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Original article

Etiological Pattern of Liver Cirrhosis: Experience at a Hospital in Southwestern Colombia

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Abstract

Introduction: Liver cirrhosis is a leading cause of morbidity and mortality worldwide and in Colombia. Its most common etiologies have traditionally been chronic alcohol consumption and viral infections due to hepatitis B and C. However, recent decades have shown a shift in the etiological profile, with an increase in metabolic dysfunction–associated liver disease and autoimmune conditions. **Objective:** To determine the etiology of liver cirrhosis in patients treated at a tertiary care hospital (Nariño, Colombia) during the period 2022–2023. **Methods:** A cross-sectional descriptive observational study was conducted. Medical records of patients treated at Fundación Hospital San Pedro with a diagnosis of liver cirrhosis were reviewed. Statistical analysis was performed using Microsoft Excel 365, and approval was obtained from the hospital ethics committee. **Results:** A total of 120 patients were analyzed, with a mean age of 66.4 years; females predominated (52.5%), and most patients were from the department of Nariño (90.83%). Clinically, 49.2% presented with ascites, 42.5% with upper gastrointestinal bleeding, and 17.5% with hepatic encephalopathy. According to the Child–Pugh classification, 39.2% were stage B and 25.8% stage C. The most frequent etiology was cryptogenic (35.8%), followed by autoimmune (25.0%) and alcohol-related (15.0%). In-hospital mortality was 11.7%. **Conclusions:** Liver cirrhosis showed a predominantly cryptogenic etiological profile, with a high burden of advanced disease at diagnosis. The high frequency of autoimmune and steatosis-associated etiologies suggests a shift from the traditional etiological pattern observed in previous decades.

Keywords

Liver cirrhosis; causality; immunity; mordibity; prevalence.

INTRODUCTION

Cirrhosis is described as a chronic disease resulting from a persistent inflammatory process that leads to fibrosis of the hepatic parenchyma accompanied by injury to the vascular architecture due to multiple causes, among which alcohol consumption, viral infections—primarily hepatitis B virus (HBV) and hepatitis C virus (HCV)—autoimmune diseases, drug-induced liver injury, cholestasis, and metabolic disorders stand out, among others[1]. Only one in three individuals with cirrhosis is aware of having the disease, as most patients remain asymptomatic until the onset of decompensation, at which point liver disease is considered advanced[2].

Once symptoms, clinical signs, or abnormal liver function tests are identified, further evaluation is performed to determine the etiology of the disease, which will guide subsequent treatment[2]. Accurate identification of the etiology, together with staging of the degree of fibrosis, is essential for determining prognosis. Without a clear etiology that allows for targeted treatment, interventions

aimed at slowing the progression of tissue damage and preventing severe complications are limited.

Chronic liver disease is the leading cause of morbidity in approximately 844 million people and is also responsible for nearly 2 million deaths annually worldwide, half of which are due to complications related to cirrhosis and the other half to chronic HCV infection[3]. The epidemiology of the disease is heterogeneous and varies according to etiology, sex, geographic location, age, and individual-specific risk factors. This condition imposes a substantial public health burden in many countries and has shown a progressive worldwide increase since 1990, partly due to population growth and aging. In Latin America specifically, deaths from liver cirrhosis doubled between 1980 and 2010[4].

Several regional studies have been conducted to clinically and epidemiologically characterize cirrhosis and to determine its etiological profile. In Colombia, MA Luján and colleagues[5] conducted a retrospective observational study that included patients diagnosed with liver cirrhosis and bacteremia at a tertiary-level hospital in the city of Medellín and described cryptogenic etiology as the leading cause of cirrhosis at 30.8%, followed by non-alcoholic fatty liver disease at 19.3%. In the city of Bogotá, JE Prieto and colleagues[6] found that the leading cause of cirrhosis was non-alcoholic fatty liver disease, accounting for 25.5%, in line with the global trend of increasing prevalence of this condition as a cause of liver cirrhosis. Similar phenomena have been described throughout Latin America, where a transition from infectious etiologies toward metabolic and autoimmune causes has been observed[7].

