World Journal of Biology Pharmacy and Health Sciences
eISSN: 2582-5542
Cross Ref DOI: 10.30574/wjbphs
Journal homepage: https://wjbphs.com/

(CASE REPORT)

Autoimmune hepatitis type 1 in a middle-aged female with ulcerative colitis: Navigating the diagnostic overlap and achieving sustained remission

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World Journal of Biology Pharmacy and Health Sciences, 2026, 26(03), 116-123

Publication history: Received on 24 April 2026; revised on 06 June 2026; accepted on 08 June 2026

Article DOI: https://doi.org/10.30574/wjbphs.2026.26.3.0343

Abstract

Autoimmune hepatitis (AIH) is a chronic, immune-mediated inflammatory liver disease that, without timely recognition and treatment, progresses to cirrhosis and liver failure. Type 1 AIH, the predominant subtype, is characterized by the presence of antinuclear antibody (ANA) and/or anti-smooth muscle antibody (ASMA), accounting for approximately 80% of AIH cases worldwide.

We report a 55-year-old Caucasian woman with a background of quiescent ulcerative colitis who presented with a six-week history of progressive fatigue, anorexia, right upper quadrant discomfort, and new-onset jaundice. Laboratory investigations revealed markedly elevated serum aminotransferases (ALT 520 U/L, AST 480 U/L), total bilirubin of 4.2 mg/dL, and significantly elevated serum immunoglobulin G (IgG) at 3,200 mg/dL. Autoimmune serology demonstrated strongly positive ANA (1:320, homogeneous pattern) and ASMA (1:160).

Magnetic resonance cholangiopancreatography (MRCP) excluded primary sclerosing cholangitis. Liver biopsy confirmed moderate-to-severe histological activity with florid lymphoplasmacytic infiltrate, interface hepatitis, emperipolesis, hepatocyte rosette formation, and early bridging fibrosis (Metavir F2). The International Autoimmune Hepatitis Group (IAIHG) simplified score was calculated at 7, confirming definite AIH Type 1. Dual immunosuppressive therapy with prednisone and azathioprine achieved complete biochemical remission by month four.

This case illustrates the diagnostic complexity of AIH Type 1 in a middle-aged woman with concurrent inflammatory bowel disease, underscoring the critical role of multidisciplinary evaluation, systematic overlap exclusion, and structured long-term surveillance.

Keywords: Autoimmune hepatitis; Antinuclear antibody; Anti-smooth muscle antibody; Cirrhosis; Liver Failure; Primary sclerosing cholangitis

1. Introduction

Autoimmune hepatitis (AIH) is a chronic, progressive inflammatory liver disease in which the immune system mounts a sustained attack against hepatocytes, driven by a complex interplay of genetic susceptibility, environmental triggers, and immune dysregulation. [1] Left unrecognized or untreated, it carries a five-year mortality exceeding 50% and a ten-

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year mortality approaching 90% in symptomatic patients. [2] Despite this, the diagnosis is frequently delayed, partly because AIH lacks a single pathognomonic test and its clinical presentation spans a wide spectrum, from asymptomatic enzyme elevation to acute liver failure. [3]

Type 1 AIH, defined serologically by the presence of ANA and/or ASMA, is the most common subtype, accounting for roughly 80% of all diagnosed cases and affecting predominantly women in their fourth to sixth decades of life. [1] Its association with other autoimmune conditions, including inflammatory bowel disease, autoimmune thyroiditis, and rheumatoid arthritis, adds further diagnostic complexity, since concurrent disease can obscure the primary hepatic picture and raise the possibility of overlapping syndromes. [4]

2. Clinical Presentation

2.1. Clinical Presentation, History, Risk Factors, Physical Examination and Initial Laboratory Work

A 55-year-old Caucasian woman with a known history of quiescent ulcerative colitis presented with a six-week history of progressive fatigue, anorexia, mild right upper quadrant discomfort, and new-onset jaundice. She denied alcohol use and had no recent new medications or herbal supplement exposure. Family history was notable for a sister with rheumatoid arthritis and a mother with hypothyroidism, suggesting a familial predisposition to autoimmune conditions. Her personal risk factors included female sex, middle age, a concurrent inflammatory bowel disease, and a positive first-degree family history of autoimmune disease.

