Випадок з практики

Severe hypertensive retinopathy secondary to malignant hypertension in systemic lupus erythematosus: a case report

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Тяжка гіпертензивна ретинопатія, що виникає внаслідок злоякісної гіпертензії при системному червоному вовчаку: клінічний випадок

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Abstract

Malignant hypertension is a medical emergency characterized by severe elevation in blood pressure with end-organ damage, including ocular involvement. Systemic lupus erythematosus (SLE) may result from lupus nephritis or vascular involvement in patients, and reports of severe hypertensive retinopathy in this setting are extremely limited. Herein, we report the case of a 30-year-old woman with SLE and poorly controlled chronic hypertension who presented with sudden bilateral vision loss. Ophthalmologic examination revealed grade IV hypertensive retinopathy with macular star exudates, a finding not previously described in similar reported cases. Optical coherence tomography demonstrated intraretinal hyperreflective deposits at the fovea without active macular edema, and fluorescein angiography showed areas of retinal ischemia. Intensive antihypertensive therapy and immunosuppressive treatment led to systemic stabilization but limited visual recovery. Only two similar cases have been reported, all presenting with hypertensive emergency, active SLE, and visual symptoms; however, the present case uniquely exhibited a macular star pattern. Early ophthalmologic recognition is essential for diagnosis, prognosis, and multidisciplinary management.

Keywords. *Hypertensive Retinopathy, Systemic Lupus Erythematosus, Lupus Nephritis, Hypertensive Crisis, Ophthalmology.*

Резюме

Ми повідомляємо про випадок злоякісної гіпертензії у 30-річної жінки з системним червоним вовчаком СЧВ, яка звернулася з раптовою двосторонньою втратою зору. Офтальмологічне обстеження виявило гіпертензивну ретинопатію IV ступеня з макулярними зірчастими ексудатами, що раніше не було описано в подібних зареєстрованих випадках. Оптична когерентна томографія показала внутрішньосітківкові гіперрефлективні відкладення у фовеа без активного макулярного набряку, а флуоресцентна ангіографія показала ділянки ішемії

DOI: https://doi.org/10.31288/Ukr.j.ophthalmol.202626670

UDC: 617.735-02:616.5-002.525.2

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Received 2025-12-21
Accepted 2026-03-11

Cite this article as: Lozada-Marquez YK, Arteaga-Rivera KM, Martínez JM, Viteri-Solorzano EJ. Severe hypertensive retinopathy secondary to malignant hypertension in systemic lupus erythematosus: a case report. Ukr J Ophthalmol. 2026;2:66-70.

сітківки. Інтенсивна антигіпертензивна та імуносупресивне терапія призвели до системної стабілізації, але обмеженого відновлення зору. Було описано лише два подібні випадки, в яких був гіпертензивний невідкладний стан, активний СЧВ та зорові симптоми; однак, цей випадок унікальним чином демонстрував макулярний зірчастий малюнок. Раннє офтальмологічне розпізнавання є важливим для діагностики, прогнозу та багатопрофільного лікування.

Ключові слова. *Гіпертонічна ретинопатія, системний червоний вовчак, вовчаковий нефрит, гіпертонічна криза, офтальмологія..*

Introduction

Malignant hypertension is the most serious presentation of arterial hypertension, although rare, and is characterized by an extreme elevation of blood pressure accompanied by acute lesions in target organs [1]. Arterial hypertension is a complication present in almost half of patients with systemic lupus erythematosus (SLE), attributed to multiple factors such as renal involvement, especially in lupus nephritis, prolonged use of corticosteroids, and predisposition to cardiovascular diseases [2].

At the ocular level, hypertensive retinopathy is a well-documented manifestation of malignant hypertension; however, its presentation with a bilateral star macula pattern is seldom described in the literature and is even rare in patients with SLE. To date, only two similar cases with this pattern have been reported in patients with SLE, highlighting the exceptional nature of this association [3, 4]. SLE-induced endothelial inflammation affects the vasculature, both renal and ocular, which could contribute to the development of hypertensive retinopathy [5, 6, 7]. Although evidence in this area is limited, the rare occurrence of this phenomenon underscores the importance of reporting cases that contribute to a better understanding of its clinical implications and possible relationship with SLE.

