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CASE REPORT

Putcher-like retinopathy revealing systemic lupus erythematosus in a young woman

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ABSTRACT

Background: To report a case of Purtscher-like retinopathy revealing systemic lupus erythematosus (SLE) in a young woman with renal failure. **Case presentation:** A 32-year-old woman with renal failure presented with a sudden bilateral decrease in visual acuity. Best-corrected visual acuity (BCVA) was 4/10 in the right eye and 1/50 in the left eye. Fundus examination revealed cotton-wool spots, Purtscher flecken, and retinal hemorrhages. Optical coherence tomography demonstrated cystoid macular edema in the left eye. The diagnosis of systemic lupus erythematosus was established based on the revised American College of Rheumatology (ACR) criteria. The patient was subsequently treated with corticosteroids and immunosuppressive therapy. **Conclusion:** Although vaso-occlusive retinopathy is a rare manifestation of SLE, it may serve as an initial presenting feature, allowing early diagnosis and prompt initiation of appropriate management.

Keywords: Purtscher retinopathy, systemic lupus erythematosus, retinal vascular occlusion.

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1. INTRODUCTION

Purtscher's retinopathy is a rare eye condition, first described by Austrian ophthalmologist Otmar Purtscher in 1910, who observed areas of superficial retinal whitening and peripapillary hemorrhages, often located around the optic nerve head in patients with severe head trauma [1,2]. Over the past 50 years, the same clinical picture has been attributed to several non-traumatic systemic conditions, namely acute pancreatitis, fat embolism syndrome, and autoimmune and connective tissue disorders, including systemic lupus erythematosus (SLE) [3]. The term “Purtscher-like retinopathy” (PLR) is the correct designation to describe this retinopathy occurring in these non-traumatic situations. The annual incidence of Purtscher retinopathy and PLR is 0.24 cases per million. Diagnosis is mainly clinical in the presence of an attributable cause [4,5]. Some authors suggest that incidence is probably underreported due to pauci-symptomatic cases that escape diagnosis.

The classic clinical presentation is characterized by sudden loss of vision, which may occur within hours of the onset of the triggering condition. The diagnosis is based on a fundus examination that reveals characteristic lesions: retinal hemorrhages, cotton wool spots (58%), and Purtscher's flecken, which are pathognomonic (50-53% of cases). The condition is bilateral in 60-75% of cases [1,4]. At present, the exact pathogenesis remains unclear. It is probably an occlusive microangiopathy, with all the characteristics suggesting microembolic occlusion of the retinal precapillary arterioles [1,2].

We report a case of PLR indicative of SLE.

2. OBSERVATION

A 32-year-old married woman with no children and a history of miscarriage, followed in the nephrology department for acute kidney injury, presented in March 2016 with a sudden bilateral decrease in visual acuity. Data collection for this case was conducted under strict anonymity to ensure patient confidentiality.

Ophthalmic examination found a best corrected visual acuity (BCVA) of 4/10 in the right eye (OD) and 1/50 in the left eye (OS). On slit lamp examination, both anterior segments were unremarkable, while the fundus examination of the OD showed dysoric retinopathy, flame-shaped hemorrhages, Purtscher's flecken, macular thickening, and localized arteriolar narrowing, without signs of active vasculitis. The optic disc and retinal periphery were normal. The OS revealed a similar but more severe appearance, with ischemic macular thickening extending to the superior temporal arcade (Figure 1).

