Araştırma Makalesi
Research Article

# Retrospective, single-center evaluation of liver cirrhosis cases in a geriatric population

GERİATRİK POPÜLASYONDA KARACİĞER SİROZLU OLGULARIN RETROSPEKTİF, TEK MERKEZLİ DEĞERLENDİRİLMESİ

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**ABSTRACT**

**Objective:** Since there is insufficient epidemiological data on liver cirrhosis, especially in the elderly population of many countries, we aimed to evaluate geriatric cases with liver cirrhosis hospitalized at our gastroenterology clinic, retrospectively.

**Materials and Methods:** The study included 99 patients aged 65 years and over who were hospitalized with liver cirrhosis in Ege University Faculty of Medicine, department of gastroenterology. The socio-demographic, clinical and laboratory data were recorded from the patient files.

**Results:** In total, 99 elderly patients (mean age 71.7 ± 5.2 years; 59% male) were included. Hepatocellular carcinoma was statistically significantly correlated with etiology, presence of ascites, the Model of End-Stage Liver Disease score and the Child-Turcotte-Pugh score (p=0.003, p=0.009, p=0.033, p=0.003, respectively). The presence of esophageal varices was statistically significantly correlated with class and scores of the Child-Turcotte-Pugh, total bilirubin, Model of End-Stage Liver Disease score and the presence of ascites (p< 0.0001, p=0.001, p=0.011, p=0.004, p=0.004, respectively).

**Conclusion:** The main etiology of liver cirrhosis was Hepatitis C virus, which accounted for almost one-third of the cirrhotic elderly inpatients at the gastroenterology department. Hepatocellular carsinoma was the leading cause of hospitalization. Elderly patients with liver cirrhosis should be carefully evaluated for cirrhosis complications. The Child-Turcotte-Pugh classification and Model of End-Stage Liver Disease scoring should be undertaken, and the suitability for liver transplantation should be considered in the follow-up and treatment of this patients.

**Keywords:** liver cirrhosis, elderly, hepatocellular carcinoma

**ÖZ**

**Amaç:** Birçok ülkenin yaşlı popülasyonunda karaciğer sirozu hakkında yeterli epidemiyolojik veri olmadığı için gastroenteroloji kliniğimizde yatan karaciğer sirozu tanılı geriatrik olguların retrospektif olarak değerlendirilmesi amaçlandı.

**Gereç ve Yöntem:** Çalışmaya Ege Üniversitesi Tıp Fakültesi Gastroenteroloji kliniğinde yatmış 65 yaş ve üzeri karaciğer sirozu tanılı 99 hasta dâhil edildi.

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Sosyodemografik, klinik ve laboratuvar verileri hasta dosyalarından kaydedilmiştir.

**Bulgular:** Çalışmaya dahil edilen 99 yaşlı hastanın yaş ortalaması 71,7 ± 5,2 yıl, % 59'u erkek idi. Hepatoselüler karsinom ile etiyoloji, asit varlığı, Model of End-Stage Liver Disease skoru ve Child-Turcotte-Pugh skoru arasında istatistiksel olarak anlamlı ilişki saptanmıştır (sırasıyla, p = 0,003, p = 0,009, p = 0,033, p = 0,003). Özofagus varisi varlığı ile Child-Turcotte-Pugh sınıfı ve skoru, total bilirubin, Model of End-Stage Liver Disease skoru ve asit varlığı arasında istatistiksel olarak anlamlı ilişki saptanmıştır (sırasıyla, p <0,0001, p = 0,001, p = 0,011, p = 0,004, p = 0,004).

**Sonuç:** Gastroenteroloji kliniğimizde yatan yaşlılarda karaciğer sirozunun ana etiyolojisi Hepatit C virüsü olup sirotik popülasyonun neredeyse üçte birini oluşturmaktadır. Hastaneye yatış nedenleri arasında ilk sırada hepatoselüler karsinom yer almaktadır. Karaciğer sirozu tanısıyla hastaneye yatırılan yaşlılar siroz komplikasyonları açısından dikkatle değerlendirilmeli, Child-Turcotte-Pugh ve Model of End-Stage Liver Disease skorlaması yapılarak, özellikle karaciğer nakli açısından uygunlukları da göz önünde bulundurularak takip ve tedavi edilmelidir.

