RESEARCH PAPER

Late-Onset Systemic Lupus Erythematosus Presenting as Pancytopenia with Thromboembolic Disease in an Elderly Patient with Multiple Comorbidities: A Clinically Annotated Case Report with Diagnostic Framework Analysis

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ABSTRACT

Objective: To describe and analyse the clinical, haematological, and serological characteristics of a case of late-onset systemic lupus erythematosus (SLE) in an elderly patient with multiple comorbidities, with emphasis on the diagnostic challenges inherent to this population, the utility of contemporary classification criteria, and the therapeutic response to targeted immunomodulatory therapy.

Methods:A single-patient observational case report with analytical review was conducted. A 68-year-old woman with pre-existing type 2 diabetes mellitus, systemic hypertension, and coronary artery disease presented with acute-onset fever, altered sensorium, progressive pancytopenia, and a preceding history of pulmonary thromboembolism with deep vein thrombosis. Full haematological, biochemical, microbiological, and immunological investigations were performed. Classification of SLE was applied using both the Systemic Lupus International Collaborating Clinics (SLICC) 2012 criteria and the 2019 European League Against Rheumatism/American College of Rheumatology (EULAR/ACR) criteria. Imaging included computed tomographic pulmonary angiography, whole-body positron emission tomography–computed tomography (PET-CT), and serial venous Doppler ultrasonography**.**

Results: Progressive pancytopenia evolved during the index hospitalisation, with nadir values of haemoglobin 7.3 g/dL, platelet count 46,400/mm³, and leucocyte count 2,240/mm³. Direct antiglobulin testing was strongly positive (3+). Serological investigations revealed: positive antinuclear antibody by immunofluorescence (3+; titre 1:100), positive anti-double-stranded DNA (anti-dsDNA) IgG antibodies, positive anti-nucleosome antibodies, intermediate anti-Smith and anti-histone reactivity, and reduced complement C3 with preserved C4. Antiphospholipid antibody testing (anticardiolipin IgG/IgM, anti-β2-glycoprotein I, and lupus anticoagulant) was uniformly negative. Both SLICC 2012 and 2019 EULAR/ACR classification thresholds were satisfied without requiring renal biopsy. Treatment with pulse intravenous methylprednisolone (1 g/day for five days) followed by oral prednisolone and hydroxychloroquine produced marked clinical and haematological recovery within seven days; platelet count normalised to 199,000/mm³ at one-month follow-up.

Conclusion: Late-onset SLE can masquerade as a constellation of common comorbid conditions in elderly patients, with each individual clinical feature obscuring the unifying autoimmune diagnosis. Validated classification criteria are indispensable tools in this setting, enabling confident diagnosis and obviating more invasive procedures. Prompt immunomodulatory therapy yields substantial clinical benefit even in the context of multimorbidity. This case reinforces the importance of incorporating late-onset SLE into the differential diagnosis of unexplained pancytopenia and recurrent thromboembolism in older adults.

Keywords: Lupus Erythematosus, Systemic; Pancytopenia; Thromboembolism; Aged; Late Onset Disorders

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1. INTRODUCTION

Systemic lupus erythematosus (SLE) is a prototypic systemic autoimmune disorder characterised by the production of pathogenic autoantibodies directed against nuclear and cytoplasmic antigens, with consequent immune complex deposition and multiorgan inflammation [1]. The pathogenesis of SLE implicates two pivotal and interrelated axes: dysregulated type I interferon (IFN-I) signalling and autoantibody generation against nucleic acid–binding proteins. In genetically predisposed individuals, environmental triggers—potentially microbial in origin—are proposed to precipitate IFN-I elaboration, autoantibody production, or both, thereby initiating the clinical disease cascade [1].

SLE classically affects women during reproductive years, with a female-to-male ratio approximating 9:1. Oestrogen-mediated augmentation of immune dysregulation is a major postulated driver of this pronounced sex predilection [2]. The clinical phenotype is highly heterogeneous, ranging from self-limited mucocutaneous manifestations to life-threatening renal, neuropsychiatric, and haematological organ dysfunction [3].

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Late-onset SLE, conventionally defined as disease onset beyond 50–65 years of age, represents a clinically and serologically distinct subgroup within the SLE spectrum, though a universally accepted age threshold has not yet been established [4]. Postmenopausal hormonal alterations and age-related dysregulation of cellular immune homeostasis have been proposed as contributors to disease onset in older individuals [5]. Compared with early-onset disease, late-onset SLE is characterised by a relative paucity of mucocutaneous and renal involvement, alongside a higher prevalence of serositis and haematological abnormalities, and an attenuated but persistent female predominance [6]. Epidemiological data from a retrospective Tunisian cohort of 342 SLE patients estimate elderly-onset disease at approximately 5.3% of all SLE cases, with a median diagnostic age of 70 years and a female-to-male ratio of 5:1 [7].

