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RESEARCH ARTICLE

SEVERE ACUTE PANCREATITIS REVEALING SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

S. Tougar, K. Zirhirhi, S. Alaoui, C. Quesbaoui and B. Charra
Department of Intensive Care Medicine, Ibn Rochd University Hospital, Faculty of Medicine and Pharmacy of Casablanca, Hassan 2 University, Casablanca, Morocco.

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Abstract

Systemic lupus erythematosus (SLE) is a systemic autoimmune disease that can affect multiple organs. Pancreatic involvement, although rare, has an estimated frequency ranging from 0.7% to 4.2%. It typically occurs in advanced stages of the disease but can exceptionally be an inaugural manifestation, as in the case reported here.The patient was a 24-year-old woman with no prior medical history, admitted for acute respiratory distress associated with abdominal pain. The diagnosis of acute pancreatitis was confirmed by a marked increase in serum lipase levels and an abdominal CT scan showing a swollen pancreas with peripancreatic collections (stage E according to Balthazar).After ruling out common causes of pancreatitis, immunological testing revealed positive antinuclear and anti-DNA antibodies, as well as decreased C3 and C4 complement fractions. The presence of pleural and pericardial effusions, combined with biological signs of active lupus, led to the diagnosis of SLE based on the 2019 ACR/EULAR criteria.

Introduction:-

Systemic lupus erythematosus (SLE) is a non-organ-specific autoimmune disease characterized by the production of anti-native DNA antibodies, sometimes associated with the secretion of antiphospholipid antibodies. The lack of organ specificity of these antibodies explains the protean nature of the disease.[1,2]

Pancreatic involvement in systemic lupus erythematosus is rare. Initially described in 1939,[3] its incidence ranges from 0.4 to 1.1 cases per 1,000 lupus patients annually.[4] Not only is it rare, but it is exceptionally an inaugural manifestation. Its pathogenesis remains mysterious and poorly understood.[5]

We report the case of a young woman with no notable medical history, hospitalized for acute gastroenteritis associated with respiratory distress. Acute pancreatitis, diagnosed during her hospital stay, was found to be the initial manifestation of SLE.

Observation:-

This is a 24-year-old female patient with no particular medical history and no known consumption of alcohol or tobacco, admitted to the medical intensive care unit for acute respiratory distress associated with abdominal pain. Her illness began 3 days prior to admission with epigastric and periumbilical pain associated with bilious vomiting and a history of chronic arthralgia in small and medium-sized joints of an inflammatory nature. The symptoms

Corresponding Author:- S. Tougar
Address:- Department of Intensive Care Medicine, Ibn Rochd University Hospital, Faculty of Medicine and Pharmacy of Casablanca, Hassan 2 University, Casablanca, Morocco.

progressed in a context of low-grade fever and general condition impairment. The condition worsened with increased pain intensity and the onset of resting dyspnea, prompting her visit to the emergency department.

At admission, the patient was conscious, with a Glasgow score of 15/15, symmetrically reactive pupils, and no sensory or motor deficits. Hemodynamically, she presented with sinus tachycardia at 120 bpm, normal blood pressure at 112/65 mmHg, a mean arterial pressure (MAP) of 68 mmHg, and preserved urine output. Respiratory examination revealed tachypnea at 28 breaths per minute without signs of respiratory distress, and SpO_2 was 87% on room air. Pulmonary auscultation showed absent breath sounds and decreased vocal fremitus on the left side. Abdominal examination found a soft abdomen with diffuse tenderness accentuated in the epigastric area and lower limb edema. The rest of the clinical examination was unremarkable.