**Anahtar Sözcükler:** karaciğer sirozu, yaşlı, hepatosellüler karsinom

Liver cirrhosis (LC) is the last stage of chronic liver diseases with different etiologies and causes more than one million deaths every year throughout the world (1). The causes of cirrhosis vary according to socioeconomic and cultural characteristics. Many factors, such as viral hepatitis, alcohol, primary biliary cholangitis, autoimmune hepatitis, non-alcoholic steatohepatitis, metabolic diseases, hemochromatosis, and Wilson's disease play a role in the etiology of LC (2). While the cause of the disease is mainly alcohol use in Western Europe and North America, viral hepatitis predominates etiology in other parts of the world. In Turkey, the most common causes of LC are reported to be hepatitis B virus (HBV) and hepatitis C virus (HCV) infections, followed by cryptogenic cirrhosis, alcohol use, and other factors (3). The symptoms and signs of LC often develop secondary to hepatocellular insufficiency and/or portal hypertension (4). Patients with LC often have life-threatening complications that can result in death without an urgent medical intervention. Most major complications, including ascites, esophageal variceal bleeding, spontaneous bacterial peritonitis (SBP), hepatorenal syndrome (HRS), portal hypertensive gastropathy, hepatic hydrothorax, hepatopulmonary syndrome, portopulmonary hypertension, cirrhotic cardiomyopathy and hepatic encephalopathy (HE) occur due to portal hypertension (5). A significant proportion of hepatocellular cancers develop on the basis of LC and are the major cause of mortality (6). Ascites is the most common presentation of LC (7-9). There are numerous methods based on clinical and laboratory data to estimate the prognosis of cirrhosis patients. An example is the Child-Turcotte-Pugh (CTP) scoring, which is associated with mortality. The rate of one-year survival is reported to be 100%, 80% and 45% at CTP Class A, B and C, respectively (10). Another example is the Model of End-Stage Liver Disease (MELD) scoring, which has been utilized as reference in liver transplant programs in the United States since 2002 (11).

Regarding the geriatric patients aged 65 and over, they are reported to be under-represented in hepatitis B and C screening approaches. However, older patients are candidates for elevated risk for advanced liver disease. (12). In the literature, there is still a lack of epidemiological data about liver cirrhosis, especially in the elderly population of many countries. Therefore, in this study, we evaluated the geriatric patients with LC hospitalized at our gastroenterology department, retrospectively.

## MATERIALS AND METHODS

Between December 2011 and January 2014, 301 cases were surveyed in the gastroenterology clinic. They included cases with liver cirrhosis, either newly diagnosed during their hospital stay, or hospitalized due to cirrhosis complications. Ninety-nine of them were aged over 65

years and complete data related to these patients were included in the study. 10.1% of 99 subjects were patients who were newly diagnosed with LC. The study was approved by the Institutional Ethical Review Board (Number: 19-11.1T/43).

The data of the patients included in the study were obtained from patient files and/or the electronic patient file system. All the data belonged to the first day of hospitalization. The patients' age, gender, reason for hospitalization, etiology of LC, viral load in cases with HBV and HCV, presence of esophageal varices, hemoglobin (Hb), leukocyte and platelet count at the time of hospitalization, prothrombin time (PT), International normalized ratio (INR), C-reactive protein (CRP), alpha fetoprotein (AFP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), creatinine, total bilirubin, direct bilirubin, albumin, globulin, MELD score, CTP score and class, presence of hepatocellular carcinoma (HCC), and presence of ascites were recorded in the case report form. In cases with ascites, the serum acid albumin gradient (SAAG), presence of spontaneous ascitic fluid infection (SAI), and the levels of albumin, total protein and leukocyte in ascitic fluid were also included in the form. The cases that died during their stay at the hospital were noted with their date of death.

Patients were diagnosed with LC using a liver biopsy (if possible), and clinical, laboratory or imaging methods. After eliminating other causes of portal hypertension, clinical diagnosis was based on esophageal varices detected by endoscopy and splenomegaly and ascites revealed by abdominal ultrasonography (USG), portal doppler USG, and/or physical examination. The CTP score was calculated using the presence of ascites, PT/INR, albumin, total bilirubin, and presence of encephalopathy (13). The MELD score was computed with the INR, bilirubin and creatinine values using the MELD calculator provided by https://www.mayoclinic.org/medical-professionals/transplant-medicine/calculators/meld-model/itt-20434705 (11).