The combination of insidious onset, atypical clinical features, and intrinsically low a priori disease probability in older populations frequently results in substantial diagnostic delay [8]. Each presenting feature—thromboembolic disease, cytopenias, proteinuria, or serositis—typically carries a plausible alternative explanation when evaluated in the context of existing comorbidities, further suppressing clinical suspicion for an autoimmune aetiology.

We present a case of a 68-year-old woman with an established cardiometabolic comorbidity burden who, following multiple hospitalisations over a one-year period, was ultimately diagnosed with late-onset SLE after the convergence of unexplained progressive pancytopenia, haemolytic anaemia, thromboembolic disease, and a comprehensive serological autoimmune profile. This report details the diagnostic pathway, application of classification criteria, and therapeutic course, with the aim of enhancing awareness of atypical late-onset SLE presentations in elderly patients with multimorbidity.

2. CASE PRESENTATION

2.1 Patient Background and Presenting Complaint

A 68-year-old woman, a resident of Tamil Nadu, India, was brought to the emergency department with a one-day history of acute-onset fever, progressive alteration of sensorium, and incoherent speech. Her established medical background included type 2 diabetes mellitus, systemic arterial hypertension, and coronary artery disease. Admission medications included torsemide 10 mg once daily and apixaban 5 mg twice daily, the latter initiated during a recent hospitalisation.

2.2 Ethics Approval

The case report was approved by the Institutional Ethics Committee (Students) (Clearance Number: SRMIEC-ST1224-1789; Date: 18 December 2024). The case was managed in accordance with institutional standards and the ethical principles of the Declaration of Helsinki.

2.3 Antecedent Hospitalisation and Thrombotic History

Two weeks prior to the index admission, the patient had been hospitalised at an external facility for a urinary tract infection. On the third hospital day, she developed acute tachycardia and oxygen desaturation. Computed tomographic pulmonary angiography confirmed acute pulmonary thromboembolism, and venous Doppler ultrasonography of the left lower limb demonstrated common femoral vein thrombosis consistent with acute deep vein thrombosis (DVT). Anticoagulation was commenced and an inferior vena cava (IVC) filter was inserted. A whole-body positron emission tomography–computed tomography (PET-CT) performed concurrently was unremarkable except for bilateral minimal pleural effusions.

2.4 Clinical Examination on Admission

On examination, the patient was pale, minimally responsive to verbal commands, and had left-sided pitting pedal oedema extending to the knee. Vital signs demonstrated: blood pressure 120/80 mmHg, heart rate 140 beats per minute, respiratory rate 30 breaths per minute, and peripheral oxygen saturation requiring supplemental oxygen at 4 L/min. The Glasgow Coma Scale score was 13/15, with bilaterally equal and reactive pupils and bilateral flexor plantar responses. Respiratory examination disclosed bilateral infrascapular coarse crepitations.

2.5 Laboratory Investigations

Initial haematological evaluation revealed thrombocytopenia (platelet count 100,000/mm^3), anaemia (haemoglobin 8.7 g/dL), and a normal total leucocyte count. Biochemical profiling demonstrated acute-on-chronic kidney injury (serum creatinine 1.6 mg/dL; baseline 0.61 mg/dL fifteen days prior), with concomitant hyponatraemia, profound hypokalaemia (1.9 mmol/L), hypochloraemia (83 mmol/L), and markedly elevated serum bicarbonate (51 mmol/L), consistent with diuretic-induced metabolic alkalosis. Hypocalcaemia (corrected calcium 7.9 mg/dL; albumin 1.9 g/dL) and hypomagnesaemia (0.8 mg/dL) were also documented. Arterial blood gas analysis confirmed severe metabolic alkalosis with partial respiratory compensation.

Urinalysis revealed 10–12 red blood cells and 20–22 white blood cells per high-power field with trace proteinuria. The urine protein-to-creatinine ratio was 0.64, urine albumin-to-creatinine ratio was 327 mg/g, and 24-hour urinary protein excretion measured 337 mg, remaining below the 500 mg threshold conventionally applied to guide renal biopsy decisions.

