Section: Miscellaneous

Original Research Article

A PROSPECTIVE STUDY CORRELATING HISTOPATHOLOGICAL FINDINGS WITH CLINICAL OUTCOMES IN PATIENTS WITH AUTOIMMUNE HEPATITIS

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ABSTRACT

Background: Autoimmune hepatitis (AIH) is a chronic immune-mediated liver disease characterized by hepatocellular inflammation, circulating autoantibodies, and distinct histopathological features. Early diagnosis and accurate assessment of disease severity are essential to prevent progression to cirrhosis and hepatic failure. Histological evaluation plays a central role not only in confirming AIH but also in determining prognosis. However, data correlating specific histopathological patterns with clinical outcomes remain limited, particularly in real-world tertiary-care settings. The aim of this study is to correlate baseline histopathological findings with biochemical response and clinical outcomes in patients with autoimmune hepatitis evaluated at a tertiary-care hospital.

Materials and Methods: This prospective study included 52 adults diagnosed with AIH based on clinical, biochemical, serological, and histological criteria. Detailed clinical assessment included demographic data, presenting symptoms, and physical examination findings. Biochemical parameters (ALT, AST, bilirubin, albumin, INR, IgG) and autoantibody profiles (ANA, ASMA, anti-LKM-1, anti-SLA) were recorded. All patients underwent ultrasound-guided liver biopsy, which was evaluated for interface hepatitis, plasma cell infiltration, lobular necroinflammation, rosetting, emperipolesis, portal inflammation, steatosis, and fibrosis stage. Various clinical outcomes like complete remission, partial response, non-response, progression to cirrhosis, portal hypertension, and hepatic decompensation were documented during follow-up.

Results: Female predominance was noted (78.85%), with fatigue (75.00%) and jaundice (65.38%) being the most common symptoms. Biochemically, ALT, AST, bilirubin, IgG, and INR were significantly associated with higher histological activity. Histopathology revealed interface hepatitis in 90.38%, plasma cell infiltration in 78.85%, and moderate to severe portal inflammation in 65.38%. Advanced fibrosis (F3–F4) occurred in 42.30% of patients and correlated strongly with poorer treatment response. Complete remission was achieved in 65.38%, while 13.46% showed non-response. Adverse outcomes included progression to cirrhosis (15.38%), portal hypertension (19.23%), and hepatic decompensation (11.54%).

Conclusion: Specific baseline histopathological features, particularly severe inflammatory activity and advanced fibrosis, are strong predictors of treatment response and long-term outcomes in autoimmune hepatitis. Integrating histological patterns with biochemical and clinical parameters enhances prognostic accuracy and supports tailored therapeutic strategies.

Keywords: Autoimmune hepatitis, histopathology, interface hepatitis, fibrosis, treatment response.

