

# Mortality Factors for Cirrhotics in an Ivorian University Hospital (Ivory Coast)

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## Abstract

**Background:** Cirrhosis, an ineluctable development of chronic liver disease, still has high mortality throughout the world despite many advances in physiopathology and therapy. This high mortality is closely related to the unpredictable course of cirrhosis with numerous complications. **Objective:** To evaluate the predictive factors of death during cirrhosis. **Materials and methods:** This is an observational, descriptive study on cirrhotic patients hospitalized in the hepatology unit of the Center Hospitalier Universitaire in Bouake (Ivory Coast) during the period from January 01, 2018 to December 31, 2019. The study focused on the data collected in the medical files (paper format) of hospitalized cirrhotics and the death register of the service. The diagnosis of cirrhosis and the death of the cirrhotic were the main criteria for judgment. The secondary criteria defined were: history of cirrhosis, reasons for consultation, clinical signs, biological signs, complications of cirrhosis and the treatment received. The relationship between the parameters was expressed as an odds ratio (OR) with a 95% confidence interval (CI) and the significance threshold fixed at  $p < 0.05$ . **Results:** The study retained 206 files of cirrhotics including 146 men and 60 women. The hospital prevalence of cirrhosis was 44.17% and the mortality rate was 42.23%. The deceased patients were mainly men with an average age of 49 years. The etiologies of cirrhosis were dominated by viral hepatitis B, chronic alcohol poisoning and viral hepatitis C, respectively 46.95%, 37.35% and 10.84%. The predictors of death were: the presence of icterus (OR = 1.89, 95%CI [1.08 - 3.30],  $p = 0.036$ ), hepatic encephalopathy (OR = 8.75, 95%CI [4.51 - 16.94],  $p < 0.001$ ) and hepatocellular carcinoma (OR = 2.23, 95%CI [1.25 - 3.98],  $p = 0.010$ ); the presence in the biology of hepatic cytolysis (OR = 2.57, 95%CI [1.35 - 4.89],  $p = 0.006$ ), severe hepatocellular insufficiency (OR = 2.57, 95%CI [1.38 - 4.77],  $p = 0.004$ ), severe renal insufficiency (OR = 2.41, 95%CI [1.09 - 5.32],  $p = 0.044$ ) and hyperleukocytosis (OR = 2.28, 95%CI [1.29 - 4.04],  $p = 0.007$ ); Child-Pugh stage C (OR = 19.64, 95%CI [9.02 - 42.74],  $p < 0.001$ ); the pres-

ence on ultrasound of large liver and variable nodules (OR = 4.16, 95%CI [2.02 - 8.58],  $p < 0.001$ ). **Conclusion:** Cirrhosis is a public health problem at the Bouake university hospiler center. Decompensated and complicated old cirrhosis, hepatic cytolysis, severe hepatocellular insufficiency, severe renal insufficiency and hyperleukocytosis, heterogeneous nodular large liver are the detrimental factors.

## Keywords

Cirrhosis, Hepatocellular Carcinoma, Mortality, CHU Bouake

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## 1. Introduction

Cirrhosis, an inevitable development of chronic liver disease, is a chronic and diffuse inflammatory disease of the liver, defined histologically by the association of fibrosis, hepatocyte necrosis and regeneration nodules [1]. In Europe, its etiology is dominated by the excessive and chronic consumption of alcohol. On the other hand, in Africa, precisely in sub-Saharan Africa, it is the viral hepatitis B and C which are the main cause [2] [3]. Mortality from cirrhosis is still high despite many advances in pathophysiology and therapy. This high mortality is closely related to the unpredictable course of enamel cirrhosis with numerous complications [4]. According to the WHO, cirrhosis and hepatocellular carcinoma are responsible for 1.4 million deaths each year worldwide [5]. The occurrence of complications related to cirrhosis would increase the mortality of this disease in hospital. Several works carried out in Europe [2], America [6] and Africa [3] have studied different aspects of cirrhotic disease. However, in Côte d'Ivoire, to our knowledge, few studies exist on the prognostic factors of cirrhosis [7] [8]. The purpose of this study concerning cirrhotics who died in hospital was to investigate the predictors of mortality from cirrhosis in hospital.

