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ORIGINAL ARTICLE

Diagnostic and prognostic evaluation of patients with autoimmune hepatitis: about 176 cases

Mohamed Lotfi BOUDJELLA[1], Mohamed El-Amine BOUDJELLA[2], Wissem BELLAGH[1], Mohamed Abderaouf KHALOUF[3], Djihane BOUTEFLIKA[4], Youcef CHADI[1], Farouk MENZOU[4], Nardjes SERIDJ[3], Nabila NAILI[5], Malika BOUALI-BENHALIMA[6], Djamila SIAHMED-BOUALI[7], Ali MEGHLAOUI[1]

ABSTRACT

Introduction: Autoimmune hepatitis (AIH) is an immune-mediated inflammatory liver disease that may present in either acute or chronic forms. It predominantly affects genetically predisposed individuals and may progress to cirrhosis in the absence of appropriate treatment. This study aimed to investigate the clinical, immunological, and prognostic characteristics of AIH in an Algerian population. **Patients and methods:** We conducted a retrospective, cross-sectional, descriptive, and analytical study involving 176 patients with a clinical suspicion of AIH who were recruited between September 2012 and December 2025 at the Immunology Unit of Blida University Hospital. Patients were diagnosed according to the International Autoimmune Hepatitis Group (IAIHG) scoring system, which incorporates immunological criteria (titers of specific autoantibodies and serum IgG levels), histological findings, and the exclusion of viral hepatitis. **Results:** Among the 176 patients diagnosed with AIH, 130 (74%) were women and 46 (26%) were men, yielding a female-to-male ratio of approximately 3:1. The overall mean age was 53.0 ± 11.6 years (59.3 ± 13.3 years in women and 37.5 ± 9.1 years in men). The most frequent laboratory abnormalities involved serum IgG (45%), albumin (23%), and IgM (19%). Anti-smooth muscle antibodies (ASMA) were detected in 129 patients (73.3%), while anti-liver kidney microsomal type 1 (anti-LKM-1) antibodies were identified in 20 patients (11.4%). Anti-LC-1 antibodies were detected exclusively in patients with AIH type 2. Regarding associated autoimmune diseases, 11 patients presented with AIH-primary biliary cholangitis (PBC) overlap syndrome confirmed by positive antimitochondrial antibodies, eight had autoimmune thyroid disease, three had systemic lupus erythematosus, four had sicca syndrome, and six had confirmed antiphospholipid syndrome. Older age at diagnosis was a significant prognostic factor for cirrhosis, as delayed diagnosis was associated with an eightfold higher risk of developing cirrhosis compared with early diagnosis. **Conclusion:** In our series, the detection of disease-specific autoantibodies remains a cornerstone in the diagnosis of AIH and is also valuable for identifying associated autoimmune disorders. Despite advances in diagnosis, AIH continues to carry a poor prognosis, underscoring the need to identify reliable biomarkers capable of predicting unfavorable outcomes.

Keywords: Autoimmune hepatitis, autoantibodies, diagnosis.

(1) Faculty of Medicine of Blida, Blida 1 University, Department of Medical Biology, EHS Organ and Tissue Transplantation, Blida. (2) Faculty of Medicine of Algiers, Benyoucef Benkhedda University, Department of Internal Medicine, Ibn Ziri University Hospital, Algiers. (3) Faculty of Medicine of Blida, Blida 1 University, Department of General Surgery, EHS Organ and Tissue Transplantation, Blida. (4) Faculty of Medicine of Blida, Blida 1 University, Department of Internal Medicine, Douera University Hospital. (5) Faculty of Medicine of Blida, Blida 1 University, Department of Forensic Psychiatry, Frantz Fanon Psychiatric Hospital. (6) Faculty of Medicine of Algiers, Benyoucef Benkhedda University, Mustapha Bacha University Hospital, Algiers. (7) Faculty of Medicine of Algiers, Benyoucef Benkhedda University, Algiers 1, Department of Internal Medicine, Mustapha University Hospital.