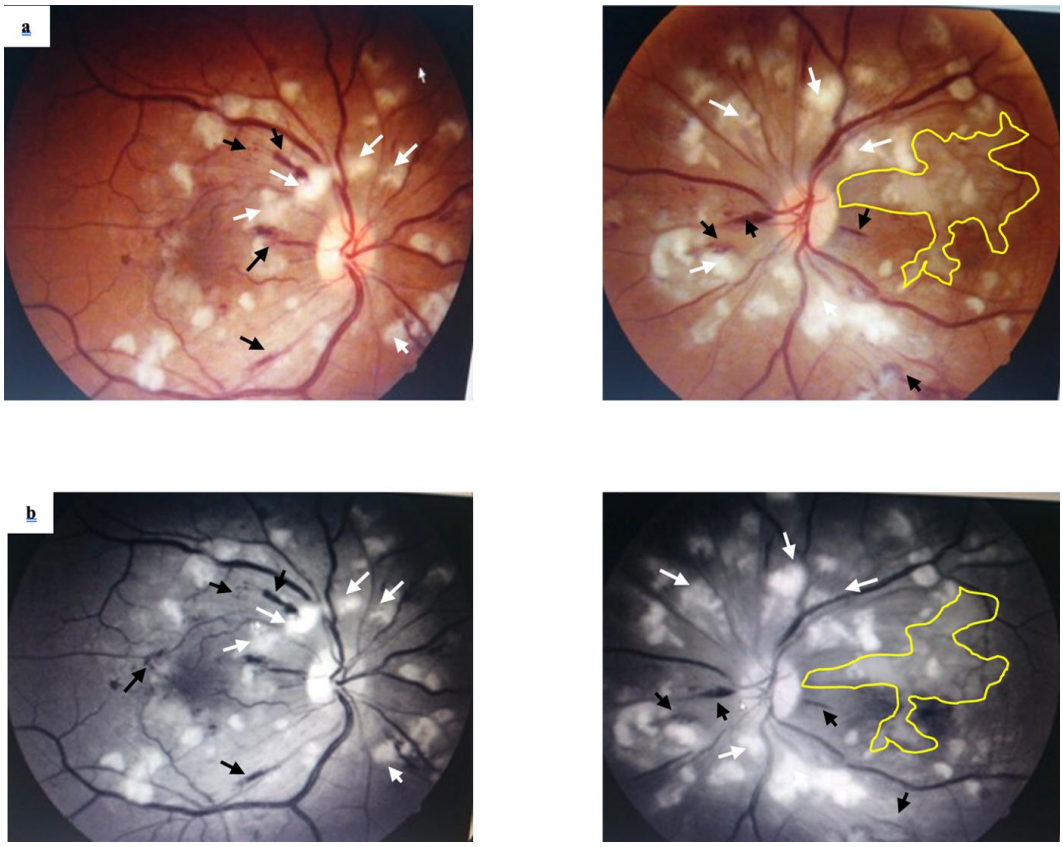

Figure 1. a. Dysoric retinopathy in the context of Purtscher-like retinopathy. Fundus photographs of the right and left eyes: multiple cotton-wool spots at the posterior pole, primarily around the optic nerve (white arrow), flame-shaped hemorrhages (black arrow), Purtscher flecken (yellow freeform shape), macular edema, and localized arteriolar narrowing in places. Note the absence of signs of active vasculitis or papillary edema. b. Red-free photographs of the same patient: the same anomalies were found in the fundus with improved visibility of the flecken.

Fluorescein angiography could not be performed due to impaired renal function. Optical coherence tomography (OCT) revealed in OS focal hyperreflective lesions of the inner nuclear layer, consistent with paracentral acute middle maculopathy (PAMM), reflecting ischemia of the intermediate and deep retinal capillary plexuses. In the OD, there was macular thickening associated with focal hyperreflective lesions of the outer plexiform and nuclear layers, without cystoid spaces. In the OS, the involvement was more pronounced, with marked macular thickening, extensive intraretinal cystoid spaces, and more diffuse PAMM signs (Figure 2). The general examination revealed a blood pressure of 220/120 mmHg, with mild left ventricular hypertrophy on echocardiography.

Laboratory work-up was abnormal, with an erythrocyte sedimentation rate of 20 mm at the first hour and 80 mm at the second hour. CRP at 12 mg/L, anemia was present with thrombocytopenia and lymphopenia in the blood count. Serum creatinine was 48 mg/L,

urea 0.45 g/L, 24-hour proteinuria 360 mg/24 hours, with signs of kidney damage on ultrasound. Immunological testing revealed the presence of antinuclear antibodies, anti-native DNA antibodies, and anti-SSA antibodies, with no antiphospholipid antibodies. Liver function tests, blood sugar and thyroid tests, coagulation tests, serology, and magnetic resonance imaging of the brain were normal.