INTRODUCTION

Autoimmune hepatitis (AIH) is a chronic, immune-mediated inflammatory liver disease characterized by hepatocellular injury in the absence of a defined viral, metabolic, or toxic cause. Clinically, it is typified by elevated serum transaminases, hypergammaglobulinaemia with raised IgG levels, circulating autoantibodies, and a characteristic histological pattern of interface hepatitis on liver biopsy. Although AIH is classified as a rare disease, it carries a substantial risk of progression to cirrhosis, hepatic decompensation, and liver failure if diagnosis and treatment are delayed, underscoring the need for early recognition and accurate risk stratification.[1] Epidemiological data suggest that the global burden of AIH has increased over recent decades, with considerable geographic variability in incidence and prevalence.[2] A large systematic review and meta-analysis reported a pooled global incidence of approximately 1.28 cases per 100,000 inhabitant-years and a prevalence of 15.65 cases per 100,000 inhabitants, with higher rates observed in regions with a high Human Development Index, in women, and in adult populations. AIH exhibits a marked female predominance, with around three-quarters of affected individuals being women, and it affects all age groups from childhood to older adults. Differences in environmental exposures, genetic background, and awareness or application of diagnostic criteria may all contribute to regional variation in disease frequency and phenotype.[3] Clinically, AIH presents with a broad spectrum of manifestations, ranging from asymptomatic elevation of liver enzymes to insidious chronic hepatitis, acute severe hepatitis, or even fulminant liver failure. Many patients present with non-specific symptoms such as fatigue, malaise, or mild right upper quadrant discomfort, while others may have jaundice, pruritus, or features of portal hypertension and decompensated cirrhosis at diagnosis. Serologically, AIH is traditionally subdivided into type 1, characterized by antinuclear antibodies (ANA) and/or anti–smooth muscle antibodies (ASMA), and type 2, defined by anti–liver kidney microsomal type 1 (anti-LKM-1) and/or anti–liver cytosol type 1 antibodies, with type 1 disease being more common in adults. Despite these classifications, overlap syndromes with primary biliary cholangitis or sclerosing cholangitis and seronegative AIH further complicate the clinical landscape.[4,5] Diagnostic assessment of AIH relies on an integrated evaluation of clinical, biochemical, serological, and histological parameters, along with exclusion of other causes of liver disease. International scoring systems and diagnostic algorithms have been developed by the International Autoimmune Hepatitis Group and incorporated into major society guidelines to standardize case definition and facilitate diagnosis in both typical and atypical presentations.[5] The European Association for the Study of the Liver (EASL) and the American Association for the Study of Liver Diseases (AASLD) recommend liver biopsy in all suspected cases, not only to confirm interface hepatitis and other characteristic features but also to stage fibrosis and assess inflammatory activity, which may guide treatment decisions and prognostication.[5] From a pathogenic standpoint, AIH is thought to result from a complex interplay between genetic susceptibility and environmental triggers that leads to a loss of tolerance to hepatocellular antigens. Disease-associated HLA class II alleles, particularly certain DRB1 haplotypes, confer increased risk and vary across ethnic groups, while non-HLA immunoregulatory genes may further modulate disease expression. Proposed mechanisms include molecular mimicry between microbial antigens and self-epitopes, dysregulated T-helper cell responses, defective regulatory T-cell function, and B-cell activation with production of pathogenic autoantibodies. These immunological processes ultimately converge on hepatocellular injury, chronic necro-inflammation, and progressive fibrogenesis.[6] Histopathology remains central to the diagnosis and characterization of AIH. The classical pattern is interface hepatitis, in which a lymphoplasmacytic infiltrate breaches the limiting plate and extends into the periportal parenchyma; plasma cells, hepatocyte rosetting, lobular necro-inflammation, and emperipolesis are also frequently observed. In addition, fibrosis staging from portal expansion to established cirrhosis provides important information on disease chronicity and prognosis. However, histological appearances may vary, especially in acute or acute-severe presentations, where centrilobular necrosis or panacinar hepatitis may predominate, potentially mimicking viral or drug-induced liver injury. Given this heterogeneity, there is growing interest in delineating which specific histological features at baseline carry independent prognostic value for treatment response and long-term clinical outcomes. Therapeutically, standard first-line management of AIH consists of corticosteroids, either alone or in combination with azathioprine, which induces biochemical remission in the majority of patients and significantly improves survival compared with the natural history of untreated disease.

Nevertheless, a substantial subset of patients exhibit incomplete response, intolerance to therapy, or treatment-refractory disease, and may require second-line agents such as mycophenolate mofetil, calcineurin inhibitors, or budesonide, or ultimately liver transplantation in the setting of advanced cirrhosis or acute liver failure. Observational data indicate that advanced fibrosis or cirrhosis at diagnosis and persistent histological activity despite biochemical improvement are associated with poorer outcomes, including higher rates of decompensation, portal hypertension, and transplant-free mortality.[7]

MATERIALS AND METHODS

This prospective study was conducted in the Department of Medicine and Pathology at a tertiary care hospital and included a total of 52 consecutively enrolled patients clinically suspected and subsequently diagnosed with autoimmune hepatitis (AIH). All eligible patients were adults of either gender who fulfilled the simplified diagnostic criteria for AIH, incorporating clinical presentation, serological markers, biochemical abnormalities, and compatible histopathological features. Patients with overlap syndromes, decompensated chronic liver disease due to other etiologies, significant alcohol intake, chronic viral hepatitis, drug-induced liver injury, or incomplete clinical records were excluded to avoid confounding effects.