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Correspondence to: Mohamed Lotfi BOUDJELLA
E-mail: mlotfii021@gmail.com

1. INTRODUCTION

Autoimmune hepatitis (AIH) is a chronic inflammatory liver disease characterized by the association of hepatic cytolysis, hypergammaglobulinemia, the presence of specific auto-antibodies (auto-Abs), and histological abnormalities. In the absence of treatment, AIH can progress to a picture of cirrhosis with severe liver failure and death. [1] AIH is a female pathology regardless of the type (75 to 80% of patients are women), occurs at any age (children and adults), and affects all ethnicities. More recent incidence values are increasing and probably more precise; this incidence is estimated at 1.5 cases in Japan, 1.68 cases in Denmark, 2 cases in New Zealand, and 3 cases in the United Kingdom per 100,000 individuals per year [1, 2, 3]. In Algeria, there are few studies on the prevalence and incidence of AIH known to date. The etiopathogenic mechanism of AIH 1 and 2 involves several factors, first infections (viral and bacterial), the use of drugs or xenobiotics; and genetic factors (HLA-A*01, HLA-B*08, HLADRB1*0301 or HLADRB1*0401/0405), the latter being at the origin of the triggering of autoimmunization. [1] AIH is characterized by anatomo-clinical and immunobiological polymorphism, but the presence of auto-Abs associated with different categories of AIH would be a valuable aid at the diagnostic and sometimes prognostic level, justifying their interest in clinical practice [1].

The initial presentation may include asthenia, jaundice, headaches, anorexia, abdominal pain, and, at an advanced stage, affected patients may present signs of cirrhosis with portal hypertension. [1] Two main entities have been described: classical type 1 AIH, which mainly affects adults, often associated with other autoimmune diseases (AD) and characterized by the presence of anti-smooth muscle auto-Abs (ASMA) of anti-f-actin specificity, anti-nuclear antibodies (ANA) and anti-SLA auto-Abs (anti-soluble liver antigen). While type 2 AIH is rare, affects children, and is characterized by the presence of anti-LKM1 auto-Abs (Liver-Kidney-Microsome), anti-CYP2D6 auto-Abs and/or anti-liver cytosol type 1 auto-Abs (anti-LC1) and often presents with a severe picture from the outset. [1, 2, 3] The treatment of AIH is based on the combination of corticosteroids and immunosuppressants, which controls the disease in most cases. A good therapeutic response is accompanied by a considerable improvement in prognosis. In some cases, liver transplantation is indicated, especially in (sub)fulminant forms resistant to high-dose corticosteroid treatment or in cases of cirrhosis accompanied by severe complications [4]. AIH accounts for approximately 5% of indications for transplantation for cirrhosis. The survival rate after transplantation is, on average, 80% at 2 years. [5] The disease can recur in 40% of cases; this recurrence, which can manifest in different forms (sometimes severe), is favored by insufficient immunosuppression or by a graft carrying the HLA DR3 haplotype. [6]

In this study, our objective was to determine the immuno-clinical characteristics and prognosis of markers associated with AIH in an Algerian population.

2. PATIENTS AND METHODS

This is a cross-sectional, retrospective, descriptive, and analytical study conducted on a sample of the Algerian population. We analyzed 2356 files of patients presenting a clinical picture suggestive of AIH recruited from September 2012 to December 2025 at the immunology unit of Blida University Hospital. In total, 176 selected patients were diagnosed with confirmed AIH, according to the diagnostic score of the International AIH Group, including immunological criteria (the titer of specific auto-Abs and the IgG level), histological criteria, and the absence of viral hepatitis.

The diagnosis of AIH was established according to the diagnostic scoring system proposed by the International AIH Group. This scoring system is based on the evaluation of several criteria: the presence and titer of specific autoantibodies (ANA, SMA, anti-LKM-1, anti-SLA), the serum immunoglobulin G (IgG) level expressed as a multiple of the upper limit of normal (ULN), liver histology findings (compatible or typical of AIH), and the absence of any viral hepatitis. Each criterion is assigned a specific number of points. A total score ≥ 7 allows a definite diagnosis of AIH, while a score ≥ 6 corresponds to a probable diagnosis.

All selected patients underwent specific serum protein assays (albumin and immunoglobulins (IgG, IgA and IgM)) by turbidimetry on a SPA Plus® automaton, a liver workup (assay of liver enzymes AST and ALT) on a Myndray 240 Pro® automaton, and a serological workup (hepatitis A, B and C). Patients also underwent an autoimmunity workup (INOVA® Kit) searching for auto-Abs specific to autoimmune liver involvement by indirect immunofluorescence (IIF) and ELISA techniques. An initial screening by IIF on triple substrate (rat liver, kidney, stomach, or LKS) followed by an ELISA test searching for anti-actin auto-Abs, anti-SLA auto-Abs and a Dot test (Immunodot Top Screen) searching for anti-LC1 auto-Abs. We also performed a search for auto-Abs specific to other ADs, such as the search for anti-nuclear auto-Abs (ANA) by IIF and ELISA, the search for anti-TPO and anti-Tg auto-Abs, and the search for anti-phospholipid Abs.