In this context, we present the clinical case of a female patient with SLE who developed a hypertensive emergency with significant bilateral visual loss. This case is relevant due to its complex interaction between its adjacent disease, associated secondary complications and ocular involvement. It underscores the importance of early recognition of atypical ophthalmic signs in SLE and the need for comprehensive, multidisciplinary management.

Case description

A 30-year-old Equatorian female with a 9-years SLE history treated with hydroxychloroquine, chronic kidney disease, and uncontrolled hypertension for the past five months, presented with a sudden vision loss and headache over the past 2 hours. Upon further questioning, she reported that mild central visual changes had been present for approximately seven days prior, which she initially attributed to the need for a change in glasses. On the day of admission, the vision deteriorated abruptly, prompting urgent evaluation. A physical examination showed a high blood pressure (243/130 mmHg), generalized pallor, and 1+ pitting edema of the lower extremities.

Laboratory results revealed leukocytosis, moderate anemia, mild elevation of C-reactive protein (CRP) and dyslipidemia with hypercholesterolemia and hypertriglyceridemia, along with decreased coagulation times. Renal profile showed a glomerular filtration rate of 19.61 ml/min, consistent with stage IV renal failure, along with electrolyte imbalances including hypokalemia, Hypocalcemia, and hypomagnesemia. Additional findings included dysproteinaemia with hypoalbuminemia and non-nephrotic range proteinuria. Immunological tests demonstrated hypocomplementemia and positive autoimmune markers (C3:64.9 mg/dl, positive ANA, anti-dsDNA: 133.49 IU/m) with normal anticardiolipin IgM levels and a lupus anticoagulant ratio of 1.2.

Axial STIR magnetic resonance imaging of the orbits (Fig. 1) showed a focal hyperintensity in the proximal retrobulbar segment of the right optic nerve. However, the official radiology report did not confirm optic nerve enhancement or definite structural damage. Neurology evaluated the patient and documented that brain MRI did not show acute or chronic lesions, and that no clear optic nerve injury was identified. Although probable optic neuritis was proposed as a working diagnosis, the neurologist recommended additional dedicated imaging for better characterization and requested ophthalmologic consultation due to persistent visual symptoms.

Before ophthalmologic evaluation, the patient had received high-dose corticosteroid and immunosuppressive

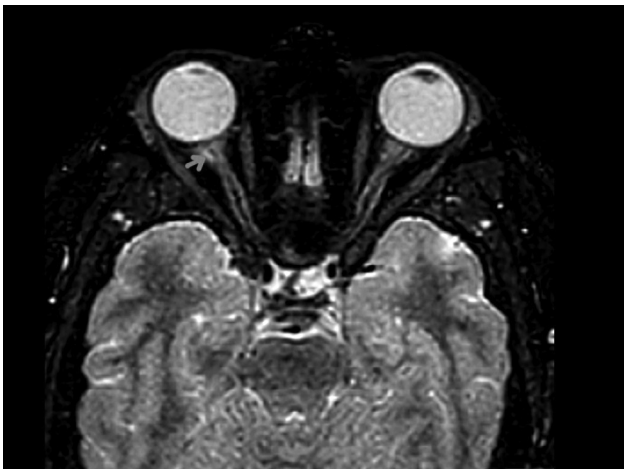

Figure 1. Orbital magnetic resonance imaging (MRI) in axial STIR sequence. A focal hyperintensity (arrow) is observed along the retrobulbar segment of the right optic nerve. The left optic nerve demonstrates normal signal.

therapy — including methylprednisolone 500 mg IV daily for 4 days, cyclophosphamide 1 g IV with premedication (dexamethasone 8 mg IV and metoclopramide 10 mg IV administered as a slow infusion), hydroxychloroquine 200 mg PO daily, and prednisone 5 mg PO daily — along with triple antihypertensive therapy (Losartan 100 mg PO QD AM, Amlodipine 10 mg PO QD PM, and Doxazosin 4 mg PO QD), without any improvement in vision.