## 2. Material and Method

This was a monocentric retrospective cohort study on cirrhotic patients hospitalized in the hepatogastroenterology unit of the Center Hospitalier Universitaire de Bouake (Ivory Coast) during the period from January 01, 2018 to December 31, 2019, over a period of 2 years. The study focused on the data collected in the medical files (paper format) of hospitalized cirrhotics and the death register of the service.

### 2.1. The Study Protocol

Two main judgment criteria have been identified:

- The diagnosis of cirrhosis posed by the combination of clinical, biological, ultrasound and endoscopic arguments, the anatomopathological examination not being carried out.

- The death retained is that which occurred in hospitalization in the cirrhotic patient during the study period, in the hepatato-gastroenterology unit of the Center Hospitalier Universitaire de Bouake.

The following parameters were the secondary endpoints. These were: a history related to cirrhosis (viral hepatitis B, old C, old cirrhosis, number of previous hospitalizations, number of previous complications, puncture of ascites fluid). Clinical signs. The length of hospital stay in days. The reason for hospitalization. Biological signs (Blood count: Hyperleukocytosis defined by the number of white blood cells  $> 10.000$  elements/mm<sup>3</sup> or leukopenia  $< 5000$  elements/mm<sup>3</sup>). Severe anemia defined by hemoglobin level  $< 8$  g/dL. Thrombocytopenia defined by the number of platelets  $< 150.000$  elements/mm<sup>3</sup>. Severe hyponatremia:  $< 125$  meq/L. Hyperkalemia  $> 5$  meq/L. The viral markers of hepatitis carried out: Ag HBs, Ac anti HBc, Ac anti HVC, Ac anti VHD. The following complications have been defined:

- Infection of ascites fluid (defined by exudative ascites with more than 250 elements/mm<sup>3</sup>).
- Externalized digestive hemorrhage in the form of hematemesis and/or melena.
- Hepatocellular carcinoma diagnosed on the basis of tumor hepatomegaly and the presence of portal thrombosis, nodules of varying sizes on radiology (ultrasound or computed tomography).
- Hepatic encephalopathy classified according to the different stages.
- Hepatorenal syndrome associating functional renal failure (urea  $> 0.45$  g/L and creatinine  $> 14$  mg/L) and refractory ascites in a cirrhotic patient with edemato-ascitic decompensation.
- Cytolysis defined by an elevation of transaminases (ALT  $> 50$  IU/L and/or ASAT  $> 50$  IU/L).
- Cholestasis defined by an elevation in bilirubinemia (total bilirubin  $> 15$  mg/L and/or conjugated bilirubin  $> 8$  mg/L).
- The treatment received was collected (antibiotics, diuretics, analgesics, lactulose, beta-blockers and paracentesis)

Sociodemographic characteristics and a history of comorbidity were the confounding factors.

## 2.2. Statistics

For input and analysis, we used SPSS® version 20 software and the figures were produced with Excel software. The analysis consisted initially of a descriptive analysis (means and frequencies) of the variables studied. In a second step, we established a predictive model of patient mortality based on the variables studied, using logistic regression. This relationship was expressed as an odds ratio (OR) with their 95% confidence interval (CI). The significance test fixed at  $p < 0.05$ .

## 2.3. Ethical Consideration

Under the investigation practices act, we protected the confidentiality of infor-

mation that was collected during the investigation by assigning an anonymity number to each investigation sheet. In addition, an authorization was requested and obtained in advance from the administrative and medical authorities of the Bouake University Hospital Center.

## 2.4. Results

From January 2018 to December 2019, the gastroenterology unit registered 781 hospitalized patients, including 345 cases of cirrhosis, *i.e.* a hospital prevalence of cirrhosis of 44.17%. Among the cirrhosis cases, 206 files were usable, including 87 deaths, representing a hospital mortality rate of 42.23%. There was a predominance of men in their fifties with a sex ratio (146/60) of 2.43. The etiologies of cirrhosis were dominated by viral hepatitis B, chronic alcohol poisoning and viral hepatitis C, respectively 46.95%, 37.35% and 10.84%. Viral B and C coinfection was found in 1.94% of cases.

The general characteristics of cirrhotic patients are summarized in **Table 1** and **Table 2**.

The death was not statistically related to age, gender, complications of ascites fluid infection and digestive hemorrhage, hyperkalemia and endoscopic signs of portal hypertension.