2.6 Imaging and Microbiological Findings

Chest radiography confirmed bilateral minimal pleural effusions. Computed tomography of the chest demonstrated bilateral mild pleural thickening with incidentally noted calcified granulomas in the liver and spleen (Figure 1). Abdominal ultrasonography revealed features suggestive of serositis with heterogeneous hepatic echotexture and mild free fluid (Figure 2). Repeat venous Doppler imaging of the left lower limb

confirmed stable common femoral vein thrombosis with extension into the superficial and deep femoral veins (Figures 3–7). Blood and urine cultures were obtained prior to antibiotic initiation; subsequent culture results identified a causative pathogen and enabled antibiotic rationalisation. Serological screening for common tropical infections—dengue, scrub typhus, and leptospirosis—was uniformly negative.

Figure 1: Axial computed tomography of the chest showing bilateral mild pleural thickening with minimal pleural effusion. Incidental calcified granulomas are noted in the liver and spleen.

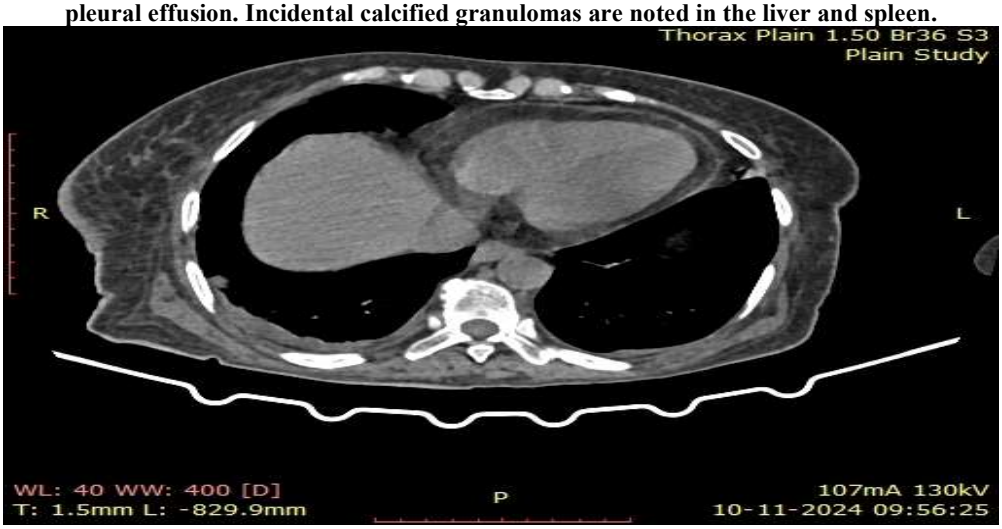


Figure 2: Abdominal ultrasonography showing heterogeneous hepatic echotexture with features suggestive of serositis.

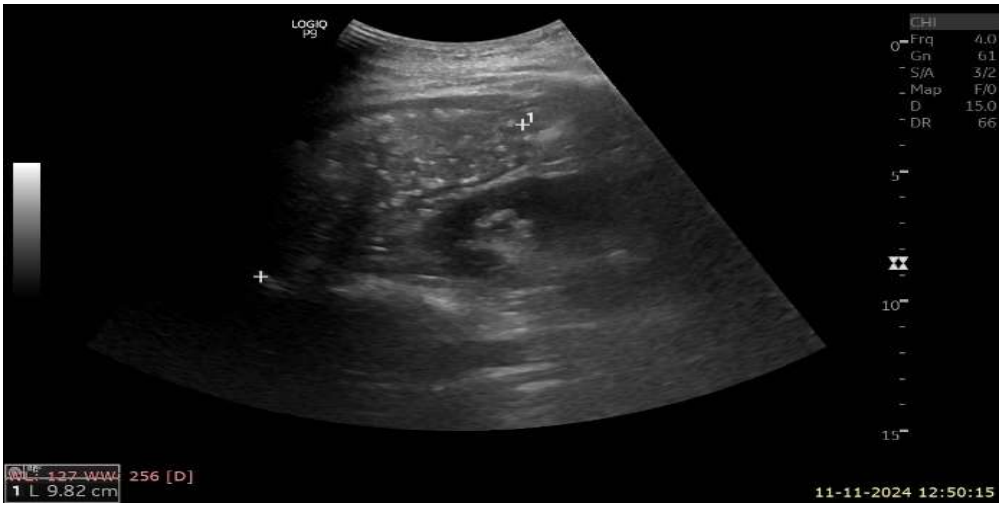


Figure 3: Venous Doppler ultrasonography of the left lower limb demonstrating non-compressible left common femoral vein (CFV) with intraluminal thrombus, consistent with deep vein thrombosis.

