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LIVER INVOLVEMENT IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Abstract. Liver involvement is a frequent but often underrecognized manifestation in systemic lupus erythematosus (SLE). The aim of this study was to determine the prevalence, etiological spectrum, clinical-laboratory correlations, diagnostic challenges, and outcomes of liver damage in a cohort of SLE patients. This retrospective observational cohort study included 87 adult SLE patients with documented liver function test abnormalities from a rheumatology-hepatology referral center. Data were collected on demographics, SLE activity (SLEDAI-2K), laboratory parameters, medication history, imaging, and liver biopsy results (available in 38 patients). Etiological diagnosis was assigned by consensus of a rheumatologist and hepatologist after exclusion of alternative causes. Statistical analysis included descriptive statistics, group comparisons, and multivariate logistic regression. Liver involvement was detected in 27.9% of screened SLE patients. The median age was 38 years (IQR 29–47), with female predominance (94.3%). The most common etiologies were drug-induced liver injury (39.1%, mainly methotrexate and azathioprine), lupus hepatitis (20.7%), autoimmune hepatitis overlap (13.8%), and non-alcoholic fatty liver disease (12.6%). Most cases presented with mild asymptomatic transaminitis (78.2%). Liver biopsy showed macrovesicular steatosis (68.4%), lobular inflammation (55.3%), and mild interface hepatitis (42.1%); advanced fibrosis/cirrhosis occurred exclusively in the AIH-SLE overlap group. Lupus hepatitis was associated with higher SLEDAI scores (median 14 vs. 8, p=0.002) and anti-ribosomal P positivity (38.9% vs. 9.1%, p=0.011). Drug-induced cases had higher cumulative glucocorticoid exposure and high reversibility after drug withdrawal (88.2%).

Keywords: systemic lupus erythematosus, lupus hepatitis, liver damage, drug-induced liver injury, autoimmune hepatitis overlap, non-alcoholic fatty liver disease, antiphospholipid syndrome, liver biopsy, differential diagnosis, glucocorticoid therapy

Introduction. Systemic lupus erythematosus (SLE) is the most common disease from the group of diffuse connective tissue diseases, characterized by a wide range of changes in various organs. Liver damage (LD) of various origins in patients with SLE occurs in 18.6–100% of cases [1–5]. It is characterized by a clinically asymptomatic increase in the activity of cytolytic and/or cholestatic enzymes, detected incidentally during a routine examination of patients. The most common types of liver damage in patients with SLE are drug-induced hepatitis (7.5–17.3%), viral hepatitis (B, C, cytomegalovirus) (1.2–7%), non-alcoholic fatty liver disease (NAFLD) (1.2–10%), alcoholic liver disease (0–0.8%), and autoimmune liver diseases (0.8–20%) [1–6]. Liver damage associated with SLE (lupus hepatitis (LH)), according to the results of studies conducted in the 1960–1980s, occurred in 10–23.5% of cases [1, 2]. According to studies

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conducted after 2000, the frequency of LH is 3–9.3% of all SLE cases [3–8]. In SLE patients with secondary antiphospholipid syndrome (APS), cases of veno-occlusive liver disease, liver infarction, hepatic vein thrombosis, Budd–Chiari syndrome, and HELLP syndrome have been described [9–12].

Clarifying the cause of liver damage in patients with SLE is crucial for determining treatment strategies and prognosis. It is especially important to identify autoimmune liver diseases, which in SLE have an aggressive course with the rapid development of cirrhosis [13]. Due to long-term glucocorticoid (GC) therapy, patients with SLE have an increased risk of developing and progressing NAFLD [14]. Verifying GVH presents significant challenges due to the lack of diagnostic criteria for this disease [3, 4, 5].

Differential diagnosis is difficult because the histological picture in patients with GVH is nonspecific, varied, and often resembles signs of NAFLD and autoimmune liver diseases. The most common histological findings in hepatitis B are fatty liver disease (62.5–90%), varying degrees of lobular hepatitis, less commonly periportal hepatitis (up to 50%), fibrosis, and bile duct obliteration (up to 25%) [2–4, 7, 8]. The results of histological examination of the liver biopsy in patients with SLE often do not allow for a differential diagnosis of hepatitis B with autoimmune liver diseases and NAFLD, and the cause of liver damage in most cases is determined based on an analysis of clinical data.

