Purtscher-Like Retinopathy as an Atypical Initial Manifestation of Systemic Lupus Erythematosus with Vaso-Occlusive Vasculitis: Case Report

Retinopatia Purtscher-*Like* como Manifestação Inicial Atípica de Lúpus Eritematoso Sistémico com Vasculite Vaso-Oclusiva: Caso Clínico

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ABSTRACT

Purtscher-like retinopathy is a rare retinal vasculopathy associated with non-traumatic systemic conditions, including immune-mediated diseases. It may present as an early manifestation of systemic lupus erythematosus (SLE), particularly in the presence of antiphospholipid antibodies. We report a 29-year-old woman who presented with bilateral visual loss. Fundoscopy showed cotton wool spots, Purtscher flecken, marked retinal edema, and intraretinal hemorrhages. Optical coherence tomography demonstrated inner retinal hyperreflectivity and cystoid macular edema, and fluorescein angiography revealed macular and peripheral nonperfusion. Laboratory testing showed positive antinuclear antibodies (1:2560), anti-dsDNA, anti-ribosomal P antibodies, low complement levels and antiphospholipid antibodies, supporting the diagnosis of SLE with probable secondary antiphospholipid syndrome. Treatment with systemic corticosteroids, rituximab, and intravitreal dexamethasone resulted in limited visual recovery. This case highlights Purtscher-like retinopathy as a severe initial manifestation of SLE and underscores the importance of early recognition to prevent significant complications.

KEYWORDS: Antiphospholipid Syndrome; Lupus Erythematosus, Systemic; Retinal Diseases; Retinal Vasculitis.

RESUMO

A retinopatia Purtscher-*like* é uma vasculopatia retiniana rara, descrita em condições sistémicas não traumáticas, incluindo doenças autoimunes. Pode constituir uma manifestação inicial do lúpus eritematoso sistémico (LES), sobretudo quando associada a anticorpos antifosfolipídicos. Relatamos o caso de uma mulher de 29 anos com perda visual bilateral. A fundoscopia revelou manchas algodonosas, Purtscher *flecken*, edema retiniano e hemorragias intrarretinianas. A tomografia de coerência ótica demonstrou hiper-refletividade das camadas internas e edema macular cistoide, e a angiografia fluoresceínica evidenciou não perfusão macular e periférica.

Os exames laboratoriais revelaram anticorpos antinucleares positivos (1:2560), anti-dsDNA, anticorpos anti-ribossoma P, complemento diminuído e anticorpos antifosfolipídicos, permitindo o diagnóstico de LES com provável síndrome antifosfolipídica secundária. A doente foi tratada com corticoterapia sistémica, rituximab e dexametasona intravítrea, obtendo recuperação visual limitada. Este caso realça a retinopatia Purtscher-*like* como manifestação inicial grave do LES e salienta a importância do reconhecimento precoce para prevenir complicações significativas.

PALAVRAS-CHAVE: Doenças da Retina; Lúpus Eritematoso Sistémico;Síndrome Antifosfolipídico; Vasculite Retiniana.

INTRODUCTION

Purtscher-like retinopathy (PLR) is a rare occlusive microangiopathy of the retina, typically presenting with sudden bilateral vision loss of variable severity, cotton wool spots, retinal whitening (Purtscher flecken), retinal hemorrhages, and optic disc swelling.[1,2] While the original Purtscher retinopathy description by Otmar Purtscher in 1910 was related to trauma-induced cases, the Purtscher-like variant occurs in non-traumatic systemic conditions, such as acute pancreatitis, fat embolism syndrome, renal failure, childbirth, and connective tissue disorders.[1-4] PLR has rarely been described in the literature as a complication of systemic lupus erythematosus (SLE) or as a distinct category of severe retinal vaso-occlusive disease in SLE.[5]

SLE is a chronic, multisystem, immune-mediated connective tissue disease with a broad range of clinical presentations, predominantly affecting women of childbearing age.[6,7] It may involve the periorbita, ocular adnexa, globe, and optic nerve.[8] Ocular involvement often includes keratoconjunctivitis sicca, the most common manifestation, but posterior segment involvement, affecting the retina, choroid, retinal vessels, and optic nerve, can result in the most severe visual complications, particularly through optic neuropathy and retinal vascular occlusion. Retinal vascular abnormalities occur in up to 29% of SLE patients, especially during periods of active systemic disease.[8,9] Among these, PLR is an exceptionally rare initial manifestation that requires prompt recognition and appropriate treatment to preserve vision and control systemic disease activity.[10]

In this report, we describe an unusual case of bilateral Purtscher-like retinopathy as the initial manifestation of SLE in a previously healthy young woman, later found to have features suggestive of secondary antiphospholipid syndrome.