Methodology: All enrolled patients underwent detailed clinical evaluation, including age, gender, symptom profile, duration of symptoms, presence of jaundice, fatigue, pruritus, abdominal pain, hepatomegaly, splenomegaly, ascites, and signs of hepatic decompensation. Baseline biochemical parameters were recorded comprising serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), total and direct bilirubin, serum albumin, international normalized ratio (INR), and immunoglobulin G (IgG) levels. Serological testing included antinuclear antibody (ANA), anti–smooth muscle antibody (ASMA), liver kidney microsomal antibody (anti-LKM-1), soluble liver antigen antibody (anti-SLA), and other autoimmune markers where indicated. Imaging with abdominal ultrasonography was performed in all patients to assess liver morphology and exclude biliary obstruction or focal lesions.

All subjects underwent percutaneous liver biopsy under ultrasound guidance using a standard 16- or 18-gauge needle. Adequate biopsy specimens containing ≥6 portal tracts were considered acceptable for evaluation. Histopathological examination was performed by experienced hepatopathologists who were blinded to the clinical outcomes. The biopsy specimens were assessed for interface hepatitis, plasma cell infiltration, lobular necro-inflammation, rosetting of hepatocytes, emperipolesis, portal inflammation, bile duct injury, steatosis, fibrosis stage, and overall histological activity. Fibrosis was graded using a standardized scoring system, and necro-inflammatory activity was assessed semi-quantitatively to ensure uniform interpretation.

All patients were evaluated for clinical outcomes following standard treatment protocols, which typically included corticosteroids with or without azathioprine based on disease severity and contraindications. Response to therapy was assessed using predefined criteria including biochemical remission, partial response, and non-response. Additional outcomes such as progression to cirrhosis, presence of portal hypertension, hepatic decompensation, treatment intolerance, and requirement for modification of immunosuppressive therapy were also recorded. Follow-up assessments included periodic clinical evaluation, liver function tests, immunoglobulin levels, and monitoring of adverse events related to therapy.

Statistical Analysis: All collected data were entered into a secure database and analyzed using Statistical Package for the Social Sciences (SPSS) version 26.0. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, depending on distribution assessed by the Shapiro–Wilk test. Categorical variables were represented as frequencies and percentages. Associations between histopathological findings and clinical outcomes were evaluated using chi-square test, Fisher's exact test, independent t-test, or Mann–Whitney U test as appropriate. Correlation analysis was performed to determine the relationship between histological scores and biochemical parameters. A p-value of <0.05 was considered statistically significant.

RESULTS

[Table 1] Baseline Demographic and Clinical Characteristics

The study population consisted of 52 patients diagnosed with autoimmune hepatitis, with a clear predominance of females (78.85%), which aligns with the well-established female preponderance observed in autoimmune disorders. Only 21.15% of the patients were males. Symptomatically, fatigue (75.00%) and jaundice (65.38%) were the most frequent presenting complaints, and both demonstrated statistically significant associations with disease severity ($p = 0.028$ and $p = 0.041$ respectively). Abdominal pain (42.31%) and pruritus (34.62%) were less common, and neither showed significant correlation with disease severity. Among clinical signs, hepatomegaly was present in more than half of the patients (57.69%) and showed a significant association with more severe disease ($p = 0.044$). Splenomegaly (36.54%) and ascites (26.92%) were also documented but did not reach statistical significance.

[Table 2] Baseline Biochemical and Serological Profile

Biochemical evaluation revealed markedly elevated liver enzymes, with a mean ALT of 181.42 ± 78.63 U/L and AST of 204.77 ± 96.51 U/L, both showing strong statistical significance when compared across histological activity grades ($p = 0.003$ and $p = 0.001$). Total bilirubin levels were also notably high (3.82 ± 2.14 mg/dL), reflecting cholestasis or hepatocellular dysfunction, with significant correlation to disease severity ($p = 0.012$). Low serum albumin (2.98 ± 0.67 g/dL) and elevated INR (1.42 ± 0.31) further indicated compromised synthetic liver function, both parameters demonstrating significant associations with higher histological activity ($p = 0.020$ and $p = 0.033$ respectively). Serologically, the majority of patients exhibited elevated IgG levels (82.69%),

ANA positivity (73.08%), and ASMA positivity (51.92%), with all three markers showing significant correlation with moderate–severe disease activity. In contrast, anti-LKM-1 and anti-SLA antibodies were relatively uncommon and did not show meaningful statistical associations.