For the diagnosis of HRS, the following major criteria defined by the International Ascites Club in 1996 were used (14): a low glomerular filtration rate (GFR) (serum creatinine of over 1.5 mg/dL or creatinine clearance of under 40 ml/min); absence of shock, underlying bacterial infection, loss of fluid, and use of nephrotoxic drugs; no improvement in renal function (serum creatinine level remaining higher than 1.5 mg/dL or creatinine clearance lower than 40 ml/min) following the termination of diuretic treatment or plasma volume support (1.5 L); a proteinuria level of below 500 mg/day; and absence of obstructive or renal parenchymal disease findings on USG. Additional criteria were defined as the urine volume being under 500 ml/day, urine sodium below 10 mEq/L, urine osmolality greater than plasma osmolality, urinary sediment less than 50 erythrocytes per field for each magnification, and serum sodium concentration lower than 130 mEq/L. For the diagnosis of HRS, all major criteria should be met. Additional criteria are not required for a diagnosis; rather, they are evaluated as supporting evidence.

Spontaneous acid infections are defined as three different clinical forms called spontaneous bacterial peritonitis (SBP), culture negative neutrocytic ascites (CNNA) and monomicrobial non-neutrocytic bacterascites (MNB). In spontaneous bacterial peritonitis, ascitic fluid culture positive and ascitic polymorphonuclear leukocyte (PMNL) count is higher than 250 cells / $mm^3$. The culture is monomicrobial. In CNNA, ascitic PMNL count is higher than 250 cells / $mm^3$ but ascitic fluid culture is negative. In MNB, ascitic fluid culture is positive, but ascitic PMNL number is less than 250 cells / $mm^3$ (9).

The diagnosis of HCC was determined based on the AFP levels by applying at least two imaging methods. The presence/absence of esophageal varices was noted using upper gastrointestinal system endoscopy. Hepatic encephalopathy was defined by employing the West Haven criteria according to the severity of signs and symptoms (15).

### Statistical analysis

IBM SPSS package program v. 18.0 was used for statistical analysis. The categorical variables were summarized using frequency tables and cross-tables, and the relationships between them were examined by chi-square analysis (or Fisher's exact probability test). Prior to

analysis, the compatibility of the numerical variables with the normal distribution was analyzed with the Shapiro-Wilk test. None of the numerical variables were found to fit a normal distribution; thus, non-parametric methods were employed to test the hypotheses. The numerical variables were first summarized using median, minimum and maximum values. The Mann-Whitney U test was used when the number of groups was two, and in the case of more than two independent groups, the Kruskal-Wallis test was utilized to determine whether there was a significant difference in terms of the related numerical variable. All hypotheses were tested by two-way analyses at $\alpha$=0.05 significance level.

## RESULTS

Ninety-nine patients included in the study, 58 (59%) were male, 68 (68.7%) were aged 65-74 years, 31 (31.3%) were aged 75-84 years, and the mean age was 71.7 ± 5.2 (65-84) years (72.3 ± 5.1 for women and 71.4 ± 5.3 for men). Table 1 summarizes the sociodemographic, clinical and laboratory characteristics of the overall study population.

The primary indication for hospitalization was HCC in 28 LC cases (28.3%), HE in 20 (20.2%), investigation of LC etiology or further examinations in 19 (19.2%), and other causes (esophageal variceal bleeding, ascites, HRS, SAI, diabetes mellitus, acute renal failure, acute cholecystitis, deep venous thrombosis, acute myeloid leukemia, pneumonia, pulmonary edema, cholangitis, and pleural effusion) in 32 (32.3%).

The etiology of LC was found to be associated with HCV in 31 cases (31.3%), cryptogenic cirrhosis in 29 (29.3%), HBV (without delta-agent) in 25 (25.3%), alcohol use in 7 (7.1%), non-alcoholic steatohepatitis (NASH) in 4 (4%), and other factors (HBV with delta-agent, primary biliary cholangitis and primary sclerosing cholangitis, and alcohol + HVC in 3 (3%).

