

# Management of Endoscopic Portal Hypertension Lesions in Cirrhotic Patients in a Country with Limited Resources: About 603 Cases in the City of Douala in Cameroon

Winnie Tatiana Bekolo Nga<sup>1,2\*</sup>, Aghoani Gilles<sup>3</sup>, Machékam-Matanga Olga<sup>2</sup>, Antonin Ndjitoyap<sup>4</sup>, Agnès Malongue<sup>1</sup>, Mathurin Kowo<sup>4</sup>, Dominique Noah Noah<sup>2</sup>, Oudou Njoya<sup>4</sup>, Firmin Ankouane Andoulo<sup>4</sup>, Luma Henry Namme<sup>1,4</sup>, Servais Albert Fiacre Eloumou Bagnaka<sup>2</sup>

<sup>1</sup>Department of Internal Medicine, the Douala General Hospital, Douala, Cameroon

<sup>2</sup>Faculty of Medicine and Pharmaceutical Sciences, University of Douala, Douala, Cameroon

<sup>3</sup>Faculty of Medicine, University of Buea, Buea, Cameroon

<sup>4</sup>Faculty of Medicine and Biomedical Sciences, University of Yaoundé I, Yaoundé, Cameroon

Email: \*winbek@yahoo.fr

**How to cite this paper:** Nga, W.T.B., Gilles, A., Olga, M.-M., Ndjitoyap, A., Malongue, A., Kowo, M., Noah, D.N., Njoya, O., Andoulo, F.A., Namme, L.H. and Bagnaka, S.A.F.E. (2023) Management of Endoscopic Portal Hypertension Lesions in Cirrhotic Patients in a Country with Limited Resources: About 603 Cases in the City of Douala in Cameroon. *Open Journal of Gastroenterology*, 13, 429-438.

<https://doi.org/10.4236/ojgas.2023.1312041>

**Received:** October 6, 2023

**Accepted:** December 12, 2023

**Published:** December 15, 2023

Copyright © 2023 by author(s) and Scientific Research Publishing Inc.

This work is licensed under the Creative

Commons Attribution International

License (CC BY 4.0).

<http://creativecommons.org/licenses/by/4.0/>



Open Access

## Abstract

**Introduction:** Portal hypertension (HTP) is a morbi-mortality factor in cirrhotic patients. It is responsible for endoscopic lesions and has digestive hemorrhage as the main complication. The objective of the study was to study the management of endoscopic lesions of portal hypertension in a country with limited resources. **Methodology:** This was a cross-sectional and analytical study conducted in 04 hospitals in the city of Douala, Cameroon, over a period of 08 years from 1 January 2014 to 31 December 2022. Included were cirrhotic patients with viral hepatitis with endoscopic lesions of PH. The data collected were sociodemographic, clinical, paraclinical, therapeutic and evolutionary. Data analysis was done using SPSS software version 25.0. Logistic regression allowed the search for prognostic factors with a significance threshold of  $p < 0.005$ . **Results:** We included 603 patient records. They were mainly male patients (56.1%) with an average age of  $47.6 \pm 6.3$  years. The Child Pugh score was ranked B in 53.7% of cases. Digestive hemorrhage was the main complication in 66.8% of cases. We had grade 2 esophageal varices in 61.5% of cases. The main treatments were prescription of propranolol (63.3%) and ligation of esophageal varices (53.3%). The average number of ligation sessions was  $2.1 \pm 1.8$  with an interval between sessions of  $28 \pm 2.8$  days. Prevention of rupture of esophageal varices was secondary in 66.5% of cases ( $n = 452$ ). The rate of hemorrhagic recurrence was 9.3%. Hospital mortality was 15.1%. Recurrence of hemorrhage was associated with  $PT < 45\%$

(OR = 1.04; 95% CI [1.01 - 1.06];  $p = 0.003$ ) and platelet levels  $< 150,000/\text{mm}^3$  (OR = 1.05; CI 95%: [1 - 1.09],  $p = 0.029$ ). Mortality was associated with a child pugh C score (OR = 9.73; 95% CI 95% [3.9 - 23.9];  $p < 0.005$ ) and hemorrhagic recurrence (OR = 2.02; 95% CI 95% [0.9 - 4.3];  $p < 0.005$ ). **Conclusion:** The management of HTP lesions was based on the prescription of beta-blockers and the ligation of esophageal varices. Factors associated with mortality were hemorrhagic recurrence, low PT and Child Pugh C score.

