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Gastroduodenal Ulcer in Cirrhotic Patients: Epidemiological, Diagnostic, and Prognostic Aspects at Libreville Teaching Hospital

Patrice Emery Itoudi-Bignoumba*, Patrick Dieudonné Nzouto, Luce Moukiringome Mamboundou, Inès Flore Maganga Moussavou, Agnès Angela Engoang, Monique Mbounja, Arlette Nsegue, Jean Baptiste Moussavou Kombila

Hepatogastroenterology Department, Libreville Teaching Hospital (CHU de Libreville), Libreville, Gabon
Email: *itoudi_emery@hotmail.com

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Abstract

Introduction: The association between peptic ulcer disease (PUD) and cirrhosis is regularly observed in practice but has never been the subject of a scientific publication in Gabon. The aim of our study was to establish the epidemiological, diagnostic, and prognostic aspects of PUD in cirrhotic patients hospitalised at the Libreville Teaching Hospital (CHUL). **Patients and Methods:** This is a retrospective, descriptive, and comparative bivariate analytical study between two groups (cirrhotic patients with PUD vs. cirrhotic patients without PUD), conducted from January 2023 to December 2024 in the hepatogastroenterology department of the CHUL. All cirrhotic patients who underwent upper digestive endoscopy were included. Clinical, biological, endoscopic, and evolutionary data were collected and analysed. Statistical analysis was performed using SPSS version 25.0 software, and Chi-2 and Student's t-tests were used for comparison, with a significance threshold of $p < 0.05$. **Results:** We included 109 cirrhotic patients divided into two groups, one with PUD (n = 35) and one without PUD (n = 74). The sex ratio was 1.06 vs 1.47. The mean age was 55.03 years ± 17.17 years vs. 48.44 years ± 15.15 years ($p = 0.013$). Hypertension and diabetes were present in 17.14% vs. 24.32% and 14.29% vs. 12.16% of patients, respectively. Abdominal pain was present in 8.57% vs. 9.46% ($p = 0.7813$). The location of the peptic ulcer was gastric in 51.43%, duodenal in 40%, and mixed in 3.857% of cases. Ascites, jaundice, and gastrointestinal hemorrhage were observed in 88.57% vs. 77.03%, 45.71% vs. 44.59%, and 40% vs. 44.59% of cases, respectively. Complications of cirrhosis were hemorrhagic shock (40% vs. 29.73%), followed by primary liver cancer (5.71% vs. 14.86%) and spontaneous infection of ascites fluid (5.71% vs. 8.11%). The aetiologies of cirrhosis were alcohol (37.14% vs. 33.78%), viral hepatitis B (31.43% vs. 22.97%), and C

(17.14% vs. 12.16%). Stress was the risk factor for PUD that showed a statistical link (p = 0.0106), whereas the search for chronic *H. pylori* infection did not. In terms of prognosis, PUD was classified as Forrest III in 68.57% of patients, Forrest IIB in 22.86%, and Forrest IIC in 8.57%. Cirrhosis was classified as Child-Pugh A in 20% vs. 28.38% (p = 0.3499), Child-Pugh B in 48.57% vs. 52.70% (p = 0.6870), and Child-Pugh C in 31.43% vs. 18.92% (p = 0.1469). There were 22.86% deaths versus 22.97%. The causes of death were hemorrhagic shock (5 deaths out of 8) versus primary liver cancer (9 deaths out of 17, with p = 0.0128). **Conclusion:** The association between peptic ulcer disease and cirrhosis is common and affects middle-aged men with comorbidities. The role of *H. pylori* remains unclear, and the prognosis is worsened by hemorrhagic shock.