[Table 3 and Fig. 1] Histopathological Findings on Liver Biopsy

Histopathological evaluation revealed that interface hepatitis, the hallmark of autoimmune hepatitis, was present in 90.38% of patients and showed a strong association with treatment response ($p = 0.002$). Plasma cell infiltration (78.85%) and lobular necroinflammation (69.23%) were also prominent, both demonstrating significant correlations with clinical outcomes ($p = 0.007$ and $p = 0.031$ respectively). More than half of the biopsies (55.77%) showed rosetting of hepatocytes, a typical feature of autoimmune-mediated liver injury, and this too reached statistical significance ($p = 0.048$). Emperipolesis was observed in 40.38% of samples but was not statistically significant. Portal inflammation of moderate to severe grade was noted in 65.38% of patients and significantly correlated with poorer treatment response ($p = 0.016$). Less frequent findings included bile duct injury (23.08%) and steatosis above 5% (19.23%), neither of which showed significant association with outcomes. Regarding fibrosis staging, 34.62% of patients were in stage F2, while 26.92% had F3 fibrosis and 15.38% had cirrhosis (F4). Stages F2, F3, and F4 each demonstrated significant associations with treatment response, indicating that advancing fibrosis negatively affects therapeutic outcomes.

[Table 4] Treatment Response and Clinical Outcomes

A majority of patients achieved complete biochemical remission (65.38%), reflecting good responsiveness to standard immunosuppressive therapy. Partial response was observed in 21.15% of cases, with a statistically significant association ($p = 0.046$), whereas 13.46% of patients exhibited non-response, which was strongly significant ($p = 0.009$), indicating a subset of patients with refractory disease. Among adverse outcomes, progression to cirrhosis occurred in 15.38% of cases during follow-up, showing a significant p-value (0.030). Hepatic decompensation developed in 11.54% of patients ($p = 0.018$), while portal hypertension was recorded in 19.23% with significant correlation ($p = 0.041$). Treatment intolerance was relatively uncommon (9.62%) and did not achieve statistical significance.

[Table 5] Correlation Between Histological Activity and Biochemical Markers

Comparison of biochemical markers between mild and moderate–severe histological activity groups demonstrated clear trends indicating more aggressive disease in the latter. Patients with moderate–severe histological activity exhibited significantly higher ALT (202.44 ± 81.92 U/L) and AST (228.31 ± 103.11 U/L) compared to those with mild activity ($p = 0.004$ and $p = 0.003$ respectively), reinforcing the link between transaminase elevation and hepatic inflammation. IgG levels were also substantially higher in the moderate–severe group (1889.72 ± 254.12 mg/dL vs. 1640.33 ± 208.76 mg/dL), with significant correlation ($p = 0.008$). Total bilirubin was almost double in the higher activity group (4.41 ± 2.33 mg/dL), indicating greater hepatocellular dysfunction, again with a significant p-value (0.011). Serum albumin was lower in the moderate–severe group (2.86 ± 0.71 g/dL), consistent with impaired synthetic function, and showed a statistically significant association ($p = 0.029$).

Table 1: Baseline Demographic and Clinical Characteristics of the Study Population (n = 52)

Parameter	Frequency (n)	Percentage (%)	p-value*
Gender			
Female	41	78.85%	—
Male	11	21.15%	—
Symptoms at Presentation			
Jaundice	34	65.38%	0.041
Fatigue	39	75.00%	0.028
Abdominal Pain	22	42.31%	0.317
Pruritus	18	34.62%	0.201
Clinical Signs			
Hepatomegaly	30	57.69%	0.044
Splenomegaly	19	36.54%	0.152
Ascites	14	26.92%	0.089

*p-value assessed for association with disease severity (mild vs. moderate–severe).

