Madurapperuma *et al.*
*Journal of Medical Case Reports* (2025) 19:132
https://doi.org/10.1186/s13256-025-05158-6

Journal of
Medical Case Reports

CASE REPORT

Open Access

# Bilateral retinal vasculitis presenting as the first manifestation of systemic lupus erythematosus: a case report

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## Abstract

**Background** Systemic lupus erythematosus is a chronic autoimmune multisystemic disease in which ocular manifestations occur in up to one-third of patients. Although retinal vasculitis is a recognized feature of systemic lupus erythematosus, it is extremely rare to present as the first manifestation. This report describes a rare case of systemic lupus erythematosus, characterized by bilateral retinal vasculitis presenting as the first manifestation, which was subsequently complicated by lupus nephritis.

**Case presentation** We report the case of a 32-year-old female patient from Sri Lanka who presented with progressive visual impairment, followed by fatigue, malaise, and arthralgia. She was initially diagnosed with retinal vasculitis. Later, she developed constitutional symptoms followed by mucocutaneous and renal manifestations consistent with systemic lupus erythematosus. Laboratory findings supported the diagnosis of systemic lupus erythematosus with positive antinuclear antibody and anti-double stranded DNA, as well as low complement levels. Renal biopsy confirmed class III lupus nephritis. The patient received treatment with corticosteroids, mycophenolate mofetil, and hydroxychloroquine, which resulted in significant improvements in visual, renal, and other clinical symptoms.

**Conclusion** It is important to consider systemic lupus erythematosus in the differential diagnosis of isolated retinal vasculitis, even in the absence of its classic symptoms. Early identification through improved diagnostic tools and revised criteria could facilitate timely intervention and improve patient outcomes.

**Keywords** Retinal vasculitis, First presentation, Systemic lupus erythematosus, Lupus nephritis

## Background

Systemic lupus erythematosus (SLE) is a chronic, multisystemic autoimmune disease characterized by widespread inflammation and potential involvement of various organ systems, including the skin, joints, kidneys, central and peripheral nervous systems, eyes, lungs, heart, and blood cells [1]. While SLE affects both men and women, it is significantly more prevalent among women, particularly during their child-bearing years [2].

Up to one-third of patients with SLE experience ocular manifestations, which can be a marker for overall systemic disease activity and are potentially associated with significant morbidity [3]. SLE can affect any ocular structure, with keratoconjunctivitis sicca due to secondary Sjögren's syndrome being the most frequent manifestation. Retinal vasculitis, the second most common eye pathology in patients with lupus, often presents with

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mild, visually non-threatening cotton wool spots [4]. However, more severe lupus retinopathy can develop, which is commonly observed in patients with active systemic disease and multiorgan involvement, suggesting a potential correlation with poorer prognosis [5].

We present a unique case of a 32-year-old female patient who developed bilateral retinal vasculitis as the first manifestation of SLE, which was subsequently complicated by lupus nephritis (LN).

## Case presentation

A 32-year-old previously healthy female patient from the North Central Province of Sri Lanka presented with gradual onset of visual impairment in both eyes for 2 months. The blurred vision was initially unilateral, involving the right eye, and within the next few weeks, it progressed to involve the left eye. Both near and far visions were impaired, however, her color vision remained unimpaired. It was later associated with persistent, mild frontal headache without aura, photophobia, or focal neurological deficits. There was no redness or pain in the eyes. Over the following weeks, her symptoms progressively worsened to a degree that affected her instrumental activities of daily living.

Eye examination revealed a best corrected visual acuity (BCVA) of 6/36 in both eyes. Fundoscopic examination revealed bilateral cotton wool spots, predominantly over the posterior pole and along the vascular arcades; arteriolar narrowing; venous segmentation; and a few areas of flame-shaped hemorrhages (Fig. 1). The pupils were normal in size without evidence of relative afferent pupillary defects. The visual fields and color vision were normal. The rest of the neurological examination results were normal. A fundal fluorescein angiogram (FFA) revealed retinal vasculitis with occlusive retinal arteriolitis and phlebitis (Fig. 2).