## Keywords

Portal Hypertension, Cirrhosis, Endoscopic Lesions, PEC, Cameroon

---

## 1. Introduction

Portal hypertension (PH) is defined as an increase in portal pressure beyond 15 mmHg, or an elevation of the port-cave pressure gradient beyond 5 mmHg [1] [2]. It is secondary to an obstacle to port-hepatic circulation [1] [3]. This obstacle occurs mainly in the liver, in the portal vein, or more rarely on the terminal part of the inferior vena cava [1]. The most common causes of portal hypertension are cirrhosis and bilharzia [4]. It causes vascular changes such as dilation of the portal vein, the formation of collateral (esophageal varices, gastric, splenic) [5]. With portal hypertension, oesophageal and gastric varices are the main endoscopic lesions [3].

The prevalence of endoscopic lesions of portal hypertension varies between 30% and 70% and one third of cirrhotic patients present at the time of diagnosis. In Ivory Coast, Outtara *et al.* in 2018, had a prevalence of 80% in cirrhotic patients [6]. In Senegal Bassène *et al.* had recovered respective prevalence of esophageal varices (VO) grade 2 and 3 of 24.3% and 75.7% [7]. In Cameroon, Tapouh *et al.* had a prevalence of 92% VO in cirrhotic patients [8]. The predictive factors of endoscopic PH lesions in cirrhotic patients are first the platelet rate  $150.109/\text{mm}^3$ , the value of elastometry 20 kPa [9]; but also the rate of prothrombin, spleen size, albumin [8] [10].

The discovery of endoscopic lesions of PH is most often done in the context of digestive hemorrhage or as part of the monitoring of cirrhosis [11]. In Cameroon, Ankouane *et al.* in 2013, had 28.9% of high digestive hemorrhages that were related to PH lesions in the city of Yaoundé [12]; and in Gabon, Itoudi *et al.* in 2019 had a prevalence of 29.5% [13]. The management is based on the Baveno consensus conference with updated in 2021, Baveno VII [9]. This consensus codifies the diagnosis and treatment of PH lesions. The diagnosis of portal hypertension in cirrhotic patients is based on a pressure gradient  $> 5$  mmHg and/or the presence of clinical signs of portal hypertension. It is considered clinically significant if the 10 mmHg gradient [9]. There are 3 levels of management (primary, secondary and tertiary) for digestive hemorrhage by rupture of esophageal varicose veins. The means of management are both medical (non-car- dis-

elective beta-blockers) and endoscopic (esophageal varicose vein ligation, TIPS) [3] [9].

Hemorrhage related to rupture of esophageal varicose veins is a significant morbi-mortality factor in cirrhotic patients. Early treatment and prevention of hemorrhagic risk contribute to lower mortality in these patients. The aim of the study was to assess the management of PH lesions in a resource-limited country like Cameroon based on Baveno's recommendations; and to investigate factors associated with patient prognosis.

## 2. Patients and Methods

### Type of study

We conducted a cross-sectional and analytical study over a period of 08 years from January 1, 2014, to December 31, 2022. It had for framework 04 sanitary structures of the city of Douala of which 02 were public (General Hospital and Hospital Laquintinie) and 02 private (Center of the diseases of the digestive tract and the Polyclinic Marie O). These structures have a digestive endoscopy unit with a technical platform allowing the realization of diagnostic and therapeutic endoscopies.

