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ANCA-Positive late-onset lupus and diffuse alveolar hemorrhage: Overlap or new clinical entity? A case report.

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Abstract

Introduction: Systemic lupus erythematosus (SLE) usually occurs in young people and manifests acutely, whereas in late cases (over 50 years of age), it is rare and insidious. The co-presence with neutrophil cytoplasmic antibodies (ANCA), characteristic of most vasculitis, is rare and leads to worse clinical outcomes.

Clinical case: A 65-year-old man diagnosed with SLE for 4 years, with mild skin and joint symptoms. In the last 3 months, he presented with asthenia, adynamia, cough with hemoptysis, edema of the lower limbs, and proteinuria in the nephrotic range. Chest CT showed alveolar occupation with a ground glass pattern; Bronchoscopy showed diffuse alveolar hemorrhage. Renal biopsy reported class IV lupus nephritis, diffuse extracapillary proliferation, and grade I interstitial fibrosis. The diagnosis of lung-renal syndrome is considered.

Conclusion: the concomitance between these autoimmune pathologies is unusual; there is a late-onset overlap syndrome between lupus nephritis and MPO-ANCA pauciimmune glomerulonephritis. In turn, as it presents more aggressive evolutions, it requires greater renal replacement therapy, plasmapheresis, and tumor necrosis factor inhibitors.

Keywords:

Lung-Kidney Syndrome, Systemic Lupus Erythematosus, Diffuse Alveolar Hemorrhage, Rapidly Progressive Glomerulonephritis, Anti-Neutrophil Cytoplasmic Antibodies, Case Report.

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Systemic lupus erythematosus (SLE) is a complex autoimmune disease characterized by a loss of immunological tolerance that leads to abnormal antibody production, complement activation, and the formation of immune complexes. These complexes are deposited in multiple tissues and organs, triggering systemic inflammation and progressive structural damage. Although its incidence is higher in women of childbearing age, approximately 2% to 12% of cases are diagnosed after age 50, constituting what is known as late-onset SLE or SLE in the elderly. This clinical variant is associated with lower inflammatory activity, a higher comorbidity burden, and atypical clinical presentations that hinder its timely recognition [1, 2].

Late-onset SLE, also called elderly-onset SLE (occurring between 60 and 70 years of age), poses a diagnostic challenge due to its subtler clinical signs and overlap with common age-related chronic diseases. Unlike classic SLE, older patients are more likely to present with constitutional symptoms, serositis, and lung involvement, while mucocutaneous and joint symptoms are usually less prominent. Serologically, these patients often have positive anti-Ro antibodies in up to 90%, anti-La antibodies in 60%, and anti-dsDNA antibodies in only 30%, which makes applying standard diagnostic criteria more difficult in this age group [3]. In this population, maintaining a high level of clinical suspicion is essential, as subclinical signs can delay diagnosis and raise the risk of permanent organ damage.

One of the systems most affected in SLE is the renal system, whose involvement clinically manifests as lupus nephritis (LN). This condition results from immune complex deposits in the glomerulus and is classified histologically, according to the ISN/RPS 2003, into six classes ranging from minimal mesangial changes to advanced glomerular sclerosis. However, in patients with SLE and renal involvement, biopsies may show atypical or non-classical nephritic patterns of lupus nephritis. Specifically, when histology reveals glomerular lesions with fibrinoid necrosis, extracapillary proliferation, crescent formation, and a lack of immune deposits, the findings suggest pauci-immune glomerulonephritis, which is more consistent with antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis than with classic LN [4, 5].

ANCA vasculitis is a heterogeneous group of necrotizing systemic vasculitides of small vessels, including microscopic polyangiitis (MPA), granulomatosis with polyangiitis (GPA), and eosinophilic granulomatosis with polyangiitis (EGPA), all characterized by pauci-immune vascular damage and positivity for ANCA antibodies, either MPO- or PR3-specific. Although these entities are considered distinct from SLE, case reports and series describe clinical, immunological, and histological overlap between the two diseases. This combination has been termed SLE/ANCA overlap syndrome and, although infrequent, poses significant clinical challenges in diagnosis, classification, prognosis, and treatment [6, 7].

