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Late-onset systemic lupus erythematosus with leukocytoclastic vasculitis, arterial hypertension, myocarditis, and lupus nephritis: a case report.

Gustavo Aroca Martínez [1,2], Valentina Pérez Jiménez [1], María De Los Ángeles Vélez Verbel [1], Andrés Cadena Bonfanti [1,2], Luis Castillo Parodi [1], Antonio Iglesias Gamarra [3], Diana Marcela Perea Rojas [2], Carlos Guido Musso [4], Rodrigo Daza Arnedo [5], Jorge Rico Fontalvo [2,6*].

1. Department of Nephrology, Clínica de la Costa, Barranquilla, Colombia
2. Department of Nephrology, Universidad Simón Bolívar, Barranquilla, Colombia
3. Department of Rheumatology, Universidad Nacional de Colombia, Bogotá, Colombia.
4. Department of Nephrology, Hospital Italiano de Buenos Aires, Buenos Aires, Argentina.
5. Department of Nephrology, IPS Caminos, Cartagena, Colombia.
6. Colombian Association of Nephrology and Hypertension, ASOCOLNEF, Bogotá, Colombia.

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*** Correspondence Author**

Email: Jorge Rico Fontalvo <jorgericof@yahoo.com>/
Dirección: Universidad Simón Bolívar , Barranquilla. Colombia. Carrera 59 No. 59-65.
Teléfono: [57] (605) 344 4333.

Abstract

Introduction: Systemic lupus erythematosus is a systemic autoimmune disease that can affect multiple organs, including vascular structures, although this involvement is not the most common. Leukocytoclastic vasculitis is a rare form of small vessel vasculitis, characterized by inflammation of the dermal venules, which clinically manifests as palpable purpura. Its association with myocarditis, hypertension, and lupus nephritis is uncommon and poses a diagnostic and therapeutic challenge.

Case Report: A 53-year-old female patient presented with purpuric lesions on the lower extremities, pretibial edema, and joint symptoms. The condition was complicated by severe arterial hypertension, myocarditis, and lupus nephritis. Skin and kidney biopsies confirmed the diagnosis of leukocytoclastic vasculitis and lupus nephritis, respectively.

Evolution: The patient was managed with intravenous methylprednisolone and subsequently with prednisolone, with significant improvement in clinical manifestations and laboratory parameters.

Conclusion: This case highlights the diversity of SLE presentations, particularly its association with leukocytoclastic vasculitis, a rare but relevant manifestation. Early management with immunosuppressants led to a favorable clinical outcome. This report emphasizes the importance of suspecting SLE in patients with atypical vasculitis and adopting a multidisciplinary approach to achieve timely diagnosis and treatment.

Keywords: Systemic Lupus Erythematosus, Leukocytoclastic Vasculitis, Myocarditis, Lupus Nephritis, Arterial Hypertension, Immunosuppressants.

Introduction

Systemic lupus erythematosus (SLE) represents the paradigm of systemic autoimmune diseases, characterized by a complex etiopathogenesis that allows involvement of virtually any parenchyma or organ [1]. Its global incidence ranges between 0.3 and 23.2 cases per 100,000 person-years, with marked predilection for the female sex and specific ethnic groups, among which Hispanic and African American populations stand out [2].

Although skin and joint manifestations are the most common, vascular involvement due to vasculitis is a critical complication, with a reported prevalence between 11% and 36%, primarily affecting small and medium-sized vessels 3, 4. Pathophysiologically, lupus vasculitis involves an inflammatory response of the vascular wall leading to tissue necrosis and potential organ damage 5. While it presents as the initial manifestation in 20% of SLE cases—90% of these being cutaneous type—its coexistence with severe multiorgan failure is less common and represents a diagnostic and therapeutic challenge 4.

Within the cutaneous spectrum, leukocytoclastic vasculitis stands out as a small vessel (dermal capillaries and venules) condition mediated by immune complex deposition. Clinically, it typically presents as palpable purpura on the lower extremities 6. However, variability in the presentation of skin lesions may mask underlying aggressive lupus activity.

In this report, we describe the case of a patient whose SLE onset manifested as an atypical dermal lesion, which rapidly progressed to severe multiorgan involvement including nephropathy, myocarditis, pleuritis, and severe systemic hypertension. The importance of early recognition of unusual cutaneous manifestations as predictors of systemic activity and the favorable response to intensive immunosuppressive management are highlighted.

Case Report

Clinical History

A 53-year-old female patient, residing in Barranquilla, Colombia, whose illness begins with the appearance of purpuric skin lesions (Figura 1) affecting the lower limbs, associated with pretibial edema, malar erythema, oral ulcers, alopecia, and arthralgias, predominantly in the proximal interphalangeal, distal interphalangeal, and metacarpophalangeal joints, accompanied by morning stiffness. The manifestations worsen one month later, with an increase in purpuric lesions and functional limitation of mobility.