### Study population and sampling

The population of cirrhotic patient studies followed in the various health structures. Diagnosis of cirrhosis was by a gastroenterologist through liver biopsy, non-invasive methods (impulse elastometrics, fibrotest/Actitest, Fib 4, NALFD fibrosis score). We included patients with endoscopic lesions secondary to portal hypertension (esophageal varicose veins, esogastric varices, gastric varices and portal hypertension gastropathy). We excluded patients whose records were incomplete or not found. Our sampling technique was non-probability based on the exhaustive recruitment of patients meeting our inclusion criteria.

### Data collection

Patients were selected using consultation and hospitalization records. Included patient records were used for data collection. The latter was done using a pre-established form respecting the anonymity and confidentiality of each patient. The data collected were sociodemographic (age and sex), history and comorbidities (hypertension, diabetes, personal history), the etiology of cirrhosis, clinical signs, results of biological analyses, indications and results of esophageal endoscopy, treatment, and course (death, hemorrhagic recurrence).

We assessed the Child-pugh score for each patient [14]. We also assessed Glasgow-Blatchford and Rockall scores in patients with digestive hemorrhage [15].

### Ethical considerations

We obtained a research authorization from the ethics committee and the various health structures (N° 3802 CEI-UDo/06/2023/T). The recommendations of the 2013 Helsinki Declaration were scrupulously respected through the design of a research protocol, the submission of the latter to the relevant institutional eth-

ics committee for evaluation and respect for the confidentiality of personal information concerning the persons involved in the research.

### Statistical analysis

The data was collected on a data sheet and then saved under the interface of the Microsoft Excel 2010 software and the SPSS software version 25.0 was used for statistical analyses. The quantitative variables were expressed in average and standard deviation and the qualitative variables in number and percentage. Logistic regression by uni- and then multivariate analyses looked for the associated factors. The odd ratio was calculated with a 95% confidence interval and a significance threshold for a  $p < 0.05$ .

## 3. Results

We collected 807 records of cirrhotic patients and included 603 with endoscopic lesions of portal hypertension, a prevalence of 74.7%. These were mainly male patients (56.1%) with an average age of  $47.6 \pm 6.3$  years. The etiologies of cirrhosis were mainly viral hepatitis B (20.1%) and C (17.7%); and alcohol (14.2%) (**Table 1**). The Child Pugh score was ranked A, B, C respectively in 1.3% and 53.7% and 45% of cases. The circumstances of discovery of the lesions were an externalized digestive hemorrhage in 66.8% of cases. Hematemesis occurred in 51.6% of cases ( $n = 208/403$ ). The Glasgow-Blatchford score was greater than 1 in all patients with digestive hemorrhage and the Rockall score was greater than 8 in 22.6% ( $n = 91/403$ ). The main clinical signs were ascites (96.5%), jaundice (85.4%), hepatic encephalopathy (59.9%) and collateral venous circulation (57.2%) (**Table 1**). Biologically, the mean hemoglobin level was  $9.1 \pm 2.6$  g/dl, and the mean platelet level was  $(149.5 \pm 40.8) \times 10^6/\text{mm}^3$  (**Table 1**). The average prothrombin rate was  $48.9\% \pm 13.1\%$  (**Table 1**). The average transaminase rate was  $30.6 \pm 7.7$  IU/L for AST (aspartate aminotransferase) and  $32.3 \pm 11.7$  IU/L for ALT (alanin aminotransferase); and the average bilirubin rate was  $36.5 \pm 9$  mg/L (**Table 1**). the mean serum albumin level was  $29.8 \pm 10$  g/l (**Table 1**). In endoscopy, we had grade II and III esophageal varices respectively in 61.5% and 20.2%; red signs in 35% of cases and gastric varices in 31.8% of cases (**Table 2**). The medical treatment was based on the prescription of propranolol (63.3%) and those of antibiotics for the prevention of ascites infection (57.9%) (**Table 3**). The main endoscopic treatment was esophageal varices ligation (53.7%) (**Table 3**). The average number of ligation sessions was  $2.1 \pm 1.8$  with an interval between sessions of  $28 \pm 2.8$  days (**Table 3**). The average number of elastics installed was  $4.1 \pm 1.9$ . The prevention of rupture of esophageal varices was secondary in 66.5% of cases and primary in 33.5% (**Table 3**). The rate of hemorrhagic recurrence was 9.3%. Hospital mortality was 15.1%. Recurrence of hemorrhage was associated with PT  $< 45\%$  (OR = 1.04; 95% CI 95% [1.01 - 1.06];  $p = 0.003$ ) and platelet levels  $< 150,000/\text{mm}^3$  (OR = 1.05; IC95%: [1 - 1.09],  $p = 0.029$ ). Mortality was associated with a Child Pugh C score (OR = 9.73; 95% CI 95% [3.9 - 23.9];  $p = 0.002$ ) and hemorrhagic recurrence (OR = 2.02; 95% CI 95% [0.9 - 4.3];  $p =$