CASE REPORT

A 29-year-old female with no significant past medical history presented to the emergency department with progressive painless visual loss. One week prior, she noted blurred vision in the right eye, which later affected the fellow eye. She reported no pain, diplopia, photopsia, floaters, trauma, or history of prior eye disease or consultations.

Her symptoms were preceded by a one-month history of productive mucous cough and, later, dysuria. Two months earlier, she had experienced a febrile illness with pharyngitis and myalgia, treated empirically with antibiotics. She denied systemic symptoms suggestive of immune-mediated disease, including arthralgias, rash, photosensitivity, oral ulcers, or alopecia. She was a non-smoker, was not taking any regular medication, and denied drug use or recent travel.

On examination, she was conscious and oriented, afebrile, and tachycardic, with normal oxygen saturation. Painless cervical and supraclavicular lymphadenopathy was noted. Pulmonary auscultation revealed fine right basal crepitations. No cutaneous or articular abnormalities were found. Neurological examination was unremarkable.

Ophthalmologic evaluation revealed visual acuity (Snellen) of 20/800 in the right eye and 20/160 in the left eye, with no improvement using a pinhole. The anterior segment was unremarkable. On pupillary examination, a relative afferent pupillary defect was detected in the right eye. Fundoscopic examination showed bilateral pale optic discs, multiple peripapillary cotton wool spots, Purtscher flecken, macular edema, and peripheral blot hemorrhages, more pronounced in the right eye (Figs. 1A and 1B). Optical coherence tomography (OCT) revealed inner retinal hyperreflectivity and cystoid macular edema in both eyes (Figs. 2A and 2B). Fluorescein angiography demonstrated extensive macular and peripheral retinal nonperfusion, without signs of neovascularization (Figs. 3A-H). These angiographic findings are consistent with widespread retinal ischemia and correlate with the extensive cotton-wool spots and retinal whitening seen on color fundus photography (Figs. 1A and 1B).

Laboratory investigations revealed anemia (Hb 10.8 g/dL), classified as hypoproliferative, without iron or vitamin B12 deficiency, but with mild folic acid deficiency. There were no signs of hemolysis. The erythrocyte sedimentation rate was elevated (73 mm/h), while C-reactive protein remained negative. Mild elevation of liver enzymes was observed (AST 141 U/L, ALT 78 U/L), along with increased lactate dehydrogenase (LDH 335 U/L). Renal function was preserved, with mild proteinuria (0.22 g/24h) and no evidence of hematuria. Serological tests showed IgG positivity and indeterminate IgM for Epstein-Barr virus and cytomegalovirus, as well as IgG and IgM positivity for herpes simplex virus; however, PCR tests performed on blood samples for all viruses were negative, excluding active in-

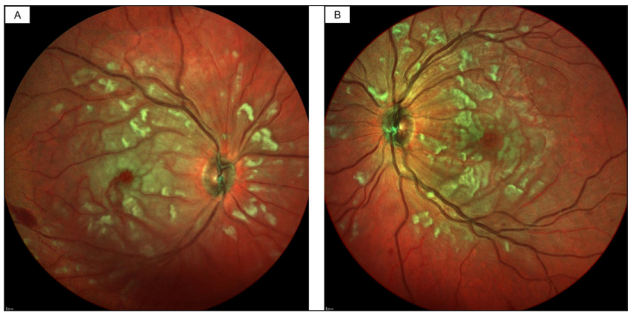

Figure 1. Fundus photography findings at presentation. (A) Multicolor fundus photograph of the right eye at presentation, showing multiple peripapillary cotton-woolspots, Purtscher flecken, macular edema, and intraretinal hemorrhages. (B) Multicolor fundus photograph of the left eye at presentation, also demonstrating extensive cotton-woolspots, Purtscher flecken, and macular involvement. Original multicolor images generated directly by the Heidelberg (Spectralis) OCTsoftware without post-processing. Apparent cropping or tilting reflects technical limitations of the in vivo acquisition.

