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Case Report
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Lupus Pleurisy Revealing Systemic Lupus Erythematosus with Severe Renal Involvement: A Case Report

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Introduction

Systemic lupus erythematosus is an autoimmune disease resulting from a multifactorial disturbance of the immune system and responsible for systemic disorders (respiratory, cardiac, cutaneous, renal, hematological, etc.).

Pleural involvement in systemic lupus erythematosus is frequent. It represents a poor prognostic factor in the evolution of this pathology and has been part of its diagnostic criteria since the 1971 primary classification of systemic lupus erythematosus [1].

We report a case of lupus pleurisy revealing systemic lupus erythematosus with severe renal involvement in an 18-year-old female patient.

Observation

The patient was an 18-year-old high-school student with no toxic habits, no comorbidities and no recent tuberculosis contagion.

She presented with minimal but progressive dyspnoea for 1 month, a dry cough, inflammatory arthralgias of the wrists and ankles with photosensitivity and hair loss. She reported no febrile sensations and was in good general condition.

On somatic examination, the patient was eupneic, with room air saturation at 95%. She was tachycardic at 110 beats per minute. All other vital signs were normal. Urinalysis revealed proteinuria and hematuria. On respiratory examination, a liquid pleural effusion syndrome was noted. She also presented with non-scarring alopecia and erythematous, papular lesions of the face and outer surface of the hands, and the rest of the clinical examination was normal. A chest X-ray taken on admission showed an elevation of the right diaphragmatic dome (Figure 1). Thoracic ultrasonography showed a medium-sized anechoic fluid effusion on the right (Figure 2).

Investigation of the pleural fluid on the right revealed exudative pleurisy with lemon-yellow fluid and lymphocytic predominance, sterile bacteriological culture and non-specific pleural biopsies. Pleural fluid was positive for anti-nuclear antibodies and anti-DNA.

At this stage, autoimmune origin was immediately the most likely given the patient's articular and renal signs, but other etiologies, notably infectious, neoplastic and vascular, were possible but less likely.

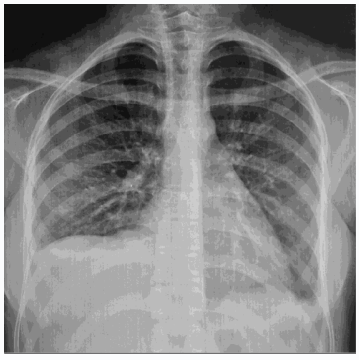

Figure 1: Chest X-ray.

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CBC showed normochromic normocytic regenerative anemia with hemoglobin 9g/dl and positive direct Coombs test, lymphopenia 500 elements/m^3, and retroviral serologies were negative. The blood immunoassay showed the following results:

- Anti-nuclear antibodies: **1200** (homogeneous, mottled appearance)

Native anti-DNA antibodies:

- Anti-soluble nuclear antigen antibodies (anti-ECT): **RNP, SSA, Sm: strongly positive**
- Anti-cardiolipin antibodies: **IgM: positive at 20.9**, IgG: negative
- C3 and C4: **low**

At the end of the renal work-up, an impure nephrotic syndrome was identified, with proteinuria at 4.4 g/l, Addis count at 40,000/min and hypoalbuminemia at 21g/l. Renal biopsy revealed segmental and focal proliferative glomerulonephritis.

The electrocardiogram was normal and cardiac echocardiography showed a pericardial effusion slide.

Applying the diagnostic criteria for systemic lupus erythematosus, the patient has an EULAR 2019 score of 46 points in front of the following criteria:

- Leukopenia (3 points)
- Hemolytic anemia (4 points)
- Non-scarring alopecia (2 points)
- Acute lupus skin disease (6 points)
- Pleural effusion (5 points)
- Proteinuria > 0.5 g/24h (4 points)
- Class 3 or 4 lupus glomerulopathy (10 points)
- Anti-cardiolipin antibodies (2 points)
- C3 and C4 reduced (4 points)
- Anti-DNA or anti-Sm antibodies (6 points)

Applying the ACR 2022 criteria, the patient was found not to have antiphospholipid syndrome (APS). And at this stage, the diagnosis of lupus pleurisy indicative of systemic lupus erythematosus with pericardial, renal (class III), hematological and cutaneous involvement was retained.

After a normal pre-therapeutic workup, corticosteroid therapy was indicated with an initial dose of 1mg/kg/d in combination with immunosuppressive treatment, notably mycophenolate mofetil (MMF): 2 g/d for 3 months with drying of pleurisy and pericarditis in the third month and persistent proteinuria.

