



## Unusual Presentation of Systemic Lupus Erythematosus with Acute Pancreatitis: A Case Report

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**Abstract:** Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease affecting any organ with diverse clinical manifestations. Lupus-related acute pancreatitis (AP) is a serious cause of SLE-induced acute abdominal discomfort, along with lupus mesenteric vasculitis. Systemic Lupus Erythematosus (SLE) is a complex and chronic autoimmune disease characterized by its ability to affect virtually any organ system, resulting in a wide array of clinical manifestations. One particularly severe complication associated with SLE is lupus-related acute pancreatitis (AP), which can cause acute abdominal discomfort. Lupus mesenteric vasculitis is another abdominal manifestation of SLE, further highlighting the systemic nature of the disease. Lupus pancreatitis is more common in females and the third decade of life, with an incidence ranging from 0.7% to 4%. This case report describes a 16-year-old Saudi female who presented to the emergency department with episodic fever, epigastric abdominal pain, sweating, loss of appetite, and diarrhoea, among other symptoms. Her laboratory test results showed leukopenia, anaemia, increased liver, and pancreatic enzyme levels, increased inflammatory markers, and hypocomplementemia. She was diagnosed with lupus pancreatitis after ruling out other potential causes. Treatment included steroids and hydroxychloroquine. The patient showed marked improvement in resolving all symptoms, emphasizing the need for prompt diagnosis and management. This case insists on timely diagnosis and appropriate management in mitigating the potentially severe consequences of lupus pancreatitis and other SLE-related complications. In conclusion, SLE is a multifaceted autoimmune condition that affects various organs, including the pancreas, leading to conditions like lupus pancreatitis. The presented case serves as a poignant reminder of the diverse clinical presentations of SLE and highlights the crucial role of prompt diagnosis and effective management in improving patient outcomes.

**Keywords:** Systemic Lupus Erythematosus, Lupus Pancreatitis, Pancreatitis, SLE-related pancreatitis, Acute pancreatitis

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## I. INTRODUCTION

SLE is an inflammatory, chronic, autoimmune disease characterized by the development of various immune complexes, numerous autoantibodies, and the involvement of multiple organ systems with a wide range of clinical manifestations<sup>1</sup>. SLE most typically affects the musculoskeletal, integumentary, and renal system<sup>2</sup>. SLE patients also have gastrointestinal (GI) symptoms; however, severe pancreatitis is uncommon<sup>3,4</sup>. The disease, unfavorable treatment interactions, or opportunistic infections can all cause gastrointestinal tract (GIT) problems. The GIT is involved in SLE in a variety of ways, including mesenteric vasculitis (0.2–9.7%), protein-losing gastroenteropathy (1.9–3.2%), intestinal pseudo-obstruction (rare), and lupus pancreatitis (0.7–4%)<sup>5,6</sup>. Acute pancreatitis (AP), one of the most frequent gastroenterological emergencies globally and a primary cause of acute hospitalization and inpatient care in gastroenterology wards, provides a substantial burden for healthcare systems<sup>7</sup>. It is an acute pancreatic inflammatory disorder that can range in severity from mild, fast-recovery forms to fulminant presentations with organ failure and local and systemic consequences. The presence of two of the three criteria-clinical ("pancreatic type") abdominal discomfort, biochemical (elevated serum amylase and lipase greater than three times the upper limit of normal), and imaging-leads to the diagnosis (findings consistent with AP on abdominal imaging Guidelines)<sup>8</sup>. SLE patients may present with subclinical pancreatitis and increased pancreatic enzymes even in the absence of overt symptoms. Lupus-induced acute pancreatitis is usually diagnosed after ruling out other causes, such as obstructive acute pancreatitis (biliary stones, pancreas divisum), toxic-metabolic (alcohol, hypertriglyceridemia, hypercalcemia), or iatrogenic (drug-induced AP)-not last, considering that some of the lupus-related medications, such as azathioprine, cyclosporine can cause SLE-related pancreatitis<sup>10</sup>. If the doctor suspects lupus pancreatitis, steroids or other immune-modifying medications may be initiated once common causes of pancreatitis have been ruled out. Plasmapheresis and intravenous gamma-globulin infusions may also be beneficial in severe cases<sup>3,11</sup>. There have been reports of 10 to 50 cases of acute pancreatitis per 100,000 people per year in western countries. However, the Arab world and the Kingdom of Saudi Arabia (KSA), in particular, have produced remarkably few reports<sup>12</sup>. In this case report, we describe another unique case of lupus pancreatitis. Our case report will add valuable data to the existing literature.