Ophthalmology assessed the patient on the seventh day of hospitalization. Her uncorrected visual acuity in both eyes was counting fingers at 1 meter, and intraocular pressure, measured by applanation tonometry, was 16 mmHg in the right eye and 21 mmHg in the left eye. No limitation or pain was observed in extraocular muscle movements; moreover, the red desaturation test and direct pupillary light reflex were symmetric bilaterally. The anterior segment examination was unremarkable. However, indirect ophthalmology revealed a well-defined optic disc with a cup-to-disc ratio of 0.4, along with a macular star pattern of hard exudates, associated with cotton-wool spots and scattered intraretinal hemorrhages in both eyes. Consequently, fundus photography was requested for documentation (Fig. 2 a-b — see cover page 3).

Optical coherence tomography (OCT) of the optic nerve (Fig. 3 — see cover page 3) showed that the peripapillary retinal nerve fiber layer (RNFL) and the ganglion cell complex were within normal limits. The measured Bruch's membrane opening area was 2.73 mm^2 in the right eye and 2.47 mm^2 in the left eye. However, macular OCT (Fig. 2 c-d — see cover page 3) revealed multiple punctate hyperreflective lesions in the parafoveal region of both eyes, corresponding to the star-shaped lipid exudates. These lesions were associated with disruption of the photoreceptor layer, external limiting membrane, and interdigitation zone, predominantly in the right eye.

Bilateral fluorescein angiography was performed, revealing a broad area of macular hypofluorescence without evidence of active leakage in the right eye (Fig. 4a), likely due to blockage from subretinal lipid deposits and alterations of the retinal pigment epithelium. In another hand, the left eye (Fig. 4b) exhibited foveal hypofluorescence with an irregular capillary network pattern and no signs of leakage.

A multidisciplinary approach was adopted for the clinical management, involving internal medicine and rheumatology specialist. Treatment focused on stabilizing blood pressure using systematic antihypertensive agents to prevent damage to other target organs (Quadruple antihypertensive therapy: Losartan 100 mg PO QD AM, Amlodipine 10 mg PO QD PM, Carvedilol 6.25 mg PO every 12 h, Doxazosin 4 mg PO QD). Topical bromfenac 0.09% eye drops were prescribed every 12 hours, along with timolol 0.5% eye drops as neuroprotective agents, also prescribed twice daily in both eyes. Concurrently, immunosuppressive and immunomodulatory therapy to control SLE-related inflammatory activity, including high-dose methylprednisolone pulses (500 mg IV daily for 4–5 days), cyclophosphamide 1 g IV, and rituximab 1 g IV, along with maintenance hydroxychloroquine 200 mg PO daily.