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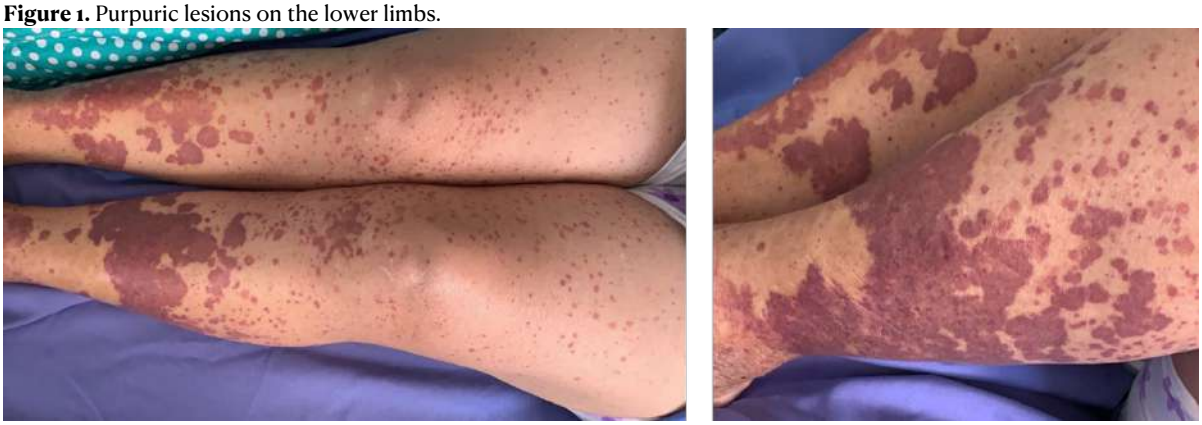
Figure 1. Purpuric lesions on the lower limbs.



Purpuric lesions on the lower limbs.

Diagnostic tests

The initial paraclinical tests were notable for marked hypocomplementemia (C3 and C4 consumed) and a positive direct Coombs test, confirming the systemic autoimmune nature. Renal involvement was critical, manifesting with massive proteinuria of 38,772 mg in 24 hours and a progressive elevation of nitrogenous waste products (Urea 109 mg/dl on day 11). The infectious profile was negative, allowing the entire clinical picture to be attributed to SLE activity. The finding of concomitant primary hypothyroidism (TSH 20.9 mIU/L) is noteworthy. During follow-up, reactive leukocytosis to immunosuppressive treatment was observed, without other significant hydroelectrolytic imbalances, except for a mild tendency toward hyperkalemia at the end of follow-up (K+ 5.3 mEq/L) (Tabla 1).

Imaging studies were performed to rule out neoplasms that could cause vasculitis. The chest X-ray showed cardiomegaly (Figura 2); the transthoracic echocardiogram reported a left ventricle of normal size with preserved contractility and a left ventricular ejection fraction (LVEF) of 65%, as well as a mild pericardial effusion. The chest tomography (Figura 3) revealed a diffuse reticular pattern, with no evidence of pneumonic consolidations or atelectasis, and no pleural effusion. The simple skull tomography showed idiopathic gangliobasal calcifications, with no signs of inflammatory activity (Figure 4). The complete abdominal ultrasound showed no abnormalities; the renal ultrasound showed kidneys of normal size and echostructure, with a preserved corticomedullary ratio; the thyroid ultrasound revealed diffuse goiter without focal lesions; and the transvaginal ultrasound showed no abnormalities. Finally, a Doppler arteriovenous ultrasound of the lower limbs was performed, which was negative for deep vein thrombosis.

