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Case Report

Unmasking lupus: acalculous cholecystitis presenting as the initial manifestation of systemic lupus erythematosus

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ABSTRACT

Acute acalculous cholecystitis (AAC) is an uncommon cause of gallbladder inflammation, typically occurring in critically ill patients. Its presentation as the initial manifestation of systemic lupus erythematosus (SLE) is exceedingly rare. We report the case of a 30-year-old female with hypothyroidism and remote history of pleural tuberculosis, who presented with acute right upper quadrant pain, fever, photosensitive rash, oral ulcers, and generalized edema. Imaging revealed gallbladder wall thickening with pericholecystic fluid in the absence of gallstones, consistent with AAC. Serological evaluation demonstrated strongly positive ANA and anti-dsDNA antibodies with hypocomplementemia, while renal biopsy confirmed Class II mesangial proliferative lupus nephritis. Additional findings included lupus pneumonitis, pleural and peritoneal serositis, and arthralgia, establishing a diagnosis of multisystem SLE. The patient responded favourably to corticosteroids, mycophenolate mofetil, and hydroxychloroquine, with resolution of systemic features and avoidance of surgical intervention. This case highlights AAC as a rare heralding manifestation of SLE and underscores the importance of considering autoimmune etiologies in atypical AAC presentations. Early recognition and prompt immunosuppressive therapy are crucial to prevent unnecessary cholecystectomy and irreversible organ damage.

Keywords: Acute acalculous cholecystitis, Systemic lupus erythematosus, Lupus

INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic, autoimmune, multisystem connective tissue disorder characterised by the production of autoantibodies against nuclear and cytoplasmic antigens, leading to immune complex formation and widespread inflammation. SLE predominantly affects women of childbearing age and has a highly variable clinical course, involving multiple organ systems, including the skin, joints, kidneys, hematologic system, and central nervous system. Gastrointestinal (GI) involvement in SLE is common, seen in up to 50% of patients at some point during the disease course. The manifestations are diverse, ranging from mild nonspecific symptoms such as nausea, vomiting, and abdominal pain to more severe complications like lupus mesenteric vasculitis, pancreatitis, protein-losing enteropathy and hepatic involvement. However, direct involvement of the gallbladder is exceedingly rare, and most commonly reported as incidental serositis.[1] Acute acalculous cholecystitis (AAC) refers to inflammation of the gallbladder in the absence of gallstones. It accounts for approximately 5-10% of all cases of acute cholecystitis and is typically seen in critically ill patients, such as those with sepsis, trauma, burns, prolonged fasting or parenteral nutrition. The pathogenesis involves gallbladder ischemia, bile stasis, and secondary infection. The occurrence of AAC as an initial manifestation of SLE is extremely rare, with only a few case reports documented in the literature. Proposed mechanisms in SLE include small vessel

vasculitis of the cystic artery, immune complex deposition leading to gallbladder wall inflammation, and less commonly, thrombosis due to antiphospholipid antibody syndrome (APLS).[2] Unlike AAC in the general population, which often requires surgical intervention, SLE-associated AAC typically responds well to immunosuppressive therapy, making early recognition essential.[3]

We present a case of a young female who presented with AAC as the initial clinical manifestation of SLE, highlighting the importance of considering autoimmune aetiologies in patients with AAC without typical risk factors, to ensure timely medical management and avoid unnecessary surgical procedures.

CASE REPORT

A 30-year-old female, known case of hypothyroidism for the past 1.5 years and with a history of treated extrapulmonary (pleural) tuberculosis 20 years ago, presented with acute onset right upper quadrant abdominal pain, low-grade fever, progressive abdominal distension for 10 days, and generalized swelling including facial puffiness.

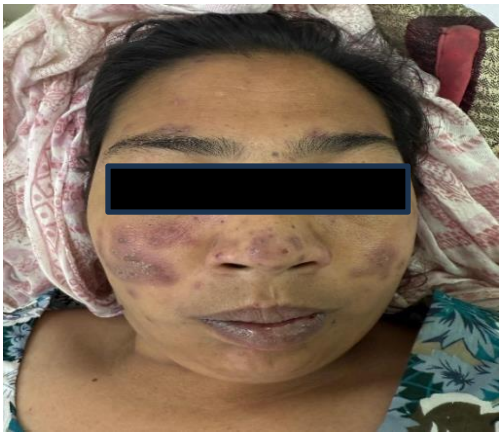

Figure 1: The discoid rash revealed irregular disc-shaped, dark erythematous plaques with hyperpigmentation on face involving tip of nose, both cheeks.