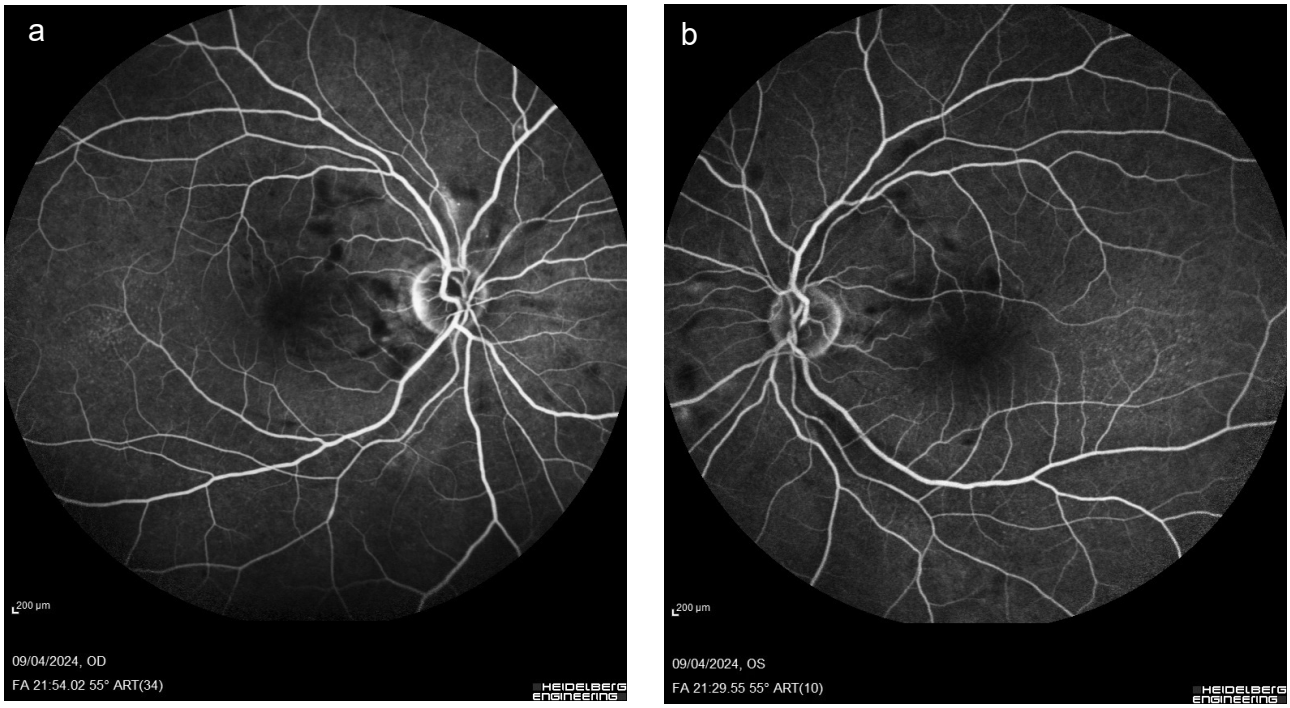

Figure 4. Late-phase fluorescein angiography shows extensive macular hypofluorescence with irregular borders and no active fluorescein leakage in the right eye (a). The left eye (b) presents foveal hypofluorescence with irregular capillary pattern and no leakage.

The final diagnosis was bilateral hypertensive retinopathy with macular star secondary to malignant hypertension triggered by reactivation of lupus nephritis due to poor adherence to SLE treatment. Discontinuation of immunosuppressive and antihypertensive therapy resulted in active lupus nephritis and a hypertensive crisis, leading to retinal microvascular damage characterized by cotton-wool spots, intraretinal hemorrhages, and lipid exudation, ultimately causing acute visual impairment.

After 15 days of hospitalization, with control of hypertension and SLE-related inflammation, the patient was discharged with follow-up by rheumatology, nephrology, and ophthalmology. Three months later, at an ophthalmology follow-up, uncorrected visual acuity was logMAR 1.3 (OD) and 0.7 (OS), after which the patient did not return for further follow-up, completing the documented course of the case.

Discussion

Although malignant hypertension is less common today due to advances in blood pressure control, it remains a critical complication in patients with SLE, particularly when renal involvement such as lupus nephritis is present [5]. This condition can lead to multiorgan damage, particularly affecting the eyes, and may manifest as hypertensive retinopathy, optic neuropathy, or choroidopathy, which in some cases can result in vision loss [1, 6, 7]. In this case, the patient developed visual loss secondary to hypertensive retinopathy, underscoring that poorly controlled systemic lupus erythematosus, particularly in the context of non-adherence to therapy, can precipitate indirect ocular injury through its complications.

Building on the clinical context of our case, malignant hypertension results from an acute, severe elevation in blood pressure that overwhelms vascular autoregulation, leading to endothelial injury, fibrin deposition, and ischemia [7]. In patients with SLE, systemic complications such as lupus nephritis and chronic hypertension amplify microvascular vulnerability,predisposing to rapid vascular decompensation. In the eye, this manifests as sudden retinal ischemia and breakdown of the blood-retinal barrier, leading to cotton wool spots, flame-shaped hemorrhages, macular edema, and optic disc swelling, while choroidal involvement results in Elschnig spots, Siegrist streaks, and subretinal fluid [7]. These acute vascular changes constitute the main pathogenic mechanism responsible for the patient's sudden vision loss. Ophthalmologic findings, including retinal vascular changes, are characteristic of sustained arterial hypertension [8] and are further influenced by endothelial dysfunction, oxidative stress, chronic inflammation, and comorbidities such as diabetes mellitus and kidney disease, which can exacerbate disease progression [9].