Keywords

Peptic Ulcer Disease, Cirrhosis, Gastrointestinal Bleeding, Prognosis, Libreville

1. Introduction

Peptic ulcer disease (PUD) is a deep, localized loss of substance in the gastric and/or duodenal wall secondary to an imbalance between the protective and aggressive factors affecting this wall [1] [2]. It affects up to 10% of the world's population, and its main causes are chronic *H. pylori* infection, the intake of gastrotoxic substances, and stress [1]-[4]. Its incidence is declining in developed countries thanks to antisecretory treatment and the eradication of *H. pylori*, while Africa remains in a paradoxical situation with a high prevalence of *H. pylori* but not a high prevalence of PUD [4]-[6]. Cirrhosis is the final stage in the progression of chronic liver disease [7] [8]. It is responsible for vascular disorders that weaken the gastroduodenal wall and thus increase the likelihood of PUD [7] [8]. In addition, the presence of PUD in a cirrhotic patient increases the risk of hemorrhage and hepatic encephalopathy, thus worsening their prognosis [7]-[9]. This association, which is increasingly frequent in African literature, has not yet been published in Gabon [10] [11]. It is in this context that we sought to determine the epidemiological, diagnostic, and prognostic profile of PUD in cirrhotic patients hospitalized at the Libreville Teaching Hospital Centre (CHUL).

2. Methods

This is a retrospective, descriptive, and comparative bivariate analytical study between two groups (cirrhotic patients with PUD vs. cirrhotic patients without PUD), conducted from January 2023 to December 2024 in the hepatology and gastroenterology department of the Libreville University Hospital. We included adult patients with cirrhosis who had undergone oesophagogastroduodenal endoscopy. The diagnosis of cirrhosis was either histological, based on the combination of

signs of portal hypertension, hepatocellular insufficiency, and hepatic dysmorphia, or by impulse elastometry greater than 14 KPa. The diagnosis of peptic ulcer was endoscopic, which revealed a mucosal defect with sharp edges and a fibrinous base extending beyond the muscularis mucosae. Simple erosive lesions, ulcerated tumors, and variceal lesions were excluded.

The variables studied were sociodemographic (age, sex, risk factors), clinical (abdominal pain, signs of hepatic decompensation), endoscopic (location, site, and Forrest classification of PUD), biological (coagulation tests, blood count, liver function, kidney function), and progressive (hemorrhagic recurrence, 6-month mortality, FORREST/Child-Pugh scores). Psychological stress was measured, taking into account acute and chronic stress. The Perceived Stress Scale score was adapted with a total ranging from 0 to 40, and a score above 20 was considered positive. These variables were collected and analysed using SPSS version 25.0 software. Comparisons of proportions and means were made using the Chi^2 and Student's t-tests, respectively. The significance threshold was set at $p < 0.05$.

3. Results

3.1. Population Characteristics

Using hospital admission records, we identified 209 hospitalizations for cirrhosis. We excluded 83 duplicates (readmissions) and 17 patients who had not undergone EOGD. The analysis, therefore, covered 109 cirrhotic patients. Patients were divided into two groups according to the presence of an EGD (Table 1). There were 35 patients (47.30%) with an EGD and 74 patients without an EGD. There was a consistent male predominance in both groups. The mean age was 55.03 (±17.17) years in the group with UGD and 48.44 (±15.15) years in the other group. This difference was statistically significant ($p = 0.013$). Low social status accounted for 45.72% of patients in the group with UGD and 60.82% in the group without UGD. This difference was not statistically significant ($p = 0.3332$). There were 74.29% single patients in the UGD group compared to 36.49% in the other group, with no statistically significant difference ($p = 0.5265$). Hypertension and diabetes were the most common comorbidities, with 17.14% vs 24.32% and 14.29% vs 12.16%, respectively.

Table 1. Comparison of sociodemographic characteristics of cirrhotic patients with PUD versus those without PUD.

