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Clinical Case Reports

Intra-Alveolar Hemorrhage Revealing Systemic Lupus Erythematosus

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Summary:

Lupus erythematosus is an autoimmune disease characterized by the production of antinuclear antibodies. It can be accompanied by pleuropulmonary manifestations, notably intra-alveolar hemorrhage, which is life-threatening.

We report the observation of a young patient, 24 years old, with no previous history, active smoker at 5P/A who presents with acute respiratory distress associated with hemoptysis with bilateral ground glass opacities. The clinical examination is unremarkable. The assessment inflammatory laboratory is positive, the blood count shows anemia and thrombocytopenia. An infectious origin is ruled out by bascilloscopies and in particular PCR. The immunological assessment came back positive in favor of SLE. The diagnosis of IAH was retained. Treatment was initiated with boluses of corticosteroids and immunosuppressants.

Conclusion:

IAH is a serious and fatal complication rarely complicating SLE. It constitutes a therapeutic emergency leading to acute respiratory failure and death.

Keywords: LES, HIA, respiratory distress

Introduction:

Lupus erythematosus is an autoimmune disease characterized by the production of anti- nuclear antibodies [1], which can be accompanied by pleuropulmonary manifestations, notably intra-alveolar hemorrhage, which is life-threatening [2].

Observation :

Young patient aged 24, hospitalized for moderate hemoptysis lasting three days. With the progressive onset of asthenia associated with respiratory discomfort. There was no fever, weight loss or extrarespiratory signs.

On admission, the patient was pale, dyspneic FR :25cycles /min, a suprasternal pull with desaturation in ambient air Spo2:85%. Pleuropulmonary auscultation finds a vesicular murmur audible in both pulmonary fields without crackling rales. Cardiac auscultation ; pace regular rapid HR: 130 beats / min without added noise or signs of right heart failure. Frontal chest x-ray revealed bilateral alveolar syndrome (fig 1).

Biological assessment: hyperleukocytosis GB 16.90 103 elements/mm3 with neutrophilic polynucleosis, lymphopenia, associated with

hypochromic microcytic anemia at 7.6 g/dl with thrombocytopenia at 22,000/mm3. Renal and hepatic functions were normal as was the hemostasis assessment.

