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Original article

Hepatocellular carcinoma: A real-life experience in a specialized center in Bogotá, Colombia

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Abstract

Introduction: Hepatocellular carcinoma (HCC) is the most frequent malignant primary liver tumor globally. In 2018, it ranked sixth and represented the fourth cause of death from cancer; the five-year overall survival is 18 %. Most cases of HCC develop in patients with cirrhosis of any etiology, especially because of hepatitis B and C viruses, alcohol, and recently nonalcoholic steatohepatitis (NASH). **Aim:** To analyze the clinical characteristics, diagnostic methods, treatments, prognostic variables, and survival. **Materials and methods:** This retrospective descriptive study was conducted on a cohort of patients diagnosed with cirrhosis and treated between January 2011 and December 2020 at a health care center in Bogotá. The diagnosis of HCC was confirmed radiologically or by biopsy. We analyzed the information descriptively with absolute frequency measures in the case of categorical variables. For continuous variables, the information was summarized with measures of central tendency (mean or median) and their relevant measures of dispersion. **Results:** We included 152 patients diagnosed with HCC, with a mean age of 69.4 years; 51.3 % were men. The leading cause of HCC was nonalcoholic fatty liver disease (NAFLD), which accounted for almost a third of cases (32 %); other causes were alcohol (15 %) and hepatitis C virus (14 %). The median manifestation of the tumor was two nodules with a size close to 4 cm. Besides, 35 % of patients had a BCLC (Barcelona Clinic Liver Cancer) stage with curative options, and 25 % received curative treatment options. The first-line systemic therapy used in this cohort was sorafenib®, used in 35 patients (33.7 %). Survival curves showed that women, Child-Pugh class A, and BCLC stage 0 had higher median survival. Multivariate analysis showed a higher risk of death for males (hazard ratio [*HR*]: 2.16; confidence interval [*CI*]: 1.24–3.76), Child-Pugh class B (*HR*: 2.14; *CI* 1.16–3.95), and Child-Pugh class C (*HR:* 7.52; *CI* 2.88–19.57). **Conclusions:** NAFLD is the leading cause of HCC in this cohort. A third of patients are diagnosed in early BCLC stages with a curative treatment option, and 25 % are treated with curative therapies. Sorafenib was the first-line therapy in advanced HCC. Overall survival after diagnosis of HCC remains low, being necessary to join forces in the follow-up of patients with cirrhosis to improve these outcomes.

Keywords

Hepatocellular carcinoma, real life, survival, sorafenib.

INTRODUCTION

Hepatocellular carcinoma (HCC) or hepatocarcinoma (HCC) is the world's most common primary liver cancer. In 2018, HCC was the sixth most diagnosed cancer and represented the fourth cause of death from cancer, with 841,000 new cases and 782,000 deaths, respectively[1]. Men's incidence and mortality rate are 2 to 3 times higher in most

regions worldwide, representing the fifth highest number of global cases and the second leading cause of death[1,2]. Overall survival at 5 years is 18%[3]. Seventy-two percent of HCC cases occur in Asia, 10% in Europe, 8% in Africa, and 5% in North and Latin America. Zonal etiological differences explain these differences in prevalence[1,4]. Between 2007 and 2013, Colombia ranked seventh in mortality with a prevalence of 2.8 to 3.2/100,000 inhabitants, responsible for more than 10,000 of the 234,763 cancer deaths[5].

Cirrhosis of any etiology has a prevalence of 85% to 95% in patients with HCC and represents a significant risk factor for tumor development[6,7]. The incidence rate of HCC in patients with cirrhosis is estimated to be 2% to 4% per year[8], while it is believed that about one-third of patients with cirrhosis develop HCC at some point in their lives[9]. Globally, approximately 90% of HCCs are associated with a known etiology[2,10], 54% of cases are attributed to chronic hepatitis B virus (HBV) infection, 31% to infection by the hepatitis C virus (HCV), and 15% to other causes such as alcohol intake and exposure to aflatoxins. However, these calculations are rough estimates that do not reflect comorbidities and underestimate the impact of non-alcoholic steatohepatitis (NASH)/metabolic syndrome[10]. Recent data from the United States show that non-alcoholic fatty liver disease (NAFLD) in its form of NASH and metabolic syndrome contribute more to the burden of HCC than any other risk factor, including HCV[11] infection, mainly due to the high prevalence of NAFLD in the general population.