A total of 2 months after the onset of her initial symptoms, while undergoing evaluation for retinal vasculitis, the patient developed a constellation of constitutional and musculoskeletal complaints. This included low-grade fever (101 °F) with chills, fatigue, and loss of appetite. She also experienced arthralgia, involving both small and large joints bilaterally, without objective signs of joint inflammation. Then, 1 week later, the patient developed an erythematous malar rash and a painless ulcer on the hard palate. It was then followed by bilateral periorbital and ankle swelling, which were worse in the morning. The sequence of these symptom presentations is illustrated in Fig. 3.

She did not have a history of chronic cough, hemoptysis, or a past or contact history of tuberculosis. She had no history of valvular or congenital heart disease, and she did not have a history to suggest inflammatory bowel disease. She denied a history of contact with household pets or livestock or recent foreign travel. She also denied high-risk sexual behaviour, unhygienic tattooing, or intravenous drug abuse.

She also did not have photosensitive rashes, increased hair loss, Raynaud's phenomenon, proximal muscle weakness, sicca symptoms, painful or red eyes, recurrent oral or genital ulcers, neck pain, or limb claudication. She had no symptoms suggesting thyrotoxicosis or diabetes mellitus. She had regular menstruation since menarche. She experienced an uneventful pregnancy 1 year prior.

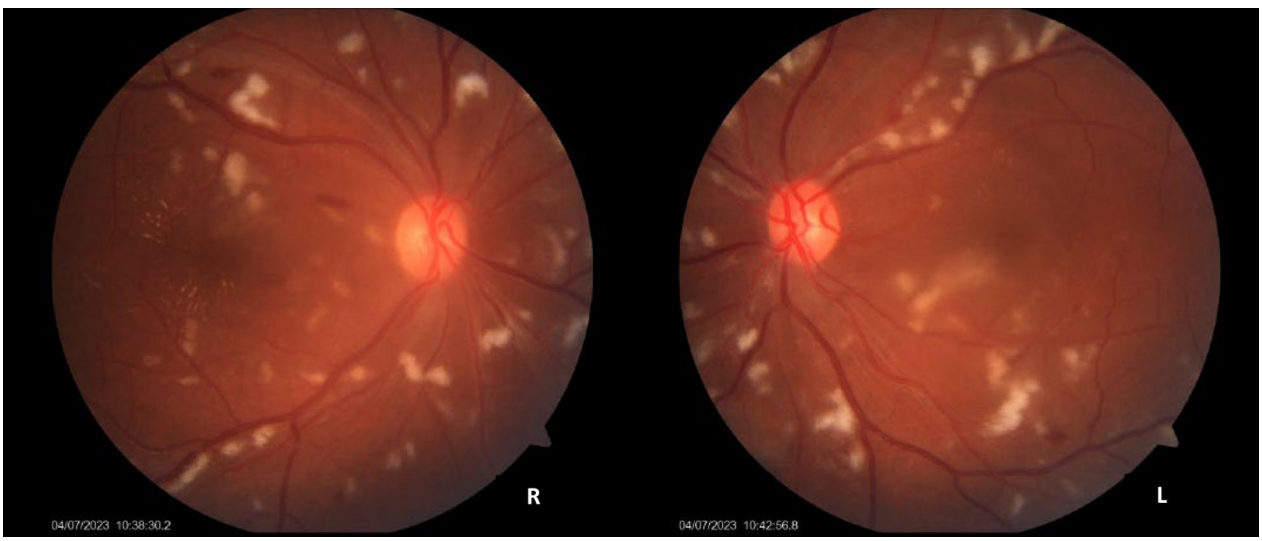


**Fig. 1** Fundal photograph at diagnosis: bilateral cotton wool spots, macular edema, arteriolar narrowing, and venous segmentation, as well as a few areas of flame-shaped hemorrhages