There was no prior history of gallstones, jaundice, or similar episodes. On examination, she had erythematous maculopapular rashes involving both cheeks and the tip of the nose, sparing the nasolabial folds, as well as involving the scalp, retroauricular areas, concha, chest and back, associated with marked photosensitivity. She also had painless ulcers over the hard palate and conjunctival chemosis. Respiratory examination revealed decreased breath sounds with fine end-inspiratory crepitations bilaterally in the infra-axillary and infrascapular regions. Abdominal examination showed tenderness in the right hypochondrium without a positive Murphy's sign. Initial hematological evaluation revealed a hemoglobin of 12.1 g/dl with a normal total leukocyte count (TLC) of 9,100/mm³ and a platelet count of 1.51 lakh/mm³. The differential leukocyte count showed mild lymphocytosis and eosinophilia (71% neutrophils, 14% eosinophils), with a normocytic normochromic (NCNC) peripheral smear, suggesting no overt cytopenias or hemolysis. Inflammatory markers were modestly elevated: ESR was 27 mm/h and CRP was 3.5 mg/l, while procalcitonin was negative supporting a non-bacterial inflammatory etiology.

Figure 2: Post treatment.

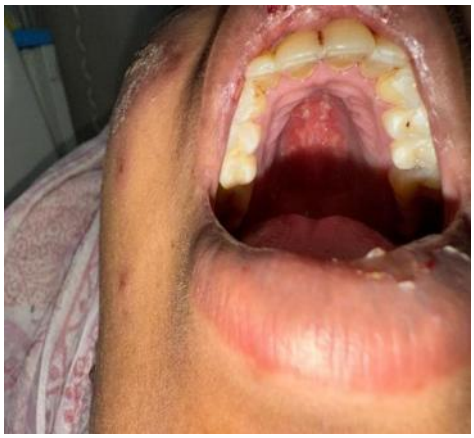

Figure 3: Oral ulcers over hard palate.

Liver and renal function tests were within normal limits, with serum urea and creatinine measuring 11 mg/dl and 0.66 mg/dl, respectively. Liver enzymes (AST 20, ALT 11, ALP 39 IU/l) and total bilirubin (0.36 mg/dl) were unremarkable. Electrolytes showed mild hyponatremia (Na 133 mEq/l) and hypokalemia (K 3.49 mEq/l), likely due to gastrointestinal losses or third-spacing from serositis.

Urinalysis revealed 2+ proteinuria, with an elevated urine albumin-creatinine ratio (UACR) of 432 mg/g and a urine protein-creatinine ratio (UPCR) of 969 mg/g. A 24-hour urinary protein excretion of 580 mg confirmed subnephrotic proteinuria. Urinary sediment was inactive, ruling out active nephritic involvement. These findings

were consistent with lupus nephritis, later confirmed on renal biopsy as Class II mesangial proliferative lupus nephritis.

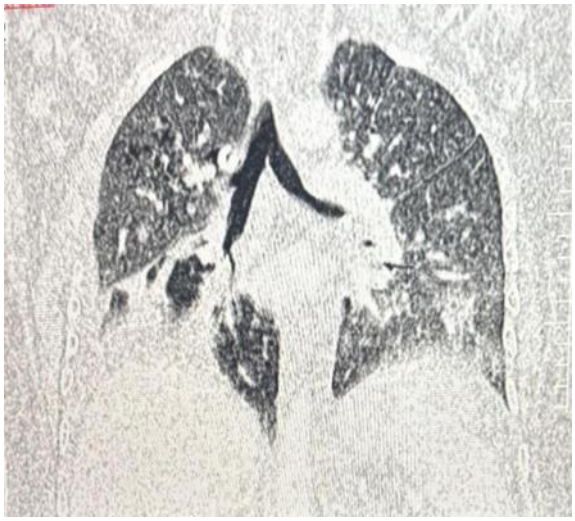

Figure 4: Left sided pleural effusion extending into fissure.