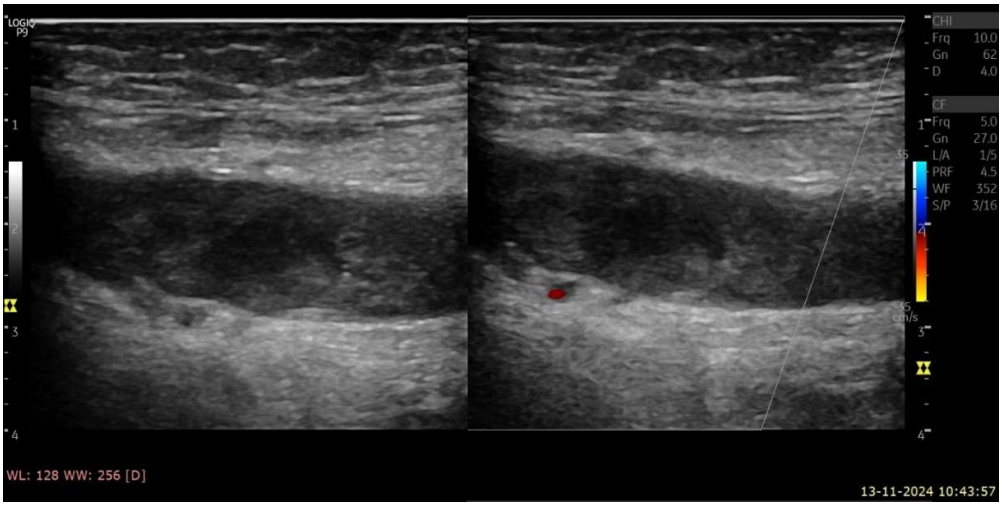


Figure 4: Colour Doppler image of the left common femoral vein showing absent/reduced flow signal in the thrombosed segment, confirming venous occlusion.

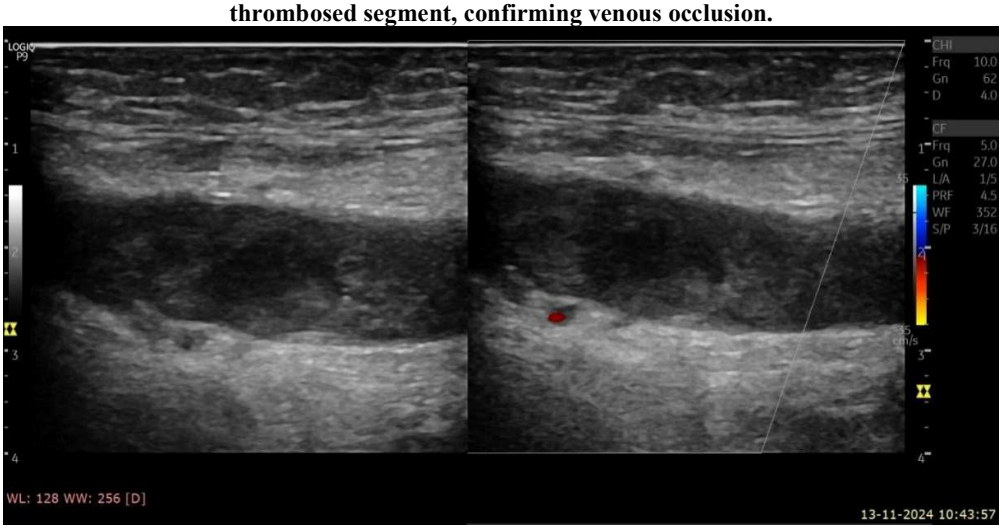


Figure 5: Longitudinal grayscale ultrasound image of the left common femoral vein demonstrating echogenic intraluminal thrombus.