Death was significantly related to a history of complicated cirrhosis and paracentesis. Clinically, the presence of hepatomegaly, signs of decompensation, jaundice, edema and ascites were statistically related to death. Biologically, the laboratory results were disturbed in patients who died with cytolysis, leukocytosis, renal and hepatocellular failure. On abdominal ultrasound, hepatomegaly with nodules of varying sizes was significantly associated with death (**Table 1**).

In multivariate, the predictors of death are summarized in **Table 3**, **Table 4** and **Table 5**.

Death was correlated with a history of paracentesis and known complicated cirrhosis. icterus, hepatomegaly, hepatic encephalopathy and hepatocellular carcinoma diagnosed during hospitalization were predictive of death. Regarding biology, death was correlated with hepatic cytolysis (ASAT > 50 IU/L and ALT > 50 IU/L), hepatocellular insufficiency (TP < 60%, Albuminemia < 30 g/L) with insufficiency renal (serum creatinine > 20 mg/L) and hyperleukocytosis (number of white blood cells > 10,000 elements/mm<sup>3</sup>). On ultrasound, hepatomegaly with nodules of varying sizes was predictive of death.

Death was correlated with the progressive stage of the disease, and the prescription of lactulose or tramadol in hospital. However, the prescription of spironolactone, beta-blockers and paracentesis iterative in hospital was associated with a good course of cirrhotic disease (**Table 5**).

## 3. Discussion

The hospital prevalence and mortality rate of cirrhosis are still high as shown in this study, like the African literature [3] [4] [9] [10]. Cirrhotic patients were

**Table 1.** Compared clinical and paraclinical characteristics of deceased and non-deceased cirrhotic patients.

Variables	Deceased n (%)	No deceased n (%)	p
<b>Sex</b>			
• M	62 (71.26)	84 (79.59)	1.000
• - F	25 (8.74)	35 (29.41)	1.000
<b>Age</b> < 50	46 (52.87)	60 (50.42)	0.83
>50	41 (47.13)	59 (49.58)	
<b>History</b>			
• Comorbidities	17 (19.54)	12 (10.08)	0.085
• <b>Known cirrhosis</b>	<b>22 (25.29)</b>	<b>9 (7.56)</b>	<b>0.001</b>
• <b>Complications</b>	<b>21 (24.14)</b>	<b>12 (10.08)</b>	<b>0.012</b>
• <b>Paracentesis</b>	<b>13 (14.94)</b>	<b>5 (4.20)</b>	<b>0.014</b>
<b>Reasons for consultation</b>			
• <b>Edemato-ascites decompensation</b>	<b>18 (20.70)</b>	<b>42 (35.29)</b>	<b>0.034</b>
• <b>Icterus</b>	<b>31 (35.63)</b>	<b>23 (19.33)</b>	<b>0.014</b>
• Abdominal pains	20 (22.99)	27 (22.69)	1.000
• Gastrointestinal bleeding	10 (11.49)	12 (10.08)	0.92
• Hepatic encephalopathy	3 (3.45)	2 (1.69)	0.72
<b>Physical signs</b>			
• Ascite	62 (71.26)	92 (77.31)	0.410
• Edema of the lower limbs	43 (49.43)	74 (62.18)	0.092
• <b>Icterus</b>	<b>48 (55.17)</b>	<b>47 (39.50)</b>	<b>0.036</b>
• Lateral venous circulation	22 (25.29)	24 (20.17)	0.483
• <b>Large liver</b>	<b>64 (73.56)</b>	<b>55 (46.22)</b>	<b>0.001</b>
• Large spleen	11 (12.64)	12 (10.08)	0.725
• <b>Lenght of hospital stay ≤ 7</b>	<b>63 (72.41)</b>	<b>84 (70.58)</b>	<b>0.896</b>
• <b>Lenght of hospital stay &gt; 7</b>	<b>24 (27.59)</b>	<b>35 (29.41)</b>	
<b>Biology</b>			
• <b>Creatinemia (mg/L) &gt; 20</b>	<b>18 (21.43)</b>	<b>12 (10.17)</b>	<b>0.044</b>
• Blood urea (g/L) > ou = 0.45	28 (33.33)	26 (22.03)	0.104
• Kaliemia (meq/L) > ou = 5	14 (20.00)	22 (18.97)	1.000
• Natremia (meq/L) < 125	5 (7.14)	3 (2.59)	0.267
• Total bilirubin (mg/L) > 10	52 (92.86)	107 (94.69)	0.897
• <b>Prothrombin nevel (%) &lt; 60</b>	<b>63 (75.90)</b>	<b>65 (55.08)</b>	<b>0.004</b>
• <b>Albuminemia (g/L) &lt; 30</b>	<b>54 (78.26)</b>	<b>72 (62.61)</b>	<b>0.040</b>
• <b>ALT (UI/L) &gt; 50</b>	<b>59 (76.62)</b>	<b>65 (56.03)</b>	<b>0.006</b>
• <b>ASAT (UI/L) &gt; 50</b>	<b>69 (89.61)</b>	<b>86 (73.50)</b>	<b>0.011</b>
• <b>White blood cells (elements/mm<sup>3</sup>) &gt; 10,000</b>	<b>45 (51.72)</b>	<b>38 (31.93)</b>	<b>0.007</b>
• Hemoglobin (g/dl) < 8	21 (24.14)	30 (25.21)	0.990
<b>Ultrasound</b>			
• Ascit	55 (87.30)	88 (85.44)	0.915
• <b>Nodule variable sizes</b>	<b>34 (53.97)</b>	<b>38 (36.89)</b>	<b>0.046</b>
• Homogeneous nodules	29 (46.03)	60 (58.25)	0.170
• Portal thrombosis	4 (6.65)	9 (8.74)	0.768
• <b>Large liver</b>	<b>50 (79.37)</b>	<b>49 (47.57)</b>	<b>0.001</b>
• Large spleen	12 (19.05)	24 (23.30)	0.652
<b>Endoscopy</b>			
• Esophageal varices	16 (84.21)	48 (84.48)	1.000
• Red signs	8 (42.10)	12 (20.69)	0.122

