranial nerve palsy, peripheral neuropathy, or cavernous sinus thrombosis.^{2,3} While the median time to onset of cerebrovascular complications is reported to be 3 weeks after ophthalmologic symptoms, delayed onset up to a year has been reported.^{4,5}

Acute posterior multifocal placoid pigment epitheliopathy has also been associated with other autoimmune diseases such as sarcoidosis, systemic lupus erythematosus, Crohn disease, and juvenile rheumatoid arthritis. Therefore, diagnostic testing for possible autoimmune conditions should be included in the APMPE evaluation. However, there are features that distinguish APMPE from these. Sarcoidosis, for example,

Figure 2. Neuroradiographic findings in case 3 (A-C) and case 4 (D-G). A, Conventional cerebral angiography reveals decreased flow and vascular beading of the right posterior cerebral artery (yellow arrows). B, Coronal reformatting computed tomography angiography shows diminutive basilar circulation and dilation of collateral vascular channels (yellow circle). C, Magnetic resonance diffusion-weighted imaging (DWI) shows ischemic infarctions throughout the posterior circulation consistent with catastrophic vasculitis (yellow arrows). D, Magnetic resonance vessel wall imaging (VWI) shows concentric perivascular enhancement consistent with vasculitis (orange arrow). E, Follow-up VWI shows resolution of perivascular enhancement with immunosuppressive therapy at 5-month follow-up. F, Vessel wall imaging shows persistent perivascular enhancement of the left middle cerebral artery after 5 months of immunosuppression (orange arrow). G, Diffusion-weighted imaging shows ischemic infarction in the left middle cerebral artery consistent with a vasculitic infarct (orange arrow).

is most often associated with anterior segment involvement, but when involved, changes in retinal pigment epithelium usually occur peripherally and are not associated with visual disturbances.⁶ Uveoretinal meningoencephalitic syndromes, including Vogt-Koyanagi-Harada (VKH) disease, Behçet disease, and primary intraocular lymphoma, should also be considered when there are both ocular and CNS symptoms. In fact, there are many overlapping features between VKH and APMPE, such as initial fundoscopic and FA findings. However, VKH disease is a chronic granulomatous panuveitis with characteristic optical coherence tomography and fundoscopic findings of multifocal serous retinal detachment.⁷

There are no treatment guidelines for APMPE. Oral steroids are variably used to treat APMPE *without* neurological complications depending upon the lesion location and severity of vision loss.⁸ Immunosuppressive therapy is typically recommended in cases with neurologic complications, but optimal treatment duration is not clear. Most authors suggest the use of long-term immunosuppression in cases with CNS vasculitis as there are several reported cases of recurrent stroke

and death with steroid taper, similar to what was seen in cases 3 and 4.²⁻⁵ This may suggest a more fulminant or persistent variant of disease. Therefore, methods to guide treatment duration and timing of steroid taper are needed.

Intracranial VWI is a likely useful adjunct to traditional imaging studies to evaluate for vasculitis.⁹ The novel use of VWI in case 4 demonstrated persistent inflammation in the left MCA, the territory of which was infarcted 5 months later during steroid taper. This imaging modality was beneficial for determining disease persistence and ultimately stroke mechanism. Vessel wall imaging sequencing could be considered in select patients as a noninvasive means of monitoring CNS vasculitic activity and potentially used to guide the treatment course, although this has not been validated in large studies.

Cerebrospinal fluid (CSF) analysis remains a valuable tool to assess CNS inflammation. Cerebrospinal fluid abnormalities have long been recognized in both patients with and without neurological manifestations of APMPE and typically consist of elevated opening pressure, lymphocytic pleocytosis, and variably elevated protein.^{4,10} While the exact

pathophysiologic mechanism of APMPE is unknown, it is known to be an inflammatory choriocapillaropathy that has been associated with many autoimmune disorders. Cerebrospinal fluid findings as described above along with the precedent flu-like prodrome further support a postinfectious, possibly autoimmune-mediated inflammatory reaction.² In our first 2 cases, these characteristic CSF findings developed up to 5 days after neurologic presentation. In these cases, symptom response and CSF cell counts were used as markers for treatment response, following which immunosuppression was discontinued without long-term recurrence. In cases 3 and 4, patients experienced recurrent stroke in the setting of steroid taper, neither benefiting from serial CSF studies to assess for ongoing signs of CNS inflammation. Sustained lymphocytic pleocytosis 1 month after symptom onset seen in case 4 raises the suspicion of ongoing CNS inflammation. That neither patient 1 nor 2 had developed cerebrovascular complications suggests either (1) they experienced a less severe disease variant or (2) a relapsing cerebrovascular event was prevented by continuing immunosuppression until CNS inflammation subsided. The absence of CSF results in patient 3 or in patient 4 at the time of representation is an important limitation to the generalizability of our observation. Meningeal tissue biopsy can add diagnostic certainty in atypical cases characterized by CNS involvement and disease recurrence and should be considered in select cases after a careful discussion of the risks and benefits.