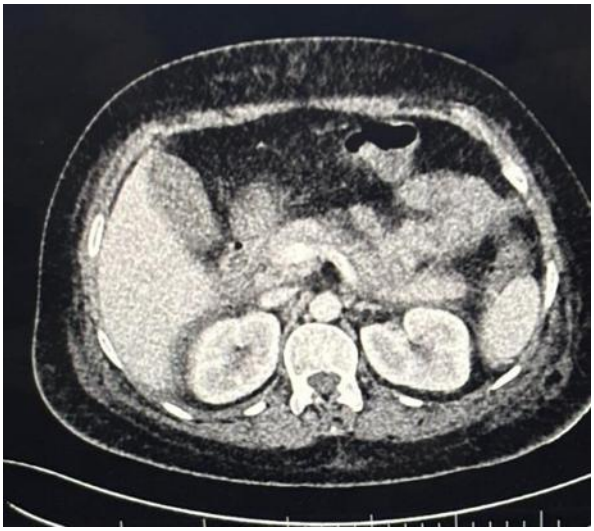

Figure 5: Inflamed gallbladder.

Endocrine evaluation was largely normal, including thyroid function tests, although anti-TPO antibodies were positive, suggesting autoimmune thyroiditis in the background. Infectious workup was negative: HIV, Hepatitis B and C, sputum AFB, gram stain, and CBNAAT were all negative. Blood, urine, sputum, ascitic, and pleural fluid cultures were sterile.

Pleural and ascitic fluid analysis revealed exudative features with elevated protein and LDH levels and low SAAG (serum-ascites albumin gradient=0.37), suggesting serositis likely of autoimmune origin. ADA levels were low, ruling out tuberculosis. ECG showed normal sinus rhythm, and chest X-ray revealed bilateral pleural effusion (left>right). Abdominal ultrasonography demonstrated gallbladder wall thickening (5 mm) with pericholecystic fluid and no gallstones, indicating acute acalculous cholecystitis. CECT of the chest and abdomen showed left-sided patchy consolidation with ground-glass opacities suggestive of non-infectious etiology such as lupus pneumonitis along with bilateral pleural effusion, ascites and post-tubercular fibroatelectatic lung changes.

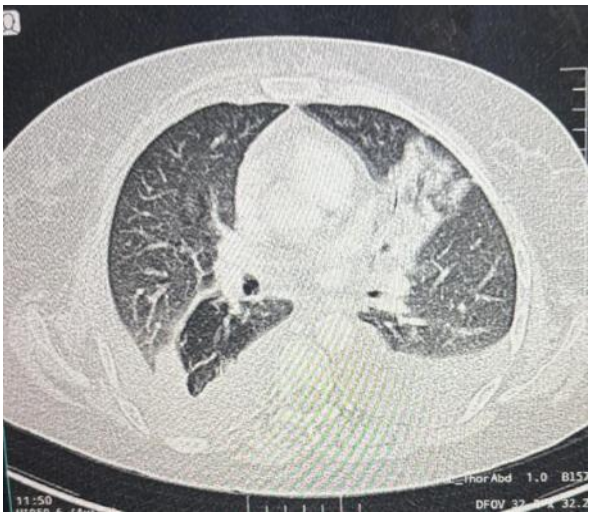

Figure 6: Lupus pneumonitis.

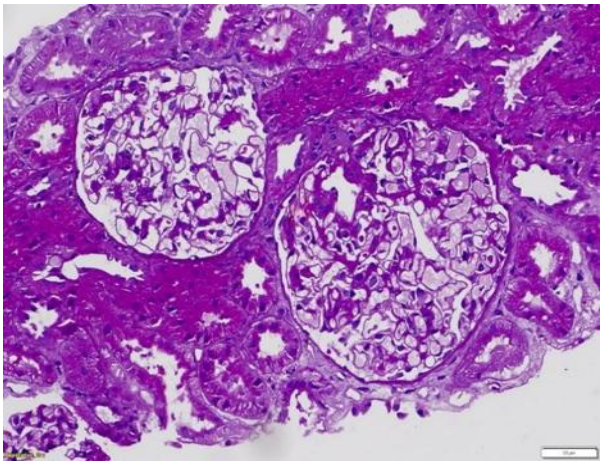

Figure 7: Light microscopy report: the kidney biopsy showed glomerulus sclerosed, viable glomeruli are enlarged mild increase in mesangial matrix. However, no increase in cellularity seen. Basement membrane shows focal splitting on silver methenamine stain. There is no evidence of proliferative activity in the form of endocapillary proliferation or crescent.