Physical examination revealed scleral icterus, mild hepatomegaly without splenomegaly, and spider angiomata on the upper chest. No asterixis or encephalopathy was noted. Initial laboratory investigations demonstrated markedly elevated serum aminotransferases (ALT 520 U/L, AST 480 U/L) with a predominantly hepatocellular pattern, elevated total bilirubin (4.2 mg/dL), and a prolonged prothrombin time (INR 1.4). Serum immunoglobulin G (IgG) was significantly elevated to 3,200 mg/dL. Complete blood count showed mild leukopenia. Alkaline phosphatases were only mildly elevated. Viral hepatitis serologies (hepatitis A IgM, hepatitis B surface antigen, hepatitis C antibody) were all negative.

2.2. Radiological Findings, Special Diagnostic Testing and Differential Diagnosis

Abdominal ultrasound demonstrated a heterogeneous liver echotexture with mild hepatomegaly, without biliary ductal dilation, focal lesions, or portal hypertension. The gallbladder and spleen were unremarkable. Given the concurrent ulcerative colitis, magnetic resonance cholangiopancreatography (MRCP) was performed to exclude primary sclerosing cholangitis (PSC); it revealed normal intra- and extrahepatic bile ducts without strictures or beading, effectively ruling out a PSC-AIH overlap syndrome.

Autoimmune serological testing was central to establishing the diagnosis. ANA returned strongly positive at 1:320 (homogeneous pattern), and ASMA was positive at 1:160. Anti-liver-kidney microsomal antibody type 1 (anti-LKM1) and anti-soluble liver antigen (anti-SLA) were negative. Serum protein electrophoresis confirmed polyclonal hypergammaglobulinemia.

The differential diagnosis included drug-induced liver injury (DILI), viral hepatitis (including EBV and CMV), Wilson disease (ruled out by normal ceruloplasmin and 24-hour urinary copper), PSC-AIH overlap (excluded by MRCP), primary biliary cholangitis (AMA negative), and nonalcoholic steatohepatitis. The serological profile, IgG elevation, and clinical context strongly favored AIH Type 1. The International Autoimmune Hepatitis Group (IAIHG) simplified scoring system was applied, yielding a pre-biopsy score of 6 (Probable AIH).

2.3. Multidisciplinary Board Discussion: Pre-Biopsy Recommendations

The case was presented to a multidisciplinary team comprising hepatology, gastroenterology, pathology, and clinical pharmacy. Given the clinical, biochemical, and serological profile highly suggestive of AIH Type 1, the team unanimously recommended proceeding with percutaneous liver biopsy to confirm the diagnosis histologically, exclude other etiologies, stage fibrosis, and complete the international autoimmune hepatitis group (IAIHG) simplified scoring. Baseline ophthalmology assessment and thiopurine methyltransferase (TPMT) genotyping were requested to determine the patient's genetic ability to metabolize azathioprine in anticipation of immunosuppressive therapy. Dermatology involvement was deferred unless treatment-related adverse effects emerged.

2.4. Pathology, Microscopic Findings, Differential Diagnosis, IHC, Molecular Testing and Final Diagnosis

The liver biopsy yielded two cores of adequate length (18 mm combined), brownish-tan in color, with no macroscopic focal abnormality. Histological sections stained with hematoxylin and eosin demonstrated a florid lymphoplasmacytic infiltrate predominantly involving the portal tracts, with conspicuous interface hepatitis (piecemeal necrosis). The infiltrate extended into the periportal parenchyma, with prominent plasma cell clusters comprising a significant proportion of the inflammatory infiltrate, a hallmark of autoimmune hepatitis. Bridging necrosis was focally present, consistent with moderate-to-severe activity. Infiltration of lymphocytes into and through the hepatocytes (Emperipolesis) was also seen (Figure 1 A, B, C). Hepatocyte rosette formation was identified, representing a classic regenerative response to periportal injury. No granulomas, steatosis, or iron deposition were identified. Reticulin staining demonstrated early bridging fibrosis consistent with Metavir stage F2 Moderate liver fibrosis.