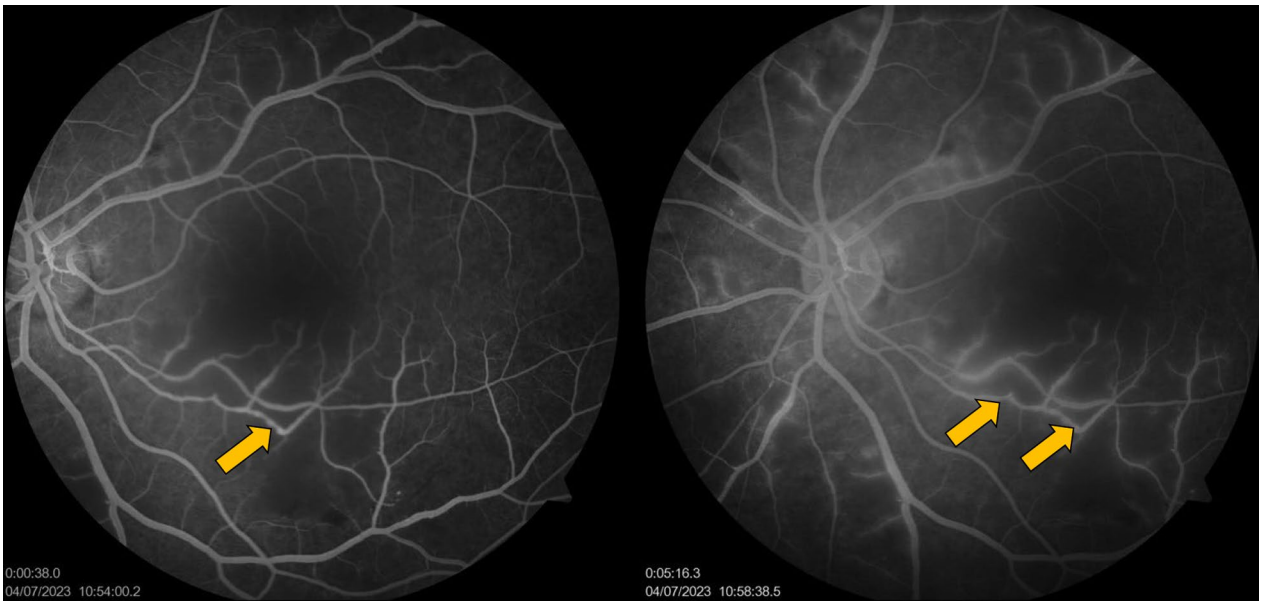


**Fig. 2** Fundus fluorescein angiogram: evidence of retinal vasculitis (yellow arrows)

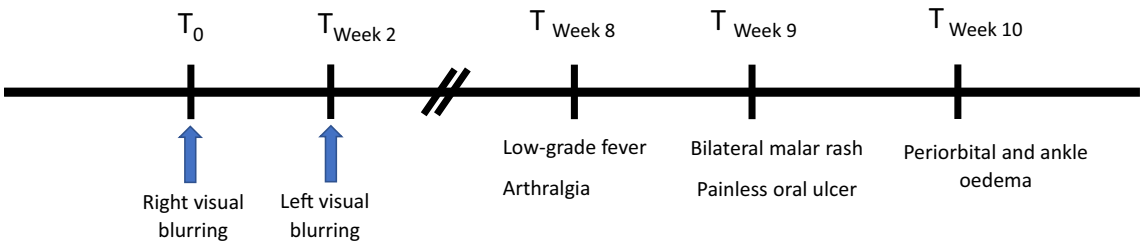


**Fig. 3** Timeline of symptom presentation

She did not have a history of recurrent miscarriages or intrauterine death. There was no family history of autoimmune diseases. She is a non-smoking teetotaler. She had good family support and no significant psychosocial stressors.