The aim of the present study is to characterize a group of patients with liver cirrhosis and determine the different etiologies present in the population of the department of Nariño treated at a tertiary-level hospital in the region during the 2022–2023 period.

METHODOLOGY

An observational, descriptive, cross-sectional, and retrospective study was conducted. Medical records of patients older than 18 years treated at Fundación Hospital San Pedro (Pasto, Nariño, Colombia) between January 2022 and December 2023 with a confirmed diagnosis of liver cirrhosis were reviewed according to the following criteria: the diagnosis of liver cirrhosis was established using a multimodal approach in accordance with the guidelines of the European Association for the Study of the Liver (EASL) and the Baveno VII Consensus. Cirrhosis was defined by fulfillment of at least one of the following criteria:

- Histological: evidence of stage F4 fibrosis according to the METAVIR classification on liver biopsy.
- Elastography: liver stiffness >15 kPa, a value with high specificity for confirming compensated advanced chronic liver disease.
- Noninvasive biomarkers: biochemical indices of advanced fibrosis such as FIB-4 >3.25 or APRI >1.0.
- Imaging and clinical findings: presence of regenerative nodules on ultrasound, computed tomography, or magnetic resonance imaging associated with unequivocal signs of portal hypertension such as splenomegaly, ascites, or esophageal varices identified on upper gastrointestinal endoscopy.

In addition, alcoholic cirrhosis was defined based on the clinical record of chronic alcohol intake (consumption >20 g/day in women and >40 g/day in men):

- Autoimmune cirrhosis: suggested by positivity for antinuclear antibodies (ANA), anti-smooth muscle antibodies (ASMA), or antimitochondrial antibodies (AMA) (titers ≥1:40), elevated total immunoglobulin G (IgG), or compatible biopsy findings (interface hepatitis and predominance of plasma cell infiltrate).
- Metabolic dysfunction-associated steatotic liver disease (MASLD): presence of steatosis on imaging associated with metabolic comorbidities (type 2 diabetes *mellitus* [T2DM], obesity, dyslipidemia) in the absence of significant alcohol consumption.
- Compatible biopsy: presence of regenerative nodules surrounded by fibrous bands (METAVIR stage F4).
- Use of APRI/FIB-4: these indices were used as diagnostic support only when clinical and radiological correlation with portal hypertension was present.

Patients with etiological profile results that included hepatitis B and C virus testing (hepatitis C virus antibodies [anti-HCV], hepatitis B surface antigen [HBsAg], antibodies against hepatitis B surface antigen [anti-HBs], antibodies against hepatitis B core antigen [anti-HBc], hepatitis B e antigen [HBeAg], viral load for HBV and HCV), autoimmunity markers (ANA, ASMA, AMA, IgG), alcohol use (clinical record and chronic intake criteria), metabolic/MASLD profile (body mass index [BMI], T2DM, dyslipidemia, hypothyroidism, liver ultrasound), storage diseases (ceruloplasmin, ferritin, transferrin saturation, total iron), and other etiologies (history of hepatotoxic drug exposure) were included. Pregnant women and patients with metastatic liver tumors were excluded. Sociodemographic, clinical, laboratory, and etiological variables were collected. Patient care was provided in the outpatient and emergency settings by hepatology, gastroenterology, and internal medicine services. Statistical analysis was descriptive, calculating measures of central tendency, means, and absolute and relative

frequencies. The study was approved by the institutional ethics committee.

RESULTS

A total of 120 medical records were analyzed. The mean age was 66.4 years with a standard deviation (SD) of 11.4 (minimum age: 28 years; maximum age: 92 years), with a slight predominance of females (52.5%). Of the patients, 90.8% were from the department of Nariño and 56.67% from the city of Pasto; 49.17% had completed only primary school education (**Table 1**). The most frequent complications were ascites (49.1%), upper gastrointestinal bleeding (42.5%) secondary to esophageal varices (75%), and hepatic encephalopathy (16.6%) (**Table 2**). According to the Child-Pugh classification, 39.2% were classified as stage B and 25.8% as stage C, reflecting a high proportion of advanced disease (**Figure 1**).