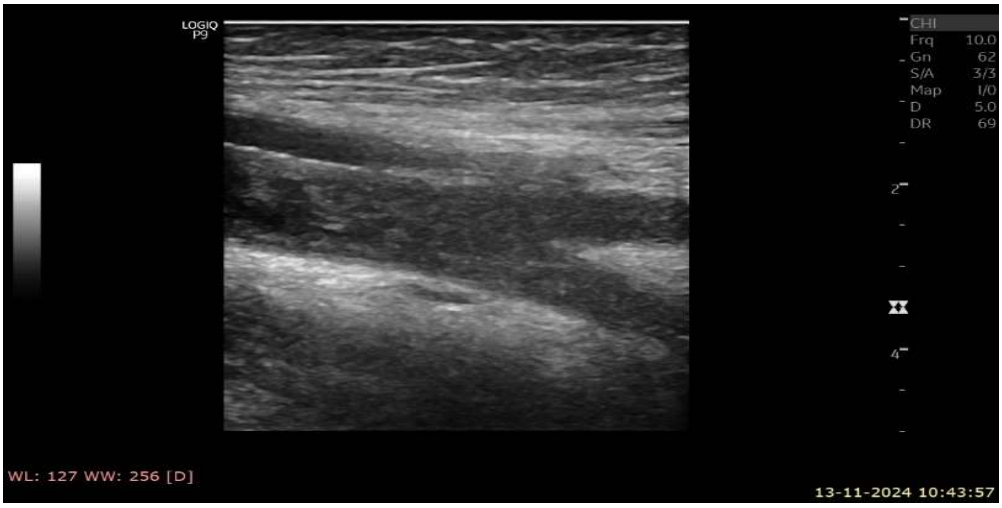


Figure 6: Venous Doppler ultrasonography showing thrombotic involvement extending into the superficial femoral vein (SFV) and deep femoral vein (DFV).

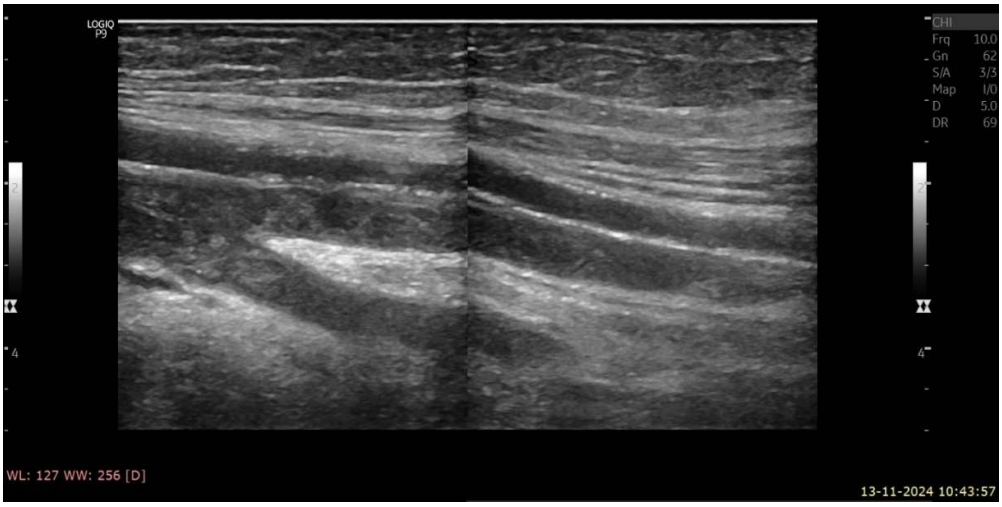
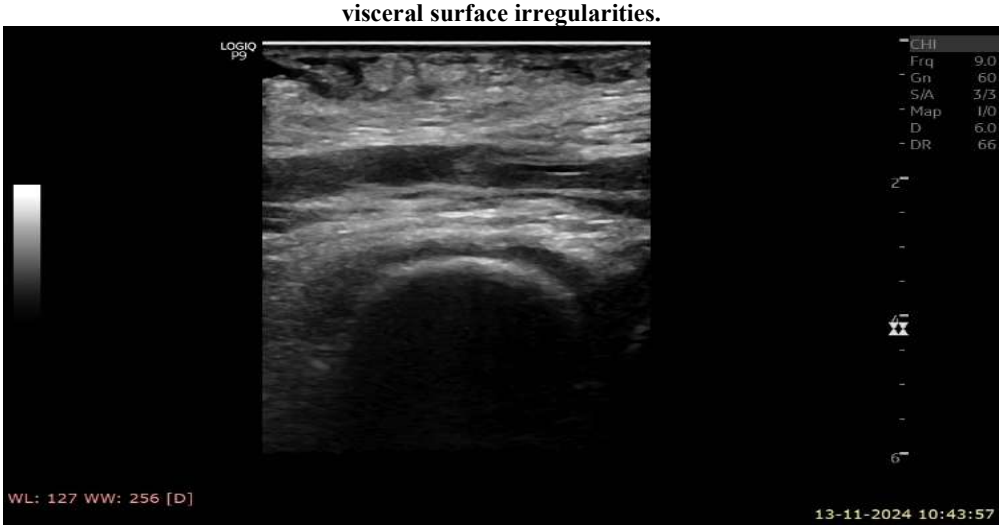


Figure 7: Abdominal ultrasonography demonstrating features suggestive of serositis with mild free fluid and visceral surface irregularities.