Variables	Cirrhosis (N = 109)				p
	With PUD (N = 35)		Without PUD (N = 74)		
	n	%	n	%	
Sex					
Female	17	48.57	30	40.54	0.429
Male	18	51.43	44	59.46	
Sex-ratio	1.06		1.47		

Continued

Age					
Mean age (years)	55.03 ± 17.17		48.44 ± 15.15		**0.013**
Social level					
Low	16	45.72	45	60.82	0.3332
Middle	17	48.57	26	35.13	
High	2	5.71	3	4.05	
Marital status					
Maried	5	14.28	33	44.59	0.5265
Single	26	74.29	27	36.49	
Widowhood	4	11.43	14	18.92	
Instruction level					
Primary	13	37.14	31	41.89	0.2041
High school	17	48.57	24	32.43	
University level	5	14.29	19	25.68	
Comorbidities					
Hypertension	6	17.14	18	24.32	0.3982
Diabetes	5	14.29	9	12.16	0.7570
Chronic renal failure	2	5.71	4	5.41	0.9474
HIV	1	2.86	5	6.76	0.4046

3.2. Diagnostic Aspects

Abdominal pain was present in 8.57% of patients in the PUD group and 9.46% in the other group (p = 0.7813). The ulcer was located in the stomach in 18 cases (51.43%), in the duodenum in 14 cases (40%), and in both the stomach and duodenum in 3 cases (8.57%). The modes of decompensation of cirrhosis were similar in both groups, dominated by ascites (Table 2). The most common complications of cirrhosis were hemorrhagic shock (40% vs 29.73%), followed by primary liver cancer (5.71% vs 14.86%) and spontaneous infection of ascites fluid (5.71% vs 8.11%), with no statistical difference between the two groups. The aetiologies of cirrhosis were similar in both groups, dominated by alcohol (37.14% vs 33.78%) and viral hepatitis B (31.43% vs 22.97%) and C (17.14% vs 12.16%). As shown in Table 2, stress was the aetiological factor that showed a statistical link (p = 0.0106). Screening for chronic *H. pylori* infection was not systematic. It was present in 6 out of 24 patients (25%) in the group with PUD and 14 out of 45 patients (31%) in the other group, with no statistical difference (p = 0.8342).

With regard to cirrhosis, its modes of decompensation were similar in both groups, with ascites (88.57% vs. 77.03%), jaundice (45.71% vs. 44.59%), and gastrointestinal hemorrhage (40% vs. 44.59%), as shown in Table 2. Cirrhosis was of viral aetiology in 48.57% of the group with PUD and 35.13% in the other group,

while it was of alcoholic aetiology in 37.14% of the group with PUD versus 33.78% in the other group. These differences were not statistically significant.

Table 2. Comparative diagnostic aspects of cirrhotic patients with PUD versus without PUD.

Variables	Cirrhosis (N = 109)				p
	With PUD (N = 35)		Without PUD (N = 74)		
	n	%	n	%	
Etiological factors of PUD					
H. pylori	6	17.14	14	18.92	0.8342
Stress	3	8.57	-	-	0.0106
Smoking	7	20.00	16	21.62	0.8464
Alcohol	20	57.14	53	71.62	0.1334
Use of NSAIDs	5	11.43	5	6.76	0.2035
Etiology of cirrhosis					
Chronic alcoholism	13	37.14	25	33.78	0.731
Hepatitis B	11	31.43	17	22.97	0.345
Hepatitis C	6	17.14	9	12.16	0.481
Both or more	5	14.29	23	31.09	0.060
Mode of decompensation					
Ascites	31	88.57	57	77.03	0.1536
Jaundice	16	45.71	33	44.59	0.9126
Gastrointestinal hemorrhage	14	40.00	33	44.59	0.2871
Hepatic encephalitis	1	2.86	5	6.76	0.4046
Classification of Child-Pugh					
A	7	20.00	21	28.38	0.3499
B	17	48.57	39	52.70	0.6870
C	11	31.43	14	18.92	0.1469
Complications of cirrhosis					
Primary liver cancer	2	5.71	11	14.86	0.1687
Ascites fluid infection	2	5.71	6	8.11	0.6545
Hemorragic chock	14	40.00	22	29.73	0.2871
Hepatorenal syndrome	1	2.86	5	6.76	0.4046
Deaths	8	22.86	17	22.97	0.3651

Alcohol consumption (alcoholism) emerges as a significant independent risk factor for peptic ulcer disease, with an odds ratio of ≈4.7. The other variables (age, gender, diabetes, NSAIDs, smoking) show no significant association in this multivariate model (Table 3).