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Table 1. Laboratory tests

Paraclinical	Day 1	Day 3	Day 6	Dia 11	Dia 14
Leucocitos 10^3 u/mm3	5.9	6.1	10.1	14.9	17.1
Neutrophils (%)	54.3	55	40.8	54.3	44.4
Lymphocytes (%)	39.4	39.2	50	38.5	48.8
Hemoglobin (g/dL)	12	12.1	12.8	13.9	12.6
Plaquetas 10^3 x mm3	322	316	442	592	558
ESR (mm/h)	22	NR	NR	NR	NR
PT (Seconds)	11.1	NR	10.3	10.1	9.3
INR	1	NR	0.9	0.8	0.8
PTT (Seconds)	26.6	NR	24.9	23.4	25.7
Blood glucose (mg/dL)	91	NR	NR	151	NR
Creatinine (mg/dL)	0.9	NR	1	0.8	NR
BUN (mg/dL)	19	NR	36	51	NR
Urea (mg/dL)	40.66	NR	77.04	109.14	NR
Sodium (mEq/L)	137	NR	NR	140	134
Potassium (mEq/L)	4.4	NR	4.4	4.7	5.3
Chloride (mEq/L)	106	NR	NR	109	110
Calcium (mg/dL)	NR	NR	NR	9.5	8.5
Magnesium (mg/dL)	NR	NR	NR	NR	2.4
AST (IU/L)	55	42	NR	NR	NR
ALT (IU/L)	28	26	NR	NR	NR
Total Bilirubin (mg/dL)	0.6 mg/dl	0.5 mg/dl	NR	NR	NR
Direct Bilirubin (mg/dL)	0 mg/dl	0 mg/dl	NR	NR	NR
Indirect bilirubin (mg/dL)	0.2	0.1	NR	NR	NR
Alkaline phosphatase (U/L)	64	NR	NR	NR	NR
Lactate dehydrogenase (U/L)	NR	318	NR	NR	NR
Albumin (gr/dL)	3.5	3.4	NR	NR	NR
C-reactive protein (mg/dL)	1.9	NR	NR	NR	NR
Urine proteins (mg/dL)	600	NR	NR	NR	NR
Complement C3 (mg/dL)	NR	40	<40	NR	NR
Complement C4 (mg/dL)	NR	8	<8	NR	NR
VDRL	NR	Non-reactive	NR	NR	NR
HIV	NR	NR	Negative	NR	NR
Cytomegalovirus	NR	NR	Negative	NR	NR
Epstein-Barr Virus	NR	NR	Negative	NR	NR
Polyomavirus	NR	NR	Negative	NR	NR
Hepatitis B	NR	NR	Negative	NR	NR
Hepatitis C	NR	NR	Negative	NR	NR
Free T4 (pmol/L)	NR	12	NR	NR	NR
TSH (mIU/L)	NR	20.9	NR	NR	NR
Rheumatoid factor (IU/ml)	NR	10.2	10.3	NR	NR
24-hour urine protein (mg)	NR	38.772	NR	NR	NR
Direct Coombs	NR	Positive	NR	NR	NR
Cryoglobulins	NR	NR	Negative	NR	NR
Cyclic citrullinated antibodies	NR	NR	<0.40	NR	NR

VSG: Erythrocyte Sedimentation Rate, **PT**: Prothrombin Time, **INR**: International Normalized Ratio, **PTT**: Partial Thromboplastin Time, **AST**: Aspartate Aminotransferase, **ALT**: Alanine Aminotransferase, **BUN**: Blood Urea Nitrogen, **HIV**: Human Immunodeficiency Virus, **T4**: Free Thyroxine, **TSH**: Thyroid-Stimulating Hormone, **VDRL**: Laboratory Test for Syphilis Detection, **NR**: Not Reported, **PT**: Prothrombin Time, **PTT**: Partial Thromboplastin Time.

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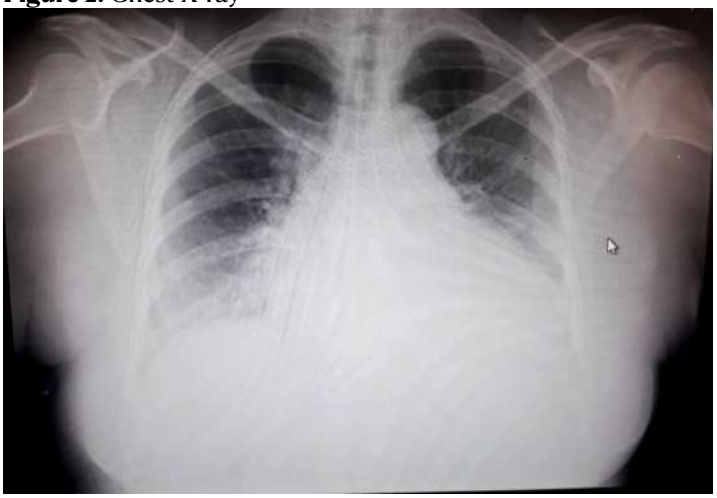
Evolution

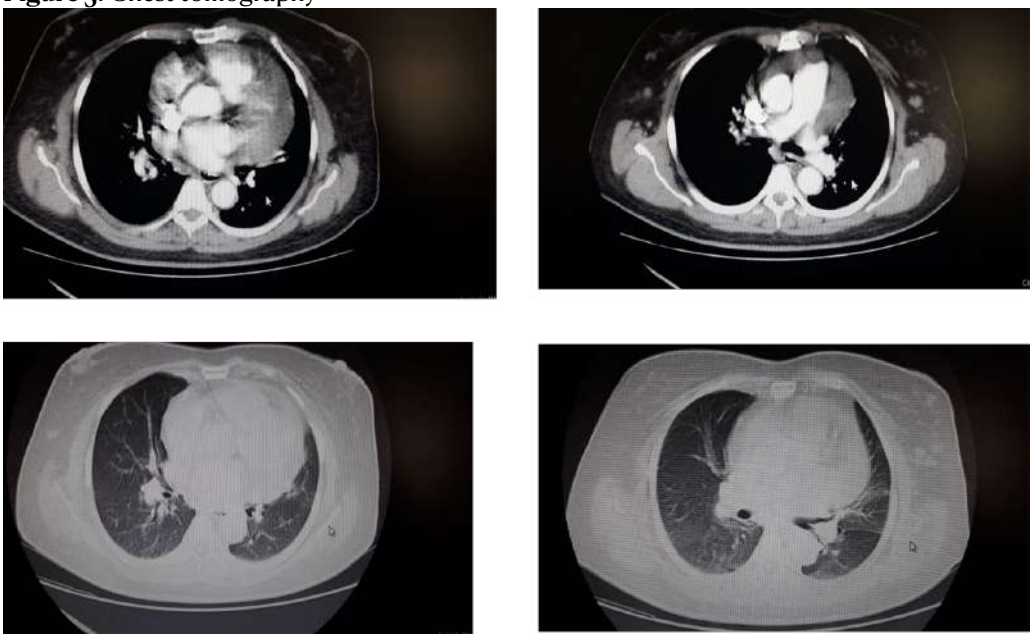
The placement of a urinary catheter was decided with partial resolution of the condition; however, uremic symptoms were added with a progressive increase in nitrogenous compounds, reaching urea values of 284 mg/dl and creatinine of 19.2 mg/dl, requiring the placement of a dialysis catheter and initiation of dialysis therapy (Tabla 1). Given the diagnosis of TBGU, antituberculous treatment was initiated and surgical resolution by urology was awaited; however, it progressed to chronic kidney disease stage V requiring chronic dialysis and surgical treatment was not possible.