Immunological testing revealed strongly positive ANA (1:640, homogeneous pattern) and anti-dsDNA antibodies. Complement levels showed low C3 with normal C4, consistent with active immune complex-mediated disease. Direct Coombs test (DCT) was positive (2+), though there was no evidence of hemolysis. Skin biopsy was non-specific, with mild chronic perivascular inflammation, but lacked definitive features of cutaneous lupus, which can often be non-diagnostic in early or treated lesions.

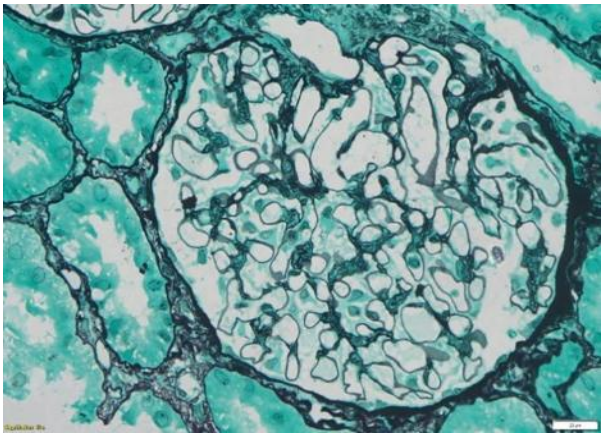

Figure 8: Electron microscopy reveals predominantly mesangial electron-dense immune complex deposits. No significant subendothelial or subepithelial electron-dense deposits are identified. Podocyte foot processes show mild focal effacement, without diffuse involvement.

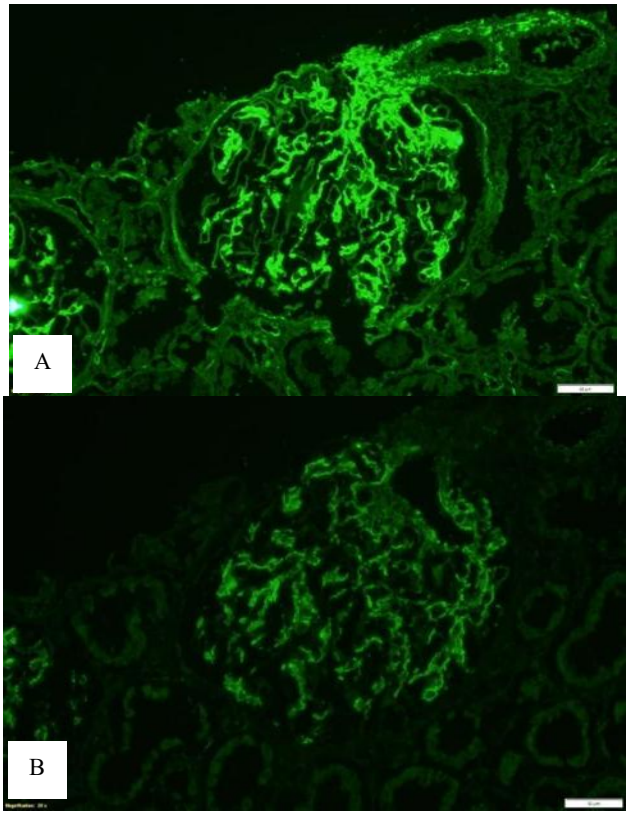

Figure 9 (A and B): Immunofluorescence shows 9 glomeruli which showed granular deposits of IGG (3+), IGM (1-2+), IGA (2-3+), C3 (+/-), C1Q (2+), kappa (2+) and lambda (2+) in the mesangium. In addition, deposits of IGG and IGA are also seen in the walls of blood vessels.

Together, the investigations supported a diagnosis of SLE with multi-organ involvement, including Class II lupus nephritis, lupus pneumonitis, pleural and peritoneal serositis, arthralgia, and acute acalculous cholecystitis as the initial manifestation. The patient was started on oral prednisolone 1 mg/kg/day, mycophenolate mofetil and hydroxychloroquine. Enalapril was initiated for proteinuria. The patient responded well to therapy, with resolution of rashes, facial puffiness, improvement in respiratory symptoms and declining proteinuria.

