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(CASE REPORT)

Recurrent Polyserositis as the Inaugural Manifestation of Systemic Lupus: A Case Report from the CHUD-Borgou, Parakou, Benin

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Abstract

Introduction: Systemic lupus (SL) is a systemic autoimmune disease with a particularly severe presentation in African populations. In tuberculosis-endemic regions of sub-Saharan Africa, its clinical overlap with extrapulmonary tuberculosis constitutes a major diagnostic pitfall, leading to harmful treatment delay. We report the case of an 18-year-old woman presenting with a severe, life-threatening, multi-organ inaugural form of SLE following an eleven-month diagnostic odyssey.

Case Report: An 18-year-old patient was admitted to the emergency department for cardiorespiratory failure and anasarca. She presented with malar rash, alopecia, Raynaud's phenomenon, polyarthritis, renal involvement (24-hour proteinuria of 1.139 g/day, hypoalbuminemia at 21.15 g/L), cardiac involvement (moderate-volume pericardial effusion without signs of poor tolerance), and pulmonary involvement (severe pulmonary hypertension). Her history revealed polyserositis eleven months earlier, mistakenly treated as tuberculosis despite a negative sputum smear. Immunological testing (antinuclear antibodies [ANA] 1:320, speckled pattern; positive anti-double-stranded DNA [anti-dsDNA] antibodies) confirmed the diagnosis of SL according to the 2019 EULAR/ACR classification criteria. Management included emergency pleural drainage and initiation of triple immunosuppressive therapy (bolus methylprednisolone, hydroxychloroquine, mycophenolate mofetil), with a favorable clinical course.

Conclusion: This case illustrates the dangerous clinical overlap between extrapulmonary tuberculosis and SLE in tuberculosis-endemic African regions, leading to harmful diagnostic delay. Any recurrent polyserositis in a young woman — especially when resistant to antituberculosis treatment — should prompt investigation for systemic autoimmune disease. A multidisciplinary approach and early, appropriate immunosuppression are critical determinants of prognosis.

Keywords: Systemic Lupus; Polyserositis; Lupus Nephritis; Parakou; Benin

1. Introduction

Systemic lupus (SL) is the prototypical systemic autoimmune disease, characterized by the production of autoantibodies directed against nuclear antigens, leading to immune complex deposition in multiple target organs. Its worldwide prevalence is estimated at 20 to 150 cases per 100,000 inhabitants, with a strong female predominance (sex ratio 9:1),

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a preferential onset between 15 and 45 years of age, and significantly higher morbidity and mortality among populations of African ancestry [1, 2].

In sub-Saharan Africa, SLE presents specific diagnostic and therapeutic challenges. Recent epidemiological data confirm a more severe clinical presentation, earlier and more frequent renal and visceral involvement, and a mean diagnostic delay of 12 to 48 months in this region [3, 4]. This delay is largely driven by the clinical overlap between SL and endemic infectious diseases, foremost among them tuberculosis (TB). In sub-Saharan Africa, extrapulmonary TB is indeed the leading cause of febrile exudative polyserositis, and empirical initiation of antituberculosis treatment for serositis of undetermined etiology remains a common practice, even in the absence of bacteriological confirmation [5, 6].

Lupus polyserositis, defined as the combination of pleural, pericardial, and/or peritoneal effusions, is an inaugural manifestation of SL in 16 to 50% of cases and represents a clinical picture of variable severity that can become life-threatening in the setting of compressive pericardial effusion, associated pulmonary embolism, or pulmonary hypertension (PH) [7, 8]. The coexistence of PH in SLE, reported in 0.5 to 17.5% of cases, is associated with significantly increased mortality, particularly among patients of African descent [9].

We report the case of an 18-year-old patient managed at the Centre Hospitalier Universitaire Départemental du Borgou/Alibori (CHUD-B/A) in Parakou, Benin, who presented with inaugural SL revealed by severe, recurrent polyserositis initially attributed to extrapulmonary tuberculosis. This case raises fundamental questions regarding the differential diagnostic approach, the consequences of treatment delay, and the management of severe SLE in a resource-limited sub-Saharan African setting.

2. Case report

2.1. Patient Information

The patient was an 18-year-old Beninese woman with no relevant personal medical, surgical, or family history. She was nulligravid and had presented with amenorrhea of undetermined etiology for ten months. She did not consume alcohol or tobacco and had no known drug allergies.