While 77 (77.8%) of the patients with LC had esophageal varices, 11 (11.1%) had no esophageal varices. The data of 11 (11.1%) people could not be reached. Twenty-five of the cases (25.2%) were classified as CTP Class A, 44 (44.4%) as CTP Class B, and 30 (30.4%) as CTP Class C. Hepatocellular carcinoma was present in 42 patients (42.4%). Ascites was detected in 64 of the cases (64.6%). Of the patients with ascites, 38 had an SAAG above 1.1 and one had an SAAG below 1.1. For the remaining ascites cases, the SAAG calculation could not be undertaken due to the unavailability of data. Eleven cases (11.1%), eight male (72.7%) and three female (27.3%), died during their stay at the hospital, and 88 patients (88.9%) were discharged. Among the patients that died, the reasons for hospitalization were HE in 6 (54.5%), HCC in 1 (9.1%), and other causes in 4 (36.4%). When the etiology of the mortality cases was examined, HCV was found in 4 (36.4%), HBV without delta-agent in 4 (36.4%), alcohol use in 1 (9.1%), and cryptogenic cirrhosis in 2 (18.2%). In addition, it was determined that three of the six cases with SAI (50%) had died.

There was a statistically significant relationship between gender and etiology in patients with LC ($p = 0.003$). The frequency of HBV (88.0%) and alcohol (75.0%) was higher in male patients while HCV (58.1%) and cryptogenic cirrhosis (55.2%) were more common in female patients.

A statistically significant relationship was found between HCC and ascites ($p = 0.009$), with the latter being less common in patients with HCC. Hepatocellular carcinoma was statistically significantly correlated with etiology, presence of ascites, MELD score and CTP score ($p=0.003$, $p=0.009$, $p=0.033$, $p=0.003$, respectively) (Table 2). The presence of esophageal varices was statistically significantly correlated with class and scores of CTP, total bilirubin, MELD score and the presence of ascites ($p<0.0001$, $p=0.001$, $p=0.011$, $p=0.004$, $p=0.004$, respectively) (Table 3).

**Table 1.** Sociodemographic, clinical and laboratory characteristics of the study population (n = 99)

| Variables | | Value |
|---|---|---|
| **Age, years*** | | 71.7 ± 5.2 |
| **Age groups, n (%)** | 65-74 years | 68 (68.7) |
| | 75-84 years | 31 (31.3) |
| **Gender, n (%)** | Male | 58 (58.6) |
| | Female | 41 (41.4) |
| **Etiology, n (%)** | Hepatitis C | 31 (31.3) |
| | Cryptogenic | 29 (29.3) |
| | Chronic hepatitis B | 25 (25.3) |
| | Alcohol | 7 (7.1) |
| | NASH | 4 (4) |
| | Others | 3 (3) |
| **Laboratory tests*** | Albumin (g/dL)* | 3.08 ± 0.66 |
| | Total bilirubin (mg/dL)** | 1.61 (0.33-17.20) |
| | ALP (U/L)** | 106.00 (34-1055) |
| | ALT (U/L)* | 36.05 ± 24.81 |
| | AST (U/L)* | 61.54 ± 48.65 |
| | GGT (U/L)** | 54 (4-1563) |
| | PT (seconds)* | 15.88 ± 3.26 |
| | Hb (g/dL)* | 11.18 ± 2.29 |
| | Platelets ($x10^9$/L)** | 103000 (5000-674000) |
| | AFP (IU/ml)* | 11.07 ± 2.33 |
| **SAAG, n (%)** | below 1.1 | 1 (1.1) |
| | 1.1 and above | 38 (38.4) |
| | Unknown | 60 (60.6) |
| **Child-Turcotte-Pugh class, n (%)** | A | 25 (25) |
| | B | 44 (45) |
| | C | 30 (30) |
| **CHILD score*** | | 8.47 ± 2.47 |
| **MELD score*** | | 13.89 ± 5.92 |
| **Precence of ascites, n (%)** | Present | 64 (64.6) |
| **Esophageal varice, n (%)** | Present | 77 (77.8) |
| | Absent | 11 (11.1) |
| | Undetectable | 11 (11.1) |
| **SAI, n (%)** | Present | 6 (6.06) |
| **HCC, n (%)** | Present | 42 (42.4) |
| | Absent | 57 (57.6) |
| **Died at hospital, n (%)** | Yes | 11 (11.1) |