## 2. CASE PRESENTATION

A 16-year-old Saudi female high school student who had no known chronic medical illnesses presented to the emergency department at King Khalid Hospital in Saudi Arabia with episodic fever for one month with a spike of 39°C on some days. The fever increases at night and is associated with epigastric abdominal pain, sweating, loss of appetite, diarrhoea that started five days ago, and generalized fatigue, which is preceded by an upper respiratory tract infection. On systemic review, she had a cough and bilateral knee and wrist joint pain and had normal vision. Her vital signs were a heart rate of 88 beats per minute, a blood pressure of 117/67 mmHg, a temperature of 36.8°C, and an oxygen saturation on room air of 98%. The abdominal examination revealed a soft and lax abdomen with epigastric tenderness. Musculoskeletal examination revealed that the knee was

bilaterally swollen and tender, as well as both wrists. Moreover, cardiopulmonary and neurological examinations were unremarkable.

### 2.1 Medical History

The fever increases at night. She denied any history of oral ulcers, hair loss, headache, palpitation, shortness of breath, chest pain, hematuria, or dysuria. She had frequent hospital visits due to fever but had not been hospitalized or undergone any surgery before. There was no history of any long-standing medication; however, she was taking paracetamol for fever. Her menstrual cycle is regular (28 days). She had no family history of chronic diseases, either congenital or acquired. On examination, she was conscious, alert, and cooperative, with an average body weight. Despite this, she was looking ill and pale with no signs and symptoms suggestive of jaundice.

### 2.2 Special tests and investigation

The laboratory test results were as follows: A complete blood count (CBC) showed leukopenia and white blood cell (WBC) levels of 3210 (normal range 4,500–11,000/L), anaemia (haemoglobin [Hb], 7.90 g/dL, normal range 13.5–17.5 g/dL), a positive direct Coombs test, and normal platelets (platelet [PLT] count, 188000/L; normal range, 150,000–450,000/L). Liver enzyme levels were increased (aspartate aminotransferase [AST], 235 IU/L, normal range 9–32 IU/L; and alanine aminotransferase [ALT], 56 IU/L, normal range 19–25 IU/L), and pancreatic enzyme levels were increased (amylase 264 U/L, normal range 30–110 U/L; and lipase 721.60 U/L, normal range 10–140 U/L), while creatinine and urinalysis results were normal. Inflammatory marker results were as follows: erythrocyte sedimentation rate (ESR) was increased (130 mm/hr, normal range 6–12 mm/hr); C-reactive protein (CRP) was 0.831. Additionally, hypocomplementemia was noted (complement 3 [C3], 0.195 g/L, normal range 0.8–1.6 g/L; C4, 0.038, normal range 0.16–0.31 g/L). Immunological test results were as follows: Immunofluorescence anti-nuclear antibody (ANA) results were positive; there was a homogenous pattern with a titer of 1/1280, and double-stranded deoxyribonucleic acid (dsDNA) results were positive with 734.8 U/ml. Other antibody test results, including lupus anti-coagulant, Anticardiolipin antibodies, Anti-beta-2-Glycoprotein-I antibodies, rheumatoid factor (RF), anti-smooth muscle (ASMA), anti-Smith, anti-histone, and cyclic citrullinated peptide (CCP) antibodies, were negative. Blood cultures and transthoracic echocardiography results were negative. COVID-19, hepatitis B and C, and human immunodeficiency virus polymerase chain reaction (PCR) results were also negative. Peripheral blood film showed no abnormal cells.

### 2.3 Diagnosis

She was diagnosed with lupus pancreatitis after ruling out other potential causes.

### 2.4 Treatment

The patient received an intravenous infusion of pulse methylprednisolone (1g) for 3 to 5 days, followed by a tapering regimen of oral prednisone with a starting dose of 40 mg daily in addition to hydroxychloroquine 200 mg daily. The patient's abdominal pain, fever, and other symptoms

were resolved four days later. Repeated lab tests improved the CBC, liver profile, amylase, and lipase levels (Table 1).