Biological investigations revealed normochromic, normocytic anemia (hemoglobin 8.8 g/dL, mean corpuscular volume [MCV] 92.7 fL, mean corpuscular hemoglobin concentration [MCHC] 32.8%) with a reticulocyte count of 23,200/mm³ and a positive Coombs test. White blood cell count was 5,390/mm³ with neutrophils at 3,410/mm³ and lymphopenia at 650/mm³. LDH was elevated at 666 IU/L. Renal assessment showed nephrotic syndrome with hypoalbuminemia (21 g/L), hypoproteinemia (52 g/L), and positive 24-hour proteinuria (1.13 g/L) but no renal failure or hematuria. Pancreatic enzymes were significantly elevated, with lipasemia>300 IU/L (more than 5 times the normal limit). Inflammatory markers showed an elevated CRP at 96 mg/L. Liver function tests revealed hepatic cytolysis (AST 215 IU/L, ALT 101 IU/L), elevated gamma-GT (417 IU/L), and alkaline phosphatase (140 IU/L). Total bilirubin was normal at 12 mg/L, conjugated bilirubin at 1.9 mg/L, and unconjugated bilirubin at 10.1 mg/L. The lipid profile was normal.

Radiological assessment included an abdominal ultrasound, which showed a normal-sized liver with no dilatation of intrahepatic or extrahepatic bile ducts. The gallbladder contained finely echogenic material with a thin wall. An abdominopelvic CT scan revealed a swollen pancreas measuring 36.3 mm in thickness at the caudal level with liquid-density peripancreatic collections and thickened peritoneal layers, consistent with Balthazar grade E pancreatitis. An MRCP found no detectable obstruction.

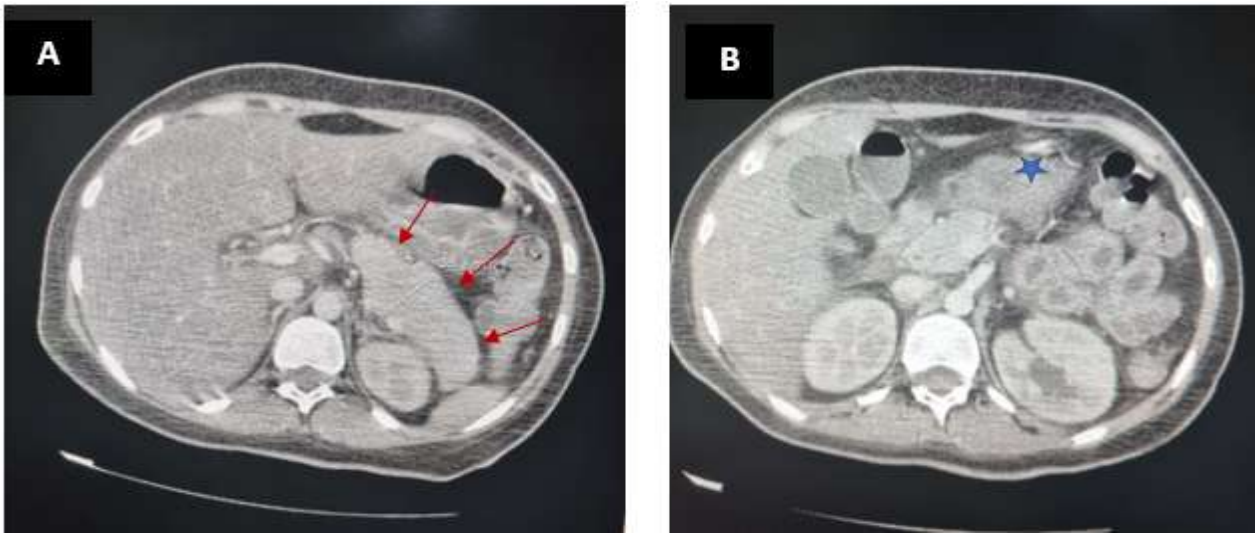

Figure 1:- Transverse section of the abdominal CT scan.
A: Swollen pancreas with caudal thickening (red arrows).
B: Peripancreatic collections (blue star), classified as stage E according to the Balthazar classification.

