Advances in Medicine and Engineering Interdisciplinary Research, 2026, Volume 4, Issue 3
DOI: 10.32629/ameir.v4i3.5464
ISSN Online: 2972-3825
ISSN Print: 2972-3833

Clinical profile of paediatric patients with autoimmune hepatitis in a tertiary care center in south-western Colombia

Carolina Aristizábal-Henao[1]*, Laura Torres-Canchala[2], Eliana Manzi[2], Verónica Botero-Osorio[2]

1. Icesi University, Colombia
2. Valle del Lili Foundation, Argentina
*Corresponding author.
Email address: caristizabalh@gmail.com

Abstract: Introduction. Autoimmune hepatitis is a progressive inflammatory liver disease that is rare in children, characterized by inflammation in liver histology, the presence of auto-antibodies and high levels of immunoglobulin G. There are few data in children and in some studies performed in Latin America, but limited in Colombia. The aim of this study is to describe the demographic, clinical, paraclinical and therapeutic data as well as, the outcome of paediatric patients diagnosed with autoimmune hepatitis in a tertiary care centre in south-western Colombia. Methods. This is a descriptive study of a retrospective cohort of paediatric patients (≤ 18 years) with a diagnosis of autoimmune hepatitis, attended in a third-level hospital in Colombia between 2011 and 2019. Statistical analyses were performed using the program Stata 14.0. Results. A total of 40 patients were included, 57.5% were female, with a median age of 10.5 years (IQR 4-13). The 62.5% presented with acute hepatitis, 20% had liver failure, a 27.5% with cirrhosis at diagnosis; 75% presented compatible liver biopsy, and 82.5% were classified as type 1 autoimmune hepatitis. All patients received a steroid plus immunomodulator; presenting biochemical remission in 75% relapse in the 22.5%. Four of the patients required liver transplantation. Conclusions. Autoimmune hepatitis is an infrequent entity, although not negligible, in children in our environment. A higher prevalence of type I autoimmune hepatitis was found. Most patients were female and presented with symptoms of acute hepatitis at the initial diagnosis. The characteristics and clinical responses, in this cohort, are similar to those described in different series in the world population.

Key words: autoimmune hepatitis; children; liver

1 Introduction

Autoimmune hepatitis (AIH) was first described in 1950 [1]. It is a progressive inflammatory liver disease characterized by the presence of inflammation with a lymphoplasmacytic infiltrate and interface hepatitis on liver histology, associated with circulating autoantibodies and high levels of immunoglobulin G [2]. This condition is rare in children [1].

It is classified into type I and type II autoimmune hepatitis, which are distinguished by the presence of certain autoantibodies (antinuclear and anti-smooth muscle antibodies in type I, and liver and kidney microsomal antibodies in type II) [3]. There is a third group of patients who are also characterized by biliary tract involvement, known as autoimmun

-e sclerosing cholangitis (formerly called overlap syndrome), and a final group defined recently, in the 1990s, corresponding to de novo autoimmune hepatitis, described in patients with a history of liver transplantation, in whom the indication was due to a different underlying condition [4,5].

Clinical manifestations vary widely. Patients may have an aggressive clinical course and present with fulminant liver failure, primarily in type II AIH, or they may present with nonspecific symptoms until developing portal hypertension and cirrhosis [6]. Early diagnosis and prompt initiation of immunosuppressive therapy are essential for remission, since, if treated appropriately, disease progression is halted and the development of complications is prevented [7].

The diagnosis of AIH is rare in children but is associated with high morbidity and mortality. Variations in race and ethnicity have influenced the clinical presentation of AIH [8]. In most studies of AIH in children, the population is predominantly Caucasian [9]. A recent study on AIH showed a higher risk of early transplantation and post-transplant recurrence in pediatric [10]. The Colombian population is primarily descended from three racial groups (Native Americans, Europeans, and Africans), resulting in considerable genetic diversity [11]. Previous studies of AIH have been conducted in Mexico, Brazil, and Argentina, showing results similar to earlier international reports, indicating a higher prevalence of type I AIH and a predominance among females [12,13,14,15]. However, data on AIH in children in Colombia are limited [16]. Most of the available findings come from adult populations or from the few studies in pediatric populations in other countries, but it is not yet known whether these findings are comparable to those in our population. Therefore, the objective of this study is to describe the demographic, clinical, laboratory, and therapeutic characteristics, as well as the outcomes, of pediatric patients diagnosed with AIH at a high-complexity center in southwestern Colombia.