Ethical approval : This study was conducted in accordance with the ethical standards of the Helsinki Declaration. All patients provided informed consent for the use of their medical records for research purposes. Data were anonymized and de-identified prior to analysis to ensure patient confidentiality.

Statistical analyses

Descriptive statistics: Quantitative data are expressed as mean ± standard deviation, accompanied by extremes. Description of qualitative variables is summarized as the number of patients and/or percentage.

Analytical statistics: GraphPad statistical software was used for data analysis. If the Chi-square test (p-value) obtained is less than the chosen risk threshold (often 5% or 0.05), the test is considered significant. OR and 95% confidence interval were determined according to Fisher's exact test. The p-value was corrected according to Yates' correction (for small samples). The difference is considered significant from $p<0.05$. Calculations were performed using GraphPad 8 software and Excel 2016.

3. RESULTS

Among the 176 patients with AIH, 130 were women (74%) and 46 men (26%) with a sex ratio of 2.8F/1H. The mean age of our 176 patients was 53.56 ± 11.62 (59.27 ± 13.32 in women and 37.48 ± 9.10 in men). Regarding the age distribution, the most affected age group for type 1 AIH was 41-60 years, accounting for 60 patients (39.47%). For type 2 AIH, the most affected age group was under 20 years, comprising 11 patients (45.83%). In the 21-40 years age group, 39 patients (25.65%) had type 1 AIH and 9 patients (37.5%) had type 2 AIH. Patients over 61 years of age were predominantly affected by type 1 AIH, with 44 patients (28.94%), compared to only 2 patients (8.33%) with type 2 AIH.

Distribution of patients according to presenting clinical signs: In our study series, 11 patients (6.25%) were asymptomatic at the time of diagnosis, and 165 patients (93.75%) presented clinical and/or biological symptoms. The most frequent symptoms were asthenia (81%), weight loss (39%), jaundice (24%), and pruritus (19%). Other symptoms related to complications were observed, such as esophageal varices (7%), anemia (5%), and ascites in 4 patients (2%). None of the 176 selected patients were positive for hepatitis B or C serology, in accordance with the diagnostic criteria. However, 17 patients (9.56%) had presented with an initial clinical picture that suggested a viral etiology, which was subsequently ruled out by serological testing. (Table 1)

Table 1. Percentage distribution of clinical signs presented by patients.

Clinical signs	AIH (N=176) n (%)	AIH 1 (N=152) n (%)	AIH 2 (N=24) n (%)
Asthenia	132 (75%)	123 (80.92%)	9 (37.5%)
Weight loss	71 (40.34%)	60 (39.47%)	11 (45.83%)
Hepatomegaly	3 (1.70%)	2 (1.31%)	1 (4.16%)
Acute hepatitis	3 (1.70%)	3 (1.97%)	0
Hepatic cytolysis	87 (49.43%)	76 (50%)	11 (45.83%)
Jaundice	42 (23.86%)	40 (26.31%)	2 (8.33%)
Pruritus	35 (19.88%)	29 (19.07%)	6 (25%)
Hepatic steatosis	5 (2.85%)	5 (3.28%)	0
Esophageal varices	12 (6.81%)	9 (5.92%)	3 (12.5%)
Anemia	9 (5.11%)	7 (4.60%)	2 (8.33%)
Ascites	4 (2.27%)	4 (2.63%)	0
Abdominal pain	34 (19.31%)	29 (17.07%)	5 (20.83%)
Hepatocellular insufficiency	38 (21.59%)	35 (23.02%)	3 (12.5%)
Liver cirrhosis	35 (19.88%)	32 (21.05%)	3 (12.5%)
Association with another AD	6 (3.40%)	6 (3.94%)	0
Viral hepatitis	17 (9.56%)	17 (11.18%)	0
Others	12 (6.81%)	11 (7.23%)	1 (4.16%)
Asymptomatic	11 (6.25%)	6 (3.94%)	5 (20.83%)

Distribution of patients according to specific protein assays: In our study series, we noted higher frequencies of IgG, albumin, and IgM disturbances (43%, 19%,and 19%, respectively). (Table 2)

Table 2. Distribution of patients according to the results of specific serum protein assays.