Table 1. Sociodemographic Characteristics of the Population Included in the Study

Variable	n	Mean ± SD
Age	120	66.4 ± 11.4
Sex	**n**	**%**
Female	63	52.50
Male	57	47.50
Origin	**n**	**%**
Nariño	109	90.8
Cauca	1	0.8
Putumayo	10	8.3
Comorbidities	**n**	**%**
Type 2 diabetes *mellitus*	36	30
Arterial hypertension	28	23.3
Hypothyroidism	28	23.3
Obesity	6	5

Author's own research.

In the prognostic evaluation using the MELD-Na score, the population was stratified according to 90-day mortality risk: 55.8% of patients presented a low risk (6% mortality), 15% an intermediate risk (19.6% mortality), and 5% were within the highest severity range (52.6% mortality) (**Figure 2**). In-hospital mortality was 11.7%; among deceased patients, 50% presented with two complications at admission, 28.57% with three complications, and 21.43% with four complications, including ascites, encephalopathy, upper or lower gastrointestinal bleeding, or spontaneous bacterial peritonitis. No outpatient follow-up data after discharge were available.

Table 2. Clinical Characteristics of the Population Included in the Study

Variable		
Laboratory Tests	**n**	**Mean ± SD**
Total bilirubin (mg/dL)	118	2.66 ± 2.97
ALT (U/L)	119	68 ± 100
AST (U/L)	119	82 ± 74.2
Alkaline phosphatase (U/L)	115	263 ± 223
GGT (U/L)	94	233 ± 334
Albumin (g/dL)	115	3.16 ± 0.89
INR	119	1.37 ± 0.5
Creatinine (mg/dL)	120	1.1 ± 1.06
Platelets (10^3/µL)	118	125.934 ± 79.661
Sodium (mEq/L)	102	137 ± 5.43
Imaging Studies	**n**	**%**
Liver ultrasound	51	42.5
Cholangiography	50	41.7
Computed tomography	13	10.8
Elastography	1	0.8
Not reported	5	4.2
Imaging Findings	**n**	**%**
Liver cirrhosis	65	54.2
Chronic liver disease	47	39.2
Hepatic steatosis	3	2.5
Not reported	5	4.2
Complications		
Ascites	59	49.1
Upper gastrointestinal bleeding	51	42.5
Hepatic encephalopathy	20	16.6

ALT: alanine aminotransferase; AST: aspartate aminotransferase; SD: standard deviation; GGT: gamma-glutamyl transferase; INR: international normalized ratio. Author's own research.

Regarding the diagnosis of cirrhosis, the predominant method was imaging studies (87.5%). Liver ultrasound and magnetic resonance cholangiography were the most frequently performed studies (42.5% and 41.7%, respectively). Elastography was performed in only 0.8% of patients, reflecting the limited availability of advanced diagnostic methods. Liver biopsy was performed in 15.8% of cases