*Variables are given as mean ± SD

**Variables are given as median (minimum - maximum)

NASH: non-alcoholic steatohepatitis, HCC: hepatocellular carcinoma, HE: hepatic encephalopathy, LC[a]: liver cirrhosis (Etiology and advanced examination), HRS: hepatorenal syndrome, SAI: spontaneous ascites infection, DM: diabetes mellitus, ALP: alkaline phosphatase, ALT: alanin aminotransferase, AST: aspartat aminotransferase, GGT: gama-glutamyltransferase, PT: protrombin time, Hb: hemoglobin, AFP: $\alpha$-fetoprotein, SAAG: serum-acid albumin gradient, MELD: model for end-stage liver disease.

Others: Hepatitis B (with delta-agent), primary biliary cirrhosis and primary sclerosing cholangitis

**Table 2.** Relationship between HCC and other variables

| Characteristics | HCC (n=42) | P value |
|---|---|---|
| **Etiology, n (%)** | | **0.003** |
| **HBV** | 18 (72) | |
| **HCV** | 14 (45.2) | |
| **Cryptogenic** | 7 (24.1) | |
| **Alcohol use** | 1 (12.5) | |
| **NASH** | 2 (33.3) | |
| **Presence of ascites, n (%)** | 21 (50) | **0.009** |
| **MELD score*** | 11 (6-26) | **0.033** |
| **CTP score*** | 7 (5-14) | **0.003** |

*median (minimum-maximum)

HCC: hepatocellular carcinoma, HB: hepatitis B virus, HCV: hepatitis C virus, NASH: Non-alcoholic steatohepatitis, MELD: model of end-stage liver disease, CTP: Child-Turcotte-Pugh,

**Table 3.** Relationship between esophageal varices and other variables

| Characteristic | Esophageal varices, (n=77) | P value |
|---|---|---|
| **CTP class, n (%)** | | **<0.0001** |
| **A** | 14 (63.6) | |
| **B** | 36 (94.7) | |
| **C** | 27 (96.4) | |
| **HCC, n (%)** | 27 (79.4) | 0.069 |
| **Presence of ascites, n (%)** | 55 (94.8) | **0.004** |
| **Total bilirubin* (mg/dL)** | 1.76 (0.33-17.2) | **0.011** |
| **Hemoglobin* (g/dL)** | 10.8 (5.1-16.9) | 0.077 |
| **MELD score*** | 13 (7-33) | **0.004** |
| **CTP score*** | 8 (5-15) | **0.001** |

*median (minimum-maximum)

CTP: Child-Turcotte-Pugh, HCC: hepatocellular carcinoma, MELD: Model of End-stage Liver Disease.

## DISCUSSION

To the best of our knowledge, this is the first study that provides comprehensive data about the epidemiology and clinical presentation of LC in an elderly population in Turkey. We retrospectively evaluated patients aged 65 years and older, who were hospitalized at our hospital gastroenterology clinic for any reason over a period of 24 months with a previous diagnosis of LC or those diagnosed with LC during their hospital stay.

Liver cirrhosis is an important health problem causing mortality and morbidity throughout the world, including Turkey. In a study conducted in Turkey, it was determined that among the causes of death in urban areas, LC ranked 19$^{th}$ in all age groups for women and men while in rural areas, it was the 13$^{th}$ leading cause of mortality for men and 17$^{th}$ for women aged over 60 years (16). In a study conducted in the United States, LC was found to be the 12$^{th}$ cause of death (6). In the current study, 11 cases (11.1%) died during their stay at the hospital (Table 1). Of these patients, 8 (72.7%) were male. Concerning the reasons for hospitalization among the mortality cases, the most common was HES at 54.5%. Furthermore, the death of half of the 6 SAI cases once again shows the severe nature of these infections.

Excessive alcohol consumption and viral hepatitis are the two leading causes of LC in the world. Due to socioeconomic and cultural differences, while viral hepatitis plays an important role in Asian countries, alcohol use is at the forefront in the United States and Western European countries. In addition, it is considered that with the increase in the incidence of obesity, NASH and related cirrhosis will become more prominent in these countries (7).