	AIH (N=176) N (%)	AIH 1 (N=152) N (%)	AIH 2 (N=24) N (%)
ALBUMIN			
NORMAL	143 (81%)	127 (84%)	16 (67%)
DECREASED	33 (19%)	25 (16%)	8 (33%)
IGG			
NORMAL	97 (55%)	82 (53%)	15 (63%)
INCREASED	79 (44%)	70 (46%)	9 (37%)
IGA			
NORMAL	154 (88%)	135 (89%)	19 (79%)
INCREASED	22 (12%)	17 (11%)	5 (21%)
IGM			
NORMAL	142 (81%)	121 (80%)	21 (88%)
INCREASED	34 (19%)	31 (20%)	3 (12%)
CRP			
NORMAL	151 (86%)	131 (86%)	20 (83%)
INCREASED	25 (14%)	21 (14%)	4 (17%)

Distribution of patients according to serological characteristics and distributionof patients according to auto-Abs detection by IIF on LKS

The indirect immunofluorescence (IIF) technique on triple substrate (rat liver/kidney/stomach or LKS) performed in our patients allowed us to detect anti-smooth muscle autoantibodies (ASMA) in 129 patients (73.29%) and anti-LKM-1 autoantibodies in 23 patients (13.06%). Twenty-four patients (13.63%) were negative for both antibodies. Regarding the titer distribution among the 152 positive patients, 26 patients (17.10%) had a titer of 1/40, 22 patients (14.47%) had a titer of 1/60, 31 patients (20.39%) had a titer of 1/80, and 70 patients (46.05%) had a titer > 1/100.

Consequently, the performance of the IIF technique on LKS was evaluated using a control sample (N=83) and allowed us to demonstrate high sensitivity and specificity for type 1 and type 2 AIH, respectively, 84.86%, 90.69%, and 95.83%, 98.57.

Distribution of patients according to the results of anti-actin Abs detection by ELISA

The ELISA technique searching for anti-actin Abs performed in 129 patients positive by ELISA allowed us to observe positivity in 91.47% (i.e. 118 patients), compared to 8.52% (i.e. 11) negative patients. Consequently, the performance of the ELISA technique was evaluated using a control sample (N=94) and allowed us to demonstrate high sensitivity and specificity in the diagnosis of type 1 AIH (91.47%, 97.87%). It should be noted that 68% of our AIH patients had positive ANA. (Table 3).

Distribution of patients according to the results of anti-LC-1 auto-Abs detection by ELISA

The ELISA technique used for the detection of anti-LC-1 auto-Abs in 24 patients with AIH-2 positive by IIF allowed us to observe positivity in 70.83% (i.e. 17) seropositive patients compared to 29.42% (i.e. 7) negative patients. Consequently, the performance of the ELISA technique was evaluated using a control sample (N=56) and allowed us to demonstrate a remarkably high specificity (96.42%) and a sensitivity estimated at 70.83%. In parallel, 58% of these same patients had positive ANCA Abs. (Table 4)

Distribution of patients according to the detection of auto-Abs specific to other ADs: As part of the search for a possible association with another AD, 11 of our patients had an overlap syndrome associated with PBC (positive anti-mitochondria auto-Abs), 8 patients had autoimmune dysthyroidism, 3 patients had associated systemic lupus erythematosus (SLE), 4 patients had associated sicca syndrome, and 8 patients with AIH had confirmed antiphospholipid syndrome (APS). The results are shown in Table 5.

Distribution of patients according to prognostic factors: Based on the presence or absence of cirrhosis at the time of diagnosis, we distinguished two groups, of which 59 out of 176 patients had histologically confirmed cirrhosis. Age remains an important parameter in the prognostic evolution towards cirrhosis, since a delayed diagnosis increases the risk of developing cirrhosis in patients 8 times higher than in patients who benefited from an early diagnosis. Among the parameters studied, only IgG and albumin levels were associated with the risk of developing cirrhosis. A low serum albumin level is associated with a high risk of developing liver cirrhosis in our AIH patients ($P<0.05$; OR = 14); likewise, the increase in IgM is associated with a high risk of developing liver cirrhosis in our AIH patients ($P<0.05$; OR = 9.8). (Table 6)

Table 3. Sensitivity, specificity, and positive and negative predictive values of auto-Abs tested in type I AIH.