2.2. Timeline and Clinical Findings

The patient was admitted as an emergency to the CHUD-B/A for acute cardiorespiratory failure associated with anasarca. On history-taking, she reported constrictive left basithoracic pain, rated 3 out of 4 on the simple verbal scale, radiating to the back, worsened by the supine position, and partially relieved by leaning forward while seated. This pain was accompanied by dyspnea that had progressively worsened over two weeks, progressing from NYHA class II to class IV, with orthopnea. The patient also reported a productive cough with moderate whitish sputum for three days, an estimated weight loss of about ten kilograms, profuse night sweats, and profound asthenia. She additionally noted a rapid, progressive increase in abdominal girth and bilateral lower-limb swelling over the preceding two weeks.

History-taking revealed that approximately eleven months before this admission, the patient had been hospitalized at a peripheral hospital for febrile polyserositis with pericarditis and pleurisy. Given the severity of the presentation, emergency pericardial drainage had been performed. Although sputum smear microscopy was negative, empirical antituberculosis treatment had been initiated for six months because of the local tuberculosis-endemic context, without bacteriological confirmation and without sustained clinical improvement. Two weeks before the present admission, a febrile flare had occurred, with progressive recurrence of peripheral edema and rapid onset of dyspnea, initially on exertion and then at rest, ultimately prompting her transfer to the intensive care unit.

On admission, her general condition was severely impaired (WHO performance status grade III). Vital signs were critical: temperature 37.6°C, respiratory rate 36 breaths/min, SpO_2 82% on room air, blood pressure 81/66 mmHg (right arm), and heart rate 130 bpm.

Skin examination revealed a butterfly-shaped malar rash sparing the nasolabial folds, diffuse non-scarring alopecia, and bilateral Raynaud's phenomenon with digital necrosis. A cutaneous ulceration was present on the right malleolus and leg.

Cardiovascular examination showed tachycardia at 130 bpm, muffled heart sounds without pericardial friction rub, spontaneous jugular venous distension, and bilateral pitting lower-limb edema. Tender hepatomegaly with hepatojugular reflux and moderate-volume ascites were also present. Pulmonary examination revealed signs of

respiratory distress (intercostal retractions, nasal flaring), bilateral basal dullness to percussion, and crackles over two-thirds of the lung fields. Joint examination revealed painful bilateral polyarthritis involving the shoulders and the right wrist. Neurological examination was normal.

Laboratory testing performed during hospitalization revealed multiple, concordant abnormalities. The complete blood count showed normocytic normochromic anemia with hemoglobin at 8.5 g/dL and MCV at 86.5 fL, without thrombocytopenia or leukocytosis. Reticulocytes were normal at 74.8 × 10^9/L, indicating an aregenerative anemia consistent with an inflammatory or central origin.

The most notable biochemical findings were severe hypoalbuminemia at 21.15 g/L (normal range: 35–53 g/L), severe hypocalcemia at 69 mg/L, hypokalemia at 3.19 mEq/L, and mild hyponatremia at 133.4 mEq/L. Renal function was preserved, with creatinine at 6.08 mg/L and urea at 0.35 g/L. Minimal hepatocellular cytolysis was noted, with AST at 52.10 IU/L, while ALT remained within normal limits at 27.77 IU/L.

Inflammatory markers revealed a marked biological inflammatory syndrome, with CRP elevated at 96 mg/L, contrasting with a procalcitonin level below 0.10 ng/mL, which formally excluded active bacterial sepsis as the main cause of the clinical presentation. Coagulation testing showed significant prolongation of the activated partial thromboplastin time (aPTT) at 190.9 seconds against a control time of 26–36 seconds, with a ratio well above 1.20, and a mildly elevated INR of 1.42, strongly suggesting a circulating lupus anticoagulant.

The electrocardiogram showed sinus tachycardia at 125 bpm, right axis deviation, and negative T waves in the antero-septal and inferior leads (Figure 1). Transthoracic echocardiography revealed a moderate-volume circumferential pericardial effusion, measuring 12 mm adjacent to the right ventricle. It also showed pulmonary hypertension, with a systolic pulmonary artery pressure (sPAP) of 48 mmHg and marked right-heart involvement (right ventricular dilation and dysfunction, with a basal RV diameter of 62 mm, TAPSE of 11 mm, and S' of 8.6 cm/s; a non-hypertrophied right ventricle; right atrial dilation with a right atrial area of 26 cm^2; dilation of the pulmonary artery trunk; and septal flattening/inversion). The left ventricle had normal morphology with preserved systolic function, with a left ventricular ejection fraction of 67% by the biplane Simpson method (Figure 2). Chest CT angiography found no evidence of pulmonary embolism but showed signs of pulmonary arterial hypertension, large bilateral pleural effusions, a pericardial effusion, right axillary lymphadenopathy, and a right basal opacity.