Radiological studies are essential for diagnosing liver tumors and contribute to their typing and staging. The non-invasive radiology diagnosis of HCC in the context of a patient with cirrhosis was accepted in 2001 when dynamic images demonstrated the typical pattern[12], which was updated in 2005[13], and which is the result of the characteristic vascular derangement that occurs during hepatic carcinogenesis[14], plus a high pretest probability of HCC before testing in the setting of cirrhosis. Typical findings include hypervascularity in the late arterial phase, defined as arterial phase hyperenhancement (APHE), according to the LI-RADS classification (*Liver Imaging Reporting and Data System*), and washout in the portal venous or portal delayed venous phases[14-16].

In patients at high risk of developing HCC (cirrhosis plus HBV or HCV, among others) and the presence of one or more lesions, the diagnosis can be made with contrast-enhanced and dynamic computed tomography (CT) or magnetic resonance imaging (MRI) with liver injury protocols, if the injury shows imaging criteria and is read as LI-RADS 5 (LR 5), is conclusive of HCC. In specialized centers, contrast-enhanced abdominal ultrasound can also be used for solitary lesions if the modality is available, although it is more widely used in Europe[15,16]. High-risk patients who do not have liver lesions can be monitored periodically by performing ultrasound with or without alpha-fetoprotein (AFP) levels every 6 months[15-17].

The *Barcelona Clinic Liver Cancer* (BCLC) staging system has been widely used for the HCC[18] approach, classifying patients into 5 categories or stages (0, A, B, C, and D) according to treatment and survival recommendations. Stages 0 and A have curative treatment options, with survival rates greater than 5 years. In stages B and C (intermediate and advanced), the therapeutic possibilities focus on slowing down the progression of the disease with survival between 1 and 5 years. Stage D (terminal) receives palliative care with survival of nearly 3 months[19].

We presented a cohort of patients diagnosed with HCC. They were monitored for 10 years in a specialized center in Bogotá, D. C., with the primary objective of analyzing the clinical characteristics, diagnostic methods, treatments, prognostic variables, and survival.

METHODOLOGY

A retrospective descriptive study of a cohort of patients treated between January 2011 and December 2020 at Centro de Enfermedades Hepáticas y Digestivas (Center for Liver and Digestive Diseases, CEHYD, by its abbreviation in Spanish) in the city of Bogotá.

As inclusion criteria, the confirmed cirrhosis and the concurrent diagnosis of HCC, confirmed radiologically or by liver biopsy, were considered. Radiologically, HCC was defined by CT or MRI before 2016 as an arterial phase hyperenhancement with portal venous or delayed phase washout reported on imaging[8,13]. Beginning in 2016, the LI-RADS liver imaging data and reporting system (standardized terminology and criteria system for interpreting and reporting liver CT and MRI exam results in patients with cirrhosis or at increased risk for HCC)[20] was used, and an LI-RADS 5 reading was required.

The medical records of the patients who met the inclusion criteria were reviewed, tabulating each patient's clinical history, laboratory data, and Child-Pugh staging. Regarding the tumor, we tabulated its cause, maximum size in cm according to the largest nodule, number of nodules, presence of vascular invasion, extrahepatic invasion, the primary treatment used, number of ablation sessions or transarterial chemoembolization (TACE), treatment duration in months with sorafenib as systemic therapy, and whether or not palliative treatment was indicated.