Table 2: Baseline Biochemical and Serological Profile

Parameter	Mean ± SD / Frequency	Percentage (%)	p-value
ALT (U/L)	181.42 ± 78.63	—	0.003
AST (U/L)	204.77 ± 96.51	—	0.001
Total Bilirubin (mg/dL)	3.82 ± 2.14	—	0.012
Serum Albumin (g/dL)	2.98 ± 0.67	—	0.020
INR	1.42 ± 0.31	—	0.033
IgG Elevated	43	82.69%	0.009
ANA Positive	38	73.08%	0.024
ASMA Positive	27	51.92%	0.041

Anti-LKM-1 Positive	5	9.62%	0.622
Anti-SLA Positive	8	15.38%	0.290

*p-value for comparison with histological activity grade (low vs. moderate–severe).

Table 3: Histopathological Findings on Liver Biopsy

Parameter	Frequency (n)	Percentage (%)	p-value*
Interface Hepatitis Present	47	90.38%	0.002
Plasma Cell Infiltration	41	78.85%	0.007
Lobular Necroinflammation	36	69.23%	0.031
Rosetting of Hepatocytes	29	55.77%	0.048
Emperipolesis	21	40.38%	0.114
Portal Inflammation (Moderate–Severe)	34	65.38%	0.016
Bile Duct Injury	12	23.08%	0.502
Steatosis (>5%)	10	19.23%	0.382
Fibrosis Stage F0–F1	12	23.08%	—
Fibrosis Stage F2	18	34.62%	0.041
Fibrosis Stage F3	14	26.92%	0.039
Fibrosis Stage F4 (Cirrhosis)	8	15.38%	0.027

*p-value for correlation with treatment response.

Table 4: Treatment Response and Clinical Outcomes

Outcome Measure	Frequency (n)	Percentage (%)	p-value
Biochemical Response			
Complete Remission	34	65.38%	—
Partial Response	11	21.15%	0.046
Non-Response	7	13.46%	0.009
Adverse Outcomes			
Progression to Cirrhosis	8	15.38%	0.030
Hepatic Decompensation	6	11.54%	0.018
Portal Hypertension	10	19.23%	0.041
Treatment Intolerance	5	9.62%	0.322

Table 5: Correlation Between Histological Activity and Biochemical Markers

Parameter	Mild Activity (n=18) Mean ± SD	Moderate–Severe (n=34) Mean ± SD	p-value
ALT (U/L)	142.67 ± 54.21	202.44 ± 81.92	0.004
AST (U/L)	165.89 ± 72.44	228.31 ± 103.11	0.003
IgG (mg/dL)	1640.33 ± 208.76	1889.72 ± 254.12	0.008
Total Bilirubin (mg/dL)	2.71 ± 1.42	4.41 ± 2.33	0.011
Albumin (g/dL)	3.19 ± 0.53	2.86 ± 0.71	0.029

