Case Report

Systemic lupus erythematosus-related acute pancreatitis: An uncommon clinical manifestation

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ABSTRACT

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease with diverse clinical manifestations, ranging from mild to life-threatening complications. Acute pancreatitis (AP) is a rare but serious manifestation of SLE, occurring in <2% of cases. SLE-related AP is often associated with high disease activity and can result in multiorgan dysfunction. We report the case of a 27-year-old female who presented with abdominal pain, joint pain, malar rash, pedal edema, and facial puffiness. Laboratory investigations revealed elevated serum amylase and lipase, with active SLE markers, including elevated antinuclear antibody and anti-dsDNA levels with hypocomplementemia. Contrast-enhanced computed tomography abdomen confirmed acute interstitial edematous pancreatitis. The patient was also diagnosed with lupus nephritis. A diagnosis of SLE-related AP with concurrent lupus nephritis was established. The patient was managed with pulse corticosteroid therapy, immunosuppressive agents, and supportive measures, leading to significant clinical improvement. SLE-related AP is a rare life-threatening complication with multifactorial pathophysiology involving autoimmunity, vasculitis, and complement activation. This case highlights the importance of recognising AP as a rare but serious manifestation of SLE. Early diagnosis and prompt immunosuppressive therapy are crucial for improving outcomes.

Key words: Acute pancreatitis, Autoimmune disease, Lupus nephritis, Systemic lupus erythematosus

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease that classically affects women of childbearing age and is characterized by autoantibody formation, deposition into tissues, and complement activation leading to end-organ damage [1]. It is characterized by multisystem involvement, with a wide spectrum of clinical manifestations ranging from mild to life-threatening complications. Common presentations include lupus nephritis, arthritis, and cutaneous lesions. Although gastrointestinal manifestations are common and are seen in about 50% of patients, involvement of the pancreas in the form of acute pancreatitis (AP) is rare, with an incidence of <2% in SLE patients [2]. AP in SLE is often associated with high disease activity and carries a poor prognosis due to its potential to progress to multiorgan dysfunction [3]. The etiology of SLE-related AP is multifactorial and can include direct autoimmune mechanisms, vasculitis, complement activation, or secondary causes such as medications, infections, or metabolic disturbances [4]. Recognizing SLE-related AP is critical, as it requires early initiation of immunosuppressive therapy alongside supportive measures, distinguishing it from other causes of pancreatitis.

This case highlights a young female presenting with an uncommon presentation of SLE-related AP complicated with lupus nephritis. It emphasizes the fact that in a young patient without any obvious cause of pancreatitis, autoimmune etiology should always be considered. It highlights the importance of teamwork, detailed diagnostic evaluation, and timely therapeutic interventions in managing such complex cases.

CASE DESCRIPTION

A 27-year-old female presented with complaints of multiple joint pain and hyperpigmentation of the face for 1 month, pedal edema for 15 days, and abdominal pain for 2 days. Joint pain was insidious in onset, gradually progressive, involving symmetrical joints, including the proximal interphalangeal joint, metacarpophalangeal joint, and elbow and knee joints, associated with early morning stiffness and partially relieved after activity. It

Access this article online	
Received - 26 March 2026 Initial Review - 10 April 2026 Accepted - 23 April 2026	Quick Response code
DOI: 10.32677/ijcr.v12i5.8192	

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was associated with hyperpigmentation of the face across the cheeks and bridge of the nose, sparing the nasolabial folds, non-pruritic and exaggerated after sun exposure (Fig. 1a). Pedal edema was insidious in onset, gradually progressive, followed by facial puffiness (Fig. 1b). She complained of abdominal pain for 2 days, acute onset, gradually progressive, in the epigastric region, radiating to the back, and relieved on bending forward. It was associated with nausea and abdominal distension, but no episodes of vomiting. She denied any history of prior cholelithiasis, alcohol abuse, abdominal trauma, recent medication changes, or the use of over-the-counter medications. There was no history of hematuria, burning micturition, jaundice, or shortness of breath. There was no significant past and family history.

On presentation, her vitals were recorded as blood pressure 110/80 mmHg, pulse rate 120 bpm, saturation 97% at room air, respiratory rate 18 breaths/min, and temperature 98.6°F. On clinical examination, she had pitting pedal edema, facial puffiness, and malar rash, sparing the nasolabial folds. On abdominal examination, the abdomen was distended with guarding and rigidity present in the epigastric region, with absent bowel sounds. Respiratory, cardiac, and nervous system examination revealed no significant abnormality.

