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(CASE REPORT)

When the lungs reveal the hidden lupus: A case of pulmonary vasculitis as the first manifestation of systemic lupus erythematosus

A. Mourhri *, S. Moatassime, M. Ijim, O. Fikri and L. Amro

Department of Pulmonology, Arrazi Hospital, CHU Mohammed VI, LRMS Laboratory, FMPM, UCA, Marrakech, Morocco.

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Abstract

Systemic lupus erythematosus (SLE) is a chronic, multisystem autoimmune disease that can affect various organs, including the lungs. Pulmonary vasculitis is a rare but severe manifestation that may acutely reveal SLE. It results from immune-mediated inflammation of pulmonary vessels, potentially leading to severe hypoxemia, respiratory failure, and diffuse parenchymal involvement. We report the case of a 36-year-old woman who presented with pulmonary vasculitis as the first manifestation of SLE, which progressed to acute respiratory distress requiring mechanical ventilation, with diagnosis confirmed by specific immunological markers. Management included intravenous methylprednisolone pulses, followed by oral prednisone and hydroxychloroquine. This case highlights the importance of early recognition of severe pulmonary manifestations of lupus to prevent life-threatening complications.

Keywords: Systemic Lupus Erythematosus; Pulmonary Vasculitis; Respiratory Failure; Hydroxychloroquine; Methylprednisolone

1. Introduction

Systemic lupus erythematosus is an autoimmune disease characterized by the production of autoantibodies directed against nuclear and cytoplasmic antigens, leading to multisystem involvement. [1,2]. Although cutaneous, articular, and renal manifestations are most common, pulmonary involvement represents a severe and sometimes initial complication of SLE [3]. Pulmonary vasculitis although rare is particular, serious and potentially life-threatening, Arising from immune complex-mediated inflammation of pulmonary vessels, causing focal necrosis and diffuse Alveolar damage [4,5]. Early recognition is critical as it may rapidly progress to acute respiratory failure requiring intensive care management [6]. We report here a case of SLE revealed by pulmonary vasculitis, highlighting the diagnostic complexity and the importance of early immunosuppressive treatment.

2. Patient and Observation

A 36-year-old women from Ouarzazat morocco , housewife, with no toxic habits but exposed to wood smoke and poultry droppings, presented with a 20-day history of progressively worsening productive cough with purulent sputum, no hemoptysis, dyspnea at Sadoul stage III, and diffuse inflammatory arthralgias. One day before admission, she developed acute respiratory distress associated with altered consciousness, in a febrile context (38.6°C) with general condition deterioration (asthenia, anorexia, weight loss).

She was initially admitted to the intensive care unit, intubated, and mechanically ventilated for one week, then transferred to our department for further management after stabilization. Pulmonary examination revealed bilateral

* Corresponding author: A. Mourhri

crackles. Chest X-ray showed dense, heterogeneous confluent opacities with blurred margins in both lung fields, associated with bilateral micronodular images.

Laboratory tests revealed leukocytosis with neutrophil predominance without lymphopenia: : GB :12660 PNN :10090 Lym :1560 Hb :10,8, and elevated CRP 174 mg/L. Liver and renal function tests were normal.

Immunology: ANA and anti-DNA antibodies were positive, while RF, anti-CCP, anti-SSA/SSB, C-ANCA, and P-ANCA were negative.

CT pulmonary angiography showed no pulmonary embolism, cardiomegaly due to right chambers with signs of pulmonary hypertension, bilateral pulmonary consolidation, and scattered branching micronodules suggesting infectious origin.

Echocardiography: Left ventricle normal size and function, LVEF 61%, no valvular disease, right atrium dilated but free of thrombus, right ventricle dilated with preserved function, moderate tricuspid regurgitation, estimated PASP 64 mmHg, pulmonary artery dilated, dry pericardium.

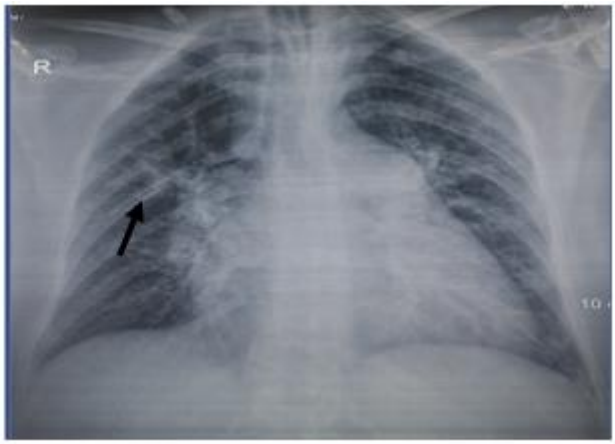

Figure 1 Dense, heterogeneous confluent opacities associated with bilateral micronodular images**.**