0.005), PT < 45% (OR = 1.53, [CI 95% (0.2 1- 8.8)], p = 0.001) (**Table 4**).

#### 4. Discussion

Three quarters of cirrhotic patients have portal hypertension lesions. This

**Table 1.** Characteristics of population study.

Variables	Frequency (%)	Mean ( $\pm$ SD)
Mean age (years)		47.6 $\pm$ 6.3
<b>Sex</b>		
Male	338 (56.1)	
Female	265 (43.9)	
<b>Etiologies of cirrhosis</b>		
Hepatitis B	121 (20.1)	
Hepatitis C	107 (17.7)	
Alcohol	86 (14.2)	
Alcohol/hepatitis B or C	83 (13.8)	
Co-infection B/C	61 (10.1)	
Auto-immune	39 (6.4)	
Autres	44 (7.2)	
Unknown	62 (10.2)	
<b>Child pugh score</b>		
A	8 (1.3)	
B	324 (53.7)	
C	272 (45)	
<b>Clinical presentations</b>		
Hemorrhage	403 (66.8)	
Ascitis	582 (96.5)	
Jaundice	515 (85.4)	
Hepatic encephalopathy	361 (59.9)	
Collateral veinous circulation	345 (57.2)	
<b>Biological paramters</b>		
Hemoglobin (g/dl)		9.1 $\pm$ 2.6
Platelets ( $10^6/mm^3$ )		149.5 $\pm$ 40.8
Prothrombin time (%)		48.9 $\pm$ 13.1
ASAT (UI/l)		30.6 $\pm$ 7.7
ALAT (UI/l)		32.3 $\pm$ 11.7
Bilirubin (mg/l)		36.5 $\pm$ 9
Albumin serum level (g/l)		29.8 $\pm$ 10

**Table 2.** Hypertension portal endoscopic lesions.

Variables	Frequency (%)
EV grade I	109 (18.3)
EV grade II	371 (61.5)
EV grade III	122 (20.2)
Gastric varices (GOV, IGV)	192 (31.8)
Red spots	211 (35)
Portal hypertension gastropathy	91 (15.2)

**Table 3.** Management of hypertension portal lesions according BavenoVII recommendations.

	Frequency	Mean ( $\pm$ SD)
<b>Type of prevention</b>		
Primary	202 (33.5)	
Secondary	401 (66.5)	
<b>Varices ligations</b>		
Average of seances	324 (53.7)	2.1 $\pm$ 1.8
Average number of elastics		4.1 $\pm$ 1.9
Delay between seances (days)		28 $\pm$ 2.8
<b>Sclerotherapy</b>	80 (13.3)	
<b>Medical treatment</b>		
Propanolol	382 (63.3)	
Carvedilol	6 (1)	
Antibiotics	349 (57.9)	
Terlipressin	171 (28.3)	