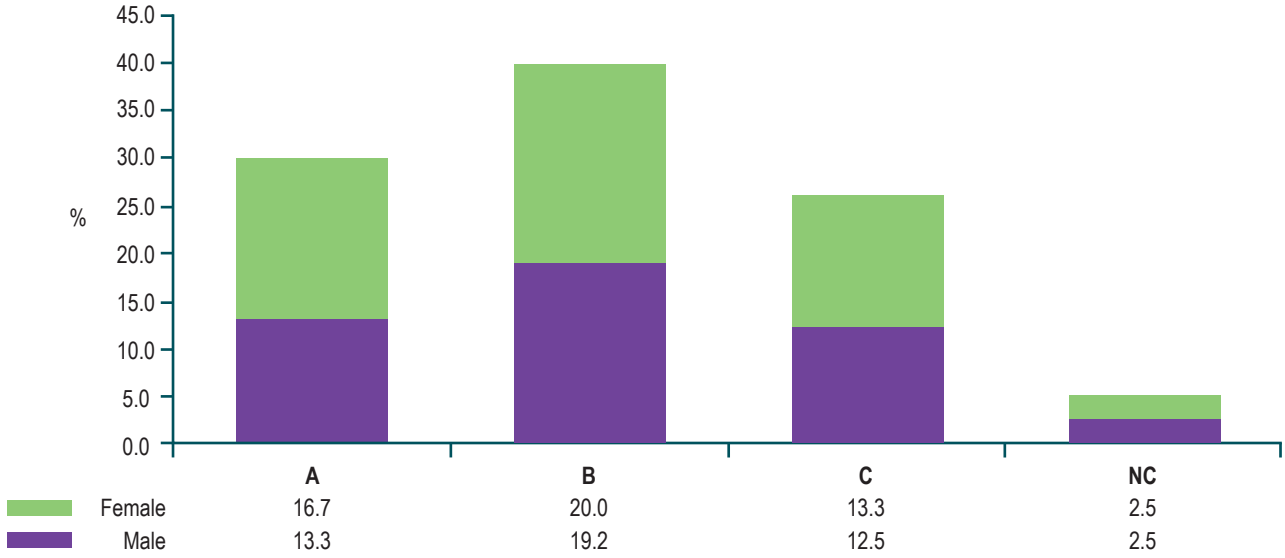


Figure 1. Percentage Distribution according to Child-Pugh Classification Stage Author's file.

(**Table 3**). In the etiological profile, the most frequent cause was cryptogenic cirrhosis (35.8%), followed by autoimmune (25.0%) and alcoholic etiologies (15.0%). Metabolic hepatic steatosis accounted for 9.2% of cases, whereas viral etiologies due to HBV and HCV were uncommon (approximately 3% to 4%). Two cases of cardiac origin and one case due to storage diseases were also identified (**Table 3**). Distribution by sex is presented in **Figure 3**.

DISCUSSION

The present study revealed a predominance of females (52.5%), a finding consistent with national reports in which the high prevalence of autoimmune diseases shifts the distribution toward this population group[5]. The mean age of 66.4 years reaffirms that cirrhosis is usually diagnosed at advanced stages due to its silent progression, with a significant impact on morbidity and mortality[1]. The high frequency of complications at admission, such as ascites (49.1%) and gastrointestinal bleeding (42.5%), demonstrates an advanced presentation of the disease. Progression to decompensated states significantly increases the risk of hospitalization and mortality[8]. These findings are consistent with the literature emphasizing that such complications reflect clinically significant portal hypertension and are associated with a greater need for early interventions. Ascites constitutes the most frequent complication of portal hypertension and is associated with poorer prognosis and increased risk of spontaneous bacterial peritonitis and hepatorenal syndrome[9,10].

Table 3. Variables related to the Etiology of Liver Cirrhosis

Variable	n	Positivity (%)
HBsAg	111	3.33
Anti-HBc	71	7.5
Anti-HBs	46	4.17
HBeAg	4	0.83
HBV viral load	4	3.33
Anti-HCV	110	4.17
ANA	75	12.5
ASMA	77	15
AMA	77	12.5
Total IgG	61	15
Ceruloplasmin	53	1.67
Ferritin	77	5.83
Transferrin saturation	62	5.83
Serum iron	69	2.5
Alcohol consumption	18	15
Liver biopsy	19	15.8

HBeAg: hepatitis B virus e antigen; AMA: antimitochondrial antibodies; ANA: antinuclear antibodies; ASMA: anti-smooth muscle antibodies; anti-HBc: antibodies against hepatitis B core antigen; HBsAg: hepatitis B surface antigen; anti-HBs: antibodies against hepatitis B surface antigen; total IgG: total immunoglobulin G; HBV: hepatitis B virus; anti-HCV: antibodies against hepatitis C virus. Author's own research.