Diagnostic pleural puncture yielded citrine-yellow fluid, with biochemical analysis showing a protein level of 40 g/L, confirming the exudative nature of the effusion according to Light's criteria. GeneXpert testing for Mycobacterium tuberculosis was negative on the pleural fluid, and cytobacteriological examination was sterile.

Twenty-four-hour urine analysis revealed significant glomerular proteinuria at 1,139 mg/24h (i.e., 1.139 g/day), with reduced diuresis at 452 mL/24h, confirming glomerular renal involvement in the setting of incipient nephrotic syndrome. Immunological testing, key to the diagnosis, showed antinuclear antibodies (ANA) at a high titer of 1:320 with a speckled pattern, and positive anti-double-stranded DNA (anti-dsDNA) antibodies — findings that, together with the clinical picture, formally confirmed the diagnosis of systemic lupus erythematosus according to the 2019 EULAR/ACR criteria.

2.3. Diagnostic Approach

Given this presentation of severe, recurrent polyserositis with multi-organ failure in a young woman, several diagnostic hypotheses were systematically considered and investigated. Relapsing multifocal tuberculosis was the main competing hypothesis, given the history of antituberculosis treatment and the local endemic context. It was ruled out based on repeatedly negative GeneXpert testing on pleural fluid, a procalcitonin level below 0.10 ng/mL excluding active bacterial sepsis, and the absence of improvement despite six months of well-conducted antituberculosis therapy. Lymphoma was considered because of the axillary lymphadenopathy seen on CT angiography, but it was not confirmed at this stage, and a lymph node biopsy was planned. Finally, isolated congestive heart failure was judged insufficient to explain the recurrent polyserositis, the mucocutaneous manifestations, and the glomerular renal involvement.

The diagnosis of SL was established based on the 2019 EULAR/ACR classification criteria, with a cumulative score well above the required threshold of ten points, as shown in Table 1. The mandatory entry criterion was met by positive antinuclear antibodies (ANA) at a titer of 1:320. Among the additional criteria, the following were retained: butterfly-shaped malar rash (+6 points), non-scarring alopecia (+2 points), bilateral polyarthritis (+6 points), pleural and pericardial serous effusions (+5 points), normocytic anemia (+3 points), glomerular proteinuria at 1.139 g/24h (+4 points), positive anti-dsDNA antibodies (+6 points), and Raynaud's phenomenon associated with prolonged aPTT

suggestive of a circulating lupus anticoagulant (+3 points). The total score, well above thirty-five points, unambiguously confirmed the diagnosis of SL.

Table 1 2019 EULAR/ACR classification criteria and their application to the patient's diagnostic score.

Domain	Criterion / Manifestation	Points	Present?
Mandatory entry criterion: ANA ≥ 1:80			
Antinuclear antibodies (ANA)	Titer 1:320 (speckled pattern)	Mandatory	YES
Additional criteria – diagnostic threshold: ≥ 10 points			
Mucocutaneous	Butterfly-shaped malar rash	+6	YES
Mucocutaneous	Diffuse non-scarring alopecia	+2	YES
Articular	Bilateral polyarthritis (shoulders, wrist)	+6	YES
Serosal	Pleural and pericardial effusions	+5	YES
Hematological	Normocytic anemia (Hb 8.5 g/dL)	+3	YES
Renal	Proteinuria > 0.5 g/24h (1.139 g/day here)	+4	YES
Immunological	Positive anti-dsDNA antibodies	+6	YES
Vascular	Raynaud's phenomenon, prolonged aPTT (lupus anticoagulant?)	+3	YES
TOTAL SCORE	35 ≥ 10 points → SL confirmed	35	

2.4. Therapeutic Interventions

Several urgent symptomatic measures were initiated on admission. Nasal oxygen therapy at 4–6 L/min was started to maintain oxygen saturation above 94%. An emergency evacuating pleural puncture was performed, relieving respiratory distress. Therapeutic anticoagulation with low-molecular-weight heparin (LMWH), subsequently switched to apixaban 5 mg twice daily, was prescribed given the right ventricular dilation and dysfunction, despite the absence of formal confirmation of pulmonary embolism on chest CT. Intravenous furosemide was administered for management of the anasarca and effusions, with strict monitoring of diuresis. The clinical course was marked by the onset of obstructive shock related to right ventricular dysfunction, requiring a 4-day stay in the intensive care unit, with a favorable outcome under vasopressor support (norepinephrine).