Our patient showed clinical improvement with prednisolone and hydroxychloroquine, thereby avoiding surgical intervention. With follow-up visits her proteinuria improved, rashes faded, edema disappeared and the general wellbeing of the patient improved. This highlights the importance of recognizing SLE in atypical AAC presentations to facilitate appropriate treatment and reduce morbidity.

DISCUSSION

AAC is an uncommon manifestation of SLE, especially when it occurs as the initial clinical presentation. While gastrointestinal (GI) involvement in SLE is relatively common affecting nearly 50% of patients over the course of their disease gallbladder involvement is rare and often underdiagnosed. AAC is characterized by inflammation of the gallbladder in the absence of gallstones and typically occurs in critically ill individuals due to gallbladder ischemia, bile stasis, and secondary infection.[3] However, when seen in the context of SLE, immune-mediated mechanisms are predominantly implicated, including small vessel vasculitis of the cystic artery, immune complex deposition and antiphospholipid antibody-related microthrombosis.[4]

In our case, a young female presented with right upper quadrant pain and fever without traditional AAC risk factors such as trauma, sepsis or total parenteral nutrition. The presence of photosensitive malar rash, oral ulcers, proteinuria and pleural effusion led to suspicion of a connective tissue disorder. Laboratory workup revealed strongly positive ANA (homogeneous pattern), elevated anti dsDNA titers and hypocomplementemia (low C3), fulfilling the diagnostic criteria for SLE.

The renal involvement in this case was confirmed by a renal biopsy revealing Class II lupus nephritis. This mesangial proliferative subtype typically presents with mild proteinuria and hematuria and has a relatively benign course with favorable prognosis if treated early. Initiation of an ACE inhibitor (enalapril) was appropriate and aimed at mitigating proteinuria and preserving renal function.

Further, the patient demonstrated serositis in the form of bilateral pleural effusion and ascites both exudative and culture-negative. Imaging showed patchy pulmonary infiltrates with ground-glass opacities in the absence of systemic infection findings suggestive of lupus pneumonitis. This reinforces the need to differentiate inflammatory SLE-related pathology from infectious etiologies, especially when immunosuppression is being considered. Though the skin biopsy was non-diagnostic for

lupus, this does not rule out the disease, as histological findings can vary with disease activity and treatment status. Clinicians should be guided primarily by clinical presentation and serological markers in such cases. Unlike conventional AAC, where cholecystectomy may be indicated, SLE-associated AAC often responds well to conservative management with corticosteroids and immunomodulators.

This case exemplifies the multi-organ involvement that can occur in SLE and demonstrates that AAC may, in rare cases, be the heralding manifestation. Awareness of this possibility is essential, especially in young women presenting with AAC and systemic symptoms in the absence of typical risk factors. Multisystem involvement such as lupus nephritis, pneumonitis, and arthritis should be thoroughly assessed and managed with a multidisciplinary approach to prevent irreversible organ damage and unnecessary surgical interventions.[5]

CONCLUSION

Acute acalculous cholecystitis can be an atypical and rare initial presentation of systemic lupus erythematosus. In such cases, high clinical suspicion, early diagnosis through immunological testing, and prompt initiation of immunosuppressive therapy are crucial for favorable outcomes.

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REFERENCES

1. Kamimura T, Mimori A, Takeda A, Masuyama J, Yoshio T, Okazaki H, et al. Acute acalculous cholecystitis in systemic lupus erythematosus: a case report and review of the literature. Lupus. 1998;7(5):3613.
2. Manuel V, Pedro GM, Cordeiro LB, de Miranda SM da RN. Acute acalculous cholecystitis in systemic lupus erythematosus: a rare initial manifestation. Rev Bras Reumatol. 2016;56(2):181-4.
3. Tian XP, Zhang X. Gastrointestinal involvement in systemic lupus erythematosus: Insight into pathogenesis, diagnosis and treatment. World J Gastroenterol. 2010;16(24):2971-7.
4. Choi YJ, Jang SA, Hong MJ, Lee WS, Yoo WH. A case of systemic lupus erythematosus initially presented with acute acalculous cholecystitis. J Rheum Dis. 2014;21(3):140-2.
5. Mohapatra S, Goldstein DR, Kumar A, Saha T, Penigalapati D. Acute acalculous cholecystitis as a presenting manifestation in systemic lupus erythematosus. Eur J Case Rep Intern Med. 2016;3:000408.

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