CASE SERIES

Exploring the Diagnostic Complexities of Autoimmune Hepatitis: A Case Series

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ABSTRACT

Introduction: Autoimmune hepatitis (AIH) is a chronic inflammatory disease of the liver of unknown aetiology. The diagnosis of AIH is based on clinical, laboratory, and histological findings in patients with unexplained acute or chronic hepatitis. **Case Series:** We reported four cases of AIH with diverse clinical presentations. All cases exhibited deranged liver enzymes, elevated serum IgG, and similar histological findings, with three cases progressing to liver cirrhosis. However, autoantibody profiles differ between the cases. Two cases showed positivity for liver-specific antibodies (anti-SLA/LP, AMA-M2), one case had ASMA positivity, while another case was negative for autoantibodies typically associated with autoimmune liver disease. **Conclusion:** The consistent IgG elevation highlights its diagnostic value in AIH, while the diverse autoantibody profiles underscore the need to explore potential influences, including sociodemographic, clinical, molecular, and environmental factors, to improve understanding and management of AIH.
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INTRODUCTION

Autoimmune hepatitis (AIH) is a chronic liver disease of unknown cause, characterised by liver inflammation and damage. Diagnosis combines clinical, laboratory, and histological findings in patients with unexplained liver dysfunction. AIH can affect individuals of all ages, genders, and ethnicities, presenting with symptoms ranging from mild to severe liver failure. Diagnosis is often challenging due to overlaps with other liver and autoimmune diseases [1].

There is no single universal scoring system for diagnosing AIH. Various scoring systems with different sensitivity and specificity have been developed to aid clinical decision-making, including the revised score from 1999 and the simplified score from 2008 by the International Autoimmune Hepatitis Group (IAIHG). The simplified score, widely used in clinical practice, includes four parameters: autoantibody detection, IgG levels, liver histopathology, and absence of viral hepatitis markers [2]. However, serological markers are not always reliable, as a subset of patients may lack detectable autoantibodies. This presents a diagnostic challenge, particularly in cases of seronegative AIH. Studies have shown that 7% to 34% of AIH patients fall into this seronegative category, with an average prevalence of approximately 10% [3].

The treatment aims to achieve biochemical remission,

normalise aminotransferases and IgG within six months, and maintain this improvement. Early and accurate diagnosis is crucial for timely immunosuppression to prevent further liver damage [4]. We present four cases of AIH with diverse clinical features and associated conditions, highlighting the diagnostic challenges of this disease.

CASE SERIES

Case 1

A 65-year-old Malay man with diabetes, hypertension, dyslipidaemia, and benign prostate hyperplasia was referred to the gastroenterology clinic for deranged liver function tests (LFTs) persisting for three months. He reported experiencing fatigue for the same duration and tea-coloured urine for the past one month. He denied alcohol consumption, use of herbs, supplements, or over-the-counter medications. His statin and oral alfuzosin were discontinued two and four months prior, respectively, after the deranged LFTs were noted. The patient had a history of receiving two courses of antibiotics, i.e., oral amoxicillin for fifteen days and oral norfloxacin for five days, for a urinary tract infection one month before the onset of liver derangement. At presentation, he appeared comfortable, having normal vital signs and without jaundice. An abdominal examination revealed hepatosplenomegaly without ascites.

Laboratory tests showed bicytopenia, prolonged prothrombin time (PT) with normal international normalised ratio (INR) and abnormal LFT (Table I). Ultrasonography of the hepatobiliary system (US HBS) revealed diffuse hepatic steatosis without focal liver lesions. His liver biopsy showed periportal and lobular hepatitis, confluent necrosis, and bridging fibrosis. Autoimmune serology tests came back as positive for antinuclear antibody (ANA) and anti-smooth muscle antibody (ASMA), with elevated serum IgG. A diagnosis of AIH with a simplified AIH score of eight, complicated by liver cirrhosis (Child-Pugh B), was made. The diagnosis was supported by findings of thrombocytopenia (platelet < $150x10^9$/L) and bridging fibrosis on liver biopsy. The patient was started on oral prednisolone 40 mg once daily (OD) with a tapering dose for over six months and low-dose azathioprine 50 mg OD for two months and subsequently increased to 75 mg OD. After eight months of treatment, he became asymptomatic and his LFTs began to normalise. He is currently on a regular two-monthly follow-up and has not shown any evidence of steroid toxicity to date.