On examination, her body mass index was 19 kg/m$^2$. She was febrile but not pale. There were bilateral erythematous rashes over the malar area of the face sparing the nasolabial folds suggestive of acute cutaneous lupus (Fig. 4). Oral examination revealed an oral ulcer over the hard palate of the mouth. She did not have redness in her eyes. Mild periorbital swelling was noted along with bilateral pitting ankle edema. Examination of the neck revealed a few subcentimeter, nontender, enlarged lymph nodes. There were no clubbing or peripheral stigmata of infective endocarditis. None of the joints were actively inflamed, nor were there any joint deformities.

Her blood pressure was 112/78 mmHg, and her pulse rate was 82 beats per minute and regular. The radial, carotid, and femoral pulses were palpable in normal volume, and there were no audible bruits or radio-radial or radio-femoral delays. The rest of the peripheral pulses were palpable. There was no tenderness in the carotid artery. Cardiac examination was normal, with no audible murmurs or pericardial rubs. The lung fields were clear. Abdominal and neurological examinations were unremarkable. There were no genital ulcers.

The initial laboratory investigations are summarized in Table 1.

Rheumatological work-up (Table 2) revealed a high antinuclear antibody (ANA) and anti-double stranded DNA (anti-dsDNA) titers with low complement levels.

The urine-protein-to-creatinine ratio (UPCR) showed a nephrotic range proteinuria. The renal biopsy confirmed class III LN. Blood and urine cultures were sterile. A transthoracic echocardiogram excluded infective endocarditis. A comprehensive workup was undertaken to exclude infectious and inflammatory aetiologies of retinal vasculitis. This included evaluation for Epstein–Barr virus (EBV), cytomegalovirus (CMV), human

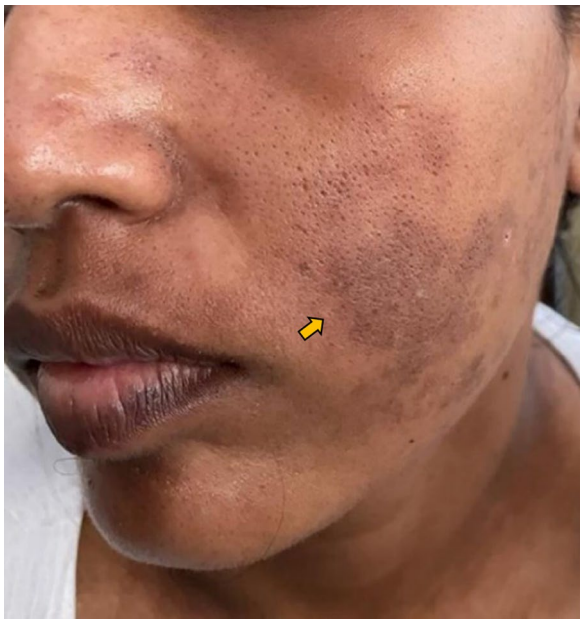

**Fig. 4** Acute cutaneous lupus: erythematous malar rash sparing the nasolabial folds (arrow)

immunodeficiency virus (HIV), hepatitis B and C viruses, toxoplasmosis, syphilis, and tuberculosis. Additionally, the rheumatologic workup excluded Behçet's disease, antineutrophil cytoplasmic antibodies (ANCA)-associated vasculitis, sarcoidosis, Sjögren's syndrome, and rheumatoid arthritis.

The patient fulfilled the 2019 European League Against Rheumatism/American College of Rheumatology (EULAR/ACR) classification criteria for SLE with a score of 37 points on the basis of the constellation of fever, leukopenia, oral ulcers, acute cutaneous lupus, arthralgia, class III LN on biopsy, low C3 and C4 levels, and anti-dsDNA antibody positivity [6]. Furthermore, the Systemic Lupus Erythematosus Disease Activity Index 2000 (SLEDAI-2 K) score of 29 points, driven by fever, visual disturbance, evidence of vasculitis, proteinuria, inflammatory rash, oral ulcers, low complement levels, and high DNA binding, categorized the disease activity as severe [7].