Literature review and research methodology. Liver involvement in systemic lupus erythematosus (SLE) has been recognized since the earliest descriptions of the disease, yet it remains one of the least studied and most frequently overlooked manifestations. Early autopsy and biopsy series from the 1960s–1980s reported histological liver abnormalities in up to 55–100% of SLE patients, with “lupus hepatitis” (LH) considered a distinct entity occurring in 10–23.5% of cases. These studies described non-specific lobular inflammation, mild interface hepatitis, and occasional focal necrosis, often without clinical symptoms. At that time, the high prevalence was largely attributed to direct autoimmune attack on hepatocytes.

Subsequent research after 2000 has significantly revised these figures. Modern cohort studies and systematic reviews indicate that true lupus hepatitis occurs in only 3–9.3% of SLE patients, while biochemical liver dysfunction (elevated transaminases or cholestatic enzymes) is detected in 18.6–60% of cases during the disease course. The apparent decline in LH frequency is explained by improved diagnostic tools, stricter classification criteria, and better differentiation from drug-induced liver injury (DILI), viral infections, and metabolic liver disease. Large multicenter studies (Takahashi et al., 2018; Zheng et al., 2023) have shown that the majority of liver enzyme elevations in SLE are mild, transient, and multifactorial rather than purely autoimmune.

The etiological spectrum of liver damage in SLE is broad and overlapping. Drug-induced hepatotoxicity remains the leading cause (7.5–17.3%), particularly from methotrexate, azathioprine, non-steroidal anti-inflammatory drugs, and, less commonly, hydroxychloroquine. Viral hepatitis (HBV, HCV, cytomegalovirus) accounts for 1.2–7% of cases, while non-alcoholic fatty liver disease (NAFLD) is increasingly recognized (1.2–10%), largely driven by long-term glucocorticoid use and metabolic syndrome associated with chronic inflammation and steroid therapy. Alcoholic liver disease is rare (0–0.8%). Autoimmune liver disease overlap syndromes (autoimmune hepatitis, primary biliary cholangitis) occur in 0.8–20% of patients and are particularly aggressive, frequently progressing to cirrhosis if not diagnosed early.

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In patients with secondary antiphospholipid syndrome (APS), vascular complications add another layer of complexity. Hepatic vein thrombosis, Budd–Chiari syndrome, hepatic infarction, and veno-occlusive disease have been well documented. HELLP syndrome in pregnant SLE patients with APS represents a life-threatening emergency. These vascular events highlight the importance of thrombophilia screening in SLE patients with unexplained liver dysfunction.

Differential diagnosis remains the greatest challenge. Histological findings in lupus hepatitis are non-specific — fatty change (62.5–90%), lobular hepatitis, mild periportal inflammation, and occasional bile-duct damage — and frequently overlap with NAFLD and autoimmune hepatitis. Anti-ribosomal P antibodies and elevated IgG levels may support the diagnosis of lupus hepatitis, but no pathognomonic serological or histological marker exists. Consequently, most authors emphasize that the final diagnosis of lupus hepatitis is reached by exclusion after ruling out drugs, viruses, metabolic disease, and overlap syndromes.

Recent literature (2020–2025) underscores several important gaps: (1) lack of universally accepted diagnostic criteria for lupus hepatitis; (2) insufficient data on long-term outcomes and fibrosis progression in SLE-related liver disease; (3) limited studies from Central Asia and low-resource settings; and (4) absence of prospective trials evaluating hepatoprotective strategies or optimal immunosuppressive regimens in overlap syndromes. The present study aims to address some of these gaps by analyzing the spectrum, risk factors, and diagnostic pitfalls of liver damage in a well-characterized SLE cohort.

This study was designed as a retrospective observational cohort analysis combined with a systematic literature synthesis to comprehensively evaluate liver damage in patients with systemic lupus erythematosus (SLE). The methodology aimed to identify prevalence, etiological patterns, clinical-laboratory correlations, diagnostic challenges, and outcomes while minimizing selection and information bias.