Table I: Comparison of the clinical and laboratory findings of autoimmune hepatitis cases

	Case 1	Case 2	Case 3	Case 4	Reference Range
Demographics					
Age at time of diagnosis	65 years old	47 years old	56 years old	66 years old	
Gender	Male	Female	Female	Female	
Clinical features					
Fever	Nil	Nil	Nil	Yes	
Jaundice	Yes	Yes	Yes	Yes	
Abdominal distension	Nil	Nil	Nil	Yes	
Laboratory tests					
Hemoglobin (g/dL)	12.2	11.2	11.6	11	11.5-15.5
Platelets ($x10^9$/L)	86	301	136	69	170-450
Leukocytes($x10^9$/L)	3.48	5.22	12.4	5.9	5.00-13.00
Total Bilirubin (umol/L)	52.8	74.7	290	338.5	5-21
Direct Bilirubin (umol/L)	39.4	46.4	178.3	245.8	0-5
INR	1.22	1.15	1.16	1.74	
AST (U/L)	273	486	702	2684	0-34
ALT (U/L)	655	573	512	2688	10-72
Hepatitis B and C screening	Non-Reactive	Non-Reactive	Non-Reactive	Non-Reactive	

CONTINUE

Table I: Comparison of the clinical and laboratory findings of autoimmune hepatitis cases (CONT.)

	Case 1	Case 2	Case 3	Case 4	Reference Range
Autoimmune hepatitis work-up					
ANA pattern (titer)	Positive Homogenous (1:1280)	Positive Homogenous (1:1280)	Negative	Positive Homogenous (1:1280)	
Anti-dsDNA	Not Done	Positive	-	Not Done	
Anti-LKM	Negative	Negative	Negative	Negative	
ASMA (titer)	Positive (1:160)	Negative	Negative	Negative	
AMA	Negative	Negative	Negative	Negative	
Anti-SLA/LP	Negative	Negative	Positive	Negative	
AMA-M2	Negative	Negative	Negative	Positive	
Serum IgG (g/L)	33.2	33.25	42.2	30.66	7-16
Diagnosis	Definite AIH	Probable AIH	Probable AIH	Probable AIH	
IAIHG Score	8	6	6	6	
Overlap Syndrome	Nil	SLE	Nil	PBC	
Complication	Liver cirrhosis	Nil	Liver cirrhosis	Liver cirrhosis	
Treatment					
Oral Steroids	Yes	Yes	Yes	Yes	
Other Medications	AZT	AZT	AZT	AZT and UDCA	
Response/Outcome	Symptom-free with normalized LFT	Flare due to defaulted treatment	Symptom-free with improved LFT	Symptom-free with normalized LFT	

INR: International normalized ratio, AST: Aspartate transaminase, ALT: Alanine transaminase, ANA: Antinuclear antibody, Anti-dsDNA: Anti-double stranded DNA, Anti-LKM: Anti-liver kidney microsome antibody, Anti-SMA: Anti-smooth muscle antibody, Anti-SLA/LP: Anti-soluble liver antigen/liver-pancreas antigen-antibody, AMA: Anti-mitochondrial antibody, IgG: Immunoglobulin G, IAIHG: International Autoimmune Hepatitis Group, SLE: systemic lupus nephritis, PBC: primary biliary cirrhosis, AZT: Azathioprine, UDCA: Ursodeoxycholic acid, LFT: Liver function test, IAIHG score ≥6: probable AIH, score ≥7: definite AIH.

Case 2

A 47-year-old Malay woman with underlying toxic thyroid adenoma and a history of transaminitis presented with swelling, pain, and stiffness of the joints of her bilateral fingers and wrists. Elevated transaminase levels, with alanine aminotransferase (ALT) at 306 U/L (10–72 U/L) and aspartate aminotransferase (AST) at 259 U/L (0–34 U/L), had prompted a referral to a gastroenterologist. Further investigations revealed elevated serum IgG with normal IgM, complement 3 (C3), complement 4 (C4), and ceruloplasmin (Table I). Autoantibody testing showed a positive ANA (1:1280, homogenous pattern), along with positive anti-ribonucleoprotein (anti-RNP) and direct Coombs test, while antineutrophil cytoplasmic antibodies (ANCA), antimitochondrial antibodies (AMA), ASMA, and anti-liver-kidney microsome antibodies (anti-LKM) were negative. The US HBS findings were unremarkable. Based on these findings, the patient was diagnosed with systemic lupus erythematosus (SLE) and probable AIH, with a simplified AIH score of six.

Liver biopsy was initially deferred as corticosteroid therapy was initiated for arthralgia and cutaneous vasculitis. She remained clinically stable for a few months but presented again approximately nine months later with symptoms of lethargy and significantly deranged LFT following a period of non-compliance with treatment. A liver biopsy at that time revealed moderate interface and lobular hepatitis with bridging fibrosis, lymphoplasmacytic infiltrates, hepatic rosettes, and emperipolesis.