Acute posterior multifocal placoid pigment epitheliopathy is a condition that needs to be recognized by neurologists due to its associated neurologic complications. Patients presenting with new-onset headache or stroke associated with visual loss should have a detailed ophthalmologic examination. Timely and appropriate initiation of steroid treatment is essential in reducing the risk of developing additional neurologic complications. Results from VWI and/or serial LPs could serve as markers of ongoing CNS inflammation, ultimately dictating duration of immunosuppression and the timing of tapering of these medications. Continued collaborative management with ophthalmology is essential to observe for progression of vision loss and the development of intraocular pathology. Additional studies will be needed to validate the use of either of these methods as markers of disease activity.

Authors' Note

All authors contributed to the content of the manuscript. Dr Tsuboyama contributed to the clinical and diagnostic evaluation of patient 3, reviewed the literature, and prepared the manuscript. Dr Chandler contributed to the clinical and diagnostic evaluation of patient 4 and to the preparation and review of the manuscript. Dr Scharf contributed to the clinical and diagnostic evaluation of patient 4 and to the preparation and review of the manuscript. Dr Scagnelli contributed to the clinic evaluation of patient 1, reviewed the literature, and prepared the manuscript. Dr Ramchandran contributed to the clinical and diagnostic evaluation of patient 1 and to the review of the manuscript. Dr Holloway contributed to the clinical diagnostic evaluation of patient 1 and prepared the manuscript. Dr Wychowski

contributed to clinical and diagnostic evaluation of patients 1 and 2, reviewed the literature, and prepared the manuscript.

Declaration of Conflicting Interests

The authors declared the following potential conflicts of interest with respect to the research, authorship, and/or publication of this article: Dr Scagnelli serves as a consultant for Teva Pharmaceuticals, Novartis Pharmaceuticals, Biogen IDEC, and Sanofi-Genzyme. Dr Holloway serves as a consultant for Milliman Inc in reviewing practice guidelines.

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References

1. Gass JD. Acute posterior multifocal placoid pigment epitheliopathy. *Arch Ophthalmol*. 1968;80(2):177-185.
2. O'Halloran HS, Berger JR, Lee WB, 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):861-868.
3. Algahtani H, Alkhotani A, Shirah B. Neurological manifestations of acute posterior multifocal placoid pigment epitheliopathy. *J Clin Neurol*. 2016;12(4):460-467.
4. Luneau K, Newman NJ, Srivastava S, Biousse V. A case of acute posterior multifocal placoid pigment epitheliopathy with recurrent stroke. *J Neuroophthalmol*. 2009;29(2):111-118.
5. Matamala JM, Feuerhake W, Verdugo R. Delayed recurrent stroke in a young patient with acute posterior multifocal placoid pigment epitheliopathy. *J Stroke Cerebrovasc Dis*. 2013;22(8):e630-e634.
6. Darugar A, Mathian A, Lehoang P, Bodaghi B. Acute posterior multifocal placoid pigment epitheliopathy as the initial manifestation of sarcoidosis. *J Ophthalmic Vis Res*. 2011;6(4):338-343.
7. Li B, Bentham RJ, Gonder JR. A case of unilateral and spontaneously resolving posterior uveitis with overlapping features of Vogt-Koyanagi-Harada disease and acute posterior multifocal placoid pigment epitheliopathy. *Springerplus*. 2016;5(1):1471.
8. Fiore T, Iaccheri B, Androudi S, et al. Acute posterior multifocal placoid pigment epitheliopathy: outcome and visual prognosis. *Retina*. 2009;29(7):994-1001.
9. Mandell DM, Mossa-Basha M, Qiao Y, et al; Vessel Wall Imaging Study Group of the American Society of Neuroradiology. Intracranial vessel wall MRI: principles and expert consensus recommendations of the American Society of Neuroradiology. *AJNR. Am J Neuroradiol*. 2017;38(2):218-229.
10. Bullock JD, Fletcher RL. Cerebrospinal fluid abnormalities in acute posterior multifocal placoid pigment epitheliopathy. *Am J Ophthalmol*. 1977;84(1):45-49.