Table 3. Factors associated with peptic ulcer disease.

Variables	aOR (adjusted Odds Ratio)	CI 95%	p-value
Age	1.01	[0.98 - 1.04]	0.70
Male gender	0.60	[0.22 - 1.62]	0.31
Diabetis	0.71	[0.13 - 3.84]	0.69
Alcohol consumption	4.71	[1.41 - 15.67]	0.012
NSAIDs	1.04	[0.25 - 4.41]	0.96
Smoking	0.24	[0.03 - 2.08]	0.20

3.3. Prognostic Aspects

In terms of prognosis, PUD was classified as Forrest III in 24 patients (68.57%), Forrest IIB in 8 patients (22.86%), and Forrest IIC in 3 patients (8.57%). Cirrhosis was classified as Child-Pugh A in 20% vs. 28.38% (p = 0.3499), Child-Pugh B in 48.57% vs. 52.70% (p = 0.6870), and Child-Pugh C in 31.43% vs. 18.92% (p = 0.1469). Table 4, showing the distribution of PUD according to the Forrest classification compared to the Child-Pugh classification, revealed that 50% of Forrest III PUDs had Child-Pugh B cirrhosis and 29.17% had Child-Pugh C cirrhosis, compared with 36.36% of Forrest II PUDs that had Child-Pugh C cirrhosis and 36.36% of Forrest II UGDs that had Child-Pugh C cirrhosis. These differences were not statistically significant.

Hemorrhagic shock was the most common complication, occurring in 40% of the UGD group and 29.73% of the non-PUD group. This difference was not statistically significant (p = 0.2871). There were 22.86% deaths in the PUD group and 22.97% in the non-PUD group. The causes of death were dominated by hemorrhagic shock in the PUD group (5 out of 8 deaths) and by primary liver cancer in the non-PUD group (9 out of 17 deaths). This difference was statistically significant (p = 0.0128).

Table 4. Relationship between Child-Pugh classification and Forrest classification.

	PUD (N = 35)				p
	Forrest III (N = 24)		Forrest II (N = 11)		
	n	%	n	%	
Child-Pugh A	5	20.83	3	27.28	0.6736
Child-Pugh B	12	50	4	36.36	0.4581
Child-Pugh C	7	29.17	4	36.36	0.6702

4. Discussion

In our series of hospitalised cirrhotic patients, the frequency of PUD was 32.11%. This high frequency had already been observed by Ky *et al.* and Voulgaris *et al.*, who found frequencies of 43.2% and 19%, respectively [12] [13]. During cirrhosis, the resulting hepatic fibrosis leads to an increase in portal pressure, which causes

damage to the digestive mucosa through hypoperfusion and hypooxygenation, creating conditions conducive to a breakdown in the balance between protective factors and factors damaging the gastric mucosa, which is conducive to the occurrence of PUDs [7]. Like Lu *et al.*, we observed a higher average age in the group with UD, confirming advanced age as a risk factor for the occurrence of PUD [14].