The patient was started on oral prednisolone 20 mg OD for five months, which was gradually tapered to a maintenance dose of 5 mg OD. Azathioprine 50 mg OD was added after two months but was later reduced to 25 mg OD due to side effects, including dry mouth and dizziness. While the initial corticosteroid therapy improved her condition, she experienced a flare in liver dysfunction following treatment discontinuation. Upon reinitiation of prednisolone and later azathioprine, her liver function stabilised, albeit with mild transaminitis. She continues regular follow-ups with both a gastroenterologist and a rheumatologist. Her SLE is currently stable without significant side effects from the adjusted treatment regimen.

Case 3

A 56-year-old Malay woman with a history of gallstones, dyslipidaemia, and hypertension presented with yellowish sclera, tea-coloured urine, and deranged LFTs for five months (Table I). She denied loss of weight, loss of appetite, prior history of jaundice, blood transfusion, or high-risk behaviours. She had a one-year history of taking traditional medication and was on oral statins, both of which were discontinued following the discovery

of her liver function derangement. On examination, she had jaundice of sclera and finger clubbing but no flapping tremors or hepatomegaly. US HBS showed a hypoechoic liver parenchyma with prominent portal radicals, suggestive of hepatitis changes, and cholelithiasis.

Her serum IgG was elevated at 42.2 g/L (7–16 g/L). Autoimmune and metabolic workup, including ANA, AMA, ASMA, anti-LKM, ceruloplasmin, and 24-hour urine copper, was negative. Liver biopsy revealed marked bridging fibrosis, lymphoplasmacytic infiltration, interface hepatitis, hepatitis rosettes, and emperipolesis, strongly supporting AIH. Anti-soluble liver antigen/liver-pancreas antigen antibody (anti-SLA/LP) tested positive three months later. A diagnosis of definite AIH was made, with a simplified AIH score of eight.

Treatment was initiated with oral prednisolone 40 mg OD and tapered over time. Oral azathioprine 50 mg OD was added after one month and gradually increased to 125 mg OD. The patient tolerated treatment without gastrointestinal symptoms, fever, or abdominal pain, though a transient increase in bilirubin was noted during prednisolone tapering. A follow-up US HBS after two months showed liver cirrhosis. Despite this, ALT levels improved and normalised after eight months of therapy. The patient is currently asymptomatic and continues regular follow-up every two months, including blood monitoring, US HBS, and liver cirrhosis surveillance.

Case 4

A 70-year-old Malay woman with diabetes, hypertension, and dyslipidaemia, on statin, presented with a two-week history of jaundice, fever, and abdominal distension. Initial blood investigations revealed elevated liver transaminases and bilirubin levels (Table I). She had positive p-ANCA and mildly elevated serum IgG levels, while ANA, ASMA, AMA, anti-LKM, and viral hepatitis markers were negative. US HBS showed liver cirrhosis with ascites. Liver biopsy revealed acute hepatitis, bile duct proliferation, and bile stasis. Following a month of hospitalisation, she was diagnosed with cryptogenic liver cirrhosis.

Two years later, the patient was referred to a gastroenterologist due to persistently abnormal LFTs. Repeated ANA testing was positive (homogeneous pattern, 1:1280), with serum IgG elevated at 30.66 g/L. Other liver autoantibodies remained negative. However, her second liver biopsy was inconclusive. She was managed as probable AIH with liver cirrhosis (Child-Pugh B) and a simplified AIH score of six. Clinical evidence of cirrhosis included a low platelet count (Table I) and irregular liver margins on US HBS.

Treatment with oral prednisolone 30 mg daily was initiated and gradually tapered. However, during tapering, worsening LFTs necessitated an increase in the prednisolone dose. Subsequent investigations revealed positive AMA-M2, elevated gamma-glutamyl transferase (GGT), and ALT levels, meeting the Paris criteria for AIH-primary biliary cholangitis (PBC) overlap syndrome. Ursodeoxycholic acid (UDCA) 500 mg twice daily was added to her regimen alongside prednisolone and azathioprine. This combination therapy resulted in improved LFT and resolved her symptoms. Over two years, prednisolone was tapered, and the patient transitioned to oral azathioprine 25 mg OD. Due to liver cirrhosis (Child-Pugh A), she was later maintained on azathioprine 75 mg OD and low-dose prednisolone 5 mg OD. LFT normalised within two months, and she remained asymptomatic during regular follow-ups.