Figure 2. Chest X-ray



Cardiomegaly is present on the chest X-ray.

Figure 3. Chest tomography



Cardiomegaly is present on the chest X-ray.

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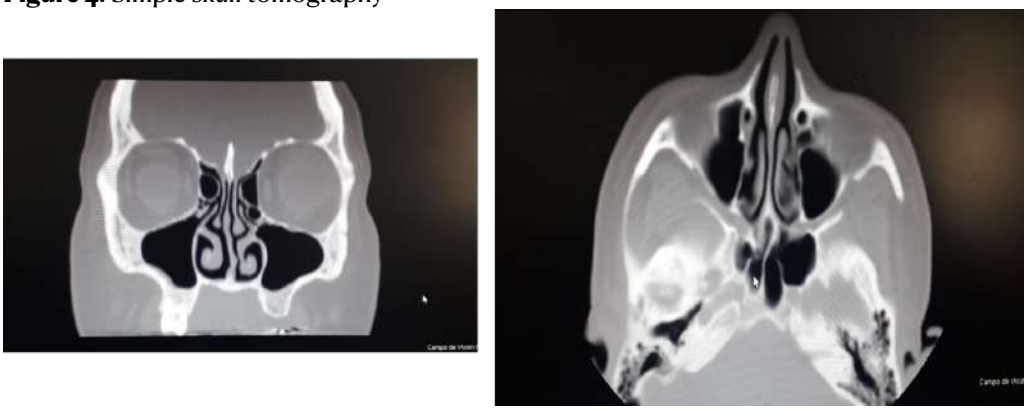
Figure 4. Simple skull tomography



Cardiomegaly is present on the chest X-ray.

An initial diagnosis of systemic vasculitis was made, and among the differential diagnostic possibilities, severe hypocomplementemic leukocytoclastic vasculitis and vasculitis secondary to systemic lupus erythematosus were considered, ruling out an infectious or immunological etiology other than lupus. Initial management included intravenous methylprednisolone at a dose of 750 mg for 3 days, followed by oral prednisolone at 1 mg/kg/day. Additionally, antihypertensive treatment was administered with oral amlodipine 10 mg daily and oral losartan 50 mg every 12 hours. Skin biopsies of the purpuric lesions and a renal biopsy were performed, the findings of which are presented in the Figura 5 and Figura 6 respectively.

Figure 5. Skin biopsy.