The patient was started on pulsed intravenous methylprednisolone (1 g per day) for 3 days followed by oral prednisolone (1 mg/kg/day) with a tapering regime. She

**Table 1** Initial laboratory investigations

| Laboratory parameter | Value | Reference range |
|---|---|---|
| Hematological | | |
| Total white cell count (cell/microL) | $3.62\times10^3$ | $4–11\times10^3$ |
| Neutrophil count (cell/microL) | $2.68\times10^3$ | $1.5–8.0\times10^3$ |
| Hemoglobin level (g/dL) | $10.9\times10^3$ | $12–16\times10^3$ |
| Platelet count (cell/microL) | $138\times10^3$ | $150–450\times10^3$ |
| Erythrocyte sedimentation rate (mm/first hour) | 88 | |
| Biochemistry | | |
| C-reactive protein (mg/dL) | 2.9 | < 6 |
| Procalcitonin (ng/mL) | 0.04 | < 0.05 |
| Serum sodium (mmol/L) | 139 | 135–148 |
| Serum potassium (mmol/L) | 4.1 | 3.5–5.1 |
| Serum creatinine (mg/dL) | 0.82 | 0.7–1.2 |
| Estimated glomerular filtration rate (eGFR) (mL/min/1.73 $m^2$) | 97 | > 90 |
| Alanine aminotransferase (U/L) | 27 | 7–56 |
| Aspartate aminotransferase (U/L) | 24 | 10–40 |
| Alkaline phosphatase (U/L) | 93 | 53–128 |
| Total protein | 6.8 | 6.6–8.3 |
| Albumin | 3.6 | 3.5–5.3 |
| Globulin | 3.2 | 2.0–3.5 |
| Serum calcium (mg/dL) | 9.4 | 8.5–10.2 |
| Serum magnesium (mg/dL) | 1.9 | 1.7–2.2 |
| Serum phosphate (mg/dL) | 3.7 | 2.5–4.5 |
| Thyroid-stimulating hormone (TSH) (mIU/L) | 2.4 | 0.55–4.78 |
| Free thyroxine (T4) (ng/dL) | 1.28 | 0.89–1.76 |
| Urinalysis | No pyuria, hematuria, or active sediment | |
| UPCR (mg/mg) | 8.86 | < 2.5 |

**Table 2** Rheumatological work-up investigations

| Investigation | Results | Reference range |
|---|---|---|
| ANA titer | 1:2560 (nuclear pattern) | < 1:80 |
| Anti-dsDNA antibodies | Positive | |
| Complement 3 (C3) (mg/dL) | 36 | 65–190 |
| Complement 4 (C4) (mg/dL) | 9 | 14–40 |
| Rheumatoid factor (U/mL) | 8 | < 20 |
| Anticyclic citrullinated antibodies | Negative | |
| Ant-Ro and anti-La antibodies | Negative | |
| Cytoplasmic antineutrophil cytoplasmic antibodies (c-ANCA) | Negative | |
| Perinuclear antineutrophil cytoplasmic antibodies (p-ANCA) | Negative | |
| Antiphospholipid panel | At diagnosis | 12 weeks later |
| Lupus anticoagulant | Negative | Negative |
| Anti-cardiolipin antibodies (IgM and IgG) | Negative | Negative |
| Anti-Beta 2 glycoprotein-1 (IgM and IgG) | Negative | Negative |