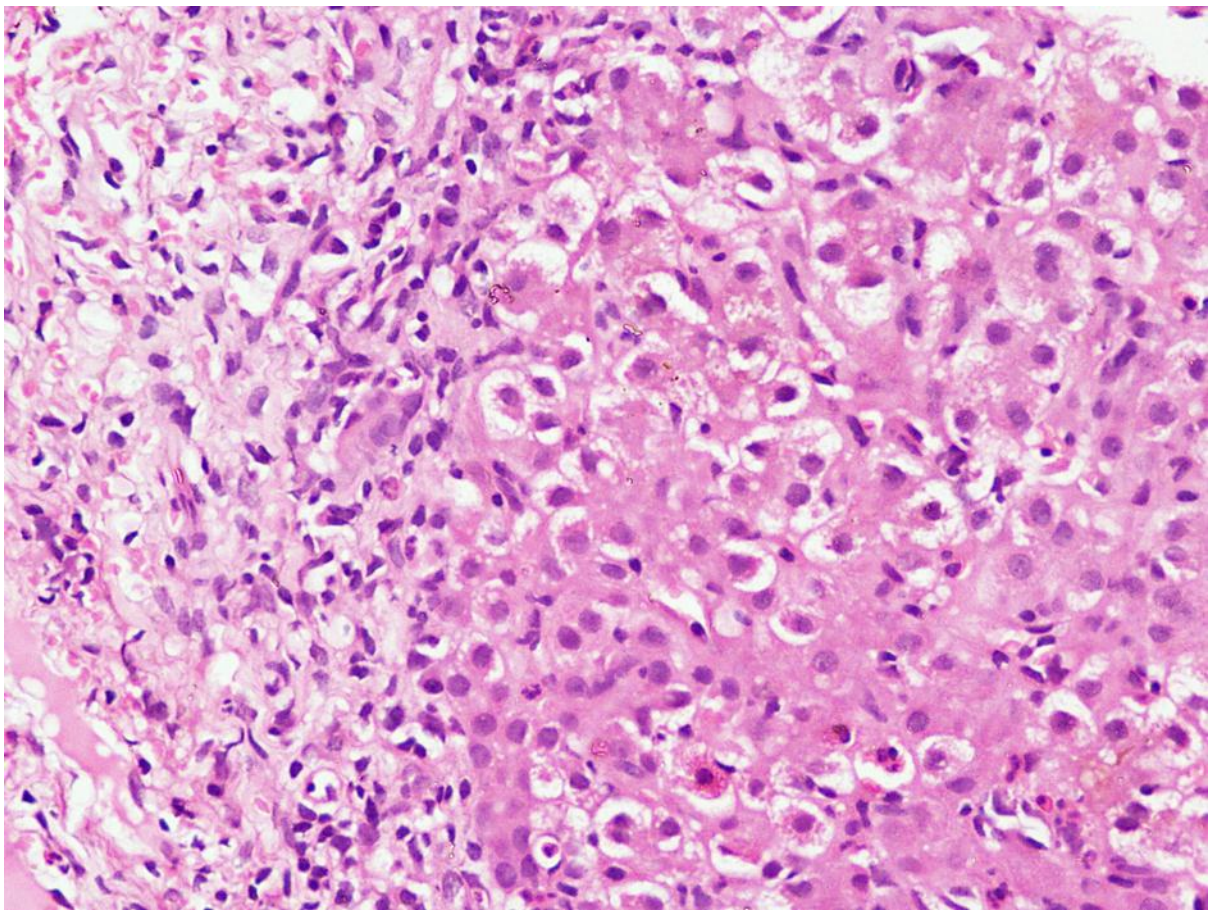

Figure 1: Microphotograph showing interface hepatitis, lobular inflammation, plasma cell infiltration and rosetting of hepatocytes.

DISCUSSION

In this prospective cohort of 52 patients with autoimmune hepatitis, we observed a clear female predominance (78.85%), which is in keeping with the established epidemiological pattern of AIH as a disease affecting predominantly women. Our female proportion is slightly higher than the 74.95% reported in a large German tertiary-center cohort of 535 AIH patients by Buechter et al. (2023), who also noted a mean age in the mid-40s and a substantial burden of autoimmune comorbidities.[7] This concordance supports the external validity of our cohort as a typical AIH population managed in a tertiary-care setting, while the somewhat higher female percentage in our study may reflect referral bias or regional genetic and environmental influences.

The symptom profile in our cohort was dominated by fatigue (75.00%) and jaundice (65.38%), with less frequent abdominal pain (42.31%) and pruritus (34.62%), and both fatigue and jaundice showed significant association with disease severity. This pattern is broadly comparable but somewhat less dramatic than in acute-onset AIH, where Aljumah et al. (2019) reported jaundice in 94% and fatigue in 44% among 70 patients, with markedly higher transaminases (mean ALT 733 U/L and AST 699 U/L) and bilirubin levels, reflecting a fulminant or sub-fulminant disease behaviour.[8] In contrast, our more moderate enzyme elevations (ALT 181.42 U/L, AST 204.77 U/L, bilirubin 3.82 mg/dL) and lower frequency of advanced decompensation suggest that most of our patients had subacute or chronic presentation rather than acute severe disease, yet still exhibited significant clinical and biochemical activity.