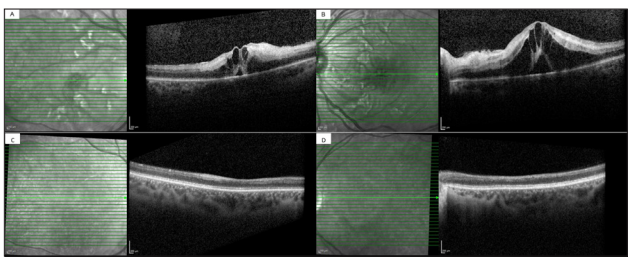

Figure 2. Optical coherence tomography findings at presentation and 19-month follow-up. At presentation, OCT of the right (A) and left (B) eyes shows cystoid macular edema with marked inner retinal hyperreflectivity. At 19-month follow-up, OCT of the right (C) and left (D) eyes demonstrates severe macular atrophy with loss of the normal foveal contour and marked loss of the inner retinal layers, consistent with irreversible macular damage. Original images generated directly by the OCT device without post-processing; any apparent cropping or tilting reflects technical limitations of the in vivo acquisition.

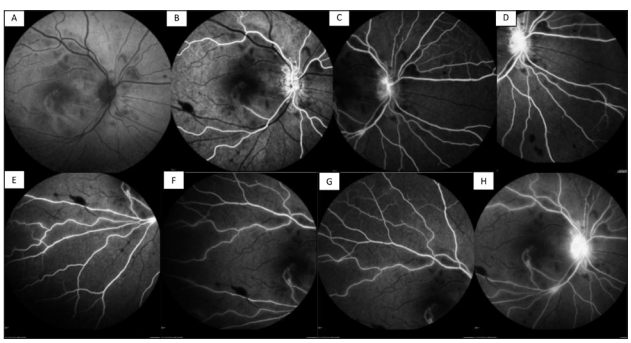

Figure 3. Fluorescein angiography at presentation demonstrating extensive macular and peripheral retinal capillary non-perfusionin the right (A–D) and left (E–H) eyes, without evidence of retinal neovascularization.

fection. Immunological studies revealed positive antinuclear antibodies at a high titer (1:2650, homogeneous pattern), positive anti-double-stranded DNA and anti-ribosomal P antibodies, and low complement levels (C3, C4, CH50). Lupus anticoagulant was positive, whereas anticardiolipin and anti-β2 glycoprotein I antibodies were negative.

A chest X-ray revealed an infiltrate in the right lower lobe. Computed tomography (CT) of the thorax, abdomen, and pelvis showed extensive cervical, supraclavicular, mediastinal, axillary, para-aortic, and iliac lymphadenopathy, including a retroperitoneal conglomerate measuring 3 cm. A subsequent CT scan of the brain and orbits was unremarkable. Brain magnetic resonance imaging (MRI) was unremarkable, ruling out Susac syndrome. Axillary lymph node biopsy showed no pathological abnormalities.

Based on these findings, a diagnosis of Purtscher-like syndrome in the context of systemic lupus erythematosus (SLE) was made.

Systemic corticosteroid therapy was initiated with oral prednisone at 1 mg/kg/day on patient's third day of hospitalization, along with prophylactic anticoagulation using enoxaparin 1 mg/kg twice daily. Concurrently, intravitreal corticosteroid therapy was initiated with a 700 μg dexamethasone implant administered in both eyes. The case was discussed at a multidisciplinary meeting, including Internal Medicine, Infectious Diseases and Ophthalmology. It was decided to increase systemic immunosuppression with intravenous methylprednisolone pulses of 1 g daily for five consecutive days, followed by resumption of oral prednisone at 1 mg/kg/day. In parallel, intraocular anti-VEGF therapy with intravitreal bevacizumab 1.25 mg/0.05 mL was initiated in both eyes and was initially administered at approximately monthly intervals. Subsequently, injections were repeated on a pro re nata basis, according to OCT and fluorescein angiography findings. Given the potential future need for cyclophosphamide therapy and its impact on fertility, the patient was referred to a Fertility Preservation Clinic. The patient subsequently received rituximab, initiated with two doses of 1 g administered 15 days apart.

Follow-up imaging demonstrated partial regression of macular edema, although inner retinal ischemia and significant retinal atrophy persisted. Follow-up fluorescein angiography was performed during the first year to monitor for retinal neovascularization and to reassess the extent of macular and peripheral ischemia, supporting the decision to continue intravitreal bevacizumab without performing peripheral laser photocoagulation. Visual acuity improved slightly in the left eye to 20/100, while the right eye remained severely impaired at 20/250.

Over the ensuing months, systemic parameters normalized. Corticosteroid therapy was progressively tapered, and the patient is currently maintained on low-dose prednisone (2.5 mg/day) in combination with scheduled rituximab infusions, administered as 1 g on day 0 and day 15, repeated every six months. No additional organ involvement has been detected. Hydroxychloroquine was initially withheld due to concerns regarding potential macular toxicity in the setting of active retinal involvement.