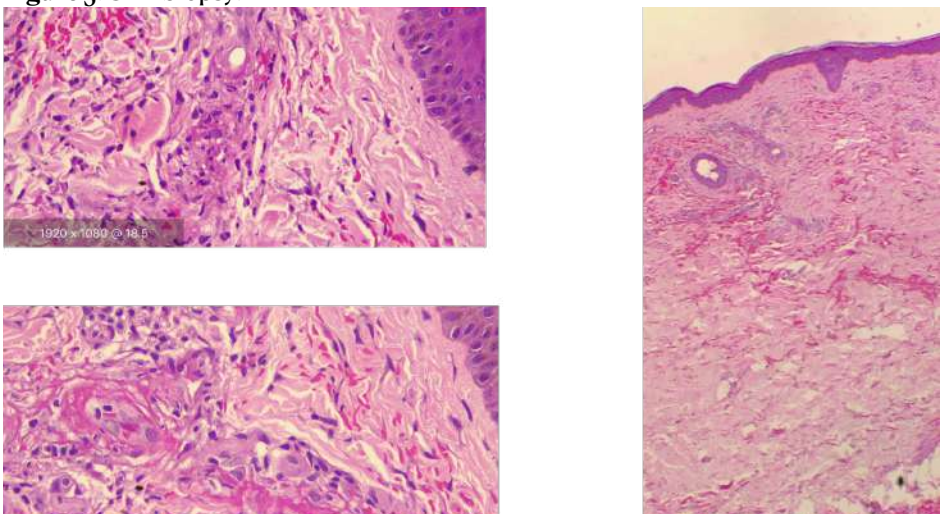
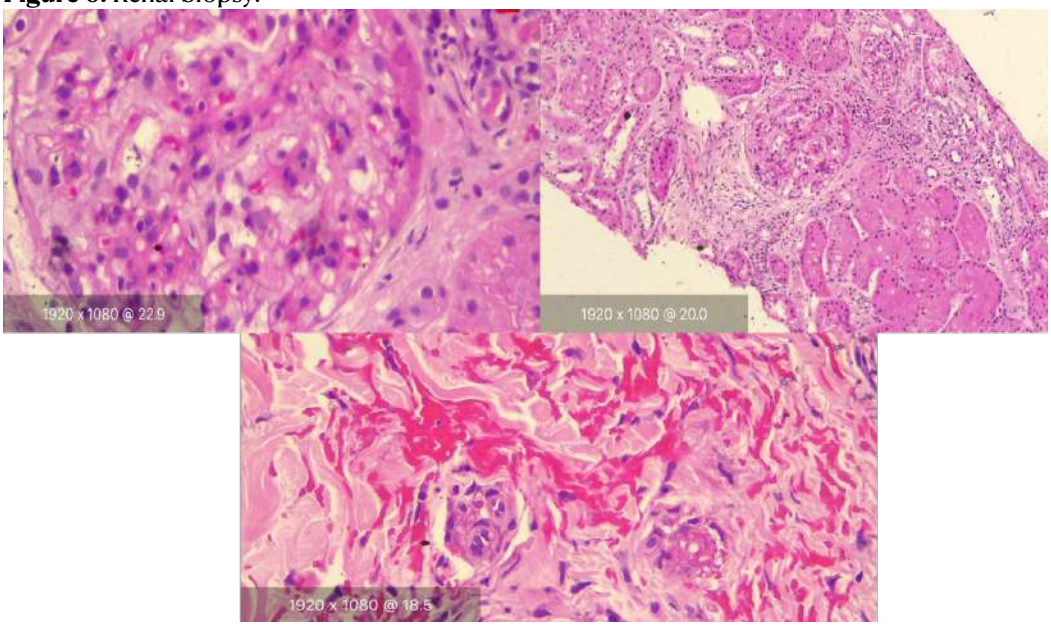


Figure 6. Renal biopsy.



Renal biopsy. A and B light microscopy. C: immunohistochemistry.

The skin biopsy showed a dermis with marked extravasation of erythrocytes due to purpura, small-caliber vessels with endothelial edema and fibrinous exudate in the vascular walls, associated with acute and chronic inflammatory phenomena, with the presence of neutrophils and leukocytoclasis. These findings suggest leukocytoclastic vasculitis (Figura 5).

In the renal biopsy, 14 glomeruli were studied, of which one showed global sclerosis. The remaining glomeruli exhibited enlargement in size, a reduction in Bowman's space, and the presence of crescents in two of them. Additionally, mesangial proliferation was identified in more than 50% of the glomeruli, with slight focal irregularities. The tubules showed mild atrophy. The interstitium was edematous, with lymphocytic inflammatory changes affecting 5% of its surface. Immunofluorescence reported positivity with a mesangial granular pattern for IgA, IgG, IgM, C3, C4, lambda, and kappa (Figura 6).

Electron microscopy confirmed the presence of immune complex deposits; at the level of the membrane, numerous subepithelial electron-dense deposits were observed, some of them intramembranous. Immune complex deposits were also evident at the mesangial level. In podocytes, loss and fusion of pedicels were observed, as well as subendothelial immune complexes. It was concluded that the patient had immune-mediated glomerulonephritis in the different compartments of the glomerulus, supporting the diagnosis of ISN/RPS class IV lupus nephritis (Figura 6). A diagnosis of class IV G (A/C) lupus nephritis was established, with an activity index of 8/24 and a chronicity index of 2/12. Additionally, extensive podocytopathy secondary to subepithelial deposits was identified, with reactive changes in podocytes, including villous transformation, hypertrophy, and detachment.

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Considering the clinical, imaging, and biopsy findings, the patient received immunosuppressive treatment with cyclophosphamide, starting with an induction dose of 500 mg every 15 days for 6 doses, along with corticosteroids. An adequate response was observed, with disappearance of the skin lesions and reduction of proteinuria, allowing partial remission 2 months after the start of treatment.

Discussion

Systemic lupus erythematosus (SLE) is a complex autoimmune disease, characterized by variable clinical manifestations, associated with multiple autoantibodies and the formation and deposition of immune complexes. This results in a clinical presentation that is difficult to define. Taking these considerations into account, the diagnosis of SLE in this case was made using the criteria proposed by the European League Against Rheumatism (EULAR) and the American College of Rheumatology (ACR) [7].