The information was analyzed using descriptive methods with absolute frequency measures in the case of categorical variables. Pearson's or Fisher's exact tests were used to evaluate the differences between the two groups. For continuous variables, the information was summarized with measures of

central tendency (mean or median) and their respective dispersion measure according to the normality of the distribution of each variable evaluated using the Shapiro-Wilk test. Additionally, the Wilcoxon rank-sum (Mann-Whitney U) test was used to assess differences between two groups for mean and median estimates, respectively.

Survival assessment was performed using Kaplan Meier analysis and univariate and multivariate Cox proportional hazards analysis. Time 0 was the date of diagnosis of cirrhosis, first decompensation, or diagnosis of HCC. The time of the event was the date of death. Patients were censored at the date of the last assessment. All analyzes were performed with the Stata version 13 statistical software package.

ETHICAL CONSIDERATIONS

This project was evaluated and approved by the ethics committee of the School of Medicine of Universidad Nacional de Colombia, Bogotá campus (minute No. 009-073 of May 13, 2021).

RESULTS

When analyzing the cohort of patients with cirrhosis and HCC, 238 were initially considered, and 86 were discarded due to inconclusive diagnosis or lack of complete clinical history data. Finally, 152 patients diagnosed with HCC were included, with a mean age of 69.4 years, 51.3% of whom were men. Clinical and laboratory characteristics are shown in **Table 1**.

The leading cause of HCC was NAFLD, in its form of NASH, which represented almost a third of the cases (32%), even more frequent in the group of women, where it reached 39% of the cases. In men, the leading cause was alcohol, followed by NASH (29.4% and 25.6%, respectively) (**Table 2**).

Regarding the tumor, the median presentation was 2 nodules close to 4 cm. Based on the BCLC system, 35% of patients had a stage with curative options (**Table 2**), and 25% received remedial treatment options. Of these, 11 patients received transplantation (**Table 3**). The first-line systemic therapy used in this cohort was sorafenib, used in 35 patients (33.7%) as primary treatment. Other 14 patients received sorafenib as secondary treatment, accounting for 49 patients treated. Of them, 15 reported side effects. Most side effects included hand-foot syndrome, diarrhea, and other gastrointestinal effects. In addition, variceal bleeding occurred in a patient treated with sorafenib (**Table 3**). As the primary non-curative treatment, palliative care was given to 17.3% of patients and 13.4% in 13.4%.

The results of the survival analysis are detailed in **Table 4**. There was evidence of 1.2, 2.4, and 3.2 deaths in 100 patients one month after the diagnosis of cirrhosis, first decompensation, and diagnosis of HCC, respectively. The survival curves from the diagnosis of HCC by Child-Pugh, BCLC stage, and primary treatment presented significant differences (**Figures 1** and **2**). The groups of women, Child-Pugh A and BCLC stage 0, showed higher median survival (**Figure 1**), as well as patients with transplant and radiofrequency ablation treatments (**Figure 2**), all with significant statistical differences.

The multivariate analysis (**Table 5**) showed an increased risk of death from the diagnosis of HCC in males (*Hazard ratio* [HR]: 2;16; confidence interval [CI]: 1.24-3 .76), fall under the B stage in the Child-Pugh (HR: 2.14; CI: 1.16 to 3.95) and Child-Pugh C (HR: 7.52, CI: 2.88 to 19.57), and having been treated with ablation (HR 4.27 CI 0.51 to 35.73), TACE (HR 10.74 CI 1.35 to 84.85), sorafenib (HR 17 .59 CI 2.31 to 133.79), and palliative care (HR 25.93 CI 3.17 to 211.48).