**Table 2.** Comparative prognostic and therapeutic characteristics of deceased and non-deceased cirrhotic patients.

Variables	Deceased n (%)	No deceased n (%)	p
<b>Complications</b>			
• Liquid ascite infection	5 (5.75)	7 (5.88)	1.000
• HCC	<b>41 (47.13)</b>	<b>34 (28.57)</b>	<b>0.010</b>
• Gastrointestinal bleeding	20 (22.99)	22 (18.49)	0.537
• Hepatic encephalopathy	<b>53 (60.92)</b>	<b>18 (15.13)</b>	<b>&lt;0.001</b>
• Hepatorenal syndrome	<b>4 (4.60)</b>	<b>0 (0.00)</b>	<b>0.031</b>
<b>Pronostic score</b>			
• Child-Pugh A	<b>0 (0.00)</b>	<b>9 (7.83)</b>	<b>0.027</b>
• Child-Pugh B	<b>26 (32.50)</b>	<b>95 (82.61)</b>	<b>0.001</b>
• Child-Pugh C	<b>54 (67.50)</b>	<b>11 (9.57)</b>	<b>&lt;0.001</b>
<b>Treatment</b>			
• Fluoroquinolone	12 (13.79)	25 (21.01)	0.25
• Tramadol	<b>69 (79.31)</b>	<b>68 (57.14)</b>	<b>0.001</b>
• Furosemide	8 (9.20)	12 (10.08)	1.000
• Spironolactone	<b>5 (5.75)</b>	<b>31 (26.05)</b>	<b>&lt;0.001</b>
• Lactulose	<b>72 (82.76)</b>	<b>73 (61.34)</b>	<b>0.002</b>
• Betablockers	<b>9 (10.34)</b>	<b>31 (26.05)</b>	<b>0.008</b>
• Blood transfusion	19 (21.84)	30 (25.21)	0.69
• Number paracentesis $\geq 2$	<b>22 (25.29)</b>	<b>54 (45.38)</b>	<b>0.005</b>

