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Acute Pancreatitis as the Initial Manifestation of Systemic Lupus Erythematosus: A Case Report

M. Aouroud[1*], Fz. Machayi[1], H. Aouroud[1], O. Nacir[1], Fz Lairani[1], A. Ait Errami[1], S. Oubaha[1,2], Z. Samlani[1], K. Krati[1]

[1]Department of Hepato-Gastroenterology, Mohammed VI University Hospital, Marrakech, Morocco
[2]Physiology Laboratory, Faculty of Medicine and Pharmacy, Marrakech, Morocco

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***Corresponding author:** M. Aouroud
Department of Hepato-Gastroenterology, Mohammed VI University Hospital, Marrakech, Morocco

Abstract | **Case Report**

Acute pancreatitis is an uncommon initial presentation of systemic lupus erythematosus (SLE). We describe the case of a 24-year-old woman hospitalized for acute epigastric pain suggestive of pancreatitis, along with fatigue and weight loss. After ruling out common causes and observing the development of pancytopenia and suggestive skin lesions, immunological testing led to the diagnosis of SLE. This case illustrates an unusual gastrointestinal onset of lupus and highlights the need for clinical vigilance in unexplained pancreatitis in young women.

Keywords: Acute pancreatitis, Systemic lupus erythematosus, Pancytopenia, Initial manifestation, Autoimmunity.

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INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic autoimmune condition characterized by diverse clinical manifestations, potentially affecting any organ. While gastrointestinal involvement is relatively rare, it can sometimes precede the diagnosis of SLE. Among such presentations, acute pancreatitis is infrequent but recognized, particularly in young females [1,2]. Potential underlying mechanisms include vasculitis, immune complex deposition, or microvascular thrombosis, especially when antiphospholipid syndrome is present [3]. We report a case of acute pancreatitis that revealed underlying SLE, followed by hematological and cutaneous manifestations.

CASE PRESENTATION

A 24-year-old female with no prior medical history presented with severe epigastric pain of four days' duration. The pain was deep, continuous, non-radiating, and associated with loss of appetite, marked fatigue, and unquantified weight loss.

On admission, her vital signs were stable. Abdominal examination revealed localized tenderness in the epigastric region without guarding or rigidity.

Laboratory results indicated elevated serum lipase levels (>3× the normal value), with no electrolyte imbalance or signs of organ dysfunction, fulfilling criteria for mild acute pancreatitis. Liver function tests revealed moderate transaminase elevation (AST 145 U/L, ALT 127 U/L), cholestasis (GGT 130 U/L, ALP 175 U/L), and total bilirubin at 9.1 mg/L. The prothrombin time remained normal (92%).

An etiologic assessment excluded alcohol use, gallstones, hypertriglyceridemia, hypercalcemia, and drug-induced causes. Abdominal ultrasound showed no biliary dilation or gallstones. MRCP identified focal hypertrophy of the left hepatic lobe with segmental enhancement but no signs of pancreatic or biliary obstruction.

Given the idiopathic nature of the pancreatitis, further evaluation included negative viral serologies (HAV, HBV, HCV, CMV, EBV) and immunological tests, including ANA, anti-dsDNA, and complement levels.

During hospitalization, the patient developed pancytopenia and unilateral brownish pruritic erythematous-squamous lesions on her left flank (Figure 1). Initially thought to be post-herpetic, a skin biopsy showed IgM and C3 deposits along the dermoepidermal junction, consistent with lupus dermatitis.

The immunologic panel confirmed SLE: ANA titer 1:640, high-titer anti-dsDNA antibodies, and low complement levels (C3 and C4). She was transferred to

internal medicine and received intravenous methylprednisolone pulses, resulting in a rapid clinical and biochemical improvement.

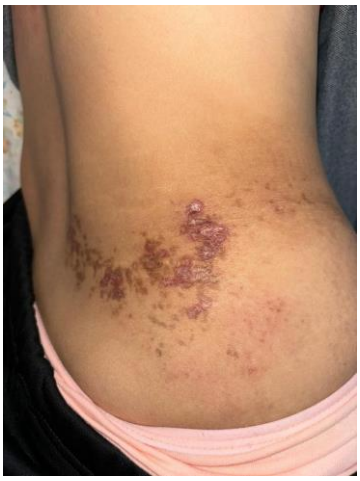

Figure 1: Healing brownish erythematous plaques on the left flank, consistent with cutaneous lupus.

DISCUSSION

Although rare, acute pancreatitis can occur in SLE, with reported incidence ranging from 0.4 to 1.1 cases per 1,000 patients annually [1]. While it may arise at any disease stage, it appears as the first symptom in 10–20% of cases [2].

The pathogenesis of lupus-related pancreatitis remains multifactorial. Possible mechanisms include vasculitis-induced ischemia of the pancreas [3,4], autoimmune injury to pancreatic tissue, or thrombosis related to antiphospholipid antibodies [5].

Standard causes of pancreatitis must be thoroughly excluded, including gallstones, alcohol, viral infections, hypertriglyceridemia, and drug-related toxicity [6]. In our patient, none of these common etiologies were found. The clinical setting and elevated lipase supported the diagnosis of acute pancreatitis of autoimmune origin.

The presentation of isolated epigastric pain without radiation was somewhat atypical. However, lupus-related pancreatitis may also manifest with classical symptoms like back pain, nausea, and vomiting [7]. Cross-sectional imaging, such as MRI, can help identify coexisting gastrointestinal involvement (e.g., mesenteric vasculitis or bowel ischemia) [8].

The secondary appearance of pancytopenia and cutaneous lesions was pivotal in the diagnostic approach. Pancytopenia affects 10–40% of SLE patients and can result from peripheral immune-mediated destruction, splenic sequestration, bone marrow suppression, or drug toxicity [9]. Histopathologic analysis of skin lesions helped confirm the autoimmune origin. Immunologic criteria (ANA, anti-dsDNA positivity, low C3/C4) met the 2019 EULAR/ACR classification standards [10].

Treatment involves standard supportive care for pancreatitis alongside immunosuppressive therapy. High-dose corticosteroids remain the first-line treatment [11]. In our case, methylprednisolone led to a rapid resolution of symptoms and biological anomalies.

In refractory or severe cases, additional immunosuppressants (e.g., mycophenolate mofetil, azathioprine, or cyclophosphamide) may be needed [12,13]. Biologic therapies such as belimumab have shown benefit in controlling active disease [14,15].

Prognosis depends on initial severity and timely initiation of therapy. Severe forms involving multiple organs carry a higher risk of mortality [16]. Long-term monitoring with repeated hematologic and immunologic assessment is essential [17].

This case reinforces the need to consider SLE as a possible etiology in unexplained acute pancreatitis in young women, especially in the presence of systemic signs. Prompt diagnosis and coordinated care are key to preventing complications [18].

CONCLUSION

Acute pancreatitis may, although rarely, be the initial indicator of systemic lupus erythematosus. In this patient, the absence of traditional causes, emergence of hematologic and dermatologic signs, and positive immunologic findings supported the diagnosis. This highlights the importance of investigating autoimmune causes in atypical pancreatitis, where early recognition and immunosuppressive treatment can be decisive in preventing severe outcomes. A multidisciplinary approach remains central to improving prognosis

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