was simultaneously treated with hydroxychloroquine (HCQ) and mycophenolate mofetil (MMF) at the discretion of the rheumatology, ophthalmology, and nephrology teams. Treatment resulted in a favorable clinical response within 1 month. The patient experienced an improvement in systemic symptoms and proteinuria, with UPCR falling below 2.5 mg/kg. Additionally, BCVA improved from 6/36 to 6/12 at 2 months, accompanied by a corresponding resolution of retinal changes (Fig. 5). A total of 3 months after initiating treatment, the patient achieved disease remission, as evidenced by a SLEDAI-2 K score of two points. Following the initial management, steroid dosages were gradually tapered off. MMF and HCQ were continued for long-term disease control. The patient underwent regular monitoring for HCQ-induced retinal toxicity. At the 1-year follow-up, the patient demonstrated excellent long-term disease control, with significant improvement in vision (BCVA improving to 6/6) and no evidence of HCQ-induced retinal toxicity. Additionally, LN remained in remission.

## Discussion and conclusion

The most common ocular manifestations of SLE include keratoconjunctivitis sicca, episcleritis, scleritis, and lupus retinopathy [8]. Although EULAR/ACR classification criteria do not include ocular manifestations as a domain, it has the highest possible effect on the disease activity according to SLEDAI [7]. Lupus retinopathy affecting 3–29% of patients with SLE is well established as a negative prognostic factor for overall survival [5]. Our case

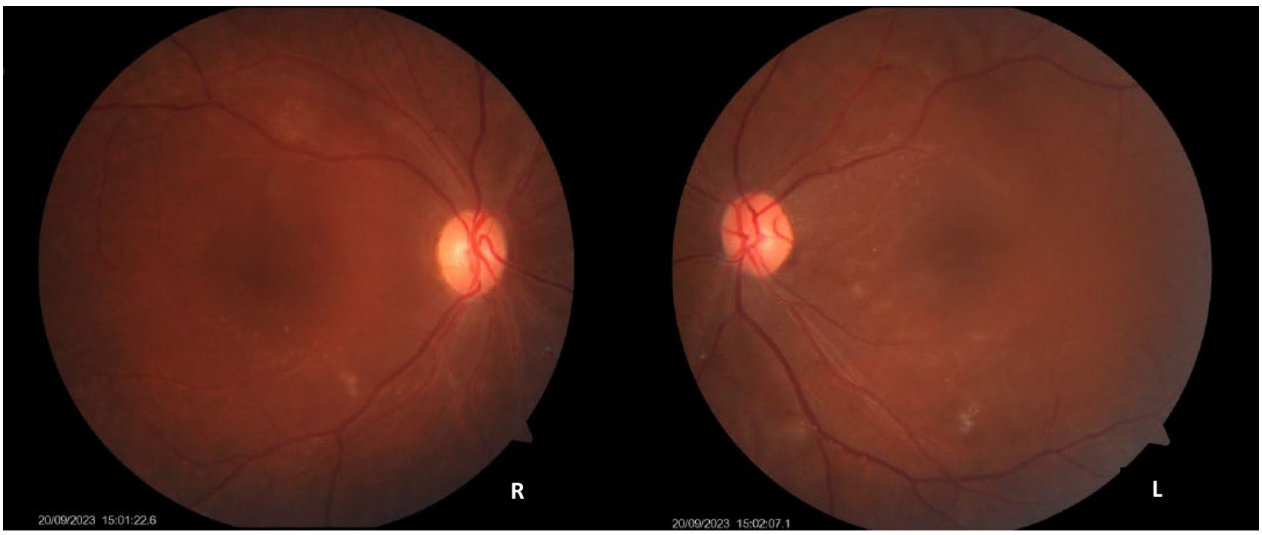


**Fig. 5** Fundal photograph after 2 months of treatment: most of the previous retinal changes improved

report describes a rare presentation of bilateral retinal vasculitis as the initial manifestation of SLE, which was then followed by the development of overt disease complicated with LN after 2 months.