Pulmonary-renal syndrome (PRS), characterized by the simultaneous occurrence of rapidly progressive glomerulonephritis (RPGN) and diffuse alveolar hemorrhage (DAH), may be the initial manifestation of this overlap syndrome. DAH in patients with SLE is often linked to severe systemic activity (SLEDAI score >12), active renal involvement, and decreased complement levels. Clinically, it can resemble severe respiratory infections, so it must be distinguished from other causes of bilateral pulmonary infiltrates, such as pneumonia, tuberculosis, pulmonary embolism, or edema due to heart failure. [8, 9]. Diagnosis is supported by radiological imaging (chest CT scan), bronchoscopy with bronchoalveolar lavage, and, in many cases, renal biopsy.

Recognizing SLE/AVA overlap syndrome is especially important in late-onset SLE, as these patients may have coexisting factors, including immunosenescence, increased infection risk, and other chronic diseases. Additionally, it has been shown that when positive MPO-type ANCA and a pauci-immune histological pattern are present, even in the absence of immune deposits, vasculitis should be considered the primary pathology, even in patients previously diagnosed with SLE [5, 10]. This distinction guides a more aggressive treatment approach, which may include pulse steroids, cyclophosphamide, rituximab, and plasmapheresis, depending on the severity of pulmonary and renal involvement. Evidence indicates that early, personalized treatment can result in renal and overall outcomes similar to those of patients with isolated vasculitis [6, 11].

This article discusses the case of a 65-year-old male patient with a previous diagnosis of mild SLE who developed progressive renal glomerulonephritis (PRGN) with diffuse alveolar hemorrhage (DAH). Immunological studies showed positive results for antinuclear antibodies (ANA), anti-dsDNA antibodies, and anti-ANCA-MPO antibodies. Renal biopsy revealed a pauci-immune pattern with crescents and fibrinoid necrosis, without immune deposits, consistent with ANCA-associated vasculitis rather than lupus nephritis. This case highlights the importance of recognizing SLE/anti-AV overlap syndrome in older adults. It emphasizes the need for specific diagnostic and treatment protocols for this subgroup, due to its high morbidity and mortality, and the challenges in early detection.

Case report

Medical record

This is a 65-year-old man with a 4-year history of systemic lupus erythematosus (SLE), presenting with mild joint and skin manifestations. Renal function was preserved, and the urinary sediment was normal, in contrast to baseline laboratory tests (Table 1), which revealed primary hypertension, hyperlipidemia, and a long history of smoking. However, in the last 3 months, the patient developed asthenia, adynamia, cough with hemoptysis, lower extremity edema, and foamy urine, prompting him to visit the emergency department of the Federico Lleras Hospital, Francia branch, where admission laboratory tests were performed (Table 2). The timeline of this case is presented in Figure 1.

Table 1. Baseline studies.

Study	Result	Reference
White blood cells (cells / mm^3)	5,600	3,600 - 11,000
Serum creatinine (mg/ dL)	0.8	0.6 - 1.2
Serum albumin (g/ dL)	4.2	3.5 - 5
24-hour proteinuria (mg)	180 mg/ dL	<150 mg/24 hours
Urinalysis	No proteinuria or hematuria	
ANAs	1:640	1:80
Anti-DNA	Negative	1/10
C3 (mg/ dL)	92	88 - 201
C4 (mg/ dL)	25	15 - 45

Table 2. Paraclinical results at admission.

Study	Result	Reference
White blood cells (Cells x10 ^{3} /mm ^{3})	7.2	3.6 - 11.0
Neutrophils (Cells x10 ^{3} /mm ^{3})	4.8	1.8-7.5
Hemoglobin (g/dl)	10.1	14-18
Platelets (Cel x10 ^{3} /mm ^{3})	276	150 – 350
Serum creatinine (mg/ dL)	1.9	0.6-1.2
BUN (mg/ dL)	22	8-23
ANA	1:5120	< 1:80
ANCA	Positive	< 1 : 20
Anti-DNA	Positive	< 1/10
C3 (mg/ dL)	41	88 - 2011
C4 (mg/ dL)	8	15 – 45
Serum albumin (g/ dL)	23	3.5 – 5
24 h proteinuria (g)	5.6	0.15
Urinalysis	Active Urinary Sediment	
PT PTT	10.8 30.9	10 - 13 seconds 25-40 seconds
Lupus anticoagulant	Negative	
Anticardiolipin IgG/IgM	Normal	< 40 units
PCR (mg/L)	14	< 10