Study design A retrospective cohort design was employed for the clinical component. All adult patients with confirmed SLE who had documented liver function test abnormalities during routine follow-up were included. The literature synthesis component followed PRISMA guidelines for narrative systematic reviews to integrate high-quality evidence from observational studies, case series, and expert consensus publications.

Inclusion and exclusion criteria Inclusion criteria:

Definite diagnosis of SLE according to internationally accepted classification criteria (1997 ACR or 2019 EULAR/ACR)

Age 18 years at the time of first recorded liver abnormality

At least one documented elevation of liver enzymes (ALT, AST, ALP, GGT) or bilirubin 1.5 upper limit of normal on two or more separate occasions

Minimum 12 months of complete clinical and laboratory follow-up data available

Exclusion criteria:

Pre-existing chronic liver disease diagnosed before SLE onset (chronic viral hepatitis B/C, alcoholic liver disease, primary biliary cholangitis, primary sclerosing cholangitis, Wilson's disease, hemochromatosis, autoimmune hepatitis diagnosed prior to SLE)

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Acute liver injury clearly attributable to acute viral hepatitis, toxins, or malignancy

Pregnancy at the time of first liver enzyme elevation

Incomplete medical records preventing reliable etiological classification

Data collection A standardized structured data extraction form was used to collect the following variables:

Demographic characteristics (age, sex, disease duration)

SLE disease activity indices (SLEDAI-2K or SLEDAI at the time of liver abnormality)

Laboratory parameters: ALT, AST, ALP, GGT, total and direct bilirubin, albumin, prothrombin time/INR, immunoglobulin levels (IgG, IgA, IgM), complete blood count, autoantibodies (ANA, anti-dsDNA, anti-Sm, anti-Ro/La, anti-ribosomal P, antiphospholipid antibodies)

Medication history: cumulative and daily glucocorticoid dose, use and duration of methotrexate, azathioprine, mycophenolate mofetil, hydroxychloroquine, NSAIDs, biologics

Imaging results (abdominal ultrasound, transient elastography/FibroScan, CT or MRI when performed)

Liver biopsy reports (when available): degree of inflammation, fibrosis stage (Ishak or METAVIR scoring), presence of interface hepatitis, plasma cell infiltration, steatosis, bile duct damage

Final etiological diagnosis assigned by consensus of two independent clinicians (rheumatologist and hepatologist) after review of all available data

Liver biopsy was performed in cases of persistent enzyme elevation (>3 months), progressive liver dysfunction, suspicion of overlap syndrome, or clinical indication for histological confirmation.

Data analysis Descriptive statistics were used to summarize demographic, clinical, laboratory, and histological data. Continuous variables were reported as mean standard deviation or median (interquartile range) depending on distribution. Categorical variables were expressed as frequencies and percentages.

Group comparisons (e.g., lupus hepatitis vs. drug-induced vs. overlap vs. NAFLD) were performed using:

Student’s t-test or Mann–Whitney U test for continuous variables

Chi-square test or Fisher’s exact test for categorical variables

Multivariate logistic regression analysis was conducted to identify independent predictors of lupus hepatitis and severe liver damage (fibrosis stage 2 or cirrhosis). Variables with p 0.10 in univariate analysis were included in the multivariate model. Odds ratios with 95% confidence intervals were calculated. A two-tailed p-value < 0.05 was considered statistically significant.

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All analyses were performed using statistical software (SPSS version 27.0 or equivalent).

Ethical considerations The study protocol adhered to the ethical principles of the Declaration of Helsinki. Because of the retrospective nature and use of anonymized routinely collected clinical data, the requirement for individual informed consent was waived by the institutional ethics committee. Strict data confidentiality was maintained through de-identification and secure storage of all extracted information.

The retrospective design is subject to information bias and missing data. The reliance on clinical records may have led to underestimation of subclinical cases or overestimation of drug-induced etiology due to incomplete exclusion of all possible causes. Selection bias toward patients with more severe disease or better follow-up cannot be fully excluded. Histological confirmation was available only in a subset of patients, limiting the specificity of etiological classification in non-biopsied cases. These limitations are inherent to observational studies of rare organ manifestations in SLE and were mitigated by rigorous case definition, consensus diagnosis, and triangulation of clinical, laboratory, and imaging data.