Serological patterns in our series—with elevated IgG in 82.69%, ANA positivity in 73.08% and ASMA in 51.92%—are consistent with classical type 1 AIH. When compared with an Egyptian single-centre experience by Alhaddad et al. (2023), who reported ANA positivity in 89.5% and ASMA in 87.7% with a similar female predominance of 73.7%, our autoantibody frequencies are slightly lower but remain within the expected range for AIH.[9] The somewhat reduced ASMA prevalence in our cohort may reflect ethnic or laboratory-method differences, but the overall pattern of hypergammaglobulinaemia with non-organ specific autoantibodies strongly supports the diagnosis in accordance with simplified IAIHG criteria.

Histopathologically, our study showed interface hepatitis in 90.38% of biopsies, plasma cell-rich infiltrates in 78.85%, lobular necroinflammation in 69.23% and hepatocellular rosetting in 55.77%, with moderate–severe portal inflammation in 65.38% and

fibrosis stage F2–F4 in 76.92%. These findings closely mirror the classical description of AIH as a chronic necroinflammatory hepatitis with prominent interface activity, plasma cells and rosettes, as summarised by Manns et al. (2015), who emphasised these lesions as key diagnostic hallmarks distinguishing AIH from other chronic liver diseases.[10] Our relatively high proportion of patients with significant fibrosis (F3 26.92%, cirrhosis F4 15.38%) underscores that many patients present after a prolonged subclinical phase, highlighting the importance of early recognition before advanced fibrosis becomes established.

Treatment outcomes in our cohort were favourable overall, with 65.38% achieving complete biochemical remission and an additional 21.15% attaining partial response, while 13.46% were non-responders. These remission rates are somewhat lower than those reported by Li et al. (2022), who, in a large series of 705 AIH patients, found complete biochemical remission in 80.7% and histological remission in 69.4% of a subset after three years of therapy, particularly in those with lower baseline IgG levels and less advanced fibrosis.[11] Compared with these data, our more modest remission rate likely reflects the inclusion of patients with higher baseline inflammatory activity and a notable proportion with F3–F4 fibrosis, reinforcing the observation that advanced disease stage and pronounced immunologic activity adversely influence the likelihood of full biochemical and histological remission.

The strong association in our study between moderate–severe histological activity and higher ALT, AST, IgG and bilirubin levels, as well as lower albumin, underlines the close link between biochemical markers and intrahepatic inflammatory burden. However, our findings must also be interpreted in light of the work by Dhaliwal et al. (2015), who studied 120 AIH patients in sustained biochemical remission and still found persisting histological activity (Ishak HAI ≥4) in 46%, which was associated with reduced fibrosis regression (32% vs. 60%) and higher all-cause mortality compared with those achieving histological remission.[12] Together, these data suggest that while our biochemical markers reliably reflect more active disease at baseline, persistent microscopic inflammation may remain even when laboratory parameters normalise, supporting the use of histology or validated non-invasive fibrosis assessments for more precise long-term risk stratification.

Despite a substantial proportion of patients with established fibrosis at baseline, our cohort showed relatively favourable intermediate-term outcomes, with complete remission in two-thirds of patients and progression to cirrhosis in 15.38%, hepatic decompensation in 11.54% and portal hypertension in 19.23% during follow-up. These observations are consistent with the broader literature, where conventional corticosteroid-based immunosuppression is reported to induce remission in the majority of AIH patients and substantially improve transplant-free survival, provided that therapy is instituted early and maintained appropriately. Krawitt (2008) highlighted that AIH is a chronic but largely treatable disease in which timely immunosuppressive therapy frequently induces remission and prevents progression to end-stage liver disease, although long-term maintenance and careful monitoring are often required to minimise relapse and treatment-related toxicity.[13] Our data support this paradigm, while also emphasising that a clinically important minority still progresses to advanced cirrhosis and complications, particularly when significant fibrosis is present at diagnosis.

CONCLUSION

This study demonstrates that specific histopathological features particularly interface hepatitis, plasma cell infiltration, and advanced fibrosis strongly correlate with biochemical response and clinical outcomes in autoimmune hepatitis. Patients presenting with higher inflammatory activity and greater fibrosis showed poorer treatment response and increased risk of complications. Integrating detailed biopsy findings with clinical and serological profiles enhances prognostic accuracy and supports individualized therapeutic planning. Early diagnosis and timely initiation of immunosuppression remain critical to improving long-term outcomes in AIH.

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