CD38 and MUM1 immunohistochemistry (IHC) revealed an abundant plasma cell infiltrate. CD3 confirmed a background T-lymphocyte-predominant portal inflammation. CK7 and CK19 showed no bile duct loss or ductopenia (excluding PBC). Copper stain was negative. IgG4 immunostain was negative (IgG4-related hepatitis excluded). No additional molecular testing was indicated. TPMT genotype returned wild-type (normal metabolizer), confirming safety for azathioprine use. Final diagnosis was autoimmune hepatitis Type 1, with moderate-to-severe histological activity (Hepatic Activity Index grade 3/4) and early bridging fibrosis (Metavir F2). IAIHG simplified score: 7 (definite AIH ≥ 6).

2.5. Multidisciplinary Board Decisions for Management After Definitive Diagnosis

Following confirmation of definitive AIH Type 1 with moderate-to-severe histological activity, the multidisciplinary team formulated a comprehensive, individualized management plan. As an induction therapy, prednisone was initiated at 40 mg/day with a structured taper over 4–6 weeks, in combination with azathioprine 50 mg/day (titrated to 1–2 mg/kg based on TPMT status and tolerability), consistent with standard dual-agent induction per AASLD (American Association for the Study of Liver Diseases) and EASL (European Association for the Study of the Liver) guidelines. Given the patient's concurrent UC, close coordination with gastroenterology was established. Mesalamine therapy for UC was continued, and colonoscopic surveillance was scheduled in alignment with IBD-related colorectal cancer surveillance guidelines. Given anticipated corticosteroid exposure, calcium and vitamin D supplementation were initiated, along with a baseline DEXA scan to assess bone mineral density.

Monitoring included Liver function tests, IgG levels, CBC, and metabolic panels, scheduled at 4-week intervals during induction and transitioning to every 3 months upon biochemical remission. The treatment response target was normalization of aminotransferases and IgG within 3–6 months. As a remission strategy, the team agreed that if complete biochemical remission were achieved and sustained for ≥ 24 months, gradual withdrawal of corticosteroids with maintenance on low-dose azathioprine monotherapy would be considered.

2.6. Outcome and Follow-Up

The patient achieved complete biochemical remission by month four, with normalization of ALT, AST, bilirubin, and IgG on dual immunosuppressive therapy. Prednisone was tapered successfully and discontinued by month eight, with the patient maintained on azathioprine monotherapy at 1.5 mg/kg/day. She tolerated therapy well without significant adverse effects. Repeating FibroScan (Ultrasound transient elastography) at twelve months demonstrated a reduction in liver stiffness, consistent with histological improvement. Ulcerative colitis remained in clinical remission throughout. After two years of follow-up, the patient remained in sustained biochemical remission. Follow-up remained vigilant for relapses, defined by aminotransferase elevation above three times the upper limit of normal, which would necessitate reintroduction of corticosteroids. Long-term hepatocellular carcinoma surveillance with semi-annual liver ultrasound and AFP was recommended, given underlying fibrosis.