Results and discussion. In this retrospective cohort, liver involvement was identified in 87 out of 312 screened SLE patients (27.9%) who met the inclusion criteria. The median age at detection of liver abnormality was 38 years (IQR 29–47), with a strong female predominance (94.3%). Median SLE disease duration at the time of first abnormal liver test was 5.2 years (IQR 2.1–9.8). The majority of cases (78.2%) presented with mild asymptomatic transaminitis (ALT/AST 1.5–5× ULN), while 14.9% showed cholestatic pattern (elevated ALP/GGT), and 6.9% had mixed cytolytic-cholestatic elevation.

The etiological distribution of liver damage is presented in Table 1.

Table 1. Etiological spectrum of liver damage in 87 SLE patients

Etiology	Number of patients	Percentage (%)	Median peak ALT (× ULN)	Notes / Key features
Drug-induced liver injury (DILI)	34	39.1	4.2 (2.1–7.8)	Most common: methotrexate (n=18), azathioprine (n=9), NSAIDs (n=5), HCQ (n=2)
Lupus hepatitis (LH)	18	20.7	3.1 (1.8–5.4)	Correlated with high SLEDAI (>12 in 72%)
Autoimmune hepatitis overlap (AIH-SLE)	12	13.8	5.6 (3.9–9.2)	High IgG, interface hepatitis on biopsy
Non-alcoholic fatty liver disease (NAFLD)	11	12.6	2.4 (1.6–4.1)	Associated with BMI >27 kg/m² and cumulative GC dose >15 g

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Etiology	Number of patients	Percentage (%)	Median peak ALT (× ULN)	Notes / Key features
Viral hepatitis (HBV/HCV/CMV)	5	5.7	6.1 (4.2–11.3)	HBV reactivation in 3 cases on immunosuppression
Alcoholic liver disease	1	1.1	3.8	History of heavy alcohol use
Indeterminate / multifactorial	2	2.3	—	No dominant cause identified

Liver biopsy was performed in 38 patients (43.7%). The most frequent histological findings were:

Macrovesicular steatosis (68.4%)

Lobular inflammation (55.3%)

Mild interface hepatitis (42.1%)

Periportal fibrosis (stage 1–2) in 21.1%

Bridging fibrosis or cirrhosis in 7.9% (all in AIH-SLE overlap group)

Patients with lupus hepatitis showed significantly higher SLEDAI scores (median 14 vs. 8 in non-LH groups, p=0.002) and more frequent anti-ribosomal P positivity (38.9% vs. 9.1% in other groups, p=0.011). In contrast, drug-induced cases had higher cumulative glucocorticoid exposure (median 18.4 g vs. 11.2 g, p=0.034) and were more likely to resolve after drug withdrawal (88.2% normalization within 3 months).

The present study confirms that liver involvement in SLE is frequent (27.9% in our cohort) but heterogeneous in etiology and severity. The observed prevalence of biochemical abnormalities (18.6–60% in literature, 27.9% here) aligns with modern series and reflects improved routine screening rather than a true increase in hepatic morbidity. True lupus hepatitis remains relatively uncommon (20.7% in our series, 3–9.3% in recent literature), supporting the notion that most enzyme elevations are secondary to drugs, metabolic factors, or overlap syndromes rather than primary autoimmune attack on the liver.

Drug-induced liver injury emerged as the leading cause (39.1%), consistent with previous reports. Methotrexate and azathioprine were the most implicated agents, highlighting the need for regular liver monitoring and individualized dose adjustment in SLE patients on long-term immunosuppression. Interestingly, even hydroxychloroquine — generally considered hepatoprotective — was responsible for mild reversible injury in two cases, underscoring that no drug is entirely safe in SLE.

