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Case Report **Hepato-Gastro-Enterology & Proctology**

Acute Hepatopancreatitis Revealing Systemic Lupus Erythematosus: About A Case

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Abstract

Digestive manifestations during systemic lupus erythematosus (SLE) are rarely reported in the literature, in particular, hepato-pancreatic manifestations. We report the case of a patient in whom SLE was revealed by acute hepato-pancreatitis with mixed acute hepatitis, acute pancreatitis fulfilling 2 diagnostic criteria (high lipasemia and Balthazar stage C pancreatitis on the abdominal CT scan). The diagnosis was retained on the ACR criteria. The evolution under corticosteroid therapy and resting of the digestive tract was favorable.

Keywords: systemic lupus erythematosus (SLE), hepato-pancreatitis, cholestasis, prothrombin level.

INTRODUCTION

SLE is a multisystem disease that primarily affects women. The diagnosis of SLE can be made if at least 4 of the 11 ACR/EULAR 2019 criteria are met [1].

SLE can affect any part of the digestive tract, from the oral mucosa to the rectum. Gastrointestinal symptoms are thought to be present in more than 50% of patients with SLE at some point in the course of the disease. [3] These manifestations attract much less attention than the other major organs. As we can see, they have been rarely reported in the literature [2, 3].

SLE remains a very rare cause of acute pancreatitis [4, 5]. As for acute hepatitis, it above all poses an etiological diagnostic problem in order to distinguish lupus hepatitis from an associated autoimmune disease.

We report here the case of a 44-year-old patient in whom lupus disease was revealed by acute hepato-pancreatitis.

OBSERVATION

Mrs. HB, 44 years old, admitted to our department for assessment of acute hepato-pancreatitis discovered fortuitously during her stay in intensive care for a state of septic shock with a pulmonary starting point. She has a history of rheumatoid arthritis on Methotrexate stopped for 3 years, then put on Prednisone at 5 mg/day with anarchic intake, and notion of 2 miscarriages.

The clinical examination revealed conjunctival sub-jaundice, erythrosis of the face in butterfly garlic, alopecia, mouth ulcers and a state of anasarca made of edema of the lower limbs, a syndrome of pleural effusion and ascites.

The biological assessment revealed mixed hepatitis with cytolysis (Transaminases 12 times the normal rate) and cholestasis (Total bilirubin three times the normal rate, Gamma-Glutamyl transferase - GGT - 100 times the normal rate, Alkaline Phosphatases 4 times the normal rate), a normal Factor V and hypoalbuminemia. 20 times normal lipase.

Hematologically, we had a prothrombin level of 100%, normochromic normocytic hemolytic anemia with hemoglobin at 11.7 g/dl, high LDH and low Haptoglobin and lymphopenia. And finally, a positive 24-hour proteinuria.

Abdominal ultrasound was performed, showing no dilatation of the intra or extra-hepatic bile

ducts, or lithiasis. On the other hand, hepatomegaly was observed.

Abdominal CT angiography revealed an appearance of Balthazar's grade C edemato-interstitial pancreatitis with a severity index calculated at 2.

Hepatic viral serologies A, B, C were negative. Positive CMV and HSV serologies with an undetectable viral load. Complement C3, C4 were collapsed. The autoimmune assessment had objectified positive antinuclear autoantibodies with mitosis > 1/160, anti-centromeres, anti-mitochondria, anti-parietal cells, and anti-endoplasmic reticulum were negative.

As for the first-line etiological assessment of acute pancreatitis, including the anamnesis, the lipid and phosphocalcic assessment and the search for the lithiasis cause, came back negative. Amyloidosis was ruled out by performing a salivary gland biopsy.

The diagnosis of systemic lupus erythematosus was thus made according to the ACR criteria (fever, arthritis, pleural effusion, skin involvement, positive proteinuria, hemolysis of autoimmune origin, leukopenia, low C3 and C4).

Faced with the picture of acute pancreatitis, the patient was kept on an empty stomach, physical means for the fever, thromboprophylaxis was started and corticosteroid therapy at a rate of 1 mg/kg/d associated with albumin therapy, with good biological and clinical evolution.

DISCUSSION

The occurrence of cytolytic hepatitis is frequent during lupus disease, reported in 25 to 50% of cases [6]. It may be a specific lupus attack, but other causes that may require specific treatment must be ruled out.