At the most recent ophthalmologic evaluation, 19 months after presentation, visual acuity remained stable (right eye 20/250; left eye 20/100), with clinical evidence of extensive ischemic retinal damage, including optic disc pallor, vascular attenuation, and thinning of the inner retinal layers (Figs. 4A and 4B). Optical coherence tomography demonstrated severe macular atrophy, characterized by complete loss of the normal foveal contour and marked

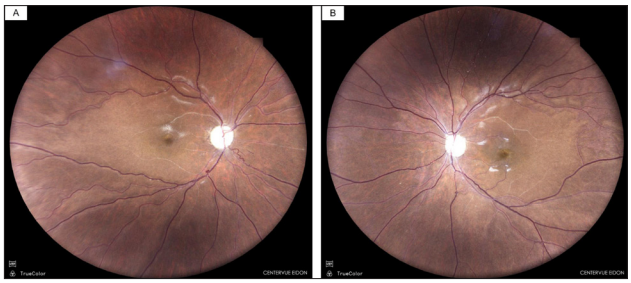

Figure 4. Fundus findings at 19-month follow-up. Color fundus photographs of the right (A) and left (B) eyes, demonstrating retinal damage, including optic disc pallor, vascular attenuation, and atrophic pigmentary alterations of the macula.

loss of the inner retinal layers in both eyes (Figs. 2C and 2D), changes that reflect long-standing, irreversible macular damage and are consistent with a poor long-term visual prognosis despite adequate systemic disease control. Intravitreal anti-VEGF injections are being administered regularly under close ophthalmologic monitoring.

Currently, the patient remains functionally independent despite significant visual impairment, with satisfactory systemic disease control maintained through multidisciplinary follow-up.

Treatment timeline:

1. Day 0 (presentation): Diagnostic workup; baseline fundus examination, OCT and fluorescein angiography; no SLE-directed therapy initiated.
2. Day 3: Start of oral prednisone 1 mg/kg/day and prophylactic enoxaparin 1 mg/kg twice daily; bilateral 700 µg intravitreal dexamethasone implants.
3. Days 4–8: Intravenous methylprednisolone 1 g/day for 5 days; continuation of anticoagulation.
4. First month: Fertility preservation counselling; first bilateral intravitreal bevacizumab 1.25 mg/0.05 mL.
5. Weeks 4 and 6: Two rituximab infusions of 1 g, 15 days apart.
6. Months 2–6: Tapering of oral prednisone; additional bevacizumab injections at approximately monthly intervals according to OCT/fluorescein angiography findings.
7. After month 6: Rituximab maintenance (1 g on days 0 and 15 every 6 months) plus low-dose prednisone 2.5 mg/day; no cyclophosphamide at any time.
8. Month 19: Latest follow-up with stable reduced visual acuity and severe macular atrophy; systemic SLE in remission.
9. After the period covered by this report: Progression of peripheral ischemia with suspected early neovascularization in the left eye on fluorescein angiography; peripheral panretinal photocoagulation proposed in both eyes because of widespread bilateral peripheral ischemia.

DISCUSSION

PLR is a rare and severe ocular complication of SLE, typically presenting with sudden visual loss, Purtscher flecken, cotton-wool spots, retinal hemorrhages, macular edema, optic disc swelling, and a pseudo–cherry red spot in the absence of trauma. The pathogenesis of PLR remains poorly understood; however, it is widely believed that microembolization, triggered by complement activation and leukocyte aggregation, causes arteriolar precapillary occlusion and microvascular infarcts in the retinal nerve fiber layer, ultimately leading to ischemic injury.3 Retinal vaso-occlusive disease correlates strongly with low complement levels and high titers of anti-dsDNA and antiphospholipid antibodies, as seen in this case.[3,12-13]

PLR may occur either as the first sign of systemic disease or during disease flares. In a case series by Wu *et al*, PLR was associated with high disease activity.[3,11] Visual prognosis is generally poor but varies widely, ranging from almost complete recovery to severe visual impairment.[11] Poor visual outcomes are usually seen in patients with optic disc pallor and macular ischemia.[3]