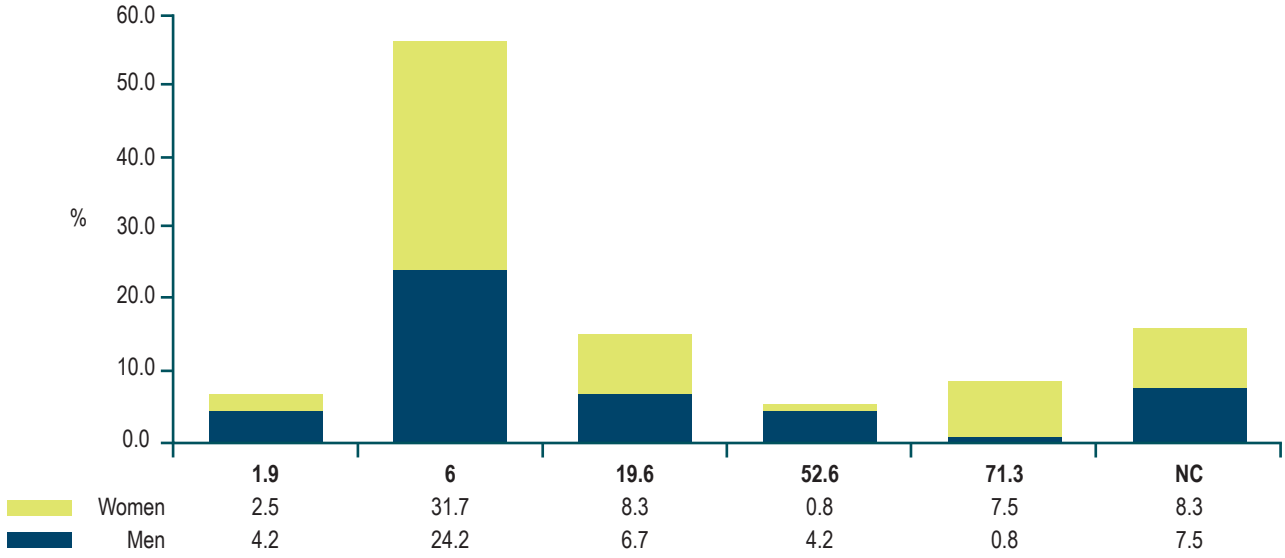


Figure 2. Percentage of 90-day mortality determined by the MELD-Na score. NC: not classifiable. Author's file.