2.7 Haematological Progression and Autoimmune Workup

As infectious triggers were excluded and the patient's clinical status stabilised, serial complete blood counts disclosed a progressive deterioration across all three haematopoietic lineages. Nadir values were: haemoglobin 7.3 g/dL, platelet count 46,400/mm³, and total leucocyte count 2,240/mm³, thereby fulfilling the operational definition of pancytopenia. Peripheral blood smear morphology was consistent with anaemia of chronic disease with no features of microangiopathic haemolysis. Direct antiglobulin (Coombs) testing was strongly positive at 3+, confirming an immune-mediated haemolytic component. Complement studies demonstrated reduced C3 with preserved C4.

Antinuclear antibody (ANA) testing by immunofluorescence assay was positive at 3+ intensity (titre 1:100). Extended autoantibody profiling (Table 1) demonstrated positive anti-dsDNA IgG, positive anti-nucleosome antibodies, and intermediate reactivity for both anti-Smith and anti-histone antibodies. The full antiphospholipid antibody panel—anticardiolipin IgG and IgM, anti-β2-glycoprotein I, and lupus anticoagulant—returned uniformly negative.

Table 1. Autoimmune Serological Profile

Parameter	Result	Reference / Note
Complement C3	Low	Normal: 90–180 mg/dL
Complement C4	Normal	Normal: 16–47 mg/dL
Direct Coombs Test (DAT)	Positive (3+)	—

ANA by Immunofluorescence	3+ (titre 1:100)	Titre ≥1:80 considered positive
Anti-dsDNA IgG antibody	Positive	—
Anti-histone antibody	Intermediate reactivity	—
Anti-Smith (anti-Sm) antibody	Intermediate reactivity	—
Anti-nucleosome antibody	Positive	—
Anticardiolipin IgG/IgM	Negative	Antiphospholipid panel
Anti-β2-glycoprotein I	Negative	Antiphospholipid panel
Lupus anticoagulant	Negative	Antiphospholipid panel

ANA-IF: Antinuclear antibody by immunofluorescence; anti-dsDNA: Anti-double-stranded DNA antibody; DAT: Direct antiglobulin test.

2.8 Diagnostic Classification

A formal diagnosis of SLE was established by satisfying both the SLICC 2012 classification criteria (requiring ≥4 criteria, including at least one clinical and one immunological domain) and the 2019 EULAR/ACR classification criteria (entry criterion ANA ≥1:80; aggregate weighted score ≥10). Table 2 details the specific criteria fulfilled under each framework. Renal biopsy was deferred given that 24-hour proteinuria remained below the 500 mg threshold and the overall clinical picture did not mandate histological confirmation for management decisions.

Table 2. Application of SLE Classification Criteria

Classification System	Criteria Met	Score / Status
SLICC 2012	Haemolytic anaemia (DAT+); Thrombocytopenia; Leucopenia; Reduced complement (C3↓); Anti-dsDNA+; Anti-nucleosome+; ANA 3+	≥4 criteria fulfilled (≥1 clinical + ≥1 immunological)
2019 EULAR/ACR	ANA ≥1:80 (entry criterion met); Haematological domain: haemolytic anaemia (+4), thrombocytopenia (+4); Complement domain: low C3 (+3); SLE-specific antibodies: anti-dsDNA (+6); Serositis: pleural effusions (+5)	Aggregate score ≥10; Diagnosis established

SLICC: Systemic Lupus International Collaborating Clinics; EULAR: European League Against Rheumatism; ACR: American College of Rheumatology.

2.9 Management and Clinical Course

Torsemide was discontinued and targeted parenteral electrolyte replacement was administered for the severe dyselectrolytaemia, attributed to prolonged diuretic therapy in the setting of reduced oral intake. The patient was commenced on pulse intravenous methylprednisolone 1 g/day for five consecutive days, followed by a transition to oral prednisolone 0.5 mg/kg/day with a planned gradual taper over 8–12 weeks. Hydroxychloroquine 200 mg twice daily was initiated concurrently. Marked clinical improvement and progressive normalisation of haematological parameters were observed within seven days of treatment initiation. At one-month outpatient follow-up, platelet count had recovered to 199,000/mm³. The patient continues under regular rheumatological review, with ongoing anticoagulation for documented DVT.

3. DISCUSSION

This case illustrates the substantial diagnostic complexity of late-onset SLE in an elderly patient whose clinical presentation was dominated by conditions that carry independent explanatory weight for each individual feature. The diagnosis was ultimately secured through the convergence of haematological, serological, and clinical findings interpreted against validated classification frameworks—a process that highlights several important themes in the management of autoimmune disease in older adults.