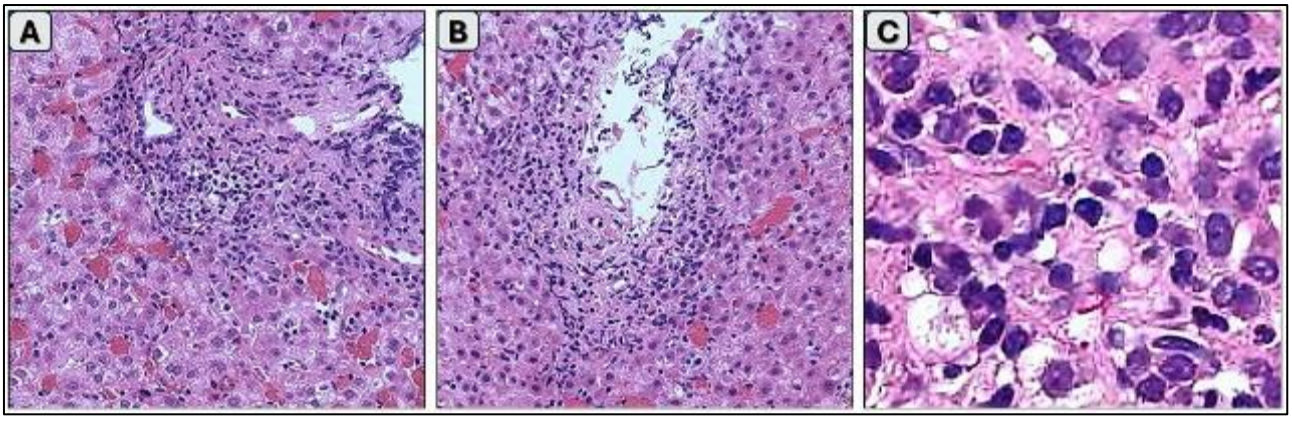

Figure 1 Microscopic findings of autoimmune hepatitis

- 1A: Low-power view showing florid lymphoplasmacytic infiltrate predominantly involving portal tracts, with conspicuous interface hepatitis (piecemeal necrosis) (H&E stain X20).
- 1B: Intermediate power view showing loss of hepatocytes around a central venule due to perivascular confluent necrosis (H&E stain X40).
- 1C: High-power view showing the infiltrate extended into periportal parenchyma, with prominent plasma cell clusters comprising a significant proportion of the inflammatory infiltrate. Infiltration of lymphocytes into and through the hepatocytes (Emperipolesis) is also seen (H&E stain X60).

3. Discussion

3.1. Background (History, epidemiology, risk factors, and WHO classification)

Autoimmune hepatitis was first described in 1950 by the Swedish physician Jan Waldenström, who identified a chronic hepatitis of young women characterized by hypergammaglobulinemia and a favorable response to adrenocorticotropic therapy. [5] In 1956, Ian Mackay introduced the term "lupoid hepatitis" following the discovery of the lupus erythematosus cell phenomenon in these patients. [2] The term "autoimmune hepatitis" was formally adopted by the IAIHG in the 1990s, which also codified the original diagnostic scoring criteria in 1993, revised them in 1999, and introduced the simplified four-parameter scoring system in 2008. [6]

AIH is a rare but increasingly recognized disease. A 2023 systematic review and meta-analysis encompassing 239 million participants across 18 countries reported a global pooled incidence of 1.28 cases per 100,000 inhabitants-years and a prevalence of 15.65 per 100,000 inhabitants, both of which have risen significantly since 1970. [7] Incidence and prevalence are highest in North America, Oceania, and high-latitude regions, and substantially higher in females than males (odds ratio 3.48; 95% CI 2.29–5.29). [7] AIH accounts for approximately 80% of cases like Type 1, which can occur at any age but shows a bimodal distribution, with peaks in adolescence and again between the fourth and sixth decades of life. [1]

Risk factors for AIH Type 1 include female sex, HLA-DR3 and HLA-DR4 haplotypes, a personal or family history of autoimmune disease, and concurrent inflammatory bowel disease, particularly ulcerative colitis. [8] The disease is classified serologically into two subtypes: Type 1 (ANA and/or ASMA positive) and Type 2 (anti-LKM1 and/or anti-LC1 positive), with Type 2 occurring predominantly in children and young adults. [1] Diagnosis is established through a synthesis of clinical, biochemical, serological, and histological criteria, formalized by the IAIHG simplified scoring system, in which a score of 6 indicates probable AIH and a score of ≥7 indicates definite AIH. [6]