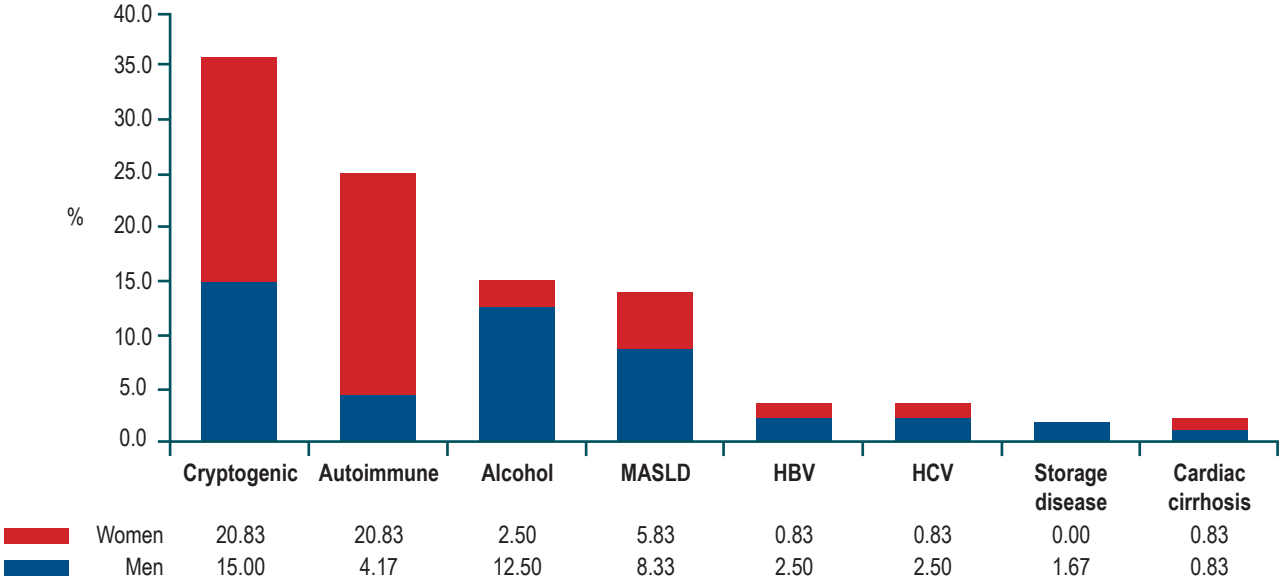


Figure 3. Etiology of Liver Cirrhosis in Patients Treated at FHSP During the 2022–2023 Period, Stratified by Sex Author's file.

The etiological distribution identified demonstrates a changing pattern in southwestern Colombia. Although cryptogenic cirrhosis was the most frequent cause (35.8%), this high percentage suggests limitations in prior comprehensive diagnostic evaluation. In the current literature, studies with rigorous follow-up usually report cryptogenic cirrhosis rates below 10%[13]. In our setting, this may be explained by the limited availability of advanced diagnostic tools: elastography was performed in only 0.8% of cases and liver biopsy in 15.8%. It is highly likely that a considerable proportion of these cryptogenic cases actually correspond to metabolic or autoimmune forms not confirmed through noninvasive tests (NITs) or histology. NITs have demonstrated diagnostic and prognostic utility in advanced fibrosis and cirrhosis, particularly in settings where biopsy is not widely available[11].

Regarding MASLD etiology, an apparent contradiction was observed: although 30% of patients had T2DM, only 9.2% were etiologically classified as MASLD. This phenomenon is characteristic of advanced stages of cirrhosis (Child-

Pugh stages B and C, which represented 65% of our sample), in which hepatic fat tends to disappear as fibrosis progresses, a phenomenon known as burnt-out NASH, making imaging or histological identification more difficult[12,15].

Autoimmune etiology accounted for 25% of cases. However, this value should be interpreted with caution; antibody positivity (ANA/ASMA) ranged from 12.5% to 15%, and only a minority of patients had biopsy confirmation. In biopsied cases, findings of interface hepatitis and hepatocyte rosettes are pathognomonic, but their absence in most of the cohort suggests that autoimmune etiology may be overestimated if based solely on limited clinical criteria[13,14].

Regarding alcohol consumption (15.0%), underreporting of intake is likely due to socioeconomic factors and the educational level of the population, in which 49.1% had only primary school education. Finally, the frequent use of magnetic resonance cholangiography (41.7%) as a diagnostic method in our cohort reflects the clinical need to rule out obstructive biliary diseases or sclerosing cholangitis in the presence of cholestatic biochemical patterns, favoring its use over computed tomography because of its greater sensitivity in evaluating the biliary tract in cases of unclear etiology.

This study has limitations inherent to its retrospective design and the limited availability of elastography. Nevertheless, it confirms an epidemiological transition toward metabolic and autoimmune causes in the region, similar to what has been observed in other developing countries, where infectious causes are being displaced[16].

CONCLUSION

This study identified a shift in the etiological pattern of liver cirrhosis in southwestern Colombia, with predominance of cryptogenic and autoimmune cases. These findings suggest the need to strengthen timely etiological diagnosis and to implement preventive strategies focused on emerging trends in chronic liver disease.

Author Contributions

NVIO: designed the research protocol, conducted data collection and analysis, and drafted the final version of the manuscript. JABM: participated as methodological advisor in the study design and critical review of the manuscript. DFTG and MBR: served as scientific advisors, providing conceptual guidance and content review. SDMM and AJVR: collaborated in data collection. All authors reviewed and approved the final version of the manuscript.

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