3.1 Diagnostic Challenges in Late-Onset SLE

The clinical features that ultimately proved central to the diagnosis of SLE—thromboembolic disease, cytopenias, proteinuria, and serositis—were each initially attributable to established comorbidities. Pulmonary thromboembolism in a patient with coronary artery disease and prolonged immobility plausibly

arises from conventional Virchow triad mechanisms, independent of lupus-associated hypercoagulability. Nevertheless, population-based data indicate that SLE confers a three- to four-fold excess risk of venous thromboembolism compared with the general population, independent of traditional cardiovascular risk factors [9]. This elevated thrombotic risk, present irrespective of antiphospholipid antibody status, likely reflects underlying immune dysregulation, endothelial injury, and platelet activation in the context of active systemic inflammation. Immune-mediated haemolytic anaemia in a patient with longstanding diabetes and chronic systemic inflammation may be readily—and erroneously—attributed to anaemia of chronic disease. The strongly positive direct antiglobulin test and the multi-autoantibody serological profile were pivotal in reframing the haematological picture from an inflammatory to an immune-mediated aetiology. Similarly, thrombocytopenia was provisionally attributed to sepsis-associated marrow suppression, and mild proteinuria was considered secondary to diabetic or hypertensive nephropathy. These sequential diagnostic pitfalls are well-described in the late-onset SLE literature [8]. Cutaneous manifestations—malar rash, photosensitivity, discoid lesions, and Raynaud phenomenon—occur with significantly lower frequency in elderly-onset SLE than in younger patients [10]. This relative paucity of the classical hallmarks that typically prompt earlier investigation further suppresses diagnostic suspicion in older cohorts. Conversely, pulmonary and pleural involvement, including serositis and interstitial lung disease, is reported with higher prevalence in late-onset disease [11], as observed in this case. The bilateral pleural effusions in the current patient, identified both on the antecedent PET-CT and on radiological and ultrasonographic imaging during the index admission, were retrospectively consistent with SLE-associated serositis rather than an isolated infectious or haemodynamic process.

3.2 Clinico-Serological Profile of Late-Onset SLE

Late-onset SLE demonstrates a distinct clinical and immunological phenotype compared with its early-onset counterpart. Mucocutaneous and renal manifestations are less prevalent, while serositis, haematological abnormalities, and pulmonary disease are more prominent; the female predominance, while retained, is attenuated, with reported female-to-male ratios ranging from approximately 5:1 to 8:1 in elderly cohorts, compared with 9:1 overall [12, 13]. The current patient exemplified this profile: haematological abnormalities (pancytopenia with immune-mediated haemolysis), serositis (bilateral pleural effusions), and multi-autoantibody seropositivity in the absence of malar rash, nephritis meeting biopsy criteria, or significant photosensitivity. Serologically, elevations in anti-Ro/SSA and anti-La/SSB antibodies have been reported with greater frequency in late-onset disease, and drug-induced SLE must always be considered in older patients receiving medications such as hydralazine, procainamide, or isoniazid, given the expected anti-histone positivity in that entity. In the present case, intermediate anti-histone reactivity was noted; however, the patient's medication list did not include classical culprit agents, and the overall serological profile—positive anti-dsDNA and anti-nucleosome with reduced complement—was not consistent with drug-induced SLE, which characteristically spares complement levels [13]. Anti-dsDNA positivity, observed here, remains a hallmark of SLE across age groups and correlates with disease activity and renal involvement.

3.3 Role of Validated Classification Criteria

The diagnostic utility of applying validated classification frameworks in the clinical, rather than exclusively research, context was well-demonstrated in this case. The SLICC 2012 criteria demand satisfaction of at least four criteria encompassing at least one clinical and one immunological domain, while the 2019 EULAR/ACR criteria employ ANA positivity as a mandatory entry criterion, followed by a weighted, domain-based scoring system with a threshold score of ten for classification. Both frameworks were satisfied in this patient, enabling confident diagnosis without recourse to bone marrow biopsy—which was under active clinical consideration given the progressive pancytopenia—or renal biopsy. This observation has practical significance: in elderly patients, invasive diagnostic procedures carry heightened procedural risk, and the availability of validated multiparameter criteria provides a clinically sound alternative pathway to diagnosis. The incremental value of the 2019 EULAR/ACR criteria, with its explicit weighting of antibody specificities and organ domain involvement, offers particular utility in complex presentations where individual findings may appear nonspecific in isolation.