## 2.5 Follow up

She was discharged home after 1 week. The patient

**Table 1. Pre and post-treatment laboratory results show an improvement after treatment**

Investigation	Pre-treatment result	Post-treatment result (4 days later)	Reference range
White blood cell	3210	5980	4,500–11,000/ $\mu$ L
hemoglobin	7.9	8.3	13.5–17.5 mg/dL
Platelet	188000	269000	150,000–450,000/ $\mu$ L
Aspartate aminotransferase	235 IU/L	46 IU/L	9–32 IU/L
Alanine aminotransferase	56 IU/L	20 IU/L	19–25 IU/L
Amylase	264	95	30–110 U/L
Lipase	721.60	158.80	10–140 U/L

The values presented in the table represent the pre-treatment and post-treatment results of the investigation conducted. The reference ranges provided are typical values for the respective parameters in a healthy individual. Any values falling outside these reference ranges may indicate abnormality and should be further evaluated by a healthcare professional.

## 2.6 Treatment and Prognosis

Abdominal pain is a common symptom in SLE, which could be due to multiple reasons, from less sinister ones like gastritis to more serious ones like acute pancreatitis. The etiology of acute pancreatitis in a lupus patient is often difficult to ascertain<sup>13</sup>. Coexisting traditional risk factors for AP, including alcohol, gallstones, hypercalcemia, and hypertriglyceridemia, should be ruled out. In the absence of a definite risk factor, especially in the setting of active manifestations of lupus in other organs, the etiology of AP is ascribed to SLE. A laboratory test is critical for the diagnosis of acute pancreatitis in a lupus patient. Liver enzyme levels, pancreatic enzyme levels, erythrocyte sedimentation rate, C-reactive protein, hypocomplementemia, Immunofluorescence anti-nuclear antibody including lupus anti-coagulant, Anticardiolipin antibodies are Anti-beta-2-Glycoprotein-I antibodies are crucial for diagnosis<sup>14</sup>. Imaging helps in making a diagnosis of AP in the absence of typical pain or elevated enzymes. Abdominal ultrasound may show evidence of bulky pancreas, peripancreatic inflammation, and increased vascularity<sup>15</sup>. Ultrasound also helps identify other causes of AP, like gallstones, structural anomalies, and local complications like pseudocyst, ascites, and abdominal collections<sup>16</sup>. AP in lupus is usually treated with immunosuppression and supportive management of pancreatitis, although not supported by high-quality evidence. Steroids are used in varying doses depending on severity<sup>17</sup>. Azathioprine or cyclophosphamide are also used along with steroids. Glucocorticoid treatment was associated with a better prognosis for SLE patients with acute pancreatitis.

## 3. DISCUSSION

While the development of renal failure and nephrotic syndrome in lupus is well-documented and seen regularly, the presentation of this 16-year-old Saudi female was unusual in that abdominal pain and pancreatitis were prominent features in her history. Another similar case of SLE-related acute

pancreatitis following the COVID-19 vaccine in a 22-year-old Saudi female was also reported in 2022<sup>18</sup>. SLE-induced pancreatitis is uncommon, particularly in children. The yearly incidence in adults is estimated to be 0.4–1.1/1000 lupus patients<sup>19</sup>. This is a rare occurrence, with fewer than 100 examples reported<sup>3</sup>. A four-year multicenter study of 232 youngsters, involving 14 centers in the UK, found that just 0.5% of them seemed to have pancreatic involvement<sup>20</sup>. Reifenstein et al. appear to have described the first instance of lupus-related pancreatitis in 1939. In 2006, 19–21 published a review of the literature on lupus-related pancreatitis during the previous 30 years. They found 77 cases, 88% of which were women, with a median age of 27. Following that, 21 stated in his study of gastrointestinal involvement in lupus that a total of 160 cases of SLE-associated pancreatitis had been documented in the literature without going into detail regarding their characteristics<sup>21</sup>. Individuals with active lupus symptoms who have pancreatitis have a considerably greater mortality risk. In one study, individuals who did not exhibit SLE symptoms at the time of pancreatitis did not die. In contrast, SLE signs were associated with 40% mortality<sup>19</sup>, especially when paired with concomitant complications such as respiratory failure (22%), ascites (19%), chronic pancreatitis (5%), infection (18%), peritoneal hemorrhage (9%), acute renal failure (14%), circulatory shock (12%)<sup>19,20</sup>, secondary diabetes mellitus (6.3%) and exocrine pancreatic insufficiency (3.2%)<sup>22</sup>. Hence, whenever a patient with SLE complains of nausea, vomiting, and stomach pain, the astute physician must have a high index of suspicion for pancreatitis. Since the first report, the pathogenesis of SLE pancreatitis has been questioned. It is multifactorial, and several etiologies are frequently implicated, such as an ischemic mechanism caused by vasculitis or thrombosis caused by an associated antiphospholipid antibody syndrome, an autoimmune mechanism caused by corticosteroids and azathioprine, or an intercurrent infectious disease. Any other known etiology of pancreatitis, such as biliary lithiasis, neoplasia, trauma, toxic (drugs, alcohol), and further viral (human immunodeficiency virus, viral hepatitis, etc.), or bacterial infections, should be excluded. Anti-smooth muscle, anti-liver-kidney microsomal, or anti-mitochondrial antibodies should be investigated<sup>23</sup>. Similarly, in our patient, acute pancreatitis was related to SLE in terms of immunological manifestations with concomitant multiorgan manifestations after excluding frequent etiologies. Clinical manifestations of our patient are very similar to those reported in the literature based on abdominal pain and