In view of the severe renal impairment, MMF was replaced by Endoxan (600 mg/15 days) with a marked improvement in renal impairment.

Progression at 6 months was marked by regression of proteinuria and complete drying of the pleurisy (Figure 3).

Discussion

Systemic lupus erythematosus is an autoimmune disease resulting from a multifactorial disturbance of the immune system and responsible for systemic disorders (respiratory, cardiac, cutaneous, renal, hematological, etc.).

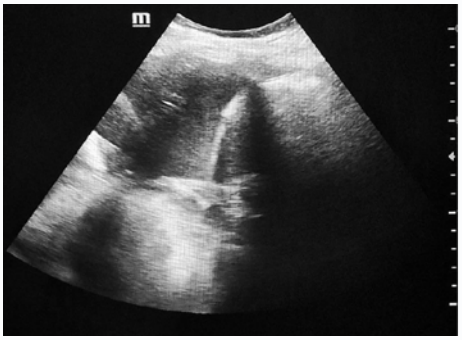

Figure 2: Thoracic ultrasound.

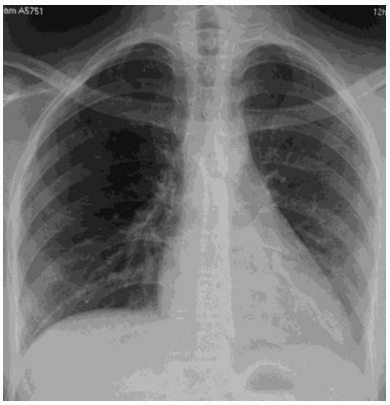

Figure 3: Chest X-ray at 6 months.

Pleural involvement in systemic lupus erythematosus is frequent. It represents a poor prognostic factor in the evolution of this pathology and has been part of its diagnostic criteria since the 1971 primary classification of systemic lupus erythematosus [1].

The worldwide incidence of systemic lupus erythematosus ranges from 0.3 to 23.7 per 100,000 inhabitants per year [2], and in Morocco one study estimates it at 6.25 per 100,000 inhabitants per year [3].

Respiratory diseases in lupus are numerous and frequent [1]:

- Pleural involvement: 45-60
- Pulmonary parenchymal damage:
- Acute lupus lung disease: 2 - 9%.
- Diffuse infiltrative lung disease: 3-9%.
- Diffuse alveolar hemorrhage: 0.5 to 5.7% (high mortality: 50-90%)

Pleural involvement is the most frequent and is associated with certain risk factors such as age of diagnosis > 30 years, lower respiratory tract infections, presence of other systemic manifestations, hypocomplementemia (C3 and/or C4) and high antibody titres [2].

Pleural involvement may be specific or nonspecific, of infectious origin given the terrain, iatrogenic or neoplastic through the chronic inflammation triggered by systemic lupus erythematosus.

Among specific pleural disorders, lupus pleurisy is the most common. It may be asymptomatic and revealed by radiological examination. It represents a diagnostic criterion for SLE. Pleural fluid study usually shows a predominantly lymphocytic or neutrophilic exudate, with glucose > 60 mg/dl and LDH <500 ui/L, low C3 and C4 levels and the presence of Hargraves cells, which are not specific for SLE, and may be found in the setting of rheumatoid arthritis, scleroderma or chronic hepatitis [4].

The anti-nuclear antibody assay is also not specific to SLE. High antibody titres can be found in other pathologies, notably transudative and neoplastic pleurisy.

Adenosine deaminase, a ubiquitous enzyme involved in the metabolism of purine bases and widely found in T lymphocytes, monocytes and macrophages activated during inflammatory processes, is also non-specific. Studies have shown positive ADA levels in tuberculous pleurisy, infectious pleurisy, lymphoma-associated pleurisy and certain autoimmune diseases [5].

Treatment is based on non-steroidal anti-inflammatory drugs, hydroxychloroquine, corticoids and immunosuppressants. Pleural symphysis is rarely indicated, given the high sensitivity to corticoids. Progression is generally favourable, with sequelae of fibrothorax in rare cases.

There are other specific pleural disorders, notably of vascular origin relating to lupus or antiphospholipid syndrome (APS).

Conclusion

Pleural involvement in systemic lupus erythematosus (SLE) is common, and can sometimes be the first symptom of SLE, revealing other conditions with a poorer prognosis, such as renal involvement. These conditions may be specific or non-specific. An infectious origin must always be ruled out first, given the dysimmune context created by the therapy or by the lupus. Treatment depends on the etiology of pleural involvement.

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