DISCUSSION

The experience of a center specializing in hepatology is presented in this study. The average age of the patients in this cohort agrees with that reported in the European guidelines (70 years)[16]. Furthermore, the age is similar to that recorded in a Latin American series (64 years)[21]. In this investigation, 51 % of patients were men, showing a ratio of almost 1:1 with women, data that contrasts with the international literature, where the ratio favors men 2 to 3 times[1,2] according to White in a series with 236,290 cases of HCC diagnosed between 2000 and 2012 in the United States, where 73% were men[22] and these figures are mainly explained by the HCV epidemic. We diagnosed more fatty liver and alcohol and proportionally less HCV, which is consistent with the trend shown in national registries[5], possibly explaining the male-female relationship found in our research.

In this cohort, the leading cause of HCC was NAFLD in its form of NASH, with a history of metabolic syndrome between 15% and 40%. In a study of cirrhosis published in 2016 with 419 patients, fatty liver was also the primary cause of cirrhosis (25%)[23]. Currently, this cohort of cirrhotic patients under follow-up reaches 1800, and fatty liver remains the primary cause[24]. In another cohort of Colombian patients, alcohol and NASH ranked as the first and second causes of HCC, respectively[25]. This is similar to data obtained in this study in males.

The Latin American series of HCC with 1336 patients shows HCC as the cause of HCV (48%), followed by alcoholic cirrhosis (22%), HBV infection (14%), and fatty liver (9%)[21]. However, it is worth mentioning that an estimated 25% of the world's adult population has NAFLD, according to a meta-analysis including more than 8 million people. The

Table 1. General Characteristics of Patients with HCC

Variable	Total n = 152 n (%)	Women n = 74 (48.6) n (%)	Men n = 78 (51.3) n (%)	Value *p*
Age at diagnosis		**Mean (SD)**		
	69.4 (9.4)	70.9 (8.4)	67.9 (10.0)	0.054*
Background		n (%)		
- Alcohol Consumption	72 (47.4)	7 (9.5)	65 (83.4)	< 0.001**
- DM	62 (40.7)	28 (37.8)	34 (43.5)	0.471***
- HTN	61 (40.1)	31 (41.8)	30 (38.4)	0.666***
- Obesity	49 (32.2)	25 (33.7)	24 (30.7)	0.691***
- Dyslipidemia	23 (15.1)	8 (10.8)	15 (19.2)	0.148***
- Coronary Disease	15 (9.8)	5 (6.7)	10 (12.8)	0.279**
Laboratories (n = 151)		**Median (IQR)**		
- Leukocytes	5610 (4490-6990)	5050 (4075-6462)	5830 (4835-7870)	0.0019****
- Neutrophils (%)	56 (50-66)	55 (50-65)	56 (49-66)	0.945****
- Lymphocytes (%)	28 (21-34)	30 (21-35)	27 (20-34)	0.436****
- Platelets	130k (91k-176k)	132.5k (91.7k-170k)	127k (90k-186k)	0.968****
- AST	61 (41.7-100.7)	61 (42-92.7)	63.5 (40.2-114)	0.504****
- ALT	49 (34-74)	48 (28-68)	52 (37-75)	0.083****
- GGT	164 (106-259)	150 (92-245)	190 (122-283)	0.032****
- Alkaline Phosphatase	166 (117-260)	158 (118-232)	190 (115-280)	0.299****
- INR	1.1 (1-1.3)	1.1 (1-1.3)	1.1 (1-1.2)	0.196****
- Total Bilirubin	1.2 (0.8-2)	1.1 (0.8-1.9)	1.3 (0.8-2.2)	0.216****
- Albumin	3.7 (3.1-4)	3.7 (3.1-4)	3.6 (3-4.1)	0.869****
- AFP	25.5 (5- 466)	20 (5-245)	27 (5-855)	0.89****
Esophageal Varicose Veins		**n (%)**		
- No	59 (38.8)	31 (41.9)	28 (35.9)	0.745***
- Small	34 (22.4)	16 (21.6)	18 (32.1)	
- Large	59 (38.8)	27 (36.5)	32 (41)	
Child–Pugh (n = 143)		**n (%)**		
- A	65 (45.5)	30 (43.5)	35 (47.3)	0.864***
- B	63 (44.0)	32 (46.4)	31 (41.9)	
- C	15 (10.5)	7 (10.1)	8 (10.8)	

*Two-sample t-test with equal variances.
**Fisher's exact test.
***X_2-Test.
**** Wilcoxon rank-sum (Mann-Whitney U) test.
ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, GGT: γ-glutamyltransferase, SD: Standard deviation, DM: Diabetes mellitus; HTN: Hypertension, INR: International normalized ratio, IQR: Interquartile range (p25-p75), Me = median.