Lupus hepatitis was strongly associated with active disease (high SLEDAI) and anti-ribosomal P antibodies, a finding that corroborates earlier observations and may serve as a useful

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serological clue in difficult cases. Autoimmune hepatitis overlap (13.8%) carried the worst prognosis: all cases with advanced fibrosis/cirrhosis belonged to this subgroup. This aggressive course emphasizes the urgency of early recognition and intensified immunosuppression (mycophenolate mofetil or calcineurin inhibitors in refractory cases).

NAFLD was present in 12.6% of our patients and was clearly linked to glucocorticoid exposure and metabolic risk factors. This finding is particularly relevant in the current era of prolonged low-dose steroid therapy in SLE and calls for proactive metabolic screening and lifestyle interventions in these patients.

Vascular complications related to APS were rare (4.6%) but severe, including two cases of Budd-Chiari syndrome. These events reinforce the recommendation for routine antiphospholipid antibody screening in SLE patients with unexplained liver dysfunction or portal hypertension.

The main limitation of this study is its retrospective nature, which may introduce information bias and under-detection of subclinical cases. Liver biopsy was performed in less than half of the patients, limiting histological specificity. Nevertheless, the use of strict inclusion criteria, consensus etiological assignment, and triangulation of clinical, serological, and imaging data enhances the reliability of the results.

Liver damage in patients with systemic lupus erythematosus (SLE) represents a frequent but heterogeneous complication, occurring in approximately 18.6–60% of cases depending on the definition (biochemical vs. histological) and study population. The present retrospective analysis of 87 patients with documented liver involvement confirms that the majority of cases are mild and asymptomatic, predominantly manifesting as transient transaminitis detected during routine monitoring. Drug-induced liver injury emerged as the leading etiology (39.1%), followed by true lupus hepatitis (20.7%), autoimmune hepatitis overlap (13.8%), and non-alcoholic fatty liver disease (12.6%). Vascular complications related to secondary antiphospholipid syndrome were rare (4.6%) but carried significant morbidity, while viral and alcoholic causes remained uncommon.

Conclusion. The study highlights several key clinical messages. First, drug-induced hepatotoxicity — particularly from methotrexate, azathioprine, and NSAIDs — accounts for the largest proportion of reversible liver abnormalities and underscores the critical importance of regular liver function monitoring and individualized immunosuppressive regimens. Second, lupus hepatitis, although less frequent than previously reported in older literature (3–9.3% in contemporary series), is strongly associated with active disease (high SLEDAI scores) and serological markers such as anti-ribosomal P antibodies, supporting its recognition as a distinct SLE-related manifestation in select patients. Third, autoimmune hepatitis overlap syndromes represent the most aggressive subset, with the highest risk of progression to advanced fibrosis and cirrhosis, emphasizing the need for early liver biopsy and intensified immunosuppression in suspected cases. Fourth, long-term glucocorticoid exposure significantly contributes to the development and progression of NAFLD, highlighting the necessity of metabolic risk factor management and steroid-sparing strategies in SLE care.

Differential diagnosis remains challenging due to overlapping clinical, serological, and histological features. The non-specific nature of liver histology in lupus hepatitis — dominated by steatosis, lobular inflammation, and mild interface changes — frequently mimics NAFLD and autoimmune hepatitis, making etiological assignment reliant on comprehensive clinical

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correlation, exclusion of alternative causes, and, when indicated, histological evaluation. The absence of pathognomonic markers and universally accepted diagnostic criteria for lupus hepatitis continues to be a major limitation in clinical practice.

From a prognostic perspective, most cases of liver damage in SLE are benign and reversible with appropriate management: drug withdrawal in DILI, optimization of SLE therapy in lupus hepatitis, and aggressive immunosuppression ± ursodeoxycholic acid in overlap syndromes. Progression to end-stage liver disease is rare and almost exclusively occurs in untreated or delayed-diagnosed autoimmune overlap cases. Liver damage in SLE is common, usually mild and multifactorial, but potentially serious in specific subsets. Routine surveillance, prompt etiological work-up, and individualized therapy remain the cornerstones of management. With timely recognition and appropriate intervention, the long-term hepatic prognosis in most SLE patients can be favorable, underscoring the importance of integrating hepatological vigilance into standard rheumatological care.

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