The spectrum of lupus retinopathy ranges from mild to severe. Mild cases typically present with cotton-wool spots, perivascular hard exudates, retinal hemorrhages, and increased vascular tortuosity. In moderately severe presentations, additional findings may include focal or generalized narrowing of arterioles and dilated, tortuous veins. The most severe form, vaso-occlusive retinopathy or retinal vasculitis, is characterized by occluded retinal arterioles leading to retinal infarction [9]. Our patient had severe retinopathy with evidence of retinal vasculitis with occlusive arteriolitis and phlebitis demonstrated in her FFA.

SLE can induce retinopathy through two primary mechanisms. One involves antiphospholipid antibodies triggering thrombosis within retinal vessels. This can lead to vasculopathy, manifesting as central retinal artery occlusion (CRAO) or central retinal vein occlusion (CRVO). The other mechanism is classic lupus retinopathy, which is caused by vasculitis. In this process, immune complexes are deposited on the endothelial lining of blood vessels, activating the complement system and promoting phagocytosis by immune cells. This releases additional inflammatory mediators, perpetuating the vascular damage [10]. Intriguingly, our patient lacked elevated antiphospholipid antibodies, which are detected in approximately 77% of patients with SLE with lupus retinopathy or optic neuropathy [11]. This suggests a possible immunologic mechanism independent of antiphospholipid antibodies contributing to the patient's retinopathy. Supporting this notion, Bashiri *et al.* reported lower C3 and C4 complement levels, along with higher ANA and anti-dsDNA levels in patients with retinopathy, potentially reflecting an immune-mediated process [12]. The similar laboratory profile observed in our case strengthens this hypothesis.

The precise timeframe for lupus retinopathy presentation in SLE remains unclear. While data on retinopathy in newly diagnosed patients are limited, Bashiri *et al.* reported a 15.8% prevalence in a recent study, although none experienced initial ophthalmologic symptoms [12]. Despite being recognized as a complication of SLE, retinal vasculitis rarely presents as the initial disease manifestation, with limited documented cases in the literature. Alhassan *et al.* described a 14-year-old girl presenting with panuveitis and bilateral retinal vasculitis as the sole initial manifestation [13]. Similarly, Palkar *et al.* reported a 15-year-old girl with purtscher-like retinopathy as the presenting symptom [14]. Barkeh *et al.* documented a 19-year-old woman presenting with optic neuritis alongside retinal vasculitis, both serving as initial features of SLE [15]. Bandyopadhyay *et al.* described a 13-year-old boy with unilateral retinal vasculitis and a photosensitive rash as the initial presentation [16]. Aldhefeery *et al.* reported a case of bilateral retinal vasculitis with antiphospholipid syndrome as the initial presentation in a 34-year-old man [17]. Donnithrone *et al.* reported two pediatric cases of retinal vasculitis in those recently diagnosed with SLE, but not as a presenting manifestation [18]. Interestingly, none of these cases demonstrated significant renal involvement at presentation or during follow-up. Additionally, all but one case involved adolescents, with the remaining adult case being a man [13–17]. Notably, our case appears to be unique in the literature, representing the first reported instance of a premenopausal adult female patient with SLE presenting with bilateral severe retinal vasculitis that subsequently progressed to involve LN.

Studies have shown a positive correlation between retinopathy in patients with SLE and higher SLEDAI scores. Supporting this association, prior studies have reported a potential link between lupus retinopathy and LN. These studies suggest that patients with retinopathy may have more severe renal involvement, as evidenced by elevated proteinuria and serum creatinine levels [19]. Our case aligns with these findings, demonstrating the presence of significant proteinuria with evidence of LN. Studies also have shown that lupus retinopathy is associated with a higher prevalence of neuropsychiatric manifestations [20, 21] and autoimmune hemolytic anemia, which were not seen in our patient [21].

Despite experiencing progressive visual impairment, the patient lacked classic SLE manifestations during the initial 2 months, posing a diagnostic challenge, hence leading to a delay in diagnosis. A heightened index of suspicion for SLE, even in the absence of established symptoms, could have facilitated a more extensive workup and potentially expedited the initiation of treatment. Early intervention with HCQ alongside immunosuppressive therapy, as supported by current evidence, could have mitigated the development of LN [22].