Physical examination

On physical examination, he presented with a blood pressure of 150/90 mmHg, a heart rate of 89 bpm, a respiratory rate of 30 rpm, and an oxygen saturation of 76%, with signs of metabolic and respiratory acidemia. Due to severe hypoxia, he was transferred to the intensive care unit, where endotracheal intubation was performed.

Diagnostic workshop

The chest computed tomography scan revealed a ground-glass opacity pattern (Figure 2), prompting a bronchoalveolar lavage, which showed diffuse alveolar hemorrhage. Active urinary sediment was also present, interpreted as rapidly progressing glomerulonephritis, leading to a diagnosis of pulmonary-renal syndrome. The biopsy revealed class IV lupus nephritis with diffuse extracapillary proliferation, grade I interstitial fibrosis, and severe atherosclerosis (Figure 3).

Treatment

Immunosuppressive treatment was started with methylprednisolone at 1 g/day for 3 days and cyclophosphamide at 1 g. Due to the patient's poor clinical progress, persistent hypoxemia, and oliguria with fluid overload, plasmapheresis using fresh frozen plasma exchange and continuous venovenous hemodiafiltration were initiated. Rituximab was considered but was unavailable, so management continued with plasmapheresis, steroids, and alkylating agents. After 12 days in the ICU, the patient did not require ventilatory support or vasopressors and continued dialysis therapy until death.