**Table 4.** Factors associated to hemorrhagic Recurrence and death of patients.

Prognosis factors	OR adjusted (CI 95%)	p-Value
<b>Death</b>		
PT < 45	1.53 (0.21 - 8.8)	0.001
Child-pugh C	9.73 (3.9 - 23.9)	0.002
Hemorrhage recurrence	2.02 (0.9 - 4.3)	0.005
GOV	0.1 (0.01 - 0.99)	0.05
Bilirubine > 15 mg/l	0.97 (0.94 - 1.01)	0.17
<b>Hemorrhage recurrence</b>		
PT < 45	1.04 (1.01 - 1.06)	0.003
Platelet < $150 \times 10^6/\text{mm}^3$	1.05 (1 - 1.09)	0.029
VO grad 2	0.79 (0.004 - 22.58)	0.935

proportion is much higher than that of Waqqas *et al.* in Pakistan whose study focused on patients who experiences a first episode of ascitis [16]. Most patients had decompensated cirrhosis as shown by child pugh scores, which are mostly classified B and C. This is similar to that of Sehounou *et al.* in Benin or Bouglouga *et al.* in Togo [17] [18]. Lesions of portal hypertension were discovered in a hemorrhagic context in most cases. It is most often the mode of discovery of cirrhosis because most patients in our context do not know they are carriers of cirrhosis. The clinical signs and biological results found reflect decompensation but also complications of cirrhosis. They are like those described in the African series [19] [20] [21].

The proportion of esophageal varices is comparable to that of Bagny *et al.* in Togo [22] higher than that of Ouattara *et al.* or Okon al in Ivory Coast [6] [21]. Hemorrhage was managed according to Baveno's recommendations [9], although it was not effective in all patients. All patients as part of their follow-up do not always have access to specialized centers with an adequate technical platform. Most patients admitted in a hemorrhagic context are lost to sight. Esophageal varices ligation may have been in just over half of patients. The high cost of a ligation kit is an obstacle to the realization of this medical procedure that is not within the reach of all budgets in limited resources country. All patients were able to have an average of 02 ligation sessions, as was in Senegal by Bassène *et al.* [7]. Due to financial limitations, many patients abandon their treatment, making monitoring and controlling the eradication of varices not always effective. Medical treatment with beta-blockers is often preferred in our context because less expensive than endoscopic treatment. Prevention was generally primary and secondary. Secondary prevention was followed by the inaugural hemorrhagic episode because it was often the mode of discovery of cirrhosis and/or portal hypertension lesions. Only a third of patients had primary prevention, which corresponds to the proportion of patients seen without hemorrhagic context. No patients received tertiary prevention. The implementation of a TIPS basis for tertiary prevention [9], necessitates the presence of sanitary structures equipped. The shortcomings of the technical platform do not allow by the realization of certain treatments.

Mortality remains very high, but it is lower than that found by Itoudi *et al.* in Gabon [19], which had a prevalence of 19.6%. Hemorrhagic recurrence was observed in one in ten patients. Factors associated with mortality were Child Pugh C score, low prothrombin time and hemorrhagic recurrence. Decompensation of cirrhosis and the occurrence of digestive hemorrhage are prognostic factors often found in the literature [22]. Hemorrhagic recurrence was associated with thrombocytopenia and low prothrombin time. Low prothrombin time was also identified by Camengo and the Central African Republic as a factor in hemorrhagic recurrence [23].

The limit of the study is its retrospective nature which does not allow to have indications on the observance of medical treatment by beta-blockers, on the era-

dication of esophageal varices after varices ligation.

## 5. Conclusion

Three quarters of cirrhotic patients had endoscopic lesions of portal hypertension. The circumstances of discovery of the lesions were mainly digestive hemorrhage. Despite the limits of the technical platform the recommendations of Baveno are respected. This is mainly a secondary prevention put in place generally after the bleeding episode. The management was based on the prescription of beta-blockers and the ligation of esophageal varices. The outcome of patients is influenced by the severity of cirrhosis and its complications.