3.2. Pathogenesis, Pathophysiology

AIH is a chronic inflammatory liver disease characterized by immune-mediated hepatitis due to the loss of immune self-tolerance. It is thought to be multifactorial. [9] In this case, the patient had multiple risk factors that predispose her to autoimmune disease, including female sex, a past medical history of ulcerative colitis, and a family history of rheumatoid arthritis and autoimmune hypothyroidism. These immune dysregulations predisposed the patient to further autoimmune disease and a loss of tolerance against hepatocyte self-antigens. [1]

This loss of tolerance leads to excess autoreactive T lymphocytes, which mediate inflammatory injury to hepatocytes through direct cytotoxicity. [9] In addition, concurrent activation of B lymphocytes and plasma cells, contribute to the hypergammaglobulinemia of IgG and the formation of specific autoantibodies such as ANA and anti-smooth muscle antibodies. [10,11]

This persistent immune-mediated injury leads to characteristic histopathologic findings, including lymphoplasmacytic infiltrates, interface hepatitis, and bridging necrosis. [10] Chronic inflammation promotes hepatic stellate cell activation, leading to excess collagen deposition, fibrosis, and, eventually, cirrhosis. [12]

3.3. Comparative Analysis of Our Case with Existing Literature.

The clinical and histopathological profile of this patient aligns closely with the established phenotype of AIH Type 1 in middle-aged women, while also illustrating several features that merit specific comparison with published cases and series.

The demographic profile of a 55-year-old Caucasian woman is entirely consistent with the recognized epidemiology of Type 1 AIH. Large registry-based studies and the 2023 global meta-analysis by Hahn et al. confirm that prevalence is highest among adults aged 65+, with a secondary peak in the fifth decade, and that female sex is associated with an odds ratio of 3.48 for disease prevalence. [7] The concurrent ulcerative colitis in our patient reflects a well-established association: AIH is recognized as an extraintestinal manifestation of IBD, and the coexistence of these two immune-mediated conditions in the same patient has been reported across multiple case series [8,13]. Importantly, our case differs from the PSC-AIH-UC overlap syndrome described by Lyu et al. (2023) [13] in that MRCP definitively excluded biliary involvement, establishing isolated AIH in the setting of quiescent UC rather than a triple overlap.

The serological profile, ANA 1:320 (homogeneous) and ASMA 1:160, with negative anti-LKM1 and anti-SLA, is characteristic of Type 1 AIH and mirrors the antibody patterns described in the structured early detection of asymptomatic liver cirrhosis (SEAL) clinical practice guidelines [8] The markedly elevated IgG (3,200 mg/dL) is consistent with the polyclonal hypergammaglobulinemia, a cardinal biochemical feature of the disease, contributing 2 points to the IAIHG simplified score in our patient, as in the majority of published cases.

Histologically, florid lymphoplasmacytic portal infiltrate, interface hepatitis, emperipolesis, hepatocyte rosette formation, and Metavir F2 bridging fibrosis, represent the classical triad of AIH on biopsy. These features align with the histopathological descriptions in the SEAL guidelines [8] and the StatPearls review by Linzay et al. (2023) [1], which identify interface hepatitis and plasma cell-rich infiltrates as the hallmarks of active AIH. The detection of emperipolesis and hepatocyte rosettes in our case is particularly notable, as these features, while not pathognomonic, substantially raise the histological specificity for AIH and are highlighted as characteristic findings in the revised IAIHG scoring literature. [6]

The treatment response in our patient, complete biochemical remission by month four on prednisone and azathioprine, followed by successful steroid withdrawal by month eight, is consistent with published response rates. The EASL guidelines report that approximately 65–80% of patients achieve remission within two years of standard dual-agent therapy [8], and the Kaiser Permanente cohort study by Bittermann et al. (2025) documented a 76% treatment response rate at six months in a large, real-world population. [8,15,16] The FibroScan improvement at 12 months further corroborates the histological reversibility of fibrosis in responding patients, a finding supported by longitudinal biopsy studies in literature [17].