In terms of diagnosis, the frequency of abdominal pain was 8.57% in cirrhotic patients with PUD, with no statistical difference compared to cirrhotic patients without UGD. Siringo *et al.* revealed this trend, with 77.5% of cirrhotic patients with PUD in Italy being asymptomatic [15]. The ulcer was located in the stomach in 51.47% of cases and in the duodenum in 40%. This gastric predominance, which appeared to be specific to Gabon compared with African data, was also observed by Ky *et al.* in Burkina Faso [12] [16]-[19]. Screening for *H. pylori* infection was not systematic, as observed by Ky *et al.* and Lawson-Ananissoh *et al.*, probably due to the clinical context and blood disorders [12] [18]. It was present in 25% of patients with PUD and 31% in the other group, far from the 79.3% *H. pylori* infection rate in a series of patients with PUD in Libreville, but already quite close to the 37.5% rate in an endoscopic series in Libreville [17] [20]. This low prevalence of *H. pylori* infection in cirrhotic patients is probably multifactorial. Indeed, the detection technique, where immunohistochemistry is rarely available in our context, underestimates the frequency of this infection [8] [20]. Furthermore, concerns about bleeding caused by biopsies limit their number [8] [9] [12]. There is also the fact that these patients are on multiple medications, including antibiotics, which can influence the results of *H. pylori* testing [8] [9] [20]. At the same time, the literature suggests that *H. pylori* infection may play a role in the pathogenesis of PU in liver cirrhotic patients [21]. Among other aetiological factors, stress was the one that showed a statistical link. In another series of patients in Libreville, stress was also identified as a risk factor associated with PUD, particularly occupational stress [17]. The clinical presentation of decompensated cirrhosis was dominated by ascites, as reported in the literature [8] [9] [12] [22]-[24]. Its aetiologies remained dominated by alcohol and chronic viral hepatitis B and C, as in the African literature [12] [22] [23].

In terms of prognosis, PUD was classified as Forrest IIC in 3 cases and Forrest IIB in 8 cases. These data are similar to those of Ky *et al.*, who observed 3 PUDs classified as Forrest IB, 2 PUDs classified as Forrest IIB, and 1 PUD classified as Forrest IIC [12]. The majority of cirrhotic patients with peptic ulcer disease had a Forrest III prognostic score. Indeed, the use of non-prescription drugs such as NSAIDs and PPIs, combined with delays in seeking medical advice due to the cost of tests, could explain these results. In addition, 31.43% of patients with PUD had Child-Pugh C cirrhosis, compared with 18.92% in the group without PUD. Although not statistically significant, this difference supports the observations of Shah *et al.*, who found that the severity of cirrhosis increases the risk of PUD [24]. Hemorrhagic shock was present in 40% of patients with PUD and 29.73% of patients without PUD, reflecting the increased severity of gastrointestinal bleeding

in cirrhotic patients with PUD, as suggested by Shah *et al.* [24]. Moreover, hemorrhagic shock was the leading cause of death in the group with PUD, whereas primary liver cancer was the leading cause of death in the group without PUD. These are the two leading causes of death from cirrhosis in Africa [11] [18] [21] [23]. Furthermore, for high-risk UGDs, PPI therapy should be continued for 72 hours, and the risk of re-bleeding should initially be assessed by repeat endoscopy. In our context, care is supported by the family despite the existing insurance system. As a result, the speed of management may compromise the prognosis [25].

These data, which are limited to one department and collected retrospectively, cannot be extrapolated to the whole country. However, SHGEED is the only hepatology and gastroenterology department in the country where five of the nine hepatologists and gastroenterologists in the public sector work. These results therefore provide a reliable profile of the activity of hepatologists and gastroenterologists treating cirrhosis. In addition, there may be a possible selection bias from excluding non-endoscoped cirrhotics. Furthermore, a case-control study would allow for a more precise determination of the association between the severity of cirrhosis and peptic ulcer disease. A larger sample size could provide more information on the frequency and endoscopic prognosis of peptic ulcer disease and the frequency of *Helicobacter pylori* in cirrhosis in Gabon.

5. Conclusion

PUD and cirrhosis are frequently associated conditions that affect older men. The role of chronic *H. pylori* infection remains to be clarified by a prospective multicentre study that will systematically investigate its presence using immunological testing of the stools of cirrhotic patients. Hemorrhagic shock remains the most common complication, associated with a poor prognosis; hence, the importance of developing an interventional endoscopy centre.

Conflicts of Interest

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

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