Table 2. Characteristics of HCC

Variable	Total n = 152 n (%)	Women n = 74 (48.6) n (%)	Men n = 78 (51.3) n (%)	Value *p*
Causes of HCC		**n (%)**		
- NASH	49 (32.2)	29 (39.1)	20 (25.6)	
- Alcohol	24 (15.7)	1 (1.3)	23 (29.4)	
- HCV	21 (13.8)	16 (21.6)	5 (6.4)	< 0.001*
- NASH + alcohol	17 (11.1)	1 (1.3)	16 (20.5)	
- Cholestasis	13 (8.5)	11 (14.9)	2 (2.6)	
- Others	28 (18.4)	16 (21.6)	12 (15.38)	
HCC data		**Median (IQR)**		
Number of nodes	2 (1-3)	1 (2-3)	1 (1-3)	0.0021**
Size in cm	3.85 (2.2-7)	3.4 (2-7.2)	4 (2.7-7)	0.34**
BCLC Stage		**n (%)**		
- 0	10 (6.5)	6 (8.1)	4 (5.1)	
- A	45 (29.6)	26 (35.4)	19 (24.3)	0.407*
- B	49 (32.2)	22 (29.7)	27 (34.6)	
- C	35 (23.0)	16 (21.6)	19 (24.36)	
- D	13 (8.5)	4 (5.4)	9 (11.5)	
Terminal Stage		**n (%)**		
- Dead	95 (62.5)	42 (56.8)	53 (67.9)	0.154*
- Live	57 (37.5)	32 (43.2)	25 (32.1)	

*Fisher's exact test.
**Wilcoxon rank-sum (Mann-Whitney U) test.

prevalence rate in South America is 31%[26]. Therefore, NASH causing HCC may also be underdiagnosed. Additionally, recent data suggest that NASH cirrhosis contributes to developing HCC and is an increasingly important risk factor for its etiology in Western countries[10,27-29]. Accordingly, Singal AG *et al.* estimated an annual incidence rate of HCC in patients with NASH cirrhosis of 1% to 2%[27]. Another fatty liver study with many patients showed an HCC incidence with a follow-up rate of 1 per 100 person/years[29].

We monitor patients with cirrhosis in our center using abdominal ultrasound and AFP every 6 months[16,17]. The therapeutic approach follows the BCLC guidelines[19]. This surveillance accounts for 35% of patients diagnosed at treatable stages. Of the 104 patients for whom treatment data were available, approximately 35% received treatment (transplantation: 10.6% and radiofrequency ablation: 25%).

About 2 thirds of patients received non-curative therapies: TACE (17.3%), systemic therapy (33.7%), and palliative treatment (13.4%). About 64% of patients received this treatment, indicating a late diagnosis. From the first decompensation event, variceal bleeding, or presence of a mass on imaging, many patients are diagnosed with cirrhosis, data supported by a median HCC survival of 9.5 months after diagnosis. The median fluctuates between 6 and 20 months in the Cancer of the Liver Italian Program (CLIP) study[30].