## Authors' Contributions

All authors contributed to the development, writing, and editing of the article.

## Conflicts of Interest

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

## References

- [1] Maeva, G., Jean-Paul, C., Carine, C.D., Aurélie, P. and Nicolas, C. (2015) Hypertension Portale: Physiopathologie, Causes, Diagnostic et Traitement. *Hépatogastro- & Oncologie Digestive*, **22**, 40-56.
- [2] Chagneau-Derrode, C. and Silvain, C. (2005) Physiopathologie de l'hypertension portale: Mise à jour. *EMC—Hépatogastro-entérologie*, **2**, 264-268. <https://doi.org/10.1016/j.emchg.2005.01.010>
- [3] Gunarathne, L.S., Rajapaksha, H., Shackel, N., Angus, P.W. and Herath, C.B. (2020) Cirrhotic Portal Hypertension: From Pathophysiology to Novel Therapeutics. *World Journal of Gastroenterology*, **26**, 6111-6140. <https://doi.org/10.3748/wjg.v26.i40.6111>
- [4] Khanna, R. and Sarin, S.K. (2014) Non-Cirrhotic Portal Hypertension—Diagnosis and Management. *Journal of Hepatology*, **60**, 421-441. <https://pubmed.ncbi.nlm.nih.gov/23978714/> <https://doi.org/10.1016/j.jhep.2013.08.013>
- [5] Gulamhusein, A.F. and Kamath, P.S. (2017) The Epidemiology and Pathogenesis of Gastrointestinal Varices. *Techniques in Gastrointestinal Endoscopy*, **19**, 62-68. <https://doi.org/10.1016/j.tgie.2017.03.005>
- [6] Ouattara, A., Coffi, D.F., Assi, C., Soro, D., Allah-Kouadio, E. and Kouacou, L. (2018) Lésions endoscopiques hautes chez le cirrhotique au Centre Hospitalier et Universitaire de Cocody [Upper Digestive Endoscopy Lesions among Cirrhotics in the University Teaching Hospital of Cocody in Cote d'Ivoire]. *Revue Internationale des Sciences Médicales*, **20**, 297-300.
- [7] Bassene, M.L., Diouf, M.L., Dia, D., Mbengue, M., Halim, A., Diallo, S., *et al.* (2012) La ligature élastique des varices œsophagiennes à Dakar (Sénégal). *Médecine et Santé Tropicales*, **22**, 166-169. <https://doi.org/10.1684/mst.2012.0046>
- [8] Tapouh, J.M., Njoya, O., Zoé, C.M., Moifo, B., Kowo, M. and Amvene, S.N. (2015) Approche non Endoscopique du Diagnostic des Varices Œsophagiennes d'Origine