The patient was managed with symptomatic treatment for acute pancreatitis, which included bowel rest and pain relief using NEFOPAM 20 mg administered intravenously three times daily. The possibility of lupus-associated pancreatitis was considered after excluding other common causes, such as lithiasic, toxic (medications or alcohol), traumatic, and neoplastic causes. Immunological testing revealed positive antinuclear antibodies at 620 UI/ml with a homogeneous appearance, positive anti-DNA antibodies at 80 UI/ml, and decreased levels of complement fractions C3 and C4 at 0.5 g/L (normal range: 0.65–1.45 g/L) and 0.006 g/L (normal range: 0.1–0.4 g/L), respectively. The remaining immunological tests were negative.

Due to respiratory deterioration, a thoracic CT scan was performed and showed bilateral pleurisy, abundant on the left side and minimally present on the right. An echocardiogram revealed a circumferential, low-volume pericardial effusion. The pleural fluid analysis showed serofibrinous and lymphocytic nature (Light's score of 0.67, greater than 0.5). The infectious cause was ruled out through appropriate microbiological sampling. A complete evacuation of the abundant pleural effusion on the left side, which was causing respiratory distress, drained 1.5 liters of yellowish fluid.

Following consultation with internal medicine specialists, the diagnosis of systemic lupus erythematosus (SLE) with hematologic, renal, articular, pancreatic, and serous involvement was established based on the 2019 ACR/EULAR criteria, with a total score of 29 points and an SLEDAI activity score of 16 points:

1. Positive antinuclear antibodies at 640 with homogeneous fluorescence (6 points).
2. Autoimmune hemolysis (4 points).
3. Articular involvement (6 points).
4. Pleural and pericardial effusions (5 points).
5. Proteinuria exceeding 0.5 g/day (4 points).
6. Complement consumption (C3 and C4) (4 points).
7. Positive anti-DNA antibodies at 80 (6 points).

Immunosuppressive therapy was initiated after a complete sterile infectious workup. The patient received five days of methylprednisolone mini-pulses at 120 mg twice daily, followed by an oral taper at 60 mg/day.

The evolution was marked by clinical and biological improvement, including reduced lipase and CRP levels and regression of pericardial, pleural, and peritoneal effusions. The patient was referred to the internal medicine department for further management.

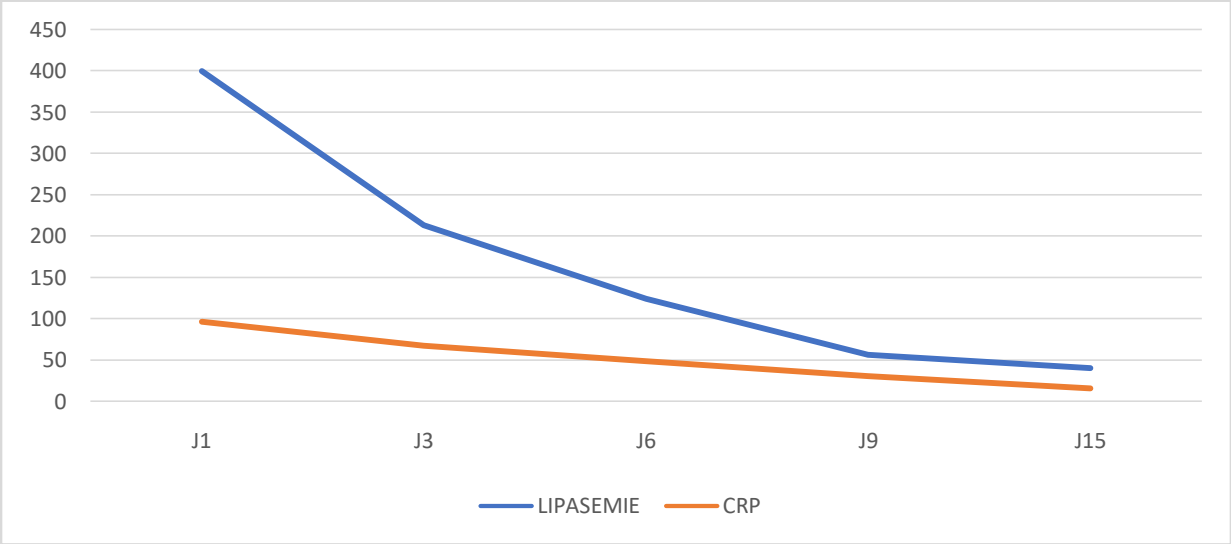

Figure 2:-Progression curve of lipase and CRP levels.

