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CASE REPORT

Bilateral Choroidal Occlusion in Antiphospholipid Syndrome Associated with Systemic Lupus Erythematosus

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Key words: anticardiolipin antibodies; antiphospholipid antibodies; choroidal vessels; systemic lupus erythematosus

Abstract This article reports a rare case of bilateral choroidal occlusion that occurred in a 24-year-old woman with antiphospholipid syndrome (APS) associated with systemic lupus erythematosus (SLE). This young lady concurred with aorta ventralis thrombosis and bilateral iliac artery occlusion when presented, and experienced a rapid deterioration of vision. She also has a history of recurrent miscarriage. Corticosteroid, immunosuppression and anticoagulation therapy were administered. Patients with APS associated with SLE are at risk for thrombotic phenomena, which may affect the ocular vessels of all sizes, including choroidal vessel. Our case alerts ophthalmologists and rheumatologists that bilateral choroidal occlusion may indeed be developed in patients with APS associated with SLE, and is a potential cause of visual morbidity.

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ANTIPHOSPHOLIPID syndrome (APS) is an autoimmune disease with moderate to high levels of antiphospholipid antibodies (aPL) in the blood. It's main clinical characteristics include arterial and venous thrombosis and recurrently spontaneous abortion.[1] APS may occur primarily in the absence of systemic disease, or accompany with a known systemic autoimmune disease, such as systemic lupus erythematosus (SLE).[2,3] In the current paper, we report a rare case of bilateral choroidal occlusion as a complication of APS associated with SLE in a woman.

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CASE DESCRIPTION

A 24-year-old female was referred to our clinic, complaining of bilateral progressively reduced visual acuity in the past 2 months. The patient had been suffering from general fatigue, inappetence and intermittent fever for 3 months, and went to see rheumatologists prior to the presence at our clinic. She had no history of malar or discoid rash, no history of photosensitivity, arthralgias, or Raynaud's phenomenon. She had an experience of four

miscarriages for unknown reason.

Clinical work-up was conducted for diagnosis. Laboratory investigation (Table 1) revealed prolonged prothrombin time (PT) and activated partial thromboplastin time (APTT). Antinuclear antibodies (ANA) was 1:80, Coombs test was positive. Levels of C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), anticardiolipin antibody (aCL), and the antibody titer to β-2-glycoprotein 1 (β2GP1) were elevated. Complement levels of C3 and C4 were reduced. Thrombocytopenia was present with an extremely low count of platelet (11×10^9/L). Analyses for anti-double-stranded DNA (anti-dsDNA) antibodies, anti-neutrophil cytoplasmic antibodies with cytoplasmic staining pattern (c-ANCA) and with peripheral staining pattern (p-ANCA), rheumatoid factor, anti-Ro (SSA) and anti-ribonucleoprotein were all negative.

Contrast enhanced abdominal CT revealed thrombosis in aorta ventralis and bilateral iliac arteries. There was no evidence of deep vein thrombosis or pulmonary embolism. No abnormity was found in chest CT, brain MRI, carotid Doppler sonography and colonoscopy.

The best-corrected visual acuity was 40/200 OD and counting fingers OS. Binocular Goldmann applanation intraocular pressures were 10 mmHg OD and 8 mmHg OS. Examinations of cornea, anterior chamber and lens were unremarkable. Clear vitreous was present. Bilateral pupils were equal in diameter with good direct and indirect reactions. Fundus examination revealed slightly pale optic discs. Multifocal choroidal infarction was bilaterally widespread, and choroidal atrophy was seen in all quadrants of both eyes. There were a few retina folds at the posterior pole of the left eye, with macular involvement. Retinal pigment epithelium atrophy in the areas of infarcted choroid was shown. No vasculitis or periphlebitis was found (Fig. 1).

Fluorescein angiography showed normal perfusion of retinal vessels in the early stage, and hypofluoresence in the areas of choroidal infarction; in the late stage, hyperfluoresence was present in the areas around the choroidal infarction and the left foveolar avascular zone (Fig. 2). The indocyanine green angiography (ICGA) of both eyes revealed large wedge-shaped and lobular areas of hypofluorescence corresponding to the location of choroidal non-perfusion lesion (Fig. 3). Optical coherence tomography (OCT) revealed that the thicknesses of bilateral choroid, especially in areas of choroidal infarction, reduced distinctly (Fig. 4).

Table 1. Results of laboratory examination of a 24-year-old patient with APS associated SLE

Items of laboratory examination	Tested value	Normal range
Prothrombin time (PT) (s)	17.8	10.4-12.6
Activated partial thromboplastin time (APTT) (s)	120	22.7-31.8
C-reactive protein (CRP) (mg/L)	76.45	⩽10mg
Erythrocyte sedimentation rate (ESR) (mm/h)	125	0-20 mm
Anticardiolipin antibody (aCL) (U/ml)	120	<10U
β-2-glycoprotein 1 (β2GP1) (U/ml)	155	<5U
Complement C3 (g/L)	0.40	0.80-1.20
Complement C4 (g/L)	0.048	0.10-0.40
Platelet count (/L)	11×10^9	$100\text{-}300\times10^9$

APS, antiphospholipid syndrome; SLE, systemic lupus erythematosus.

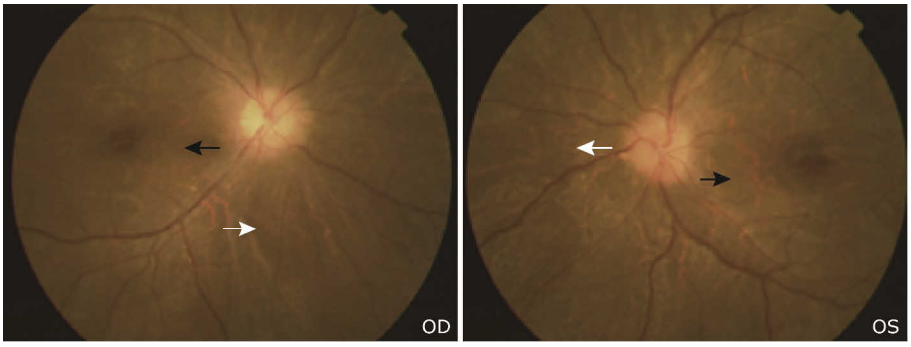

Figure 1. Fundus photography of both eyes.
Slightly pale optic discs, bilaterally multiple areas of choroidal atrophy (white arrow), a few retina folds at the posterior pole (black arrow), and atrophy of the retinal pigment epithelium.

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