3.4 Prognosis, Damage Accrual, and Therapeutic Considerations

Late-onset SLE generally exhibits lower mean disease activity scores and less severe acute organ involvement compared with early-onset disease; paradoxically, however, a higher damage accrual index has been reported in late-onset cohorts, attributable predominantly to premorbid comorbidity burden rather than lupus-specific end-organ injury [14]. This creates a therapeutic dilemma: the requirement for effective immunosuppression must be carefully balanced against the heightened risks of infection, glucocorticoid-associated osteoporosis, metabolic dysregulation, and cardiovascular toxicity inherent to elderly patients with multimorbidity.

In the present case, prompt initiation of pulse glucocorticoid therapy in combination with hydroxychloroquine produced a rapid and clinically meaningful haematological response, consistent with reports of favourable short-term outcomes following treatment in elderly-onset disease. Hydroxychloroquine, with its well-established cardioprotective and immunomodulatory properties, and comparatively favourable safety profile, represents

an appropriate cornerstone of long-term maintenance therapy in this population. Anticoagulation for documented DVT was maintained, and an IVC filter had been inserted during the antecedent hospitalisation.

It is noteworthy that antiphospholipid antibody testing was uniformly negative in this patient. While antiphospholipid syndrome is an important secondary diagnosis to consider in SLE-associated thromboembolism, the absence of antiphospholipid antibodies does not exclude a lupus-related hypercoagulable state, as alternative mechanisms—including endothelial activation, complement-mediated prothrombotic states, and innate immune dysregulation—may contribute to thrombotic risk in seronegative individuals [9].

4. CONCLUSIONS

This case demonstrates the formidable diagnostic complexity that characterises late-onset SLE in elderly patients with multimorbidity. Each clinical feature—thromboembolic disease, progressive pancytopenia, serositis, and mild proteinuria—bore ready alternative attribution in the context of pre-existing cardiometabolic disease, rendering the unifying autoimmune diagnosis non-intuitive. Diagnosis was ultimately achieved through systematic application of both the SLICC 2012 and 2019 EULAR/ACR classification criteria, which together provided a structured and evidence-based framework that precluded the need for more invasive procedures.

Several clinically important lessons emerge from this case. First, unexplained progressive pancytopenia in an elderly patient—particularly when accompanied by a positive direct antiglobulin test and hypocomplementaemia—should prompt dedicated autoimmune evaluation irrespective of the apparent sufficiency of alternative diagnoses. Second, thromboembolic disease in the context of SLE carries an excess risk independent of antiphospholipid antibody status, and a negative antiphospholipid panel does not exclude a lupus-related hypercoagulable contribution. Third, validated classification criteria are not merely research instruments; their structured application in clinical practice can expedite diagnosis, reduce unnecessary invasive investigations, and enable timely initiation of life-altering therapy.

Prompt glucocorticoid and hydroxychloroquine therapy produced rapid, clinically significant haematological recovery in this case, underscoring the treatability of late-onset SLE when recognised. Clinicians in general medicine, geriatrics, haematology, and nephrology should maintain late-onset SLE within the differential diagnosis of unexplained cytopenias, recurrent thromboembolism, and multisystem illness in older adults, even when established comorbidities appear to provide competing explanations for individual clinical features. Systematic vigilance in this population represents an opportunity to reduce the diagnostic delay and its associated morbidity that currently characterise this distinct and underrecognised clinical entity.

DECLARATIONS

Ethics Approval and Consent to Participate

This case report does not involve human experimentation or interventional research beyond standard clinical care; formal ethics committee approval was therefore not required. The case was managed in accordance with institutional standards and the ethical principles of the Declaration of Helsinki.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and all accompanying images. Consent documentation is available upon request by the editorial office.

Competing Interests

The authors declare no competing interests.

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Authors' Contributions

VV was the treating clinician, contributed to case identification, data collection, and manuscript drafting. SG conducted the systematic literature review and contributed to manuscript writing. TAV supervised clinical management, provided critical intellectual revision of the manuscript, and approved the final version for submission. All authors read and approved the final manuscript.

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LIST OF ABBREVIATIONS

ANA: Antinuclear antibody
APLA: Antiphospholipid antibodies
anti-dsDNA: Anti-double-stranded DNA antibody
DAT: Direct antiglobulin test
DVT: Deep vein thrombosis
EULAR/ACR: European League Against Rheumatism/American College of Rheumatology
IFN-I: Type I interferons
IVC: Inferior vena cava
PET-CT: Positron emission tomography–computed tomography
SLE: Systemic lupus erythematosus
SLICC: Systemic Lupus International Collaborating Clinics

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