Triple immunosuppressive therapy was initiated. First, intensive corticosteroid therapy with intravenous methylprednisolone pulses of 500 mg for two consecutive days was administered, followed by oral prednisone at 0.3 mg/kg/day. Second, hydroxychloroquine 200 mg/day was introduced after a normal baseline fundoscopic examination. Finally, mycophenolate mofetil (MMF) was started at progressively increasing doses up to 2 g/day as induction therapy for lupus nephritis. Vasodilators were also added to control digital ischemia and pulmonary hypertension. Gastric protection with pantoprazole 40 mg/day and calcium supplementation were systematically combined with corticosteroid therapy.

2.5. Follow-up and Outcome

The short-term clinical course was overall favorable under this combined therapeutic strategy. A clear and rapid improvement in dyspnea was observed from the third day after pleural drainage, with oxygen saturation rising to 95–97% on 4 L/min oxygen therapy. The anasarca progressively and completely resolved within ten to fourteen days under the combined effect of furosemide and immunosuppression. Hemodynamic parameters stabilized, with progressive normalization of blood pressure to 130/100 mmHg and of heart rate.

Follow-up echocardiography showed complete resolution of the pericardial effusion but persistence of pulmonary hypertension with signs of chronic cor pulmonale. The digital necrotic lesions stabilized under anticoagulation and vasodilator therapy. Nutritional status progressively improved, with weight gain. Additional immunological work-up, including complement C3 and C4, anti-Sm antibodies, anticardiolipin antibodies, and anti-β2-glycoprotein I antibodies, is currently being analyzed to further characterize the immunological profile of the disease and to investigate for

associated antiphospholipid syndrome. A renal biopsy is planned to obtain a precise histological classification of the lupus nephritis, likely class III or IV according to the 2018 ISN/RPS classification, in order to guide maintenance therapy.