Initial investigations included an Electrocardiogram showing sinus tachycardia and a chest X-ray, which showed no abnormality. Relevant blood investigations, urine examination, and ultrasonography of the abdomen were done (Table 1).

A provisional diagnosis of SLE with lupus nephritis with AP was made. Contrast-enhanced computed tomography (CT) abdomen was performed to evaluate the cause of AP, which was suggestive of a bulky and altered pancreas with adjacent fat stranding and multiple peri-pancreatic intercommunicating pockets of fluid collection, suggestive of acute interstitial edematous pancreatitis with modified CT severity index 6/10 (Fig. 2). Hence, the diagnosis of SLE-related AP with lupus nephritis was made considering the elevated levels of autoantibodies and pancreatic enzymes, and after excluding other frequent etiologies of pancreatitis.

The patient was started on pulse therapy of methylprednisolone for 3 days. The patient was kept nil per oral and symptomatic treatment was continued with IV fluids, opiates, and antiemetics. The patient improved symptomatically after 3 days with no complaints of abdominal pain. Blood investigations showed significant improvement. Liver function tests were improving with serum glutamic-oxaloacetic transaminase 65 U/L, serum glutamic pyruvic transaminase 44 U/L. Serum amylase and lipase levels decreased to 213 U/l and 149 U/l, respectively. The patient was then started on Tablet Prednisolone 40 mg once daily. She was also started on Tablet hydroxychloroquine 200 mg twice daily and Tablet Mycophenolate mofetil 500 mg twice daily. The patient was then discharged, continued on follow-up, and planned for a renal biopsy.

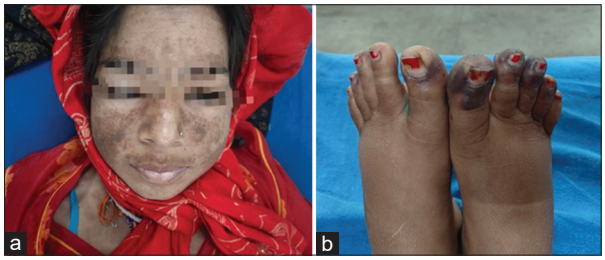

Figure 1: (a) Malar rash with sparing of nasolabial folds; (b) Pedal edema with blackish discoloration of toes

Table 1: Laboratory investigations

Investigation	Value
Hemoglobin	9.3 g/dL
White blood cells	16,100/mm^3
Platelets	51,000/mm^3
Serum glutamic-oxaloacetic transaminase	866 U/L
Serum glutamic pyruvic transaminase	150 U/L
Alkaline phosphatase	276 U/l
Total bilirubin	2.2 mg/dL
Direct bilirubin	2.05 mg/dL
Urea	46.8 mg/dL
Creatinine	0.8 mg/dL
Serum amylase	1,235 U/L
Serum lipase	2,304 U/L
Serum cholesterol	170.0 mg/dL
Triglycerides	147.0 mg/dL
High-density lipoprotein cholesterol	45.0 mg/dL
Low-density lipoprotein cholesterol	86.0 mg/dL
Very-low density lipoprotein cholesterol	30.4 mg/dL
Sodium	131 mmol/L
Potassium	4.7 mmol/L
Calcium	9.2 mg/dL
Antinuclear antibody	2.0
ds DNA	>150 IU/l
C3	31 mg/dL (decreased)
C4	8 mg/dL (decreased)
Anticardiolipin IgG	2.87 GPL/mL
Anticardiolipin IgM	3.44 MPL/mL
Lupus anticoagulant	0.6
Beta-2 glycoprotein IgG	3.0 U/mL
Thyroid-stimulating hormone	2.04
Urine routine and microscope	Proteinuria (2+), Hematuria (3+)
Urine albumin-to-creatinine ratio	1,107 mg/g
Ultrasound abdomen	Bulky and altered texture of pancreas with altered echotexture of liver.

IgG: Immunoglobulin G, IgM: Immunoglobulin M

DISCUSSION

SLE is a multisystem autoimmune disease with a wide spectrum of clinical manifestations. Among these, AP

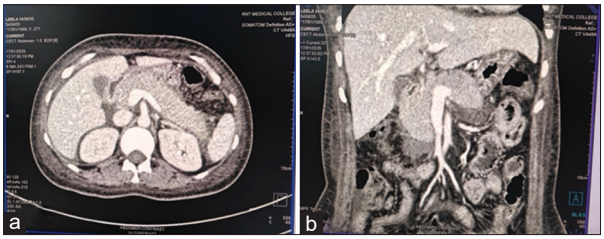

Figure 2: (a) Bulky and altered pancreas with adjacent fat stranding (transverse section); (b) Bulky and altered pancreas with adjacent fat stranding and multiple peri-pancreatic intercommunicating pockets of fluid collection (longitudinal section)

is a rare but potentially life-threatening complication, occurring in approximately 0.4% to 1.1% of SLE patients [2]. This case of a 27-year-old female highlights the diagnostic and therapeutic challenges associated with SLE-related AP, further complicated by nephritis.