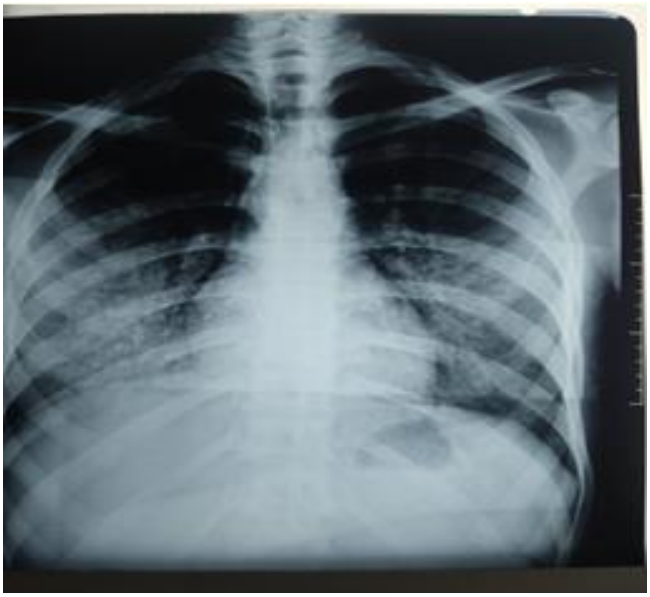

Figure 1: a front chest x-ray showing bilateral alveolar syndrome

The microbiological research was negative, as was the search for the COVID 19 Virus by PCR and HIV serology. The D-Dimer level was normal at 800. The chest CT scan showed ground glass opacities at the level of the parenchymal windows associated with intralobular non-septal thickenings extended to both pulmonary fields without regional predilection more marked on the right associated with centrilobular nodules suggesting an intra-alveolar hemorrhage. The mediastinal windows did not reveal lymphadenopathy in the thymic and Baréty compartments (fig 2). Echocardiography found good left ventricular function, the absence of mitral stenosis therefore excluding a cardiac origin. A fibroscopy with bronchoalveolar washing to confirm the hemorrhage supplemented with bacteriological samples could not be carried out due to the worsening of respiratory distress. In order to eliminate vasculitis although there were no disturbances in urinary function with absence of proteinuria and hematuria, an assay of ANCA and anti-glomerular basement membrane antibodies was carried out. but negative income. On the other hand, the rate of antinuclear antibodies (ANN) was positive at 1200 with anti nDNA Ab positive, anti-nucleosome Ab positive and anti-histone Ab positive. The diagnosis of systemic lupus erythematosus was made and treatment with corticosteroids was started at high doses.

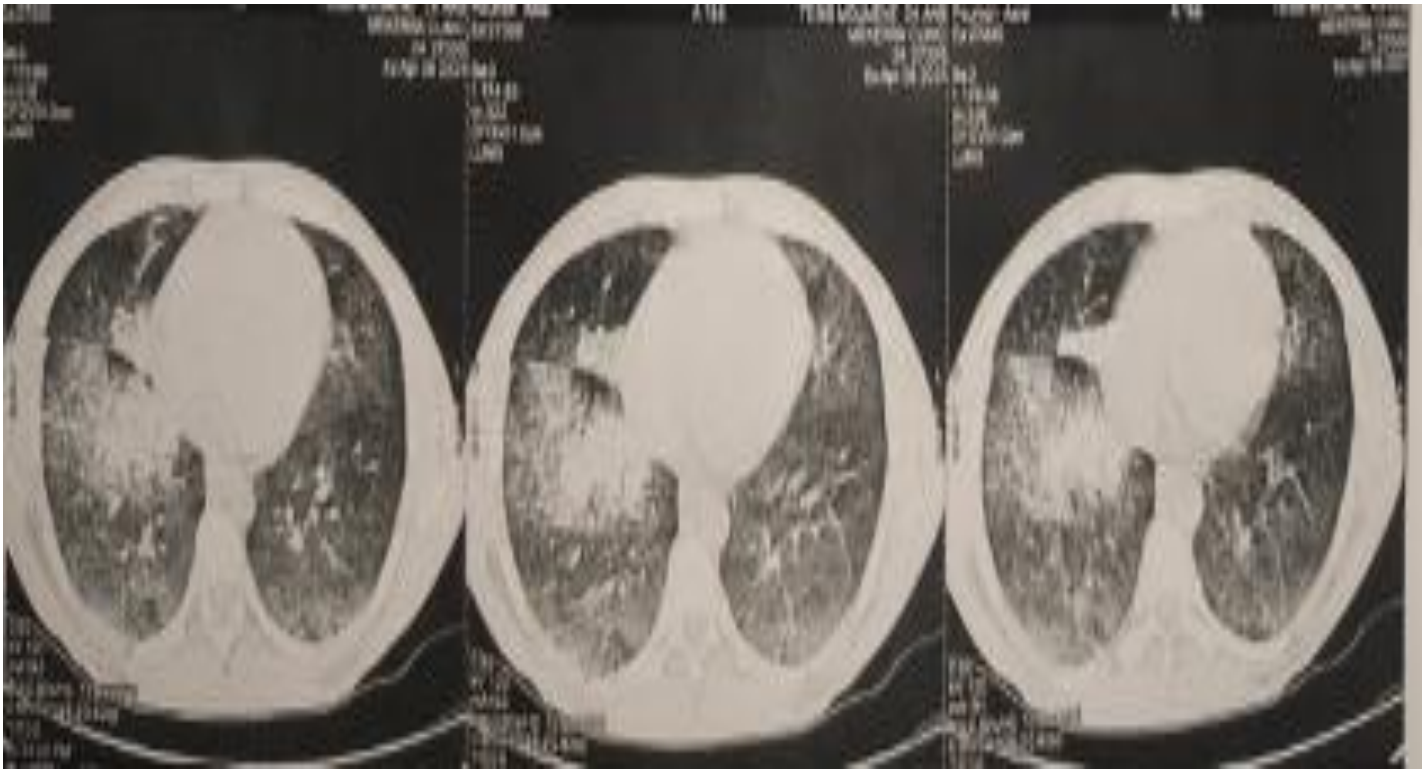

Figure 2: Thoracic CT: polished glass appearance of bilateral seat suggesting intra-alveolar hemorrhage

Discussion:

Intraalveolar hemorrhage is a rare but serious pathology that can be life-threatening.

clinical arguments, imaging but especially on bronchoalveolar washing.