Skin lesions in patients with SLE appear in more than 70% of cases during the course of the disease, with the most frequent being malar rash (40%), alopecia (24%), and oral ulcers (19%) 8. Additionally, up to 36% of cases present with associated vasculitis, of which 29% manifest with skin lesions 9, which have typically been associated with active disease and a poor prognosis 10.

Histopathologically, lupus-associated vasculitis is primarily characterized by affecting small vessels, although it can also involve medium-sized vessels. Morphologically, vasculitic lesions in small vessels can present in lupus as well as in other autoimmune entities, with palpable purpuric areas and findings of leukocytoclastic vasculitis in the pathology [11].

Cutaneous leukocytoclastic vasculitis is a non-specific manifestation of SLE, which can occur in 6.3% of cases. Its clinical presentation forms vary and include palpable purpura, petechiae, urticaria, splinter hemorrhages, necrosis, ulceration, livedo reticularis, microinfarcts, fingertip nodules, and Janeway spots 9, 12, 13. In this case, it typically presented as palpable purpuric lesions localized to the lower extremities.

Since leukocytoclastic vasculitis can arise in the context of infections by Mycobacterium, Staphylococcus aureus, Chlamydia, Neisseria, and HIV, as well as malignant diseases and the use of certain medications, these etiologies were ruled out, as was done in this case. Similarly, immunological tests were performed to exclude other secondary causes, leading to the diagnosis of SLE 14, 15.

On the other hand, in this case, an active urinary sediment was identified, suggestive of lupus nephritis, which was confirmed by renal biopsy showing class IV lupus nephritis. This is consistent with the natural progression of the disease, in which up to 50-70% of patients with SLE may develop lupus nephritis, with a high incidence of proliferative nephritis that can sometimes be associated with vasculitic phenomena [3, 17, 18].

Regarding management, it should be individualized based on the severity of the cutaneous episode. However, there are no specific treatment regimens for leukocytoclastic vasculitis. Currently, the use of systemic steroids in combination with immunosuppressive and steroid-

sparing drugs is proposed. In cases of visceral involvement, the use of cyclophosphamide and azathioprine has been suggested [19], [20]. In this case, it was managed with cyclophosphamide, using the EUROLUPUS regimen for the treatment of class IV lupus nephritis and leukocytoclastic vasculitis, achieving a satisfactory response [21].

Conclusions

This case describes a patient who presented with late-onset lupus. The clinical presentation suggests an overproduction of circulating immune complexes that severely affected the microvasculature of the skin, kidneys, and heart, with nephritis and carditis accompanied by pericardial and pleural effusion. SLE is a disease with variable presentation, and cutaneous manifestations, such as the atypical lesions presented in this case, often appear during the course of the disease. Among these manifestations, vasculitis of medium and small vessels should be suspected, as it carries an increased risk of morbidity and mortality. Given its association with lupus activity, it is necessary to rule out involvement of other organs. In this case, the timely diagnosis of class IV lupus nephritis allowed for appropriate treatment and an initially satisfactory response.

Abbreviations

ACR: American College of Rheumatology.
SLE: Systemic lupus erythematosus.

Supplementary Information

Supplementary materials have not been declared.

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Not declared.

Author Contributions

Gustavo Aroca Martínez: Conceptualization, data curation, research, visualization, original drafting.
Valentina Pérez Jiménez: Conceptualization, data curation, research, visualization and writing of the original draft.
María De Los Ángeles Vélez Verbel: Conceptualization, formal analysis, methodology, project management, resources, software, supervision, validation, drafting – review and editing.
Andrés Cadena Bonfanti: Conceptualization, data curation, research, visualization and writing of the original draft.
Luis Castillo Parodi: Conceptualization, data curation, research, visualization and writing of the original draft.
Antonio Iglesias Gamarra: Conceptualization, data curation, research, visualization and writing of the original draft.
Diana Marcela Perea Rojas: Conceptualization, data curation, research, visualization and writing of the original draft.

Carlos Guido Musso: Conceptualización, curación de datos, investigación, visualización y redacción del borrador original.
Rodrigo Daza Arnedo: Conceptualización, curación de datos, investigación, visualización y redacción del borrador original.
Jorge Rico Fontalvo: Conceptualización, análisis formal, metodología, administración del proyecto, recursos, software, supervisión, validación, redacción – revisión y edición.

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Consent for publication
The authors have the patient's authorization for the publication of the images and the clinical case.

Conflicts of interest
The authors declare that they have no conflicts of interest.

Use of generative AI
The authors declare that they have not used generative AI in this document.