**Table 3.** Clinical factors predictive of death.

Factors	Odds ratio	IC à 95%	p
<b>History of cirrhosis</b>			
• Paracentesis: yes vs No	4.01	[1.37 - 11.70]	0.014
• Complications: yes vs No	2.83	[1.31 - 6.14]	0.012
• Known cirrhosis: yes vs No	4.13	[1.80 - 9.52]	0.001
<b>Clinical signs</b>			
• Icterus: yes vs No	1.89	[1.08 - 3.30]	0.036
• Large liver: yes vs No	3.24	[1.78 - 5.88]	<b>&lt;0.001</b>
<b>Complications diagnosed in hospital</b>			
• HCC: yes vs No	2.23	[1.25 - 3.98]	0.010
• Hepatic encephalopathy: yes vs No	8.75	[4.51 - 16.94]	0.001

**Table 4.** Paraclinical factors predictive of death.

Factors	Odds ratio	IC à 95%	p
<b>Biological signs</b>			
• ALT (UI/L): >50 vs ≤50	2.57	[1.35 - 4.89]	0.006
• ASAT (UL/L): >50 vs ≤50	3.11	[1.34 - 7.20]	0.011
• Creatinemia (mg/L): >20 vs ≤20	2.41	[1.09 - 5.32]	0.044
• Prothrombin nevel (%): <60 vs ≥60	2.57	[1.38 - 4.77]	0.004
• Albuminemia (g/L): <30 vs ≥30	2.15	[1.08 - 4.27]	0.040
• White blood cells (elements/mm <sup>3</sup> ): >10,000 vs ≤10,000	2.28	[1.29 - 4.04]	0.007
<b>Ultrasound signs</b>			
• Large liver: yes vs No	4.16	[2.02 - 8.58]	<0.001
• Nodules of varying sizes: yes vs No	2.01	[1.06 - 3.79]	0.046

**Table 5.** Prognostic and therapeutic factors predictive of death.

Factors	Odds ratio	IC à 95%	p
<b>Pronostics factors</b>			
• Child-Pugh C: yes vs No	19.64	[9.02 - 42.74]	0.001
<b>Treatment</b>			
• Tramadol: yes vs No	2.88	[1.53 - 5.42]	0.001
• Spironolactone: yes vs No	0.17	[0.06 - 0.47]	0.000
• Lactulose: yes vs No	3.02	[1.55 - 5.90]	0.002
• Betablockers: yes vs No	0.33	[0.15 - 0.73]	0.008
• Number of paracentesis: ≥2 vs <2	0.41	[0.22 - 0.74]	0.005

generally men in their fifties as reported in several studies in Africa and Europe [2] [10] [11]. Patients very often consult at the edemato-ascitic decompensation stage as reported by our results and other African studies [12]. This observation testifies to the delay in consulting cirrhotic patients, most often at the decompensation stage where the presence of ascites reflects the development of cirrhosis [9] [10] [13]. In this study the etiology of cirrhosis was dominated by viral hepatitis B and alcohol. Chronic alcoholism takes an important place in the etiology of chronic liver disease in Africa due to the excessive consumption of alcohol most often adulterated by our young and often idle populations as reported by other African authors [8] [10] [14]. Ultrasound anomalies are dominated by nodular hepatomegaly with nodules of varying sizes [3] [9]. Indeed, abdominal ultrasound allows, in addition to the non-invasive diagnosis of cirrhosis [15], to strongly suspect hepatocellular carcinoma in 26% of cases when the liver is heterogeneous with nodules of variable sizes [9]. Digestive fibroscopy (FOGD) was poorly performed (37.38%) due to its high cost and the clinical in-

stability of hospitalized cirrhotic patients. The endoscopic anomalies were dominated respectively by grade 2 and 3 esophageal varices (78%) with the presence of red signs in 26% of the cases and a mosaic gastropathy (49.35%). These results are consistent with African literature [9] [16]. The complications of hepatocellular carcinoma and hepatic encephalopathy diagnosed in the hospital were the same as those described by African and European authors [1] [17] [18] [19]. In multivariate analysis, concerning sex, the results differ according to the authors. Some authors do not find a link between the male sex and death in the cirrhotic patient [10] [18] [19] as in our work. On the other hand, other authors have found an association between death and the male sex [7] [12] [17].