Figure 1. Case timeline

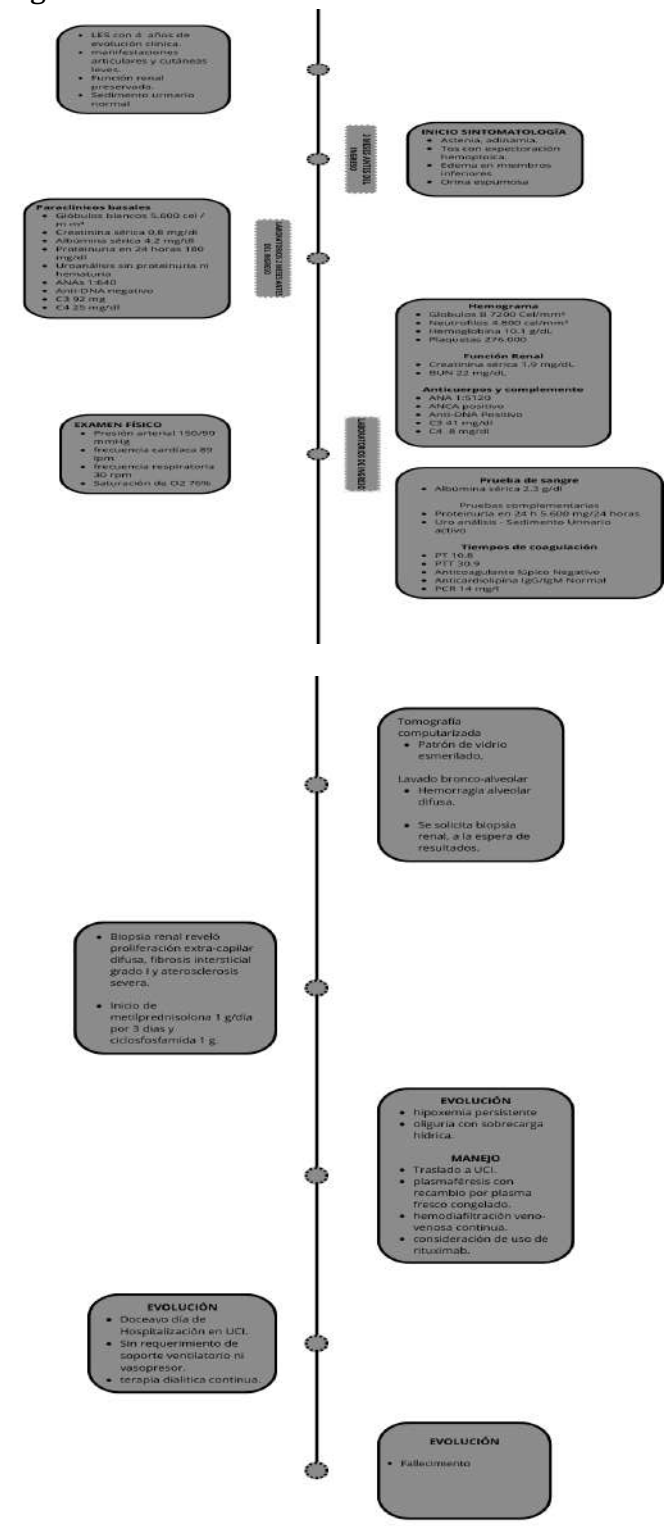
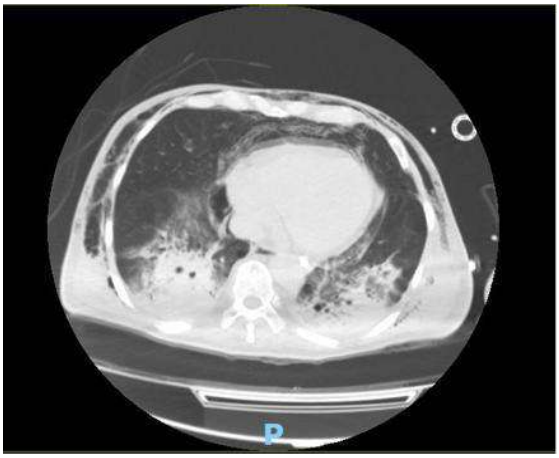
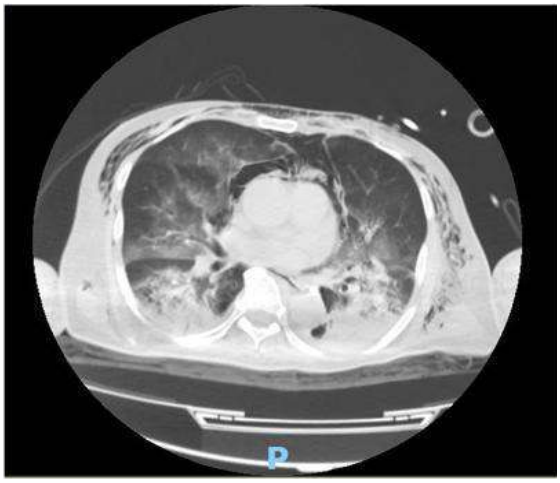


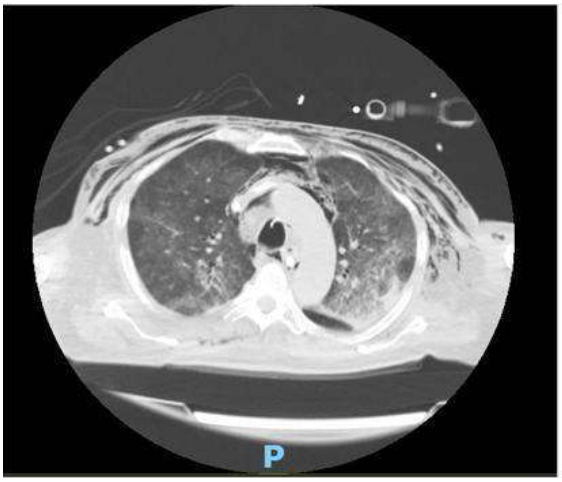
Figure 2. Chest CT scan.



A . ventricular cut.