2 Patients and methods

2.1 Study design, inclusion criteria, and data collection

Cali is Colombia's third-largest city and has a population of approximately 2.22 million [8]. The Fundación Valle del Lili is a tertiary-care hospital affiliated with the Icesi University School of Medicine and serves as one of the referral centers in southwestern Colombia, serving a population of approximately ten million people. It has a pediatric hepatology unit that treats around four hundred patients diagnosed with liver disease each year. This study was a non-risk research project conducted in accordance with the guidelines of the Declaration of Helsinki and approved by the institution's Ethics Committee.

This is a descriptive study of a cohort of patients from the electronic medical records database. Patients aged 18 years or younger with a diagnosis of AIH validated by the simplified criteria for AIH diagnosis established by the International AIH Group (IAIHG) (Table 1) were included; these patients were treated in outpatient clinics, the emergency department, and during hospitalization. Patients who used intravenous drugs or received a transfusion prior to diagnosis, those with fewer than two clinical follow-up visits, and those with incomplete clinical, biochemical, and histological data were excluded. Data were collected by searching the hospital's electronic medical record system for pediatric patients with a diagnosis corresponding to the code K754 for autoimmune hepatitis, according to the International Classification of Diseases (ICD-10). Patients were followed up until their last visit to the institution.

Table 1. Simplified criteria for the diagnosis of AIH from the International AIH Group (IAIHG)

Elevated IgG	
IgG above the upper limit of normal	1 point
IgG 10-fold above the upper limit of normal	2 points
Autoantibodies	
ANA or SMA ≥ 1:40	1 point
ANA or SMA ≥ 1:80	2 points
LKM ≥ 1:40	2 points
Liver Histology	
Histology compatible with AIH	1 point
Histology typical of AIH	2 points
Absence of viral hepatitis	2 points

ANA: antinuclear antibodies; SMA: anti-smooth muscle antibodies; LKM: liver-kidney microsomal antibodies; IgG: immunoglobulin G; AIH: autoimmune hepatitis. Score ≥ 6 = Probable AIH. Score ≥ 7 = Definite AIH.

2.2 Clinical characteristics and diagnosis

Demographic, clinical, laboratory, and histological variables, as well as medical history, were collected. The type of presentation at the time of diagnosis was determined, classifying patients as asymptomatic (defined as patients with abnormal liver function tests without associated symptoms), acute hepatitis (patients with suggestive symptoms such as fever, jaundice, nausea, abdominal pain, and a pattern of hepatocellular damage on laboratory tests), acute liver failure (patients with symptoms of acute hepatitis associated with coagulopathy or the development of encephalopathy), and hemorrhage (for patients who presented with gastrointestinal bleeding) [1,3].

The results of the liver biopsy were also included, classified as "incompatible," "compatible," or "typical" based on histological characteristics and IAIHG criteria, as analyzed by a clinical pathologist.

AIH was classified as type I if antinuclear antibodies (ANA) and/or anti-smooth muscle antibodies (SMA) were positive, as type II if liver and kidney microsomal antibodies (LKM-1) were positive, and as seronegative in the absence of antibodies [1,2,3]. In addition, immunoglobulin G levels were determined, and transaminase levels and liver function tests were included at the time of diagnosis and at the last follow-up for each patient.

2.3 Treatment and clinical outcomes

Regarding treatment, the management received by each patient was identified, and patients were classified as receiving steroids alone, steroids plus an immunomodulator, or an immunomodulator alone. Patients who required a liver transplant as part of their treatment were also included.