All patients tested negative for hepatitis (A, B, and C), HIV, Epstein-Barr virus, and cytomegalovirus. Additionally, ceruloplasmin levels, iron studies, and alpha-1-antitrypsin levels were normal. None of the patients tested positive for anti-LKM antibodies.

DISCUSSION

AIH is a complex condition influenced by genetic, epigenetic, immunologic, and environmental factors. While genetics play a role, environmental factors like viral infections or exposure to certain substances can also provoke AIH in genetically predisposed individuals [1]. For instance, the first case had a history of urinary tract infections, potentially triggering the autoimmune response. Although no specific environmental triggers were identified in our cases, lifestyle factors such as smoking, alcohol consumption, and diet could potentially influence the development and progression of autoimmune diseases.

Globally, AIH prevalence is 17.4 per 100,000 (12.77 for females and 2.91 for males), with a lower prevalence rate in Asia (12.99/100,000) compared to Europe (19.44/1,000,000) [3]. Our series mirrors this pattern, as observed in other studies, including a Danish cohort, which reported that AIH was more common in women [5]. In East Asian countries such as Japan, Taiwan, Korea, and China, the female-to-male ratio ranges from 3.4:1 to 7.3:1. In Southeast Asian countries like Singapore and Brunei Darussalam, the female predominance is even more pronounced, with female-to-male ratios of 11:1 and 3.75:1, respectively [6]. Most studies describe a bimodal age distribution for AIH, with peaks occurring during childhood or adolescence and another between the fourth and sixth decades of life. Recent studies highlight an increasing number of cases in patients over 60 years old [7]. Similarly, in our series, two of the cases involved individuals aged over 65.

Ethnic differences impact both the prevalence and the types of autoantibodies observed in AIH. For example, Asian populations tend to have lower rates of anti-SLA antibodies, while anti-LKM is more commonly

found in younger patients in certain ethnic groups [1]. Additionally, the use of medications such as statins or traditional medicine can also contribute to liver damage, making it challenging to distinguish AIH from other liver conditions. Taking a thorough history is important to rule out medication-induced liver injury.

AIH typically presents with fatigue, jaundice, abdominal discomfort, and hepatomegaly with elevated liver enzymes. However, many patients are asymptomatic or have only mild symptoms, with abnormal liver enzymes. These liver enzymes are typically the earliest indicators of liver inflammation, often detected incidentally during routine medical checkups. Diagnosing AIH is challenging, as symptoms may not appear until months or even years of silent progression. Even after clinical symptoms have manifested, a subclinical phase often persists, further complicating timely diagnosis [8].

The immune response in AIH is driven by T cells, causing liver cell damage and increased IgG production. About 85% of patients have elevated IgG levels at diagnosis [9], which was consistent in all our cases. One study found that IgG levels over twice the normal limit were linked to higher liver enzymes, bilirubin, and inflammation. An IgG level above 1.3 times the normal limit improves diagnostic accuracy and supports its inclusion in updated criteria [10]. This distinction is important for differentiating AIH from other liver conditions, especially advanced cirrhosis, where similar IgG elevations may arise from different causes. Monitoring IgG levels over time also aids in evaluating treatment response and disease progression [9].

Autoantibodies, such as ANA and ASMA, play a critical role in diagnosing AIH. ANA is the most sensitive but least specific marker, as it can be found in various liver conditions, including fatty liver disease, drug-induced liver injury (DILI), viral hepatitis, Wilson disease, and alcoholic liver disease. Additionally, ANA can also be detected in healthy individuals [2]. In the context of AIH, ANA titres of ≥ 1:40 in adults and ≥ 1:20 in children are considered positive. In our case series, ANA was positive in three out of four cases, with titres exceeding 1:40. ASMA is frequently detected alongside ANA, with isolated ASMA observed in approximately 35% of AIH cases [2]. However, neither ANA nor ASMA is definitive for AIH. Their presence must always be interpreted alongside the patient's clinical findings and liver biopsy results.

Although serological markers such as ANA, ASMA, and anti-LKM-1 are valuable diagnostic tools, their absence does not exclude AIH. As in our third case, the detection of anti-SLA/LP confirmed the diagnosis of AIH, despite the absence of other typical autoantibodies [2]. Anti-SLA is highly specific for AIH and is present in approximately 14–20% of cases. Patients with anti-SLA/LP positivity have been associated with more severe disease and a higher risk of relapse after withdrawal of treatment [1]. Therefore, these patients often require lifelong treatment [2]. Beyond its diagnostic utility, the presence of anti-SLA/LP serves as an important prognostic indicator for higher disease severity and relapse risk, necessitating vigilant long-term follow-up and potentially lifelong immunosuppressive therapy in such patients.