	Patients	Controls
Detection of anti-smooth muscle auto-Abs by IIF technique on LKS		
Positive	129 (85%)	8 (10%)
Negative	23 (17%)	78 (90%)
Total	**152**	**86**
Sensitivity	84.86%	
Specificity	90.69%	
PPV*	94.16%	
NPV**	77.22%	
Detection of anti-Actin auto-Abs by ELISA technique		
Positive	118 (91.47%)	2 (2.12%)
Negative	11 (8.52%)	92 (97.87%)
Total	**129**	**94**
Sensitivity	91.47%	
Specificity	97.87%	
PPV	98.33%	
NPV	89.32%	
Detection of ANA by IIF and ELISA techniques		
Positive	103 (67.76%)	3 (10%)
Negative	49 (32.23%)	27 (90%)
Total	**152**	**30**
Sensitivity	67.76%	
Specificity	90%	
PPV	97.16%	
NPV	35.52%	

***PPV:** Positive predictive value ***NPV:** Negative predictive value

Table 4. Sensitivity, specificity, and positive and negative predictive values of auto-Abs tested in type II AIH.

	Patients	Controls
Detection of anti-LKM-1 auto-Abs by IIF technique on LKS		
Positive	23 (95.83%)	1 (1.42%)
Negative	1 (4.16%)	69 (98.57%)
Total	**24**	**70**
Sensitivity	95.83%	
Specificity	98.57%	
PPV	95.83%	
NPV	98.57%	
Detection of anti-LC-1 auto-Abs by ELISA technique		
Positive	17 (70.83%)	2 (3.57%)
Negative	7 (29.16%)	54 (96.42%)
Total	**24**	**56**
Sensitivity	70.83%	
Specificity	96.42%	
PPV	89.47%	
NPV	88.52%	
Detection of ANCA by IIF techniques		
Positive	14 (58.33%)	3 (4.28%)
Negative	10 (41.66%)	67 (95.71%)
Total	**24**	**70**
Sensitivity	58.33%	
Specificity	95.71%	
PPV	82.35%	
NPV	87.01%	

Table 5. Distribution of patients according to the results of auto-Abs detection found in other ADs.

Auto-Abs specificity	N (%)
ANA screening	
Negative	38 (21.59%)
Positive	114 (64.77%)
ANA identification	
- Sm	2 positives
- SSA	3 Positives
- SSB	4 Positives
- RNP	3 Positives
- Jo-1	Negative
- Scl-70	Negative
Anti-native DNA	3 Positives
Detection of anti-TPO Abs	5 (2.84%)
Detection of anti-Tg Abs	6 (3.40%)
Detection of anti-parietal cell Abs	3 (1.70%)
Detection of ANCA	17 (9.65%)
c-ANCA pattern	2 (1.13%)
p-ANCA pattern	15 (8.52%)
Detection of APL	
- Anti-cardiolipin Abs IgG type	1 (0.56%)
- Anti-cardiolipin Abs IgM type	6 (3.40%)
- Anti-βgpI Abs IgG type	0
- Anti-βgpI Abs IgM type	7 (3,97%)

Table 6. Distribution of patients according to biological parameters and the risk of developing cirrhosis in patients with AIH.

Parameters	AIH without cirrhosis (N=117)	AIH with cirrhosis (N=59)	p	OR	95% CI
Mean Age	47 ± 13.23	44 ± 11.58	NS	-	-
AIH (N=176)	51 ± 11.89	49 ± 10.59	NS	-	-
AIH 1 (N=152)	23 ± 7.89	19 ± 6.72	NS	-	-
AIH 2 (N=24)					
Sex					
Male (N=46)	35	11	NS	-	-
Female (N=130)	82	48	NS	-	-
Diagnostic delay	2.23 ± 1.45	5.83 ± 1.88	<0.05	8.16	1.22 – 4.32
Albumin	92	12	<0.05	0.06	0.27 – 0.52
Normal (N=104)	25	47	<0.05	14	1.89 – 3.59
Decreased (N=72)					
IgG	78	10	<0.05	0.10	0.38 – 0.62
Normal (N=88)	39	49	<0.05	9.8	1.59 – 2.6
Increased (N=88)					
Anti-smooth muscle auto-Abs	85	44	NS	-	-
Positive (N=129)	32	15	NS	-	-
Negative (N=47)					
Anti-Actin auto-Abs					
Positive (N=118)	78	40	NS	-	-
Negative (N=58)	39	19	NS	-	-
Anti-LKM-1 auto-Abs					
Positive (N=23)	15	8	NS	-	-
Negative (N=153)	102	51	NS	-	-
Anti-Nuclear auto-Abs					
Positive (N=105)	70	35	NS	-	-
Negative (N=71)	47	24	NS	-	-
Association with other ADs	4	7	NS	-	-
PBC (N=11)	3	5	NS	-	-
APS (N=8)	1	3	NS	-	-
Sicca syndrome (N=4)	1	2	NS	-	-
Biermer's disease (N=3)					