Biochemical remission was defined as a reduction in blood transaminase levels to age-appropriate normal levels, and partial remission as a decrease in transaminase levels that did not reach normal levels. A lack of response to treatment was considered in patients who failed to achieve at least a 25% reduction in transaminase levels [1,3]. Adverse events related to the treatment regimen were also described.

The onset of cirrhosis, portal hypertension with esophageal varices, and gastrointestinal bleeding was classified as complications. Additionally, patients who experienced a relapse were identified; a relapse was defined as an elevation of transaminase levels to more than three times the upper limit of normal after achieving remission with treatment [1,3].

2.4 Statistical analysis

An exploratory analysis of the data was performed to verify and correct outliers and missing data. Once the quality and completeness of the database were ensured, a univariate analysis was conducted to determine the distribution of the numerical variables. The normality of the variables was determined using the Shapiro-Wilk test; those with a p-value > 0.05 were considered normally distributed and were presented with means and standard deviations, while those that were

not normally distributed were presented with medians and interquartile ranges.

3 Results

Between January 1, 2011, and December 31, 2019, sixty patients diagnosed with autoimmune hepatitis were treated. Of these, twenty met the exclusion criteria, and the analysis was performed on the remaining forty. Figure 1 describes the selection process.

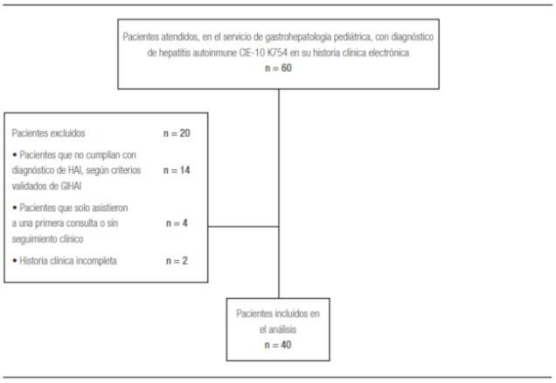

Figure 1. Selection diagram for patients with autoimmune hepatitis

Table 2 describes the clinical and diagnostic characteristics of patients with AIH. The median age at diagnosis was 10.5 years (interquartile range [IQR] 4–13); twenty-three patients (57.5%) were female; seven patients (17.5%) had a history of another autoimmune disease, of whom two had thyroiditis, and the remaining five had systemic lupus erythematosus (n = 1), type I diabetes mellitus (n = 1), ulcerative colitis (n = 1), juvenile rheumatoid arthritis (n = 1), and IgA nephropathy (n= 1). The median follow-up was 30.1 months (IQR 11.7–55.3).

Table 2. Clinical, laboratory, and diagnostic characteristics

Characteristic	Patients with AIH, n = 40
Age at diagnosis (years), median (IQR)	10.5 (4–13)
Female sex, n (%)	23 (57.5)
Follow-up duration (months), median (IQR)	30.1 (11.7–55.3)
History of autoimmune disease, n (%)	7 (17.5)
Clinical presentation, n (%)	
Asymptomatic	6 (15)
Acute hepatitis	25 (62.5)
Liver failure	8 (20)
Bleeding	1 (2.5)
Liver biopsy findings, n (%)	
Inconsistent with AIH	1 (2.5)
Compatible with AIH	30 (75)
Typical of AIH	9 (22.5)
Autoantibody profile, n (%)	
Positive for both ANA and SMA	19 (47.5)
ANA positive alone	10 (25.0)