The patient initially attributed her slowly progressive visual changes to the need for new corrective lenses. When she experienced sudden and severe vision loss, she sought emergency care. Ophthalmologic examination revealed cotton wool spots, flame-shaped hemorrhages and macular star, supporting the diagnosis [7, 10]. Neuroimaging and ophthalmologic assessments excluded autoimmune optic neuropathy, inflammatory vasculitis, and vascular occlusion. Findings, lack of visual response to immunosuppressive therapy, and retinal patterns confirmed acute hypertensive ocular injury as the cause.

Hypertensive retinopathy in patients with SLE is a rare and poorly documented manifestation. In the literature, we found two similar cases: one involving a 38-year-old Korean patient [3] and another 14-year-old Brazilian patient [4]. Both cases shared common features with our patient, such as hypertensive crisis, active lupus activity, and visual disturbances, but also presented differences in comorbidities and ophthalmologic findings, such as optic disc edema. These variations reflect the heterogeneity in the presentation and progression of hypertensive retinopathy in SLE, suggesting that additional factors may influence its clinical manifestation.

Treatment of malignant hypertension secondary to SLE requires a comprehensive and multidisciplinary approach, combining antihypertensive therapy with immunotherapy targeting lupus nephritis- a key contributor to morbidity and mortality. In this case, brimonidine was prescribed despite normal intraocular pressure due to its neuroprotective effects on the retina [11]. Implementing a well-rounded treatment strategy is crucial to improving visual outcomes and overall prognosis.

This case is strengthened by the thorough documentation of clinical progression and detailed ophthalmologic findings, enabling a meaningful correlation between retinal changes and the patient's systemic condition. However, it also has limitations, including its nature as a single case report, the absence of follow-up evaluations, and the lack of visual field testing-each of which could have contributed valuable information regarding long-term visual outcomes.

In conclusion, vision loss in patients with uncontrolled systemic lupus erythematosus — especially with complication such as lupus nephritis and malignant hypertension — reflects systemic vascular injury affecting the eyes. Prompt evaluation for hypertensive retinopathy is essential, highlighting ocular involvement as an early indicator of systemic instability and the need for multidisciplinary management.

Author Contributions

KLM: Conceptualization; writing – drafting; writing – design; Investigation. KAR: writing - review and revision; writing – design. JMM: writing - review and revision; Supervision. EVS: Supervision, Visualization; writing - review and revision. All authors read and approved the final version of the manuscript.

Funding

The authors declare that no funds, grants, or other support were received during the preparation of this manuscript.

Conflict of Interest

The authors declare that they have no conflicts of interest that could influence their opinion on the subject matter or materials described and discussed in this manuscript.

Acknowledgements

During the preparation of this work the authors used ChatGPT in order to improve the clarity and quality of the English language. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

Disclaimers

The views expressed in this case report are solely those of the authors and do not necessarily reflect the official policy or position of their affiliated institutions.

Ethics Statement

This case report was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from the patient for publication of the clinical information. Patient anonymity has been preserved.

Data Availability Statement

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study. All relevant clinical information is included in this published case report.

Abbreviations

SLE – Systemic lupus erythematosus; CRP – C-reactive Protein; C3 – Complement component 3; ANA – Antinuclear antibodies; anti-dsDNA – Anti–double-stranded DNA antibodies; OCT – Optical coherence tomography; RNFL – Retinal nerve fiber layer; logMAR – Logarithm of the minimum angle of resolution; OD – Oculus dexter; OS – Oculus sinister; PO – Orally / by mouth / oral administration; QD – Once daily; AM – Morning; IV – Intravenous.

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