are reported in most of the patients<sup>19,21</sup>. Elevating serum amylase and lipase levels is the most common laboratory abnormality in lupus pancreatitis (Tian & Zhang, 2010)<sup>17</sup>. In addition, other significant biochemical abnormalities noted were hypoalbuminemia (78%) and liver function test abnormalities (65%). Leukocytosis is relatively uncommon, occurring in only about 15% of the patients, whereas anaemia, leukopenia, and thrombocytopenia have been found in 81%, 59%, and 48% of cases in the literature, respectively<sup>19</sup>. Similarly, our patient had elevated serum amylase and lipase levels with hypoalbuminemia, anaemia, leucopenia, thrombocytopenia, and elevated liver function tests (AST and ALT). The positivity for ANA and anti-ds DNA serum titers confirmed SLE. Steroid treatment for acute pancreatitis in SLE patients is debatable due to steroid-induced toxic effects,<sup>24</sup> but this is viewed as a minor concern. A recent research outlining the hallmarks of corticosteroid-associated pancreatitis discovered 451 cases on a systematic review, with 5% of patients developing pancreatic injury within 3 days of starting moderate to high doses of corticosteroids (Dwivedi et al., 2019)<sup>9</sup>. Since steroids' immunosuppressive effect can enhance prognosis in patients with acute pancreatitis, new studies recommend using steroids during the acute episode of SLE-related pancreatitis.<sup>19,22,25</sup> One study found that plasma exchange therapy had a positive outcome; therefore, this strategy might be considered. Immunosuppression may also be harmful and should be avoided in SLE individuals who suffer from pancreatic damage<sup>26</sup>. Furthermore, several authors have observed a significant use of antibiotics during AP episode<sup>27</sup>, even though the current guidelines state that antibiotics are restrictive in acute pancreatitis<sup>28</sup>. The prognosis of AP can be fatal due to necrosis and hemorrhagic forms or specific complications<sup>29</sup>, such as ketoacidosis developed in some patients. In literature, even if an acute complication does not occur, patients develop severe uncontrolled diabetes<sup>30</sup>. To avoid such severe complications, some authors insist on the importance of the systemic screening of AP to make early diagnosis and treatment<sup>31</sup>. We believe that this case study provides a better understanding of SLE-related pancreatitis.

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and highlights the importance of timely diagnosis and management to prevent severe morbidity and mortality.

## 4. CONCLUSION

In conclusion, lupus-induced pancreatitis is a rare occurrence, particularly in children, and its presentation can be atypical. The literature reports several cases of lupus as a primary etiology for pancreatitis, although the exact pathophysiology is still uncertain. Since pancreatitis may be the earliest clinical symptom of SLE, lupus screening is suggested in individuals with idiopathic pancreatitis, particularly in younger females. It is important to appropriately treat these patients with high vigilance, as failure to do so has been demonstrated to increase mortality. Treatment of SLE pancreatitis is debatable, but steroids and other immune suppressants may improve the overall prognosis of lupus pancreatitis. Plasma exchange therapy has shown positive outcomes, and antibiotics should be used restrictively. Overall, this report highlights the importance of considering lupus pancreatitis as a potential diagnosis in patients with systemic lupus erythematosus who present with abdominal symptoms, even if there are no overt symptoms of pancreatitis, and highlights the need for further research in the Arab world regarding acute pancreatitis.

## 5. AUTHORS' CONTRIBUTION STATEMENT

All authors analyzed and interpreted data; also, they have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

## 6. INFORMED CONSENT

Informed Consent was obtained from the patient.

## 7. CONFLICTS OF INTEREST

Conflicts of interest declared none.

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