Although there is no standard ophthalmic therapy, early and aggressive systemic treatment, including corticosteroids and immunosuppressants, may improve outcomes in some cases.[12,13] Although Purtscher-like retinopathy is predominantly an ischemic vaso-occlusive process, inflammation and breakdown of the blood–retinal barrier also contribute to macular edema and further microvascular damage, which justified the use of intravitreal dexamethasone as adjunctive local anti-inflammatory and anti-edematous therapy in this case. Intravitreal anti-VEGF agents have been used to manage macular edema and neovascularization associated with retinal ischemia. Although their efficacy in reversing ischemic damage is limited, combining these injections with properly timed retinal laser photocoagulation can help prevent further progression of ischemic areas and improve clinical outcomes.[11,14] In the present case, intravitreal anti-VEGF therapy with bevacizumab was initiated in both eyes even in the absence of demonstrable neovascularization on fluorescein angiography. Laser photocoagulation is an accepted therapeutic option in ischemic retinal vasculopathies, including PLR and severe vaso-occlusive lupus retinopathy, particularly for the prevention or regression of neovascularization, as illustrated by reports of recurrent PLR complicated by neovascularization and vitreous hemorrhage treated with panretinal photocoagulation.[15] However, panretinal or wide-field peripheral photocoagulation may further reduce peripheral visual fields in eyes with pre-existing extensive ischemia, and visual acuity often remains poor despite neovascular regression. In our patient, peripheral retinal laser photocoagulation was not performed because the extensive ischemic areas were already associated with markedly constricted visual fields. Additional ablation of peripheral retina was considered likely to cause further functional visual field loss without improving central visual acuity. This decision was made in favour of close monitoring and repeated intravitreal anti-VEGF injections as a preventive strategy to reduce the risk of secondary neovascularization from retinal ischemia while preserving the remaining peripheral visual function as much as possible. During the period covered by this report, serial fluorescein angiography confirmed persis-

tent ischemia without development of neovascularization, which guided the choice of ongoing anti-VEGF therapy and close observation rather than immediate peripheral laser treatment. Subsequently, outside the period formally covered by this case report, fluorescein angiography suggested progression of peripheral ischemia with suspected early neovascularization in the left eye, and peripheral panretinal photocoagulation was therefore proposed in both eyes, given the presence of widespread peripheral ischemia bilaterally.

Rituximab has been increasingly recognized as an effective treatment for severe or refractory SLE, including ocular manifestations, with some reports suggesting benefit in PLR.[3,12,13] Our patient received rituximab shortly after corticosteroid pulses and fertility preservation, achieving systemic disease control.

In this case, rituximab was selected early in the disease course due to the severity of retinal involvement, the presence of high disease activity markers, and the need to avoid cyclophosphamide given fertility concerns, as cyclophosphamide is associated with a dose- and age-dependent risk of premature ovarian failure and infertility in premenopausal women with SLE, whereas rituximab is considered safe for fertility preservation.[16-19] Cyclophosphamide was therefore not administered at any point during follow-up. The patient received two induction doses of 1 g, 15 days apart, and is currently planned to receive maintenance infusions every six months, with the regimen to be reviewed based on disease activity and tolerance. This approach has proven effective in sustaining systemic remission to date. Hydroxychloroquine, while a mainstay of SLE treatment,[16] was initially withheld due to concerns regarding potential macular toxicity in the setting of active retinal ischemia.[20] Its initiation is being considered as immunosuppression is gradually tapered, with close adherence to current ophthalmologic screening recommendations.[20]

This case is consistent with previously reported clinical presentations in the literature and underscores the importance of prompt ophthalmologic evaluation in patients with visual symptoms and suspected immune-mediated disease. Multidisciplinary collaboration is essential to optimize diagnosis and management.[14]

In conclusion, this case highlights the importance of considering PLR as a potential presenting feature of systemic lupus erythematosus, particularly in young patients with acute bilateral visual loss and signs of retinal vaso-occlusion. The presence of cotton-wool spots, retinal hemorrhages, and macular ischemia in the absence of trauma should prompt an urgent systemic workup. Despite prompt initiation of high-dose corticosteroids, intraocular therapy, and rituximab, visual outcomes may remain poor due to irreversible ischemic injury. This highlights the need for early recognition, aggressive multidisciplinary intervention, and continued investigation into optimized treatment strategies. As PLR may represent the first manifestation of SLE, ophthalmologists play a key role in facilitating timely diagnosis and systemic management to prevent further morbidity.

CONTRIBUTORSHIP STATEMENT / DECLARAÇÃO DE CONTRIBUIÇÃO

AMS: Conceptualization, drafting of the text, and critical revision for important intellectual content.

KS, JC, CC, and VP: Critical revision for important intellectual content.

All authors approved the final version to be published.

AMS: Conceptualização, redação do texto e revisão crítica do conteúdo intelectual importante.

KS, JC, CC e VP: Revisão crítica do conteúdo intelectual importante.

Todos os autores aprovaram a versão final a ser publicada.

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