SMA positive alone	4 (10.0)
LKM positive	2 (5.0)
Elevated IgG	13 (32.5)
AIH subtype classification, n (%)	
Type 1 AIH	33 (82.5)
Type 2 AIH	2 (5.0)
Seronegative AIH	5 (12.5)
Clinical treatment response, n (%)	
Complete biochemical remission	30 (75.0)
Partial remission	10 (25.0)
Initial and post-treatment laboratory parameters, median (IQR)	
Baseline ALT (U/L)	551 (10–1631)
Post-treatment ALT (U/L)	25 (13–61)
Baseline AST (U/L)	502 (210–1600)
Post-treatment AST (U/L)	28 (25–50)
Total bilirubin (mg/dL)	2.8 (1.11–6.7)
Direct bilirubin (mg/dL)	1.9 (0.5–5.8)
GGT (U/L)	116 (63–180)
INR	1.16 (1.04–1.44)
Complications, n (%)	25 (62.5)
Cirrhosis	11 (27.5)
Portal hypertension with varices	10 (25.0)
Gastrointestinal bleeding	4 (10)
Relapse, n (%)	9 (22.5)

ANA: antinuclear antibodies; SMA: anti-smooth muscle antibodies; LKM: liver and kidney microsomal antibodies; IgG: immunoglobulin G; AIH: autoimmune hepatitis; ALT: alanine aminotransferase; AST: aspartate aminotransferase; GGT: gamma-glutamyl transferase

3.1 Clinical and diagnostic characteristics

Twenty-five patients (62.5%) presented with acute hepatitis, and eight patients (20%) with liver failure; six patients (15%) were asymptomatic and were detected, for the most part, through routine liver function tests. One patient in this cohort presented with upper gastrointestinal bleeding secondary to esophageal varices caused by portal hypertension. All patients underwent liver biopsy: thirty (75%) had histological findings consistent with AIH, and nine (22.5%) had typical AIH. One patient did not have histological findings consistent with AIH but was managed under this diagnosis based on clinical and laboratory findings, meeting the GIAIH criteria.

Thirty-five patients (87.5%) tested positive for antibodies, with ten patients (25%) testing positive for ANAS, four patients (10%) testing positive for SMA, and nineteen patients (47.5%) testing positive for both ANAS and SMA. Only two patients (5%) tested positive for LKM-1, which correlates with the presentation type of AIH. Thirty-three patients (82.5%) were classified as type I AIH, five patients (12.5%) as seronegative AIH, and two patients (5%) as type II AIH (Table 2).

The median baseline alanine aminotransferase (ALT) level was 551 U/L (IQR 10–1631), and the post-treatment level

was 25 U/L (IQR 13–61). For aspartate aminotransferase (AST), the baseline median was 502 U/L (IQR 210–1600) and the post-treatment median was 28 U/L (IQR 25–50), with a greater difference observed in patients who achieved biochemical remission compared to patients with partial remission. These results are shown in Figure 2.

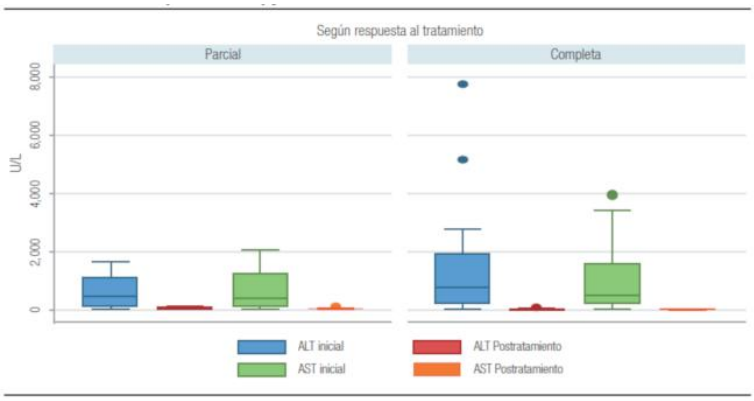

Figure 2. Baseline and post-treatment AST and ALT levels. Baseline and post-treatment levels of AST (aspartate aminotransferase) and ALT (alanine aminotransferase) are shown, according to the clinical response group: patients with partial response and patients with complete biochemical response

3.2 Treatment and clinical outcomes

At the time of diagnosis, all patients received treatment with steroids (prednisolone) plus an immunomodulator; thirty-six patients (90%) were treated with azathioprine, and the remaining four (10%) with mycophenolate mofetil (two patients due to concomitant bone marrow aplasia at the time of diagnosis and the other two due to fulminant liver failure, which required a liver transplant within the first week).