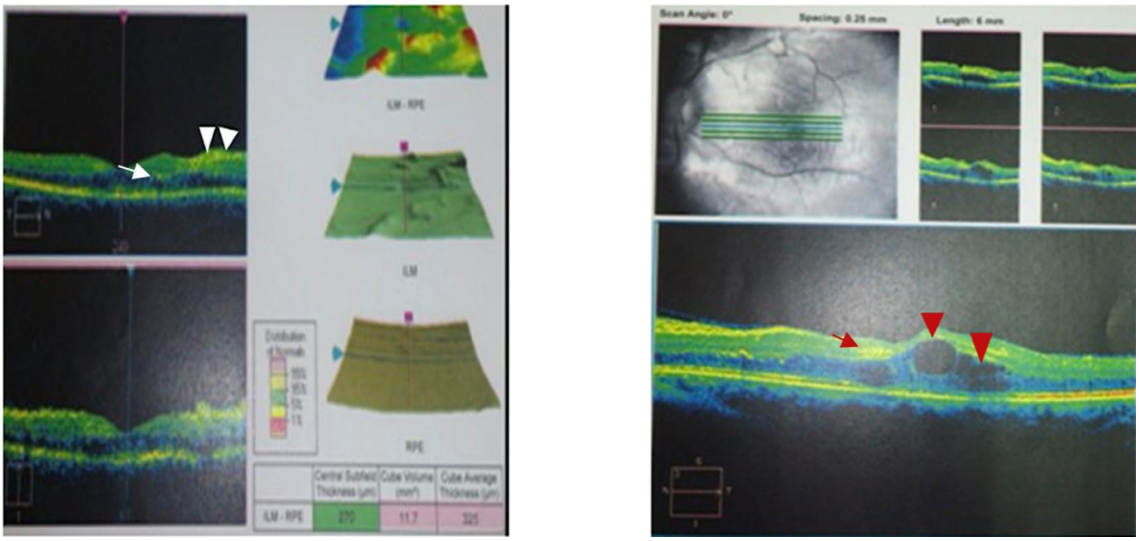

Figure 2. Spectral-domain macular OCT of the same patient: hyperreflectivity of the inner layers in the papillomacular area, particularly the nerve fiber layer, corresponding to the presence of cotton-wool spots (white arrowhead). In the OD, macular thickening associated with focal hyperreflective lesions of the outer plexiform and nuclear layers (white arrow), without cystoid spaces. In OS focal hyperreflective lesions of the inner nuclear layer, with paracentral acute middle maculopathy (PAMM), in the OS (red arrow) and extensive intraretinal cystoid spaces (red arrowhead).

At the end of these investigations, the diagnosis of Purtscher-like retinopathy revealing a flare of SLE was made based on the presence of more than four diagnostic criteria according to the lupus classification criteria proposed by the American Rheumatism Association.

Treatment of the flare-up was initiated with prednisone 1 mg/kg/day, tapered very slowly, combined with adjuvant treatment with immunosuppressants and control of arterial hypertension. The outcome was favorable, with progressive visual recovery beginning on day 8 and reaching the final level at 3 weeks: BCVA remained between 4/10 and 6/10 in the OD and improved from 1/50 to 4/10 in the OS. Blood pressure and kidney function stabilized.

3. DISCUSSION

PLR is a rare condition that threatens visual prognosis and for which there are no recommendations or consensus on diagnosis or treatment. Although it manifests as a sudden decrease in visual acuity, some authors believe that PLR is often asymptomatic, which is why its actual incidence is uncertain [1,2,6].

The association between PLR and SLE was first characterized by Wu et al. [7]. According to the systematic review by Hashem Abu Serhan et al., SLE accounts for 13% of the etiologies of PLR, followed by acute pancreatitis (11.9%), without the exact pathogenesis being known [4]. Meng et al. confirmed that this involvement occurs almost exclusively in patients with active disease and a high SLEDAI score [8].

The diagnosis is based mainly on the clinical characteristics of the fundus, but also on retinal imaging such as fluorescein angiography or optical coherence tomography. The diagnosis is retained in the presence of at least three of the following elements proposed in the systematic review by Miguel et al. [6] and adopted by Abu Serhan et al. [4]; with retinal abnormalities limited to the posterior pole and in the absence of direct ocular trauma: Purtscher flecken, flame-shaped or dot hemorrhages, cotton-wool spots, an identifiable etiology, and concordant ancillary tests. All of these criteria were met in our patient. Malignant hypertension (220/120 mmHg) at diagnosis raises the question of its role versus PLR in the clinical picture. However, PLR is supported by Purtscher flecken, absence of classic hypertensive signs (papilledema, macular star, nicking), and PAMM on OCT.

The most widely accepted theory currently is that microembolization causes arteriolar precapillary occlusion and microvascular infarcts of the retinal nerve fiber layer. Histopathological studies have demonstrated signs of retinal vascular occlusion with edema of the inner layers of the retina, the subretinal space, and a sudden transition to normal retina [7].

Our patient had positive anti-double-stranded DNA and anti-SSA antibodies in the absence of antiphospholipid antibodies. The classic literature readily attributes lupus PLR to the associated antiphospholipid syndrome, considered a major factor in microthrombosis and the severity of retinal damage [9]. This configuration, recently highlighted by Wang and Bao (2024), suggests that antiphospholipid antibodies are not the only thrombogenic factor during lupus [10]. Anti-double-stranded DNA antibodies could play a specific role in thrombosis through platelet activation. In addition, our case clinically illustrates the convergence of several mechanisms on the retinal microvascular bed: deposition of immune complexes related to lupus activity, endothelial microthrombosis in the absence of aPL, and malignant arterial hypertension secondary to lupus nephropathy and the activation of the renin-angiotensin-aldosterone system. This convergence, theoretically described by Meng et al., is rarely illustrated in the literature with such a clinical conjunction[5]. Our observation, therefore, supports the emerging hypothesis of a role of other autoantibodies in the pathophysiology of lupus LPR and underlines the multifactorial nature of retinal microvascular lesions during lupus.

4. CONCLUSION:

Although rare, the association between PLR and SLE is possible and may be a precursor to the disease. It is distinguished from other forms of lupus-associated retinal vasculitis by its specific clinical and pathophysiological features.

Competing interests: The authors declare that they have no competing interest.

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