4. DISCUSSION

AIH is a disease of immunological origin localized to the liver; it remains rare but potentially serious, linked to inappropriate activation of the immune response against hepatocytes. AIH is distinguished by its chronicity, its polymorphic clinical expression, and its risk of evolution towards terminal liver failure with cirrhosis in the absence of treatment.

AIH results from a loss of immune tolerance, with the production of auto-Abs and lymphocyte activation, leading to hepatic inflammation on histology. [7]

In all published series, as in our study, AIH preferentially affects women (sex ratio approximately 3 to 4 women to 1 man) but can also occur in children and the elderly, depending on the type of AIH I or II. The first type preferentially affects adults after 40 years, while the second type affects children according to our study. [8, 9]

AIH is a disease that generally evolves in an insidious and progressive manner. The circumstances of discovery vary from one patient to another and can be asymptomatic and can exceed 10 years when the diagnosis is purely biological and/or immunological [10]. This variation in the frequency of asymptomatic patients is linked to the stages of the disease at which patients were diagnosed.

AIH is diagnosed based on non-specific clinical signs, such as asthenia (85% of cases), jaundice of variable intensity (80%), hepatomegaly (80%), or right hypochondrium pain (50%), and more rarely asthenia, headaches, or anorexia (especially for AIH-1). AIH can also present with an acute picture (30% to 50% of cases) mimicking viral hepatitis A or E associated with fever, nausea, vomiting, abdominal pain, and anorexia, followed by the appearance of jaundice with dark urine and discolored stools.

In 50% of cases, AIH is associated with extrahepatic manifestations such as arthralgia, thyroid disorders, or insulin-dependent diabetes. In 10% of cases, AIH may present with cirrhosis with portal hypertension, especially when the diagnosis is made late. [11]

Biologically, hepatic cytolysis with elevated transaminase levels (between 5 and 10 times the normal value) and polyclonal hypergammaglobulinemia (2 to 3 g/dL), predominantly of IgG, is classically observed. An elevation in alkaline phosphatase levels is detected in 80% of patients, isolated or associated (2 to 4 times the normal value). It is common to incidentally discover an increase in transaminases in an otherwise asymptomatic child. [12 - 14]

The mode of revelation was asthenia in our study, the most represented symptom in 75% of our patients, followed by cytolysis syndrome and weight loss (49% and 40%). In 20% of our patients, the revealing picture included one or more complications (jaundice, hepatocellular insufficiency) and less frequently ascites and hepatomegaly (about 2%). [15, 16]

Immunologically, in the literature, anti-actin antibodies are present in more than 85% of AIH-1 with a specificity exceeding 80%. The search for anti-SMA Abs remains mandatory (by indirect immunofluorescence on triple substrate, dot -blot, or ELISA), although the presence of a non-actin anti-SMA Ab is of no interest in AIH-1. [17]

ANA are found in 70% of AIH-1, and their detection is performed by IIF; the most common patterns are homogeneous and speckled (about 35% each). They are present in isolation in 15% of AIH-1, whereas the antigenic targets are multiple, such as ribonucleoproteins (RNP), chromatin, histone proteins, lamins, and soluble nuclear proteins, which are described in this liver pathology. [17, 18]

Other auto-Ab specificities in AIH-1 are described, such as anti-SLA auto-Abs, present in 15 to 30% of AIH-1, with excellent specificity (greater than 99%). These auto-Abs could be of poor prognosis; they have been described in two main situations: de novo AIH and recurrent AIH-1 after liver transplantation. [17, 18]