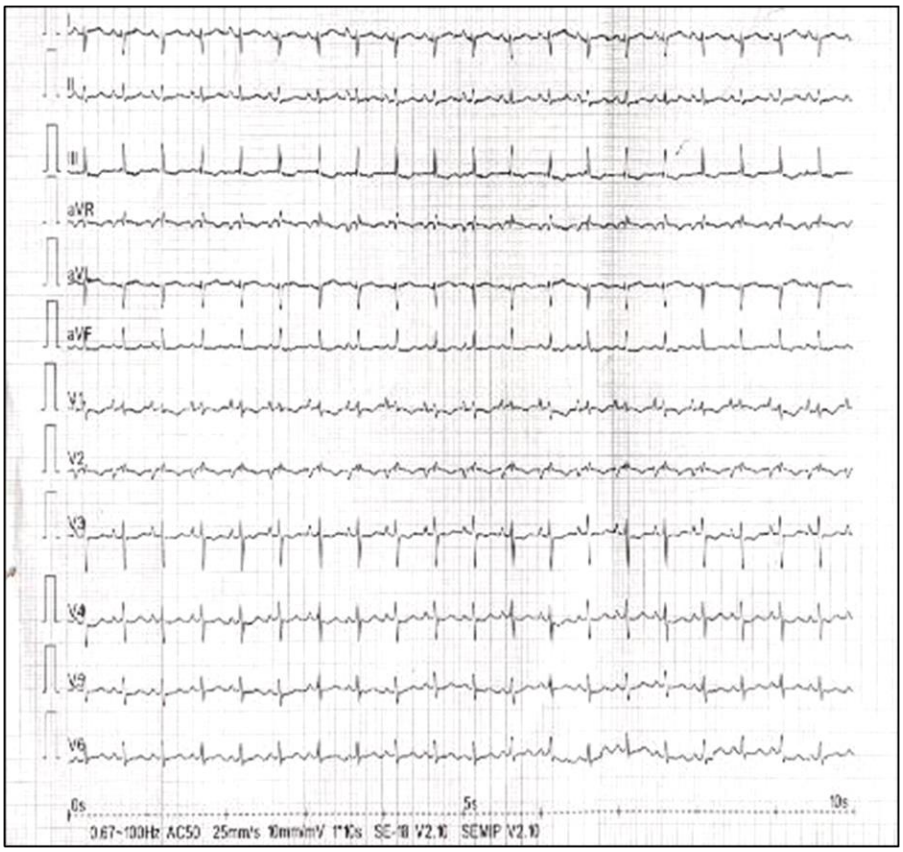

Figure 1 Electrocardiogram showing sinus tachycardia at 125 bpm, right axis deviation, and negative T waves in the antero-septal and inferior leads.

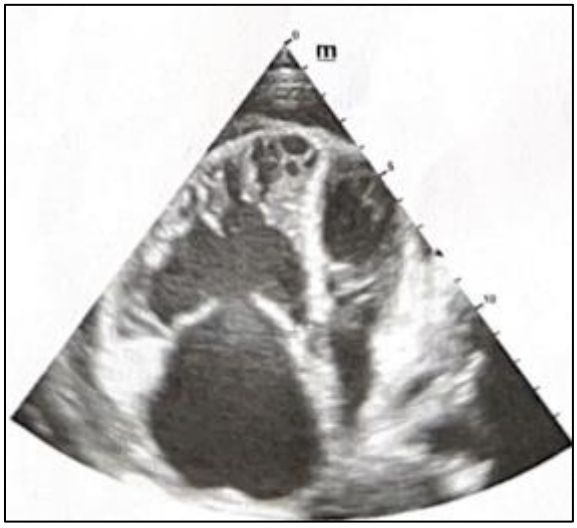

Figure 2 Echocardiographic image showing a moderate-volume circumferential pericardial effusion, measuring 12 mm adjacent to the right ventricle, and right-heart chamber dilation.

3. Discussion

This case corresponds to the most classic epidemiological profile of SLE in sub-Saharan Africa: a young woman in the peri-pubertal period (18 years old), with no identified family history, presenting with a severe inaugural form with multi-organ involvement. Recent data from the African literature confirm that patients of African ancestry develop SLE on average four to five years earlier than Caucasian patients, with an increased frequency of renal, serosal, and neuropsychiatric involvement [1, 2]. The ten-month amenorrhea observed in our patient constitutes a potentially early sign, possibly reflecting hypothalamic-pituitary axis dysfunction related to lupus activity or to malnutrition [10]. This manifestation, often overlooked in daily clinical practice, should systematically alert clinicians to the possibility of an underlying systemic disease in a young woman of childbearing age.

The main diagnostic challenge in this case lies in the clinical overlap between extrapulmonary tuberculosis and SLE, which accounted for an eleven-month diagnostic delay. In sub-Saharan Africa, tuberculosis remains the leading cause of exudative pericarditis and citrine pleural effusion, and the coexistence of fever, polyserositis, and impaired general condition is often sufficient to justify empirical antituberculosis treatment, even without bacteriological confirmation [5, 6]. In our patient, this empirical antituberculosis treatment, continued for six months despite an initially negative sputum smear, allowed the autoimmune disease to progressively worsen toward life-threatening multi-organ involvement.

The discriminating features that should have prompted earlier consideration of an autoimmune etiology included recurrence of serositis despite well-conducted antituberculosis treatment, the presence of suggestive mucocutaneous manifestations (malar rash, alopecia, Raynaud's phenomenon), bilateral polyarthritis, glomerular renal involvement, and prolonged amenorrhea. In line with recent consensus recommendations, ANA testing should be systematically included in the work-up of any recurrent polyserositis resistant to antituberculosis treatment or associated with extrapulmonary signs [6, 7].

Our patient presented with a severe form of SLE according to the 2023 EULAR criteria [11], with simultaneous involvement of six target organs: cutaneous, articular, serosal, renal, pulmonary vascular, and peritoneal. The coexistence of pulmonary embolism, PH, and digital necrosis reflects major endothelial dysfunction and a state of systemic thrombophilia inherent to active SLE, likely amplified by the presence of a circulating lupus anticoagulant, strongly suggested by the marked prolongation of the aPTT to 190.9 seconds [12]. Lupus-associated PH is a rare but serious complication, with a multifactorial pathophysiology combining recurrent thromboembolic events, primary inflammatory vasculopathy, and autoantibody-mediated endothelial dysfunction [8, 9]. Its prevalence is estimated at 0.5 to 17.5% in SLE, with five-year mortality reaching 40 to 60% in African series [9].