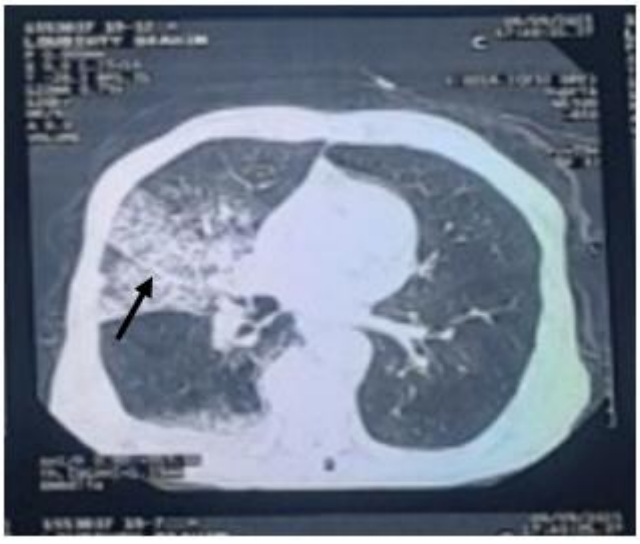

Figure 2 Bilateral pulmonary consolidation foci

The diagnosis of pulmonary vasculitis secondary to SLE was established. The patient received intravenous methylprednisolone pulses (1 g/day for 3 days), followed by oral prednisone and hydroxychloroquine (Plaquenil).

The patient showed a progressive clinical improvement under corticosteroid and hydroxychloroquine therapy, with gradual resolution of respiratory distress and normalization of oxygen saturation. Follow-up chest imaging demonstrated a marked regression of pulmonary infiltrates and no new lesions. She was discharged in good general condition and remained stable during outpatient follow-up, confirming a favorable clinical and radiological outcome.

3. Discussion

Pulmonary vasculitis is a rare but severe manifestation of systemic lupus erythematosus (SLE) and can be the initial presentation of the disease [1,2]. It results from immune-mediated inflammation of small- and medium-sized pulmonary vessels, triggered by circulating immune complexes and autoantibodies, particularly anti-dsDNA, leading to endothelial injury and focal parenchymal necrosis [3,4].

3.1. Clinical manifestations

Patients usually present with progressive respiratory symptoms such as cough, dyspnea, and sputum production, which may evolve to acute respiratory failure, as observed in our patient [5]. Associated systemic symptoms include fever, asthenia, anorexia, and inflammatory arthralgias, reflecting systemic disease activity [6]. Acute respiratory distress and altered consciousness are markers of severity requiring ICU care.

3.2. Imaging and laboratory investigations

Chest imaging is essential for diagnosis. Radiographs may show heterogeneous and micronodular bilateral opacities, while CT scans can reveal consolidation, branching micronodules, and occasionally ground-glass opacities, consistent with vascular and parenchymal involvement [7]. CT angiography rules out pulmonary embolism and identifies secondary pulmonary hypertension [8].

Immunologic testing confirms the diagnosis of SLE and guides treatment. Positive ANA and anti-dsDNA antibodies, with negative RF, anti-CCP, anti-SSA/SSB, C-ANCA, and P-ANCA, strongly support SLE with pulmonary vasculitis [9].

3.3. Therapeutic management

Treatment is based on rapid suppression of immune-mediated inflammation. High-dose intravenous methylprednisolone pulses control acute inflammation and stabilize respiratory function [10]. Oral prednisone maintains remission and prevents relapse. Hydroxychloroquine is essential as long-term therapy, reducing systemic immune activity, joint flares, and mortality [10]. Mechanical ventilation may be required in severe cases to ensure adequate oxygenation and prevent prolonged hypoxemia [9].

3.4. Prognosis and follow-up

The prognosis of pulmonary vasculitis in SLE depends on early diagnosis and appropriate immunosuppressive therapy. Severe complications include respiratory failure, secondary pulmonary hypertension, opportunistic infections, and pulmonary fibrosis [10]. Regular imaging and laboratory follow-up are recommended to monitor treatment response and detect early relapse.

3.5. Risk factors for severity

- Severe initial pulmonary involvement with respiratory distress and hypoxemia
- Presence of pulmonary hypertension
- Delayed diagnosis or inadequate initial treatment
- Secondary infections due to immunosuppression [10]
- Early recognition of pulmonary manifestations and prompt immunosuppressive therapy are essential to improve prognosis and reduce mortality.

4. Conclusion

Pulmonary vasculitis revealing SLE is a medical emergency requiring rapid and appropriate management. The combination of specific immunological markers and thoracic imaging allows early diagnosis, while corticosteroids and hydroxychloroquine improve prognosis and prevent serious complications. This case highlights the importance of considering SLE in any unexplained pulmonary vasculitis.

Compliance with ethical standards

Disclosure of Conflict of Interest

The authors declare no conflict of interest.

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