Since 2006, sorafenib has been approved as systemic therapy for HCC in Colombia[31]. Sorafenib was the only therapy available until the second half of 2018 when rego-

Table 3. Treatment of HCC

Variable	Total n = 104 n (%)	Women n = 49 n (%)	Men n = 55 n (%)	Value *p*
Main Treatment				
- Transplant	11 (10.6)	3 (6.1)	8 (14.5)	
- Radiofrequency ablation	26 (25)	18 (36.7)	8 (14.5)	
- TACE	18 (17.3)	10 (20.4)	8 (14.6)	0.051*
- Sorafenib	35 (33.7)	13 (26.5)	22 (40)	
- Palliative	14 (13.4)	5 (10.2)	9 (16.4)	
		Me (IQR)		
Radiofrequency ablation sessions	1 (1-2)	1 (1-2.5)	1 (1-1.5)	0.3106**
Sessions with TACE	1 (1-2)	1 (1-2.2)	2 (1-2)	0.591**
Months with sorafenib	4 (2.25-8)	4 (3-10)	4 (2-8)	0.779**
Side effects of sorafenib	**n = 15**	**n = 7**	**n = 8**	
- Hand-foot syndrome	6 (40.0)	4 (57.1)	2 (25)	
- Diarrhea	2 (13.3)	1 (14.3)	1 (12.5)	
- Other dermatological	1 (6.6)	0	1 (12.5)	0.627*
- Bleeding during intake	2 (13.3)	0	2 (25)	
- Other gastrointestinal	3 (20.0)	1 (14.3)	2 (25)	
- Others	1 (6.6)	1 (14.3)	0	

*Fisher's exact test.
** Wilcoxon rank-sum (Mann-Whitney U) test.

Tabla 4. Survival Analysis

	Incidence rate * 100 (95%CI)	Median survival (months)	Percentile 25%-75% Percentile (months)
Cirrhosis Diagnosis	1.2 (0.9 to 1.4)	49.8	17.1 – 158.9
First Decompensation	2.4 (2.0 to 3.0)	26.3	8.8-63.3
HCC Diagnosis	3.2 (2.6 to 3.9)	15.9	6.4-50.2

rafenib was approved as a second-line treatment[32]. In this study, 35 patients received sorafenib as their primary treatment and 14 as a second or third option, with an average of 6.8 months of use for the 49 patients. Average survival rates between 4.6 and 12 months are mentioned in the literature[33-35]. Of the 14 patients with combined therapies, 8 had previously received radiofrequency ablation (5 later received TACE), and 6 had previously received TACE. Fifteen patients (30.6%) reported the usual side effects[31,34] in the following order a) hand-foot syndrome, b) gastrointestinal issues, and c) diarrhea.

Interestingly, 2 patients with platelets above 100,000 and regular INR experienced variceal bleeding while taking sorafenib. The first patient had large varicose veins with red dots that were not initially ligated due to administrative problems. The second patient, who had previously ligated varicose veins, bled at the beginning of the treatment, with unclear bleeding, after which he received sorafenib for 12 months without new episodes. On the other hand, although 54.5% of the patients had varicose veins, this was