Acute pancreatitis (AP) is a rare complication of SLE, occurring in 0.8% of patients screened for pancreatitis and 8% of patients with abdominal pain [7, 8].

Hepatitis and pancreatitis all have a similar female predominance with a wide range of age distribution [3].

The occurrence of acute hepatitis concomitant with pancreatitis is uncommon and generally related to viral infection A, B, E or CMV [9, 10]. In a patient infected with HIV, drug toxicity is often the cause [11]. The association of acute hepatitis and lupus pancreatitis has been reported only exceptionally [12, 13].

Liver involvement in SLE usually occurs during a disease flare and is often asymptomatic, limited to biological disturbances [14, 15]. Clinically, patients with lupus hepatitis may present with fatigue, malaise, anorexia and nausea. Jaundice, hepatomegaly, and splenomegaly can be observed on physical examination [16]. Biologically, we can observe an increase in transaminases, Alkaline Phosphatases (ALP), GGT, bilirubin, sedimentation rate, C-reactive protein (CRP) and serological markers of Lupus. Low levels of complement C3, C4 and positive autoantibodies to Ribosomal P antigen in some patients [17, 18].

Lupus hepatitis is therefore a combination of diagnostic criteria for SLE, biological abnormalities, with or without morphological abnormalities. Provided that all other causes of liver disease are excluded, including alcohol, hepatotoxic drugs, viral, autoimmune and metabolic causes. Because lupus hepatitis is essentially a diagnosis of elimination [19].

Histologically, SLE can cause chronic infiltration of inflammatory cells in the liver leading to fibrosis [20-22]. Although cirrhosis of the liver has been reported in patients with lupus hepatitis [17, 23, 24], it is rather a rare complication.

Lupus hepatitis responds well to systemic corticosteroid therapy [25]. Azathioprine has been used as a steroid-sparing agent to prevent relapses, especially in chronic active hepatitis or in people with high ALT levels at diagnosis [26]. Mycophenolate Mofetil can be considered in cases of lupus hepatitis refractory to Tacrolimus, Azathioprine and Cyclophosphamide [27].

Pancreatic involvement, on the other hand, generally occurs within the first 2 or 3 years after the diagnosis of SLE [28]. The etiopathogenesis of lupus-associated pancreatitis remains unknown [30] and is thought to be multifactorial [30-32].

Abdominal pain was the most common symptom of SLE-related AP, being present in almost all cases. Additionally, more than half of the patients reported nausea, vomiting, and fever. Laboratory parameters related to the diagnosis of pancreatitis or its complications revealed elevated amylase and lipase levels, supporting the diagnosis of AP. Levels of hypocalcaemia and hypertriglyceridaemia varied across studies. Around 50% of patients may present with hypoalbuminemia. Anemia of various mechanisms was observed in up to three quarters of the patients included. In addition, 20% to about 50% of patients had thrombocytopenia. Along with disease activity, complement consumption was observed in virtually all cases.

For the positive diagnosis approach, any other known etiology of pancreatitis must be excluded, such as cholelithiasis, neoplasia, trauma, a toxic cause (drugs, alcohol) [33], other viruses (human immunodeficiency virus, viral hepatitis, etc.) or

bacteria. As well as the search for anti-smooth muscle, anti-liver-kidney microsome or anti-mitochondria antibodies [34].

The relationship between corticosteroids and pancreatic lesions in SLE is not clearly established [32] and there are no specific recommendations for glucocorticoids [35]. However, there are studies that do not consider BP as a side effect of corticosteroids [36] that the outcome of SLE-related pancreatitis is favorable with glucocorticoids [33]. In terms of dosage, high doses [33] or even pulsed steroid treatment [30] have been considered in cases of SLE-related pancreatitis [37] and have also been associated in some studies with a better prognosis [29]. It should be noted that one study reported good results of plasmapheresis [32] and therefore this approach could be considered for SLE-related pancreatitis.

Reported mortality for SLE-related pancreatitis is high, even up to 37% [38].

CONCLUSION

Isolated hepatic and pancreatic damage to the neck of SLE is rare, and exceptional when associated.

The diagnosis is based on a range of arguments and remains essentially a diagnosis of elimination. The treatment is mainly based on corticosteroids while taking into account their possible harmful effect on pancreatic damage reported in the literature.

The association of pancreatic involvement with hepatic involvement during SLE makes the prognosis reserved.

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