Renal involvement is confirmed by glomerular proteinuria at 1.139 g/24h associated with severe hypoalbuminemia at 21.15 g/L, a picture consistent with proliferative lupus nephritis, class III or IV according to the 2018 ISN/RPS classification. This renal involvement, which occurs in 40 to 60% of lupus patients of African descent, represents the main long-term prognostic determinant and justifies the urgency of immunosuppressive induction therapy [1, 13].

The therapeutic strategy adopted is fully consistent with the 2023 EULAR [11] and 2024 KDIGO [13] recommendations for severe SLE with active lupus nephritis. Mycophenolate mofetil at 2 g/day is currently the first-line treatment for proliferative lupus nephritis; its choice was particularly appropriate in this 18-year-old patient because of the preservation of ovarian reserve compared with cyclophosphamide, and its demonstrated superior efficacy in subjects of African ancestry in several randomized trials [13, 14]. The use of prednisone at an intermediate dose of 0.3 mg/kg/day is consistent with the new 2023 EULAR recommendations, which advocate a glucocorticoid-sparing strategy to limit long-term metabolic and infectious adverse effects [11]. Hydroxychloroquine, recommended in all patients with SLE regardless of disease activity, is particularly relevant here given its immunomodulatory effects, cardiovascular protection, flare reduction, and antithrombotic effect in the setting of pulmonary embolism and PH [11, 15]. Therapeutic anticoagulation with apixaban was justified by the severe RV dilation and dysfunction secondary to systemic PH, as well as the prothrombotic state associated with glomerular involvement. The prolonged aPTT is suggestive of a lupus anticoagulant, whose formal confirmation will require two positive assays 12 weeks apart, according to the 2023 ACR/EULAR criteria [12].

The initial choice of therapeutic anticoagulation with apixaban nevertheless warrants discussion. The 2019 EULAR recommendations for the management of antiphospholipid syndrome (APS) advocate vitamin K antagonists as the reference treatment in patients with a high-risk antiphospholipid antibody profile, particularly in the presence of a positive lupus anticoagulant; direct oral anticoagulants are considered only as second-line therapy in this setting and are formally discouraged in triple-positive patients, given the increased risk of recurrent thrombosis demonstrated by

the randomized RAPS and TRAPS trials [16]. In our patient, the marked prolongation of the aPTT together with the elevated INR was strongly suggestive of a circulating lupus anticoagulant, without formal confirmation of APS at the time of the therapeutic decision. The choice of apixaban, motivated by the emergency context and the absence of a need for close laboratory monitoring in the intensive care unit, therefore partially departs from international standards and illustrates the tension between pragmatic management in a resource-limited setting and optimal recommendations. A switch to a vitamin K antagonist, targeting an INR between 2 and 3, should be considered if the positivity of the lupus anticoagulant is confirmed on follow-up testing.

Several challenges specific to the African context deserve emphasis. The inaccessibility of modern biologic therapies such as belimumab or anifrolumab, recommended for resistant or corticosteroid-dependent forms in high-income countries, remains a major obstacle in most sub-Saharan countries because of their prohibitive cost [2, 14]. There is a pressing need to train African clinicians in the practical application of the 2019 EULAR/ACR criteria and to make basic immunological tests (ANA, anti-dsDNA, complement C3/C4) available in regional referral hospitals. Finally, renal biopsy, essential for precise histological classification of lupus nephritis and for guiding maintenance therapy, must be further developed and made more accessible in qualified African centers [13].

4. Conclusion

Systemic lupus erythematosus can present as catastrophic multi-organ failure, particularly in young women in sub-Saharan Africa. This case exemplifies the harmful consequences of the clinical overlap between extrapulmonary tuberculosis and SLE in endemic regions: an eleven-month diagnostic delay, a detrimental therapeutic odyssey, and progression to life-threatening multi-organ involvement.

Compliance with ethical standards

Acknowledgments

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Disclosure of conflict of interest

The authors declare that they have no financial or personal conflicts of interest related to this article.

Statement of informed consent

Written consent was obtained from the patient for the publication of this case report and the associated clinical, paraclinical, and imaging data.

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