Thirty patients (75%) achieved complete biochemical remission, and ten patients (25%) achieved partial remission. Nine patients (22.55%) experienced a relapse of the disease: three due to discontinuation of treatment, two following a reduction in the immunosuppressive dose, one in whom an Ebstein-Barr virus infection was documented, and in the remaining three, the cause of relapse could not be clearly determined.

Both for patients who discontinued treatment and those whose immunosuppressive dose had been reduced, the same regimen they had previously been on was resumed, thereby achieving an adequate response, with the exception of one patient in the treatment-discontinuation group (who had been treated with azathioprine + prednisolone), who required a switch to second-line treatment with mycophenolate mofetil+ prednisolone. In the remaining patients, immunosuppression was adjusted, resulting in clinical improvement.

Among patients receiving immunomodulatory therapy with azathioprine, adverse effects were observed in eight patients (20%); of these, six presented with cytopenias (leukopenia, anemia, and thrombocytopenia), one with abdominal pain and vomiting, and the remaining patient with alopecia. In all of these cases, the regimen was switched to mycophenolate mofetil.

Four patients (10%) required liver transplantation, all with a diagnosis of type I AIH; two of these patients presented with fulminant liver failure requiring immediate transplantation, while the other two required transplantation 9 months and 4 years after diagnosis, respectively, due to portal hypertension refractory to endoscopic management. Of the transplant recipients, graft dysfunction occurred in three of them, at 7, 8, and 36 months post-transplant, respectively. Only one of them experienced disease recurrence, which was managed by adjusting immunosuppression. Two other patients are currently enrolled in a transplant protocol, both also diagnosed with type I AIH and complications secondary to cirrhosis and portal hypertension.

Among the complications observed in this patient cohort, cirrhosis was documented in eleven patients (27.5%), and portal hypertension and esophageal varices in ten patients (25%); of these, four experienced episodes of gastrointestinal bleeding. None of our patients died.

4 Discussion

AIH is a rare condition in children, although not negligible in our setting. There is limited epidemiological data available for this population, and much of the existing knowledge is derived from studies in adults. This study represents one of the first published cohorts of pediatric patients with AIH in Colombia.

There was a predominance of type I AIH (82.5%), as well as a higher prevalence among females (57.5%). These findings are similar to those reported in various publications worldwide. Costaguta et al. described a series of pediatric patients in Argentina in which 90% presented with type I AIH, with a higher prevalence among females [9]. This is consistent with the findings reported by Porta et al. in the largest cohort of patients reported in Brazil (and also for all of Latin America), with a sample of 828 patients, where 89.6% were classified as Type I AIH and the condition was predominant among females [10]. It is worth noting that these findings are not only comparable to the data presented in Latin American cohorts, but they also align with what has been reported in the global literature, as evidenced by the largest series of Asian patients with AIH described by Lee Way et al., in which 59% of the patients were female and the majority (56%) were classified as Type I AIH. 11 However, this percentage is significantly lower than those described above.

The literature reports a predominance of AIH in the white, Caucasian population, but it is clear that patients of mixed race are also affected, a group that predominates in Colombia and the rest of Latin America. Various human leukocyte antigen (HLA) polymorphisms have been reported, with HLA DQ2 and DR52 identified as risk factors for the development of AIH in the Latin American population and HLA DR3 and DQ2 in European and Asian populations [12]. Furthermore, it has been determined that the presence of these polymorphisms partly determines the clinical presentation and course of the disease, thus explaining the variability among patients [13]. It would be important to conduct such studies in our population to compare these findings.

AHI has a broad spectrum of presentation, ranging from asymptomatic patients to those who present with fulminant liver failure [6]. In our study, 62.5% of patients presented with symptoms of acute hepatitis, a finding consistent with what has been described in the global literature [9,11,14].