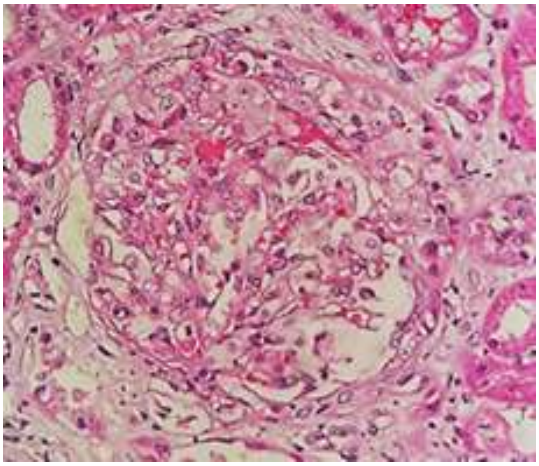


B. atrial cut.

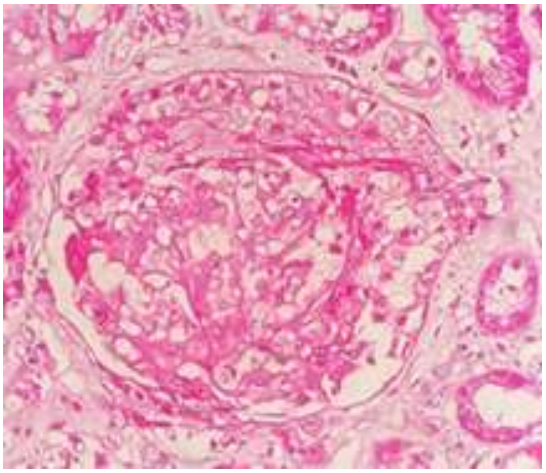


C. Cut in the aortic arch.

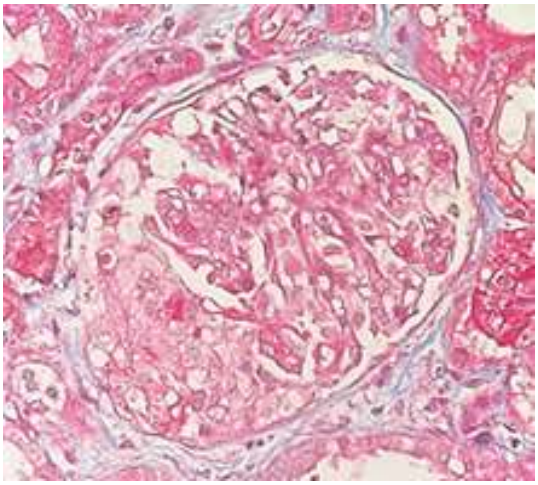
Figure 3. Glomeruli with extracapillary proliferation.



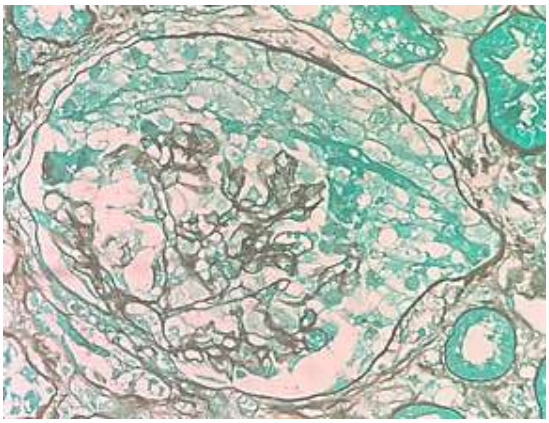
A. HE staining of cell crescent.



B. PAS staining of fibrocellular crescent.



C. Trichrome with fibrinoid necrosis.



D. Jones silver methenamine stain with capillary loop rupture. Immunofluorescence was negative.

Discussion

The pathophysiology of SLE involves multiple genetic, immunological, and environmental factors that contribute to its development, accounting for the clinical and laboratory variability observed among patients. In the lungs, between 20% and 90% of SLE patients experience respiratory symptoms at some point during their illness, with dyspnea being the most common. These symptoms may include cough, fever, hemoptysis, and, less often, pleuritic chest pain. It is estimated that up to 60% of patients may have respiratory symptoms as an initial sign, which is linked to increased mortality. Imaging often shows unilateral or bilateral alveolar infiltrates, typically in the lung bases, along with pleural effusion. Blood gas analysis often shows hypoxemia, whereas the complete blood count may be nonspecific. It is important to consider infectious processes in the differential diagnosis [1, 2].