When the etiology of our patients was examined (Table 1), the first five factors were found to be HCV (31.3%), cryptogenic cirrhosis (29.3%), HBV (25.3%), alcohol use (7.1%), and NASH (4%). Similarly, in the current study, a viral etiology was detected in 56.6% of our cases and alcohol use in 7.1%. In a study conducted by Ökten et al. (3), viral hepatitis (55.1%), cryptogenic cirrhosis (16.4%), and alcohol consumption (12.4%) were found responsible in the etiology of LC, which is consistent with the order of causes in our study. Ökten et al. (3) showed that among the viral agents causing cirrhosis, HBV ranked first and HCV second. In contrast, HCV was the most common cause of LC in our study. In the literature, HBV is usually the leading factor in the etiology of LC in the majority of Asian countries (18-21). These results are not consistent with our study.

In our study, HCC was the most common reason for hospitalization (Table 1), which is not consistent with the literature from Turkey and other parts of the world. Considering that our clinic is a general reference center, we attribute this finding to most patients presenting to our hospital with HCC being admitted for a short term for transarterial chemoembolization and radiofrequency ablation. Hepatitis C virus was present in 42.4% of our cases and absent in 57.6% (Table 1). We found a statistically significant relationship between HCC and LC etiology ($p = 0.003$). In patients with HCC, the incidence of HBV was the highest (42.9%), followed by HCV (33.3%). These results are consistent with the strong correlation between HBV and HCC prevalence reported worldwide (20, 22).

In this study, interestingly and contrary to expectations, an inverse relationship was found between the prevalence of HCC and the CTP ($p = 0.003$) and MELD ($p = 0.033$) scores, and this relationship was statistically significant for both scores. The frequency of HCC was found to be less in cases with high CTP and MELD scores. The possible reasons for this situation are that the HCC cases were admitted to our clinic overnight for radiofrequency ablation and transarterial chemoembolization, and early-stage HCC patients diagnosed during follow-up were referred from an external center to our clinic as a reference center.

97 % of the participants who had ascites and had undergone puncture and had complete data were found to have a SAAG value of 1.1 or above while only 3% of them had less than 1.1 SAAG. These findings are in agreement with the literature (23).

Esophageal varices were detected in 77.8% of our patients, with hospitalization due to esophageal variceal bleeding accounting for 9.1% of the total sample. Considering that our clinic is a reference center, it can be

stated that cases associated with esophageal variceal bleeding mostly present to our clinic for follow-up and treatment.

The expected findings of the study were CTP Class A being seen at a higher rate in cases without esophageal varices and CTP Class B being more common in those with esophageal varices ($p<0.0001$), higher MELD ($p=0.004$) and CTP ($p=0.001$) scores and elevated total bilirubin level ($p=0.011$) in cases with esophageal varices, and esophageal varices being more common in cases with ascites ($p=0.004$). Furthermore, hemoglobin levels were lower in patients with esophageal varices due to non-apparent bleeding. However, there was no statistically significant difference between the presence of esophageal varices and hemoglobin values.

This study has potential limitations. First, the retrospective nature of our study is a limitation. Secondly, as this is a single center study, our results do not reflect the whole Turkish population. Thirdly, 30 patients over 65 years of age were not included in the study, because of missing data. Additionally, higher obesity rates in women in our country might partially play a role in higher cryptogenic cirrhosis rates among women in our study. However, body mass index values of the patients were not available in our hospital records. Besides, the prevalence of NASH is likely to be under-reported in this study. Some patients were diagnosed at a time when the role of NASH in liver cirrhosis was not routinely recognized.

Hepatitis C virus was the primary etiology of LC in the elderly population admitted to our clinic and accounted for almost one-third of the cirrhotic cases. Hepatocellular carcinoma is the leading cause of hospitalization. This result can be attributed to our clinic being a general reference center, to which most HCC patients are referred for short-term hospitalization for transarterial chemoembolization and radiofrequency ablation. According to the CTP classification, Classes B and C were mostly seen in the majority of LC cases, suggesting that the LC of the patients admitted to our clinic was at a more advanced level. Ascites was detected in the majority of our LC patients, most of whom had an SAAG of 1.1 or above. Elderly patients hospitalized with the diagnosis of LC should be carefully evaluated for cirrhosis complications, CTP and MELD scoring should be performed, and the suitability of this patient group for liver transplantation should be considered in the follow-up and treatment processes.

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