An association was described with other autoimmune diseases, such as systemic lupus erythematosus, thyroiditis, type I diabetes mellitus, and ulcerative colitis, which were also described by Porta et al. [10] and Costaguta et al. [9]. In our cohort, other associated conditions were identified, such as juvenile rheumatoid arthritis and IgA nephropathy.

The treatment provided to all patients follows the recommendations of international AIH guidelines [1,7]. Ten percent of the patients required a liver transplant, which is consistent with what has been reported in the literature [21,22]. In the majority of patients (75%), complete biochemical remission was achieved, a figure very similar to that found in the largest cohort of patients with AIH in Brazil, where 76.2% achieved biochemical remission [15,16,17]. The relapse rate was 22.5%, a percentage much lower than that reported in the vast majority of studies [12,13,14,15,16,17,18,19,20,21,22,23]. In our series, there were no deaths.

It is clear that a delayed diagnosis and a delay in treatment are risk factors for complications and poorer clinical outcomes in these patients. Social factors also play a role, as was observed in this cohort; these include poor treatment adherence—which was identified as a clear cause of relapse—and graft dysfunction in transplant patients, a finding also reported in other studies [12, 13, 15].

Among the complications described, cirrhosis was found in 27.5% of patients, a figure consistent with Latin

American series from Argentina and Brazil, which report rates of 23% and 22.4%, respectively [13, 15], but differs from findings reported on other continents, such as those described by Gregorio et al. in England and Saadah et al. in Australia, where the prevalence of cirrhosis is reported to be as high as 69% [23,24].

In this cohort, relapse occurred in 22.5% of patients, a lower percentage than that reported in the literature [15,25], with lack of adherence identified as one of the causes, as previously described [13,15]. This should lead us to provide greater support and follow-up for patients in whom a risk factor for treatment discontinuation is identified, or who may be at risk due to their social circumstances. Relapse was also observed in three patients, in whom no clear cause could be identified; this was attributed to an active phase of their disease.

4.1 Limitations and strengths

This study has several limitations, and its interpretation must take into account that it was conducted at a single referral center for liver diseases and that, as a retrospective study, data collection was limited to information available in medical records. Nevertheless, this study has strengths. It is the largest study conducted in the country on AIH, providing a deeper understanding of the clinical outcomes of these patients at the local level, thereby helping to make more targeted decisions to ensure better outcomes. Multicenter studies are needed to evaluate this condition and obtain results that can be extrapolated to other population groups.

5 Conclusion

AIH is a rare condition in children, but it is not negligible in our setting. A higher prevalence of type I AIH was found; most patients presented with symptoms of acute hepatitis, and a higher percentage of the population was female. It was evident that the clinical characteristics and response observed in this cohort of patients are similar to those described in various series involving populations worldwide.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

References

[1] Squires RH. Autoimmune hepatitis in children. Curr Gastroenterol Rep. 2004;6:225-30.

[2] Heneghan MA, Yeoman AD, Verma S, et al. Autoimmune hepatitis. The Lancet. Elsevier BV. 2013;382:1433-44.

[3] Mieli-Vergani G, Vergani D, Baumann U, et al. Diagnosis and Management of Pediatric Autoimmune Liver Disease: ESP-GHAN Hepatology Committee Position Statement. J Pediatr Gastroenterol Nutr. 2018;66:345-60.

[4] Roberts EA. Autoimmune hepatitis from the paediatric perspective. Liver Int. 2011;31:1424-31.

[5] Mieli-Vergani G, Vergani D. Paediatric autoimmune liver disease. Arch Dis Child. 2013;98:1012-7.

[6] Wang Q, Yang F, Miao Q, et al. The clinical phenotypes of autoimmune hepatitis: A comprehensive review. J Autoimmun. 2016;66:98-107.

[7] Della Corte C, Sartorelli MR, Sindoni CD, et al. Autoimmune hepatitis in children: An overview of the disease focusing on current therapies. Eur J Gastroenterol Hepatol. 2012;24:739-46.