Paradoxically, in this case, high titers of antinuclear antibodies (ANA 1:5120) were observed, as well as ANCA positivity, primarily PR3, despite the patient having inactive rheumatic disease. These findings suggest the possibility of an overlap syndrome between systemic lupus erythematosus (SLE) and ANCA-associated vasculitis. This overlap could explain the severity of both the pulmonary involvement and the glomerulonephritis observed. In this regard, Nasser et al. have reported that the prevalence of ANCA is significantly higher in SLE patients who experience relapses than in those who do not, even when ANCA levels remain stable before the clinical event [1, 3].

This finding indicates a potential pathogenic role for these autoantibodies in the active manifestation of the disease, especially in severe cases such as lung-kidney syndrome, as documented here. This evidence supports the hypothesis of dual immune activation that may have worsened multi-organ progression in this patient [13].

In general, male patients with systemic lupus erythematosus (SLE) have been observed to have a lower frequency of photosensitivity but a higher incidence of serositis, a later onset of the disease, and

a higher mortality rate compared to women [14]. In this case, the multi-organ dysfunction suggests expanding the diagnostic approach to consider the possibility of underlying vasculitis as a marker of clinical severity. It is important to highlight the findings of the study conducted in Barcelona, Spain, by Ramos-Casals et al., which analyzed 670 patients with SLE, of whom vasculitis was identified in only 11% of cases. This subgroup showed a female-to-male ratio of 8.5:1 and a mean age of presentation of 37.8 years, emphasizing the infrequent but clinically significant nature of this complication, particularly in male patients with severe or atypical forms of the disease. Therefore, a high index of suspicion is essential during initial evaluation.

Regarding ANCA-associated vasculitis (ANAV), these include three main clinical entities: granulomatosis with polyangiitis (GPA), formerly known as Wegener's granulomatosis; microscopic polyangiitis (MPA); and eosinophilic granulomatosis with polyangiitis (EGPA), formerly called Churg-Strauss syndrome. These conditions present a broad clinical spectrum, from mild skin manifestations to severe cases with multi-organ involvement. Although they share ANCA-associated immunopathological mechanisms, they differ in organ involvement, predominant antibody type, relapse frequency, and clinical prognosis [15].

Diffuse alveolar hemorrhage (DAH) is a rare but serious complication of SLE. It typically presents with dyspnea, fever, and cough, which can be mistaken for pneumonia or other lung infections. DAH is linked to high scores on the lupus activity index (SLEDAI > 12), and often coincides with symptoms like lupus nephritis (66–100%), arthritis (15–75%), and neuropsychiatric issues (20–60%) [14].

Histopathologically, three patterns have been described in DAH: pulmonary capillaritis (with neutrophilic infiltrate and alveolar capillary damage), "soft" pulmonary hemorrhage (accumulation of blood and fibrin without inflammation), and diffuse alveolar damage (septal edema and hyaline membranes). Frequently, patients present with bilateral infiltrates that respond rapidly to corticosteroids and poorly to antibiotics, indicating a non-infectious origin. Although "soft" hemorrhage predominates, factors such as infection, aspiration, or cardiac or renal failure can also contribute; therefore, early diagnosis and treatment are key given its high mortality rate [12].

Lupus nephritis is a serious complication of systemic lupus erythematosus, affecting 30–50% of patients and serving as the initial manifestation in 10%. It is linked to high morbidity and mortality, with histological transformation occurring in 10–45% of cases, usually to more severe forms. Minimal change disease (MCD) is seen in 2–25%, characterized by mild proteinuria and normal urinary sediment. The mesangial form (10–20%) presents with proteinuria, mild microhematuria, and immune deposits in the mesangium. Focal segmental glomerulosclerosis (FSGS) manifests with microhematuria and proteinuria, progressing to the diffuse form in 20%; it is classified as IIIA, IIIA/C, and IIIC based on the damage proportion (>50%). Proliferative GN is among the most common and severe forms, associated with nephrotic syndrome (60–90%), microhematuria (70%), hypertension (40%), and chronic kidney disease (50%). A renal biopsy is recommended in cases of significant proteinuria or microhematuria, impaired renal function, suspected thrombotic microangiopathy, or histological changes. [12].