The death in men could be explained by the increased risk of developing HCC, higher alcohol consumption and a high prevalence of HBV. These factors could worsen the prognosis of cirrhosis regardless of the etiology [20]. The age of onset of death less than 50 years has been found in several studies in Africa [10] [18], unlike in Europe [19] [20] where death occurs after 50 years. In this study, the history of complicated and decompensated cirrhosis was predictive of death. Hepatic encephalopathy testifying to severe hepatic insufficiency is strongly implicated in mortality during cirrhotic disease [17] [18]. Jaundice has been reported by several authors to be a predictor of death [10] [18] [21]. Hepatomegaly could be related to cirrhosis itself or to the most dreaded complication of cirrhosis: hepatocellular carcinoma (HCC). The occurrence of HCC on cirrhosis reduces the short-term survival of the cirrhotic patient [1] [17]. The occurrence of ascites in cirrhotics is known to be a factor of poor prognosis, especially infected ascites [10] [14] [18]. Some authors have shown that infection with ascites fluid significantly reduces survival in cirrhotics [8] [17]. The digestive hemorrhage was found mainly in the event of death ( $p = 1.000$ ) not significantly. According to several studies, digestive hemorrhage is frequently cited as a detrimental factor in cirrhosis [10] [18]. The prognostic score studied was that of the Child-Pugh classification. The prognostic value of Child Pugh's score in cirrhosis has been the subject of several publications [7] [19] [22]. The evolution of cirrhosis was significantly linked to the Child-Pugh prognosis stage with a very pejorative stage C ( $p < 0.001$ ; OR = 19.64; CI = 9.02 - 42.74) according to the data in the literature [7] [14] [17]. Regarding biology, the results were generally disturbed in the cirrhotic patients who died in our study. We found an independent association between severe renal impairment (creatinine  $> 20$  mg/L), hepatic cytolysis, hepatic impairment and death. Biological balance abnormalities have been frequently found in several studies concerning the development of cirrhosis. According to Attia in a study carried out in Ivory Coast, the patients having a serum creatinine  $> 15$  mg/L on admission died more than those whose creatinine is  $\leq 15$  mg/L with a risk of death multiplied by 4.5 of cirrhotic in case renal failure [7] [13]. Other authors in France and Morocco [18] [19] have also implicated renal failure in the mortality of the cirrhotic patient. Claire *et al.* have shown that an ASAT value  $\geq 80$  IU/L was a factor in poor prognosis [1]. Hepa-



tocellular insufficiency (HCI) with severe hypoalbuminemia and hypoprothrombinemia was associated with death in several studies [7] [14] [18]. Indeed, the impact of the HCI on the survival of the cirrhotic has long been proven so much that it is an important element of the classification of Child-Pugh [7]. Dyskalemia has been reported in several studies as a predictor of death [8] [13] [18]. Observation which was not made in this study. Hyperleukocytosis is a reflection of sepsis frequently found in cirrhotic patients [17], due to a global dysfunction of innate and adaptive immunity inducing a greater susceptibility to bacterial infections [23]. The presence of signs of portal hypertension on upper gastrointestinal endoscopy is associated with high mortality [14] [17]. For some authors, it is rather the rupture of esophageal varices which is implicated in mortality in cirrhotic patients [10]. In our study, no endoscopic signs were significantly linked to death. This observation could be explained on the one hand, by the very low percentage (37.38%) of esogastroduodenal fiberoscopy (OGDF) carried out in order to look for signs of portal hypertension and on the other hand, by the systematic prescription of beta-blockers as part of the primary prevention of digestive haemorrhage in cirrhotics in the event of VO. In fact, the prevention of rupture of esophageal varices by beta-blockers improves survival in cirrhotics [8].

The limit of this study is mainly related to the relatively high cost of certain examinations and the insufficient technical platform which did not allow a more exhaustive exploration of certain data. Nevertheless, this study made it possible to highlight in a significant way the predictive factors of mortality in cirrhotic patients in an Ivorian hospital service.

#### 4. Conclusion

Cirrhosis is a public health problem at the Bouaké University Hospital Center (Ivory Coast) with hospital prevalence and mortality still high. The diagnosis is made at the stage of complications and they are dominated by hepatocellular carcinoma and hepatic encephalopathy. In single or multivariate analysis, the history of complicated cirrhosis, the presence of signs and treatment related to hepatocellular carcinoma, hepatic encephalopathy, biochemical activity, severe hepatocellular and renal failure were correlated with death. An early diagnosis of cirrhosis and its complications, as well as regular monitoring, will help detect and correct these factors. This also involves screening upstream for viral hepatitis and raising awareness about excessive alcohol consumption.

#### Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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