The diagnosis of IAH is considered in the face of the classic clinical triad associating dyspnea, hemoptysis and anemia, with radiological pulmonary infiltrates. Each of these signs may be missing. Hemoptysis, rarely abundant due to its distal nature, is only present in two thirds of cases [3], is the cause of acute respiratory failure Bronchial fibroscopy is the key examination, making it possible to eliminate a local cause of bleeding or an infectious origin but also to confirm the diagnosis of the ' HIA by bronchoalveolar washing (BAL). Taking the hemorrhagic or pinkish appearance of the fluid in particular the GOLD score. The cytological examination of the BAL shows the presence of countless red blood cells. Siderophages are often present, but may be missing, particularly in cases of acute and recent IAH (less than 72 hours) [4]. Bronchial endoscopy was not performed in our case, given the importance of respiratory distress.

In our patient the diagnosis of IAH was made based on clinical and radiological arguments.

, with or without autoimmune origins. Immune causes include ANCA vasculitis: granulomatosis with polyangiitis and connective tissue disease and mainly systemic lupus erythematosus. The diagnostic approach is based on examination. clinical and in particular the search for extra-respiratory signs such as renal damage causing pneumorenal syndrome. Confirmation of the diagnosis was obtained by immunological assessment [5,6]. Furthermore, it is necessary to eliminate other non-immune etiologies such as pulmonary embolism, cardiac overload, overdose of anticoagulants, a neoplastic cause, infectious origin including leptospirosis and finally idiopathic hemosiderosis [7].

In our patient, after eliminating non-immune causes by history, cardiac ultrasound, bacteriological and biological assessment, we focused on an autoimmune cause. Indeed, the immunological assessment came back positive in favor of systemic lupus erythematosus. It is a systemic disease whose severity is linked to certain severe visceral damage (kidney and central nervous system) [1]. Pulmonary damage is frequent and varied and can occur during the evolution of SLE or reveal it. Their prevalence is 20-90% according to studies [8-9]. Among these respiratory manifestations, pleurisy, non-specific interstitial pneumonia and intra-alveolar hemorrhage (IAH). IAH was first described by Osler in 1904 [2]. It is a rare complication with a prevalence between 1-5.4% but potentially serious with a mortality rate of 50-95%[10] L HIA generally occurs during the evolution of lupus disease for which the diagnosis has already been established, with an average duration of 1.8 to 4.5 years[10] As it can be precipitated by high doses of corticosteroids during an attack. renal. However, it is indicative of the disease in 20% of cases [10], which makes its diagnosis more difficult. Lupus HIAs have the particularity of often very rapid onset within a few days [11] as in our patient while the HIA of vasculitis develops over one to several weeks.

The occurrence of IAH is linked to lesions such as capillaritis and microangiitis [10]

The treatment of lupus IAH is not well codified. Corticosteroid therapy remains the standard treatment, administered intravenously in high doses by bolus of 15 mg/kg for 3 days without exceeding 1 g with a relay of 1 mg/ kg at least during the first week, associated with immunosuppressive treatment such as cyclophosphamide. Plasmapheresis whose undeniable effectiveness is reserved for corticosteroid forms resistant. The spontaneous evolution of lupus IAH is unfavorable marked by death occurring during the first 48 hours from the onset of symptoms. However, early diagnosis and adequate therapeutic management can reduce the mortality rate by 20%.

Conclusion:

Lupus intra-alveolar hemorrhage is a rare but very serious complication. It is a therapeutic emergency. because it can quickly lead to fatal asphyxiating

acute respiratory failure in the absence of early and appropriate treatment.

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