The treatment of retinal vasculitis in patients with SLE is primarily related to treating the disease itself. Since our patient had a severe disease activity of SLE, she was given intravenous methylprednisolone pulses followed by oral glucocorticoids and MMF. The decision to initiate HCQ presented a therapeutic challenge due to the patient's retinal vasculitis and its associated increased risk of HCQ-induced retinal toxicity. Following a multidisciplinary discussion with the ophthalmology team, the potential benefits of HCQ for long-term disease control were deemed to outweigh the potential risks of worsening maculopathy. Therefore, HCQ was

initiated with close monitoring for retinal toxicity. Fortunately, at the 1-year follow-up, the patient did not exhibit any signs of HCQ-induced retinal toxicity.

This case underscores the critical importance of considering SLE in the differential diagnosis of retinal vasculitis, especially when classic systemic manifestations of the disease are lacking. It also highlights the potential for retinopathy to be the initial presenting manifestation of SLE, even in patients who appear otherwise healthy. Recognizing the possibility of such atypical presentations allows clinicians to maintain a heightened index of suspicion for SLE. This vigilance can be twofold in its benefit. First, it can expedite diagnosis of SLE, leading to earlier intervention and improved patient outcomes. Second, it can aid in early prognostication, prompting close monitoring for renal, neuropsychiatric, and hematologic complications.

The current SLE classification criteria established by the EULAR/ACR do not include lupus retinopathy, although the SLEDAI-2 K score acknowledges its impact on disease activity [7, 8]. While our case adds to a growing body of knowledge, highlighting the potential for retinal manifestations to precede systemic involvement, the overall evidence base remains limited. However, considering this emerging evidence, future revisions of the SLE classification criteria could warrant reevaluation of the inclusion of lupus retinopathy, particularly retinal vasculitis, as a potential diagnostic domain to increase diagnostic sensitivity.

**Abbreviations**

| | |
|---|---|
| SLE | Systemic lupus erythematosus |
| ANA | Antinuclear antibody |
| LN | Lupus nephritis |
| BCVA | Best corrected visual acuity |
| FFA | Fundal fluorescein angiogram |
| ESR | Erythrocyte sedimentation rate |
| CRP | C-reactive protein |
| Anti-dsDNA | Anti-double stranded DNA |
| C3 | Complement 3 |
| C4 | Complement 4 |
| UPCR | Urine-protein-to-creatinine ratio |
| c-ANCA | Cytoplasmic antineutrophil cytoplasmic antibody |
| p-ANCA | Perinuclear antineutrophil cytoplasmic antibody |
| HIV | Human immunodeficiency virus |
| HCQ | Hydroxychloroquine |
| MMF | Mycophenolate mofetil |
| EULAR | European League against Rheumatism |
| ACR | American College of Rheumatology |
| CRAO | Central retinal artery occlusion |
| CRVO | Central retinal vein occlusion |
| SLEDAI | Systemic lupus erythematosus disease activity index |

**Acknowledgements**
The authors would like to acknowledge the nephrology team led by Dr. A.L.M. Nazar at the National Hospital of Sri Lanka for their input in diagnosing and managing of lupus nephritis.

**Author contributions**
CPM collected information, followed up with the patient, performed the literature review, and drafted the manuscript. HK revised it. All the authors were involved in the diagnosis and management of the patient. All authors have read and approved the final manuscript.

**Funding**
Not applicable.

**Availability of data and materials**
Not applicable.

## Declarations

**Ethics approval and consent to participate**
Not applicable.

**Consent for publication**
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

**Competing interests**
The authors declare that they have no competing interests.

Received: 29 April 2024 Accepted: 22 February 2025
Published online: 21 March 2025

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## Publisher's Note