Author information
Gustavo Aroca-Martínez: Medical degree from Universidad del Norte. Specialist title in nephrology from Universidad Militar Nueva Granada. Internal medicine physician and nephrologist. Treating physician at Clínica de la Costa, Universidad Simón Bolívar, Barranquilla, Colombia
Email: garoca1@clinicadelacosta.co
https://orcid.org/0000-0002-9222-3257

Valentina Pérez Jiménez: Medical degree from Universidad Simón Bolívar.
Place of work. Clínica de la costa, Universidad Simón Bolívar, Barranquilla, Colombia
Email: Dra.valentina.perez.jimenez@gmail.com
https://orcid.org/0009-0002-5263-0795

María de los Ángeles Vélez: Medical degree from Universidad Libre. Specialist degree from Universidad Simón Bolívar, internal medicine physician.
Place of work. Universidad Simón Bolívar
Email: mvelez99@hotmail.com

https://orcid.org/0000-0002-8853-1871

Andrés Cadena Bonfanti: Medical degree from Universidad CES.
Specialist degree in internal medicine and nephrology, University of Texas.
Place of work. Clínica de la Costa, Universidad Simón Bolívar, Barranquilla, Colombia.
Email: acadena@clinicadelacosta.co
https://orcid.org/0000-0003-3903-1915

Luis Castillo Parodi: Medical degree in Nephrology from Universidad Militar. Place of work. Universidad Simón Bolívar
https://orcid.org/0009-0001-8138-7889

Antonio Iglesias Gamarra: Medical degree from the National University.
Specialist title in Internal Medicine, Rheumatologist: Harvard Medical School
Place of work. National University
Email: iglesias.antonio1@hotmail.com
https://orcid.org/0000-0002-6734-5471

Diana M Perea Rojas: Medical degree from Simón Bolívar University.
Place of work. Simón Bolívar University
Email: dianaperea1822@gmail.com
https://orcid.org/0000-0001-8821-6428

Carlos Guido Musso: Medical degree from the University of Buenos Aires.
Specialist in internal medicine, nephrologist
Place of work: Hospital Italiano de Buenos Aires
Email: drcarlosmusso@gmail.com
https://orcid.org/0000-0001-8666-1130

Rodrigo Daza-Arnedo: Physician from Universidad Libre de Barranquilla. Nephrologist from Universidad Militar. Works at IPS Caminos, Cartagena, Colombia.
Email: rodrigoandres_2@hotmail.com
https://orcid.org/0000-0002-6295-4972

Jorge Rico-Fontalvo: Medical degree from Universidad Militar Nueva Granada, Bogotá. Colombia. Nephrologist from Universidad de Antioquia.
Email: jorgericof@yahoo.com
Research Professor, Simón Bolívar University, Barranquilla, Colombia.
Orcid: https://orcid.org/0000-0002-2852-1241.

References

1. Oku K, Atsumi T. Systemic lupus erythematosus: nothing stale her infinite variety. Mod Rheumatol. 2018 Sep;28(5):758-765. doi: 10.1080/14397595.2018.1494239. PMID: 29947275.

2. Gergianaki I, Bortoluzzi A, Bertsias G. Update on the epidemiology, risk factors, and disease outcomes of systemic lupus erythematosus. Best Pract Res Clin Rheumatol. 2018 Apr;32(2):188-205. doi: 10.1016/j.berh.2018.09.004. Epub 2018 Sep 27. PMID: 30527426.

3. Fanouriakis A, Tziolos N, Bertsias G, Boumpas DT. Update on the diagnosis and management of systemic lupus erythematosus. Ann Rheum Dis. 2021 Jan;80(1):14-25. doi: 10.1136/annrheumdis-2020-218272. Epub 2020 Oct 13. PMID: 33051219.

4. Fukuda S, Nishikawa K, Ohta R. Progressive Muscle Weakness and Dysphagia in Late-Onset Systemic Lupus Erythematosus: A Case Report. Cureus. 2025 Feb 19;17(2):e79327. doi: 10.7759/cureus.79327. PMID: 40125233; PMCID: PMC11928338.

5. Jennette JC, Falk RJ, Andrassy K, Bacon PA, Churg J, Gross WL, Hagen EC, Hoffman GS, Hunder GG, Kallenberg CG, et al. Nomenclature of systemic vasculitides. Proposal of an international consensus conference. Arthritis Rheum. 1994 Feb;37(2):187-92. doi: 10.1002/art.1780370206. PMID: 8129773.

6. Younger DS, Carlson A. Dermatologic Aspects of Systemic Vasculitis. Neurol Clin. 2019 May;37(2):465-473. doi: 10.1016/j.ncl.2019.01.017. Epub 2019 Mar 16. PMID: 30952419.

7. Aringer M, Costenbader K, Daikh D, Brinks R, Mosca M, Ramsey-Goldman R, et al. 2019 European League Against Rheumatism/American College of Rheumatology Classification Criteria for Systemic Lupus Erythematosus. Arthritis Rheumatol. 2019 Sep;71(9):1400-1412. doi: 10.1002/art.40930. Epub 2019 Aug 6. PMID: 31385462; PMCID: PMC6827566.

8. Hasbún Z MT, Rollan MP, Chaparro R X, Fischer S C, Castrillón V A, González B S, Reculé F. Bullous systemic lupus erythematosus: An uncommon manifestation in pediatric population. Andes Pediatr. 2021 Jun;92(3):428-433. English, Spanish. doi: 10.32641/andespediatr.v92i3.2729. PMID: 34479250.