In our study, anti-SMA auto-Abs were present in 85% of cases, with remarkable sensitivity and specificity, respectively 85% and 91%, and anti-Actin auto-Abs were present in 91% of cases, with very high sensitivity and specificity, respectively 91% and 98%. This can be explained by the recruitment modalities of our patients from the internal medicine department, with confirmed cases, and who have few associated diseases. Currently, the majority of patients have regular follow-up and optimal care that has brought very interesting evolutionary stability, thanks to recent and adapted therapeutic protocols. [18, 19]

While the search for ANA by IIF in our study confirmed their frequencies published by several teams (68%), with high sensitivity and specificity, respectively, 68% and 90%. [19]

Regarding AIH-2, anti-LC1 auto-Abs are present in 85% with modest specificity since they are present in viral C infections, while anti-formiminotransferase cyclodeaminase auto-Abs have a variable presence between 30 and 70%. And unlike AIH-1 markers, the prognostic value is certain, especially since anti-LKM1 and anti-LC1 auto-Ab titers vary according to the stage of the disease and the treatment used. [17, 19]

Other auto-Abs have also been identified in AIH, particularly ANCA auto-Abs. However, these antibodies are present in 50 to 80% of primary sclerosing cholangitis (PSC) and in inflammatory bowel disease without PSC. [18]

In our study, the search for anti-LKM-1 and anti-LC1 auto-Abs showed their diagnostic value; these two auto-Abs were positive in 96% and 71% in our series, with a specificity exceeding 96% for both. A study on a larger sample size could support our results. For ANCA-type auto-Abs, 58% were positive, results that corroborate the literature and which deserve to be studied in depth to demonstrate their origin and role in the pathophysiology of AIH-2. [17 - 19]

It seems that the immune dysregulation in patients with AIH is not limited to the liver; it can affect other organs, which explains why nearly half of patients with AIH develop at least one other AD in the context of overlap syndromes or others. The most frequently observed are autoimmune thyroid disorders, celiac disease, and Sjögren's syndrome. [20]

In our series, the search for an association with other ADs gave results that correlate with the literature data, since 11 of our patients had an overlap syndrome associated with PBC (positive anti-mitochondria auto-Abs), 8 patients had autoimmune dysthyroidism, 3 patients had associated systemic lupus erythematosus (SLE), 4 patients had associated sicca syndrome, and 6 patients with AIH had confirmed antiphospholipid syndrome.

Finally, for factors involved in unfavorable progression to cirrhosis, several studies have agreed that delayed diagnosis and management is the main factor for cirrhosis in AIH (more than 30% of patients already have cirrhosis at the time of diagnosis). In the absence of immunosuppressive treatment, chronic inflammation progressively destroys liver cells and causes fibrosis progressing to cirrhosis. [21]

Other risk factors promoting cirrhosis or its worsening are persistent elevation of transaminases, persistent elevation of IgG even after treatment, treatment interruption, persistent decrease in serum albumin concentrations, and early forms. These factors expose patients to chronic inflammatory states that promote destruction of liver tissue architecture. [2, 22 - 24]

In our study, our results confirm the involvement of increased IgM and persistently decreased serum albumin concentrations, as well as diagnostic delay in predicting the occurrence of cirrhosis in patients with AIH.

Limitations

This study has several limitations that should be acknowledged. First, its retrospective and single-center design may limit the generalizability of our findings to broader or different populations. Second, while our sample size of 176 patients is substantial, it remains relatively small for robust multivariate analyses. Third, potential selection bias is inherent to the referral nature of a tertiary care center. Finally, we were unable to collect data on treatment response and long-term outcomes, which are important prognostic factors.

5. CONCLUSION

Our study confirms the female predominance; it mainly affects adults between 41 and 70 years old with a mean age around 53 years and an immuno-clinical polymorphism similar to that reported by most studies. In all cases, the presence of a wide diversity of auto-Abs makes it a disease that is difficult to diagnose. Anti-SMA, anti-actin, and ANA auto-Abs constitute the key element in the diagnosis of type I AIH, while anti-LKM1 and anti-LC1 auto-Abs are associated with type II AIH. This finding is also reported by our study, and their diagnostic input is also very useful for the clinician. The prognostic factors of value in the face of this type of disease, whose management is not easy, are mainly the diagnostic delay, the IgG level, and the serum albumin level.

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