Discussion:-

During SLE, the frequency of gastrointestinal involvement is estimated to be around 30% of cases[6]. Acute pancreatitis has been reported with a variable frequency ranging from 0.7% to 4.2%, likely underreported[7]. In most published cases, pancreatitis occurs in patients with long-standing lupus disease, multiple visceral complications, and those on steroid, diuretic, or immunosuppressive treatments, whose role in the onset of pancreatitis is well known in the literature[8]. Although rare, pancreatitis has been known to reveal SLE in a few cases[9], as seen in this observation.

The etiology of pancreatic involvement in SLE is multifactorial and often difficult to pinpoint. Besides common causes of acute pancreatitis in non-lupus patients (biliary diseases, alcohol, medications, trauma, neoplasms, infections), proving a lupus-related origin is usually not possible, even through autopsy studies. It is more the

exclusion of other possible etiological factors and the improvement of symptoms under corticosteroids that indirectly supports a lupus-related cause[10]. Pathogenesis remains unclear, but vascular lesions are considered the most plausible mechanism[11]. Histological studies from autopsies have revealed mainly vascular lesions, including thrombosis and vasculitis[12].

In our case, the absence of typical causes of pancreatitis, along with clinical and laboratory signs pointing to lupus disease, allowed us to fulfill the diagnostic criteria of ACR/EULAR 2019, thus the improvement under corticosteroid treatment provided further evidence supporting the diagnosis of lupus-related pancreatitis.

In the context of SLE, the diagnosis of pancreatitis is generally based on clinical symptoms, predominantly abdominal pain reported in 88% of cases, which may radiate to the back in only 23% of cases, followed by nausea, vomiting, and a febrile syndrome, with diarrhea being less common[13]. Laboratory results, such as abnormal pancreatic enzyme levels and imaging findings, further confirm the diagnosis. However, there are rare cases of silent pancreatitis in which CT imaging alone can reveal the condition[14]. In our case, the symptoms were dominated by persistent epigastric pain, associated with nausea, vomiting, and watery diarrhea, occurring in a febrile context with fever reaching 38°C. Lipase levels were more than five times the normal range, and CRP was elevated to 96 mg/L. Abdominal CT revealed stage E pancreatitis according to the Balthazar classification.

The management of pancreatitis associated with SLE, like other causes of acute pancreatitis, involves pain control, intravenous rehydration—particularly during the first 24 hours, correction of electrolyte and metabolic imbalances, and dietary restrictions. While some studies have suggested that glucocorticoids may reduce mortality in acute pancreatitis in SLE patients, others limit the use of systemic glucocorticoids to patients with clear evidence of active SLE[15,16]. In this case, after establishing the diagnosis of lupus-related pancreatitis, corticosteroid therapy was initiated—first with a brief course of methylprednisolone followed by oral administration. The patient showed significant clinical and biological improvement.

Conclusion:-

Acute pancreatitis is a rare but potentially serious complication of systemic lupus erythematosus (SLE). Exceptionally presenting as a revealing manifestation of lupus, this case highlights the importance of considering SLE in the differential diagnosis of unexplained pancreatitis, especially in patients exhibiting evocative systemic signs.

Management relies on a multidisciplinary approach, combining symptomatic treatment of pancreatitis with immunosuppression tailored to lupus activity. This case also underscores the importance of regular follow-up to prevent complications and assess treatment efficacy. Early recognition and optimal management are crucial to improving the prognosis of patients with lupus-related pancreatitis.

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