Within the diagnostic protocol for suspected diffuse alveolar hemorrhage (DAH) or autoimmune lung diseases, the determination of antineutrophil cytoplasmic antibody (ANCA) levels, bronchoscopy, chest computed tomography and urinary sediment analysis are recommended [12].

For the therapeutic management of systemic lupus erythematosus (SLE)-associated kidney-lung syndrome (GLLS), the use of aggressive immunosuppressive therapies is crucial due to the condition's high severity. Plasmapheresis has proven effective for quickly removing antibodies, immune complexes, and inflammatory mediators, and is especially recommended in critical cases. Although its full efficacy is not yet proven, studies such as those by Enaldino et al. have shown benefits in diffuse alveolar hemorrhage (DAH), and the randomized clinical trial by Lewis et al. (1992)—which included patients with lupus nephritis—found no significant differences in mortality or renal failure when comparing standard treatment with or without plasmapheresis, but it did show that its use does not increase risks, supporting its use in selected situations [16].

This strategy is complemented by glucocorticoids, such as pulsed methylprednisolone followed by oral prednisone, which are essential for inducing remission by reducing inflammation and suppressing antibody production. In refractory cases or those with a high autoimmune burden, rituximab—an anti-CD20 monoclonal antibody—has shown effectiveness in ANCA vasculitis and has been proposed for SLE with severe involvement, such as lupus nephritis or autoimmune hemorrhagic fever (AHF), especially when there is a poor response to cyclophosphamide or related toxicity. Although standardized guidelines for its use in PRS are not yet available, its safety profile and therapeutic potential make it a relevant option in refractory cases [16].

Conclusion

The overlap syndrome between late-onset systemic lupus erythematosus (SLE) and ANCA vasculitis is a rare but serious condition requiring rapid diagnosis and intensive treatment. The coexistence of clinical and serological manifestations typical of both diseases characterizes it. In late-onset SLE, lupus nephritis is common and can lead to high morbidity and mortality if not treated appropriately. However, the case described here presents with an acute and unusual onset, with rapidly progressive glomerulonephritis and kidney-lung syndrome, likely due to concurrent ANCA vasculitis. Early diagnosis of lupus nephritis allows for timely treatment, resulting in a better therapeutic response and a lower risk of irreversible kidney damage. Furthermore, type III or IV proliferative nephritis has been documented to be frequently associated with antidiuretic hormone (ADH), high anti-dsDNA titers, low complement levels, and high lupus activity, as well as the development of lupus nephritis in patients with SLE.

Abbreviations

SLE: Systemic Lupus Erythematosus.
NL: Lupus nephritis
ANA: Antinuclear antibodies
dsDNA : native DNA
GN: Glomerulonephritis.
SLiCC : Systemic Lupus International Collaborating Clinics

Supplementary information

Supplementary materials have not been declared.

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Authors' contributions

José Lucas Daza: Conceptualization, data curation, research, visualization, original draft writing.
Juan Camilo García Peralta: Conceptualization, data curation, research, visualization, original draft writing.
Manuela Alejandra Olarte Osma: Conceptualization, data curation, research, visualization, original draft writing.
Francisco Javier Vega Pérez: Conceptualization, data curation, research, visualization, original draft writing.
María Alejandra Falla Macias: Conceptualization, data curation, formal analysis, project management, software, validation, visualization, writing – review and editing.
Verónica Piedad Remache Otañez : Conceptualization, data curation, research, visualization, original draft writing.
Vanesa Villavicencio Cerón: Conceptualization, data curation, research, visualization, original draft writing.
Aldrin Diógenes Sosa Alvarado : Conceptualization, formal analysis, methodology, project management, resources, software, supervision, validation, writing – review and editing.
All authors read and approved the final version of the manuscript.

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Not applicable.

Statements

Ethics committee approval and consent to participate

Not required for clinical cases.

Consent for publication

The authors have the patient's written permission to publish. The document is available for review if requested by the editor.

Conflicts of interest

The authors declare no conflicts of interest.

Author information

None

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