[8] Wong RJ, Gish R, Frederick T, et al. The Impact of Race/Ethnicity on the Clinical Epidemiology of Autoimmune Hepatitis. J Clin Gastroenterol. 2012;46:155-61.

[9] Radhakrishnan KR, Alkhouri N, Worley S, et al. Autoimmune hepatitis in children.

[10] Palle SK, Naik KB, McCracken CE, et al. Racial disparities in presentation and outcomes of paediatric autoimmune hepatitis. Liver Int. 2019;39:976-84.

[11] Maldonado Gómez H, Sepúlveda CE, Subdirector R, et al. República De Colombia Departamento Administrativo Nacional De Estadística. DANE. 2009:28-38. 1997;40:283-6.

[12] Nares-Cisneros J, Jaramillo-Rodríguez Y. Hepatitis autoinmune en niños: Evolución de 20 casos del norte de México. Rev Gastroenterol Mex. 2014;79:238-43.

[13] Costaguta A, González A, Pochettino S, et al. Incidence and Clinical Features of Autoimmune Hepatitis in the Province of Santa Fe (Argentina). J Pediatr Gastroenterol Nutr. 2018;67:e107-e110.

[14] Goldberg AC, Bittencourt PL, Oliveira LC, et al. Autoimmune Hepatitis in Brazil: An Overview. Scand J Immunol. 2007;66:208-16.

[15] Porta G, Carvalho E de, Santos JL, et al. Autoimmune hepatitis in 828 Brazilian children and adolescents: clinical and laboratory findings, histological profile, treatments, and outcomes. J Pediatr, Rio J. 2019;95:419-27.

[16] Suárez D, Andrade R, Vera JF, et al. Clinical and histopathological characterization of children with autoimmune hepatitis at a referral center in Bogotá, Colombia. Rev Colomb Gastroenterol. 2019;34:364-9.

[17] Mileti E, Rosenthal P, Peters MG. Validation and Modification of Simplified Diagnostic Criteria for Autoimmune Hepatitis in Children. Clin Gastroenterol Hepatol. 2012;10:417-21.e2.

[18] Lee WS, Lum SH, Lim CB, et al. Characteristics and outcome of autoimmune liver disease in Asian children. Hepatol Int. 2015;9:292-302.

[19] Al-Chalabi T, Underhill JA, Portmann BC, et al. Impact of gender on the long-term outcome and survival of patients with autoimmune hepatitis. J Hepatol. 2008;48:140-7.

[20] Jossen J, Annunziato R, Kim HS, et al. Liver transplantation for children with primary sclerosing cholangitis and autoimmune hepatitis: UNOS database analysis. J Pediatr Gastroenterol Nutr. 2017;64:e83-e87.

[21] Perkins JD. Techniques to ensure adequate portal flow in the presence of splenorenal shunts. Liver Transplant. 2007;13:767-8.

[22] Van Gerven NMF, Verwer BJ, Witte BI, et al. Relapse is almost universal after withdrawal of immunosuppressive medication in patients with autoimmune hepatitis in remission. J Hepatol. 2013;58:141-7.

[23] Saadah OI, Smith AL, Hardikar W. Long-term outcome of autoimmune hepatitis in children. J Gastroenterol Hepatol. 2001;16:1297-302.

[24] Gregorio GV, Portmann B, Reid F, et al. Autoimmune hepatitis in childhood: A 20-year experience. Hepatology. 1997;25:541-7.

[25] Ferreira AR, Roquete MLV, Toppa NH, et al. Effect of treatment of hepatic Histopathology.

Abbreviations

AIH: Autoimmune hepatitis

IQR: Interquartile range

ICD-10: International Classification of Diseases, 10th Revision

IAIHG: International Autoimmune Hepatitis Group

ANA: Antinuclear antibodies

SMA: Anti-smooth muscle antibodies

LKM-1: Liver-kidney microsomal type 1 antibodies

ALT: Alanine aminotransferase

AST: Aspartate aminotransferase

HLA: Human leukocyte antigen