9. Miyagawa F, Ogawa K, Hashimoto T, Asada H. A Case of Systemic Lupus Erythematosus with Cutaneous Leukocytoclastic Vasculitis Mimicking Bullous SLE. Case Rep Dermatol. 2021 Sep 20;13(3):464-469. doi: 10.1159/000519022. PMID: 34720919; PMCID: PMC8525288.

10. Khil J, Nguyen TM, Troxell ML, Zheng S. Systemic Lupus Erythematosus and ANCA-Associated Vasculitis Overlap Syndrome: A Case Report. Kidney Med. 2022 Sep 16;4(11):100544. doi: 10.1016/j.xkme.2022.100544. PMID: 36353648; PMCID: PMC9637818.

11. Calle-Botero E, Abril A. Lupus Vasculitis. Curr Rheumatol Rep. 2020 Aug 26;22(10):71. doi: 10.1007/s11926-020-00937-0. PMID: 32845454.

12. Biglione B, Zhong C, Iriarte C, Cucka B, Hoang MP, Kroshinsky D. Cutaneous Leukocytoclastic Vasculitis as the Presenting Sign of Systemic Lupus Erythematosus. Am J Med. 2023 Apr;136(4):368-371. doi: 10.1016/j.amjmed.2022.11.008. Epub 2022 Dec 8. PMID: 36502957.

13. Hsieh FN, Yu-Yun Lee J. Leukocytoclastic Vasculitis Concurrent With Bullous Systemic Lupus Erythematosus Manifesting Striking Wood-Grain and Wi-Fi Sign-like Purpuric Lesions. J Clin Rheumatol. 2019 Oct;25(7):e104-e105. doi: 10.1097/RHU.0000000000000717. PMID: 29652701.

14. Fraticelli P, Benfaremo D, Gabrielli A. Diagnosis and management of leukocytoclastic vasculitis. Intern Emerg Med. 2021 Jun;16(4):831-841. doi: 10.1007/s11739-021-02688-x. Epub 2021 Mar 13. PMID: 33713282; PMCID: PMC8195763.

15. Bečvář R. Secondary vasculitis - omitted manifestation of many diseases. Cesk Patol. 2020 Spring;56(2):83-88. English. PMID: 32493024.

16. Rojas-Rivera JE, García-Carro C, Ávila AI, Espino M, Espinosa M, Fernández-Juárez G, Fulladosa X, Goicoechea M, Macía M, Morales E, Porras LFQ, Praga M. Consensus document of the Spanish Group for the Study of the Glomerular Diseases (GLOSEN) for the diagnosis and treatment of lupus nephritis. Nefrologia (Engl Ed). 2023 Jan-Feb;43(1):6-47. doi: 10.1016/j.nefroe.2023.05.006. Epub 2023 May 19. PMID: 37211521.

17. Horisberger A, Griffith A, Keegan J, Arazi A, Pulford J, Murzin E, et al.; Accelerating Medicines Partnership Rheumatoid Arthritis and Systemic Lupus Erythematosus (AMP RA/SLE) Network; Brenner MB, Anolik JH, Raychaudhuri S, Hacohen N, James JA, Davidson A, Petri MA, Buyon JP, Diamond B, Zhang F, Lederer JA, Rao DA. Blood immunophenotyping identifies distinct kidney histopathology and outcomes in patients with lupus nephritis. J Clin Invest. 2025 Jun 19;135(16):e181034. doi: 10.1172/JCI181034. PMID: 40536813; PMCID: PMC12352894.

18. Santacruz-Sandoval E, López-Bonilla J, Guevara-Calderón LA, Nieto-Aristizábal I, Ruiz-Ordoñez I, Cañas CA, Santos VA, Tobón GJ, Aguirre-Valencia D. Clinical Characteristics and Outcomes of Patients With ANCA-Associated Vasculitides in a Colombian Hospital. J Clin Rheumatol. 2022 Mar 1;28(2):e491-e497. doi: 10.1097/RHU.0000000000001775. PMID: 35192595.

19. Ansari A, Larson PH, Bates HD. Vascular manifestations of systemic lupus erythematosus. Angiology. 1986 Jun;37(6):423-32. doi: 10.1177/000331978603700601. PMID: 3729068.

20. Sebastian J, Mathew M, Sharsty V, Ramesh M. Leukocytoclastic Vasculitis Following COVID-19 Vaccination: A Case Report. Hosp Pharm. 2022 Aug;57(4):564-567. doi: 10.1177/00185787211067379. Epub 2021 Dec 29. PMID: 35898243; PMCID: PMC9310316.

21. Dong Y, Shi J, Wang S, Liu Y, Yu S, Zhao L. The efficacy of immunosuppressive drugs induction therapy for lupus nephritis: a systematic review and network meta-analysis. Ren Fail. 2023;45(2):2290365. doi: 10.1080/0886022X.2023.2290365. Epub 2023 Dec 12. PMID: 38087473; PMCID: PMC11001324.

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