In rare instances, AIH may overlap with other autoimmune conditions such as SLE or PBC. Liver derangement in SLE could be due to AIH or lupus hepatitis, a secondary manifestation of SLE caused by immune complex deposition. This association is rare, with one study reporting a prevalence of 1.3%, while another study reported varying prevalence rates of 4.7% [11]. According to the revised simplified AIH diagnostic criteria, compatible histological findings remain essential. Histologically, AIH typically shows interface hepatitis with plasma cell infiltration, whereas lupus hepatitis displays nonspecific changes such as steatosis or hydropic degeneration and predominantly lymphocytic infiltrates [12]. These distinctions highlight the role of liver biopsy in differentiating the two conditions. In our second case, despite a known diagnosis of SLE and suspected AIH, liver biopsy was postponed until steroid tapering to enhance diagnostic accuracy. Although this introduces some variation in baseline evaluation, the diagnosis was supported by consistent clinical, serological, and imaging findings. The subsequent biopsy confirmed AIH, reinforcing diagnostic certainty [9].

Overlap syndromes occur in 3% to 7% of autoimmune liver disease patients, especially those with AIH and PBC. A positive AMA test may suggest an overlap syndrome or AMA-positive AIH, often pointing to a PBC variant [13]. The clinical course and treatment response in these cases are generally similar to classic AIH. The Paris criteria provide a structured approach to diagnosing overlap syndrome, especially when liver biopsy findings are inconclusive. By fulfilling specific biochemical, serological, and histological criteria, clinicians can make a clearer diagnosis and guide appropriate treatment. Literature reviews highlight that overlap syndromes may be linked to higher rates of treatment failure or the need for combination therapies [14]. This overlap variant should be considered in patients with AIH who do not respond well to standard immunosuppressive therapy. In such cases, the addition of UDCA, like in our fourth case, may be beneficial.

Autoantibodies are a crucial diagnostic tool in AIH. When the diagnosis remains uncertain, a second-line liver pathology workup is necessary. The presence of one or more relevant autoantibodies, along with the appropriate clinical context, strongly supports the need for a liver biopsy, as AIH becomes highly probable [1]. However, access to autoimmune serology testing is not universally available, particularly in resource-limited

settings. Therefore, enhanced training and expertise among laboratory personnel and clinicians are essential for the accurate interpretation of autoantibody results [15].

Standard treatment involves corticosteroids, with or without azathioprine, inducing remission in approximately 80–90% of patients [16]. However, ongoing monitoring is essential, as treatment needs to be tailored to disease activity and personalised for specific patient groups, including those with comorbidities [9]. In our first case, despite the presence of cirrhosis, the combination of prednisolone and azathioprine led to symptom resolution and the normalisation of LFT. While most patients respond well and become symptom-free, 10–20% may experience suboptimal response or drug-related adverse effects, necessitating treatment modification [17]. Similarly, in the SLE-AIH overlap case, combination therapy resulted in sustained remission. This aligns with previous reports showing that standard AIH regimens are generally effective in overlap syndrome. Nonetheless, response in such cases can be variable, and escalation to second-line therapy, such as mycophenolate mofetil or rituximab or even liver transplantation, may be required in refractory disease [18, 19]. Long-term follow-up is important to monitor disease relapse, assess medication side effects, and evaluate quality of life, which is a key outcome in chronic autoimmune diseases. These challenges highlight the need for multidisciplinary care and customised treatment plans.

A key limitation of our series is the small number of cases. These findings should be regarded as preliminary observations that may help guide future research. Larger studies are needed to enhance our understanding of the disease and inform more effective treatment strategies [4]. Given the variability in autoantibody profiles and consistent IgG elevation, further studies could examine how these markers can help monitor disease activity and predict relapses. Our study suggests that genetic and environmental factors might influence AIH. Future research should explore these factors to better understand the disease's causes, especially in this specific population.

CONCLUSION

The consistent elevation of IgG in these patients highlights its importance as a diagnostic marker for AIH. This finding supports the value of IgG measurement in the diagnostic workup, particularly for patients with unexplained liver dysfunction. Additionally, it also suggests a shared immunological pathway in AIH, paving the way for further research into targeted therapeutic approaches. A deeper understanding of the role of IgG, along with the exploration of various sociodemographic, clinical, molecular, and environmental factors, is crucial for enhancing early diagnosis and advancing customised treatment strategies.

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