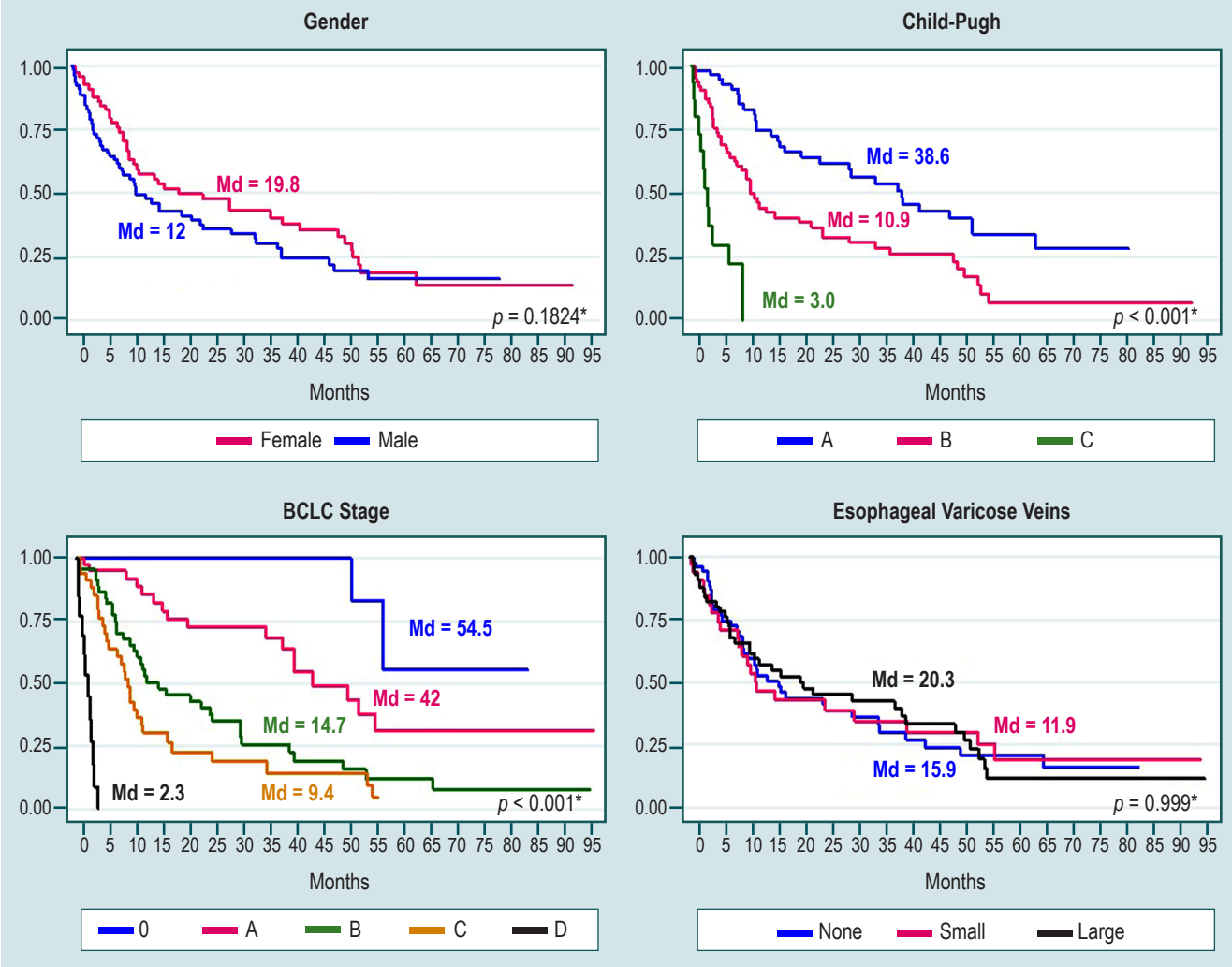

Figure 1. Survival curves of the risk of death from the diagnosis of HCC by gender, Child-Pugh, BCLC stage, and esophageal varicose veins. Kaplan-Meier survival estimates. *Log-rank test for equality of survival functions. Md: median.