4. What have we learned from This Case?

The case reported here is clinically instructive on several levels. A 55-year-old woman with known ulcerative colitis presented with acute hepatocellular injury and jaundice, a constellation that could plausibly represent drug-induced liver injury, viral hepatitis, or a PSC-AIH overlap. The systematic exclusion of these alternatives, the application of the IAIHG simplified scoring system, and the histopathological confirmation of definite AIH Type 1 together illustrate the diagnostic rigor this condition demands.

This case carries several practical observations that extend beyond the individual patient. The first is diagnostic: new-onset jaundice with a hepatocellular injury pattern in a middle-aged woman with concurrent inflammatory bowel disease should prompt early consideration of AIH, even when the clinical picture initially suggests other diagnoses. The six-week prodrome of fatigue and anorexia, the markedly elevated IgG, the strongly positive ANA at 1:320, and the absence of biliary pathology on MRCP collectively constituted a pre-biopsy profile that should have raised AIH Type 1

to the top of the differential. Clinicians working with IBD patients are expected to recognize that abnormal liver enzymes in this population do not automatically point to PSC; isolated AIH is a distinct and treatable entity that demands its own diagnostic pathway.

The second observation concerns the indispensable role of liver biopsy. The pre-biopsy simplified score of 6 established probable AIH, but histology elevated the score to 7, confirmed the severity of activity, staged the fibrosis, and excluded other diagnoses, including IgG4-related hepatitis and primary biliary cholangitis. No surrogate marker replaces tissue in this disease.

Third, the TPMT genotyping performed before initiating azathioprine exemplifies the precision medicine approach that modern AIH management demands. Identifying wild-type metabolizer status permitted full-dose azathioprine with confidence, contributing directly to the rapid and sustained remission achieved.

Finally, the case reinforces that sustained biochemical remission is an achievable and durable goal. The two-year remission on azathioprine monotherapy, with ongoing HCC surveillance given the underlying F2 fibrosis, reflects the long-term vigilance that AIH requires even after clinical success.

Abbreviations

- Autoimmune hepatitis (AIH)
- Antinuclear antibody (ANA)
- Anti-smooth muscle antibody (ASMA)
- Magnetic resonance cholangiopancreatography (MRCP)
- Primary sclerosing cholangitis (PSC)
- International autoimmune hepatitis group (IAIHG)
- Structured early detection of asymptomatic liver cirrhosis (SEAL)

5. Conclusion

This case demonstrates that AIH Type 1 can present as the sole hepatic diagnosis in a patient with inflammatory bowel disease, without biliary involvement, and that this distinction carries direct therapeutic consequences. Additionally, it illustrates the sequential and structured application of the IAIHG simplified scoring system, MRCP-based overlap exclusion, and IHC-confirmed histopathology as an integrated diagnostic pathway that should be reproducible across clinical settings.

For the medical community, this case adds to the growing body of evidence supporting early biopsy, TPMT-guided immunosuppression, and long-term surveillance as the cornerstones of AIH management in patients with complex autoimmune backgrounds.

Compliance with ethical standards

Acknowledgments

Special thanks to Kush Kanjia, Talina Fuentes, and Malhar Kanojia for their assistance in reviewing the final manuscript. Additionally, we appreciate the assistance of Grammarly's language editor and the Manus 1.6 program, which provided valuable writing support by identifying and correcting errors in grammar, spelling, punctuation, and writing style, ultimately enhancing the manuscript.

Disclosure of conflict of interest

All authors make the following declarations:

- Payment/services information: All authors have declared that they received no financial support from any organization for the submitted work.
- Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

This work did not involve any studies on human or animal subjects performed by the authors. It was a retrospective review of archival pathology material collected during routine clinical care, and all data were fully de-identified before analysis.

Statement of informed consent

Informed consent for the publication of this case was obtained from the patient.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

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