The exact pathophysiology of SLE-associated AP remains unclear, but several mechanisms have been proposed. Autoimmune-mediated inflammation, vasculitis, complement activation, and microthrombosis are thought to contribute to pancreatic injury in SLE [4]. Hypocomplementemia (reduced C3 and C4 levels), elevated anti-dsDNA antibodies, and the absence of other common etiologies of pancreatitis in this patient strongly suggest an autoimmune etiology. Studies have shown that SLE-related AP often occurs during active disease and is associated with higher disease activity indices, hypocomplementemia, and proteinuria, all of which were observed in this case [3,5]. Similarly, another cohort study by Charles reported that pancreatitis in SLE patients is associated with hypocomplementemia and a higher disease activity index [6]. The patient's elevated serum amylase and lipase levels, combined with CT imaging findings, confirmed the diagnosis of acute interstitial edematous pancreatitis. Common etiologies, including gallstones, alcohol use, hypertriglyceridemia, hypercalcemia, and drug-induced pancreatitis, were systematically excluded, thereby supporting an autoimmune etiology.

This case was further complicated by lupus nephritis, as evidenced by significant proteinuria and elevated urine albumin-to-creatinine ratio, which is a well-documented manifestation of SLE, affecting up to 50% of patients during their disease course [7]. Renal biopsy is crucial for subclassification and in deciding the immunosuppressive therapy, as planned in this case.

Corticosteroids remain the cornerstone of therapy for SLE-related AP. However, steroid treatment was considered controversial because of the risk of steroid-induced pancreatitis, but this concern is regarded as minimal, and the immunosuppressive effect of steroids can significantly improve prognosis and is recommended for SLE-related autoimmune pancreatitis [8]. This patient responded well to intravenous methylprednisolone, followed by oral prednisolone, which resulted in symptomatic improvement. Early nutritional support and supportive measures, including intravenous fluids, analgesics, and antiemetics, are essential components of AP management. Yang *et al*. highlighted that prompt treatment with immunosuppressive agents significantly improves outcomes in SLE-related AP, as observed in this case [3]. Immunomodulators, such as hydroxychloroquine and mycophenolate mofetil, were initiated to control systemic inflammation and prevent disease flares [9]. A similar case has been reported from Peru by Rodriguez *et al*., involving a 21-year-old female with known SLE and lupus nephritis, receiving corticosteroids and pulse cyclophosphamide therapy. She presented with AP, and after exclusion of other common etiologies, SLE was considered the underlying cause. Her laboratory findings revealed elevated anti-dsDNA levels along with reduced complement levels (C3 and C4), indicating active disease. Similar to our case, the patient showed significant clinical improvement following pulse methylprednisolone therapy [10].

The prognosis of SLE-related AP is generally poorer than that of idiopathic pancreatitis due to the risk of recurrent flares and multiorgan involvement. This patient's favorable response highlights the importance of early diagnosis and aggressive treatment. Close monitoring during follow-up is necessary to assess disease activity, prevent relapses, and address potential complications, including infections and medication-related side effects.

CONCLUSION

This case highlights the rare occurrence of AP in SLE, complicated by lupus nephritis. Early recognition of AP in SLE, guided by clinical, laboratory, and imaging findings, exclusion of other etiologies, and timely initiation of immunosuppressive therapy are crucial in improving outcomes. Immunosuppressive therapy, particularly corticosteroids, alongside supportive care, forms the cornerstone of management. Integrated care and close follow-up are essential to improve outcomes and minimize complications in such complex cases.

ACKNOWLEDGMENT

I am deeply grateful to the Department of Medicine of RNT Medical College, Udaipur for providing me the opportunity to write this rare case report. I am also grateful to the anonymous peer reviewers for their dedicated time and expertise.

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Funding: Nil; Conflicts of interest: Nil.

How to cite this article: Mahur HK, Jain N, Mittal D, Agarwal A, Labana D, Bankal Y. Systemic lupus erythematosus-related acute pancreatitis: An uncommon clinical manifestation. Indian J Case Reports. 2026;12(5):308-311.