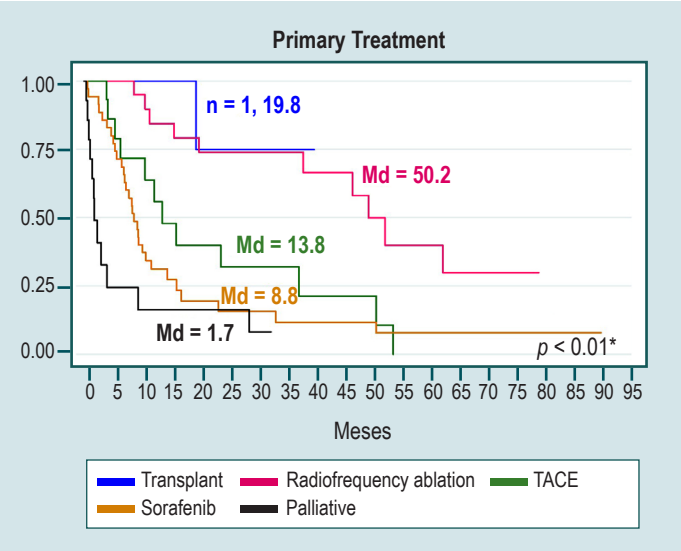

Figure 2. Survival curves of the risk of death from diagnosing hepatocarcinoma by primary treatment. Transplantation = 1 patient died after transplantation. Kaplan-Meier survival estimates. *Log-rank test for equality of survival functions. Md: median.

Table 5. Univariate and Multivariate Analysis of the Risk of Death from the Diagnosis of HCC.

	Univariate Analysis HR (95%CI)	Multivariate Analysis HR (95%CI)
Gender		
- Women	Reference	Reference
- Men	1.31 (0.87 to 1.97)	2.16 (1.24 to 3.76)*
Child-Pugh al diagnóstico HCC		
- A	Reference	Reference
- B	2.23 (1.41 to 3.54)*	2.14 (1.16 to 3.95)*
- C	11.39 (5.52 to 23.50)*	7.52 (2.88 to 19.57)*
Estadio BCLC		
- 0	Reference	
- A	2.75 (0.63 to 11.97)	
- B	7.03 (1.67 to 29.55)*	***
- C	10.89 (2.56 to 46.34)*	
- D	146.92 (28.79 to 749.58)**	
Várices esofágicas		
- No	Reference	Reference
- Pequeñas	0.98 (0.58 to 1.68)	0.64 (0.29 to 1.37)
- Grandes	0.99 (0.62 to 1.57)	1.18 (0.65 to 2.13)
Por tratamiento principal		
- Trasplante****	Reference	Reference
- Radioablación	2.20 (0.27 to 17.37)	4.27 (0.51 to 35.73)
- TACE	6.47 (0.83 to 49.95)	10.74 (1.35 to 84.85)*±
- Sorafenib	9.40 (1.27 to 69.19)*	17.59 (2.31 to 133.79)*±
- Paliativo	22.25 (2.88 to 171.79)*	25.93 (3.17 to 211.48)*±

*p < 0.05.
** Imprecise estimator due to the number of patients in this group (n = 13).
***Variable not included in the multivariate analysis due to imprecise estimators.
****1 patient died after the transplant. ± Inaccurate estimators due to the number of patients included.

not statistically significant in the overall survival of the patients. A study in Italy identified tumor deep vein thrombosis (DVT) as the strongest independent predictor of bleeding (HR: 15.4; 95 % CI: 1.84-129.6)[35], but none of the 2 patients in this study had it. The meta-analysis by Dai *et al.*, with 4720 patients who received sorafenib to treat HCC[36], showed a significant increase in the risk of low-grade bleeding events (relative risk [RR]: 1.99; 95% CI: 1 59-2.49; $p < 0.00001$), the second patient in our series could be in this group. Thus, we could say that one patient (2%) experienced low-grade bleeding associated with sorafenib in this cohort.

We acknowledge the limitations of the study as it is retrospective. However, this is a cohort of patients with HCC monitored for an extended period in Bogotá, drawing attention to the etiology, fatty liver, a frequent condition in our population. In the future, this condition could change the guidelines on its screening and follow-up as a risk factor for the development of HCC.

CONCLUSIONS

In this cohort, the leading cause of HCC is NAFLD, more than a third of patients are diagnosed in early BCLC stages with a curative treatment option, and 25% are treated with curative therapies. Sorafenib was the first line of treatment for advanced HCC. However, overall survival after diagnosis of HCC remains low, and it is necessary to join efforts in the follow-up of patients with cirrhosis to improve early diagnosis rates.

Disclosure

The authors declare that there are no conflicts of interest in this study.

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