- Cirrhotique dans une Population d'Afrique Noire Subsaharienne. *Health Sciences and Diseases*, **16**.
- [9] De Franchis, R., Bosch, J., Garcia-Tsao, G., Reiberger, T., Ripoll, C., Abraldes, J.G., *et al.* (2022) Baveno VII—Renewing Consensus in Portal Hypertension. *Journal of Hepatology*, **76**, 959-974.
- [10] Doffou, S.A., Assi, C., Hamidine, I., Bangoura, D., Kouamé, D., Yaogo, A., *et al.* (2022) Valeur prédictive négative du ratio taux de plaquettes sur diamètre de la rate pour exclure la présence de varices œsophagiennes chez le cirrhotique d'origine virale B. *Revue de Médecine et de Pharmacie*, **11**, 1208-1212.
- [11] LaBrecque, D., Khan, A.G., Sarin, S.K., Le Mair, A.W., Gonvers, J.J., Dite, P., *et al.* (2014) Varices Oesophagiennes. World Gastroenterology Practice Guidelines.
- [12] Andoulo, F.A., Nonga, B.N., Noah, D.N., Kowo, M., Babagna, I.D., Talla, P., *et al.* (2013) Aetiology and Risk Factors of Acute Upper Gastrointestinal Hemorrhage: Analysis of 613 Cases in Yaounde, Cameroun. *Port Harcourt Medical Journal*, **7**, 175-182.
- [13] Bignoumba, P.E.I., Moussavou, I.F.M. and Kombila, J.B.M. (2019) Hémorragie Digestive Haute au Centre Hospitalier Universitaire de Libreville : Aspects Cliniques et Prise en Charge Réelle : À Propos de 210 Patients. *Health Sciences and Diseases*, **20**, 20-22.
- [14] Durand, F. and Valla, D. (2005) Assessment of the Prognosis of Cirrhosis: Child—Pugh versus MELD. *Journal of Hepatology*, **42**, S100-S107.  
<https://doi.org/10.1016/j.jhep.2004.11.015>
- [15] Badel, S., Dorta, G. and Carron, P.N. (2011) Hémorragie digestive haute : Utilité des scores pronostiques. *Revue Médicale Suisse*, **305**, 1574-1578.
- [16] Shabbir, W., Namooos, K., Aslam, M. and Hameed, M.A. (2023) Frequency of Esophageal Varices in Cirrhotic Patients Presenting with New Onset Ascites. *Rawal Medical Journal*, **48**, 26-29.
- [17] Sehonou, J., Cossou Gbeto, C., Dodo, L.S.R., Wollo, B.A., Agbodande, K.A., Azon-Kouanou, A., *et al.* (2019) Cirrhose hépatique dans le service de médecine interne du CNHU de Cotonou (2011-2014) : Aspects épidémiologiques, cliniques et évolutifs. *Journal de la Société de Biologie Clinique du Bénin*, **27**, 36-41.
- [18] Bouglouga, O., Bany, A., Djibril, M.A., Lawson-Ananissoh, L.M., Kaaga, L., Redah, D., *et al.* (2012) Aspects épidémiologiques, diagnostiques et évolutifs de la cirrhose hépatique dans le service d'hépatogastroentérologie du CHU Campus de Lomé. *Journal de la recherche scientifique de l'Université de Lomé*, **14**, 1-7.
- [19] Bignoumba, P.E.I., Nzouto, P., Alilangori, T., Moussavou, I.F.M., Saibou, M., Nguema, A.G.E., *et al.* (2020) Cirrhose Décompensée : Aspects Épidémiologiques, Pronostiques et Évolutifs à Propos de 167 Patients. *Health Sciences and Diseases*, **21**, 60-62.
- [20] Bagny, A., Bouglouga, O., Djibril, M.A., M'ba, F., Lawson, A.L., Redah, D., *et al.* (2013) Lésions endoscopiques hautes chez le cirrhotique au CHU-campus de Lomé (Togo) [Upper Endoscopic Lesions in Cirrhotic Patients at the University Hospital of Lomé (Togo)]. *Annales Africaines de Médecine*, **6**, page.
- [21] Okon, A.J.B., Diakité, M., Aké, F., Koffi, K.O.C., Koné, A. and Koulibaly, Y. (2020) Lésions endoscopiques chez les cirrhotiques noirs africains. *Revue de Médecine et de Pharmacie*, **10**, 1047-1057.
- [22] Noah Noah, D., Eloumou Bagnak, S.A.F., Andoulo, F.A., Bilounga, J.N. and Namme, H.L. (2016) Complications and Prognosis of Cirrhotic Patients at the Dou-

ala General Hospital in Cameroon. *Journal of Applied Medical Sciences*, **5**, 43-52.

- [23] Camengo Police, S.M., Diemer, H., Koffi, B., Boua-Akélélo, N.P., Mbeko Simaleko, M.M., Longo, J., *et al.* (2016) Facteurs de risque d'hémorragies digestives par rupture de varices œsophagiennes en République Centrafricaine. *Acta Endoscopica*, **46**, 384-388. <https://doi.org/10.1007/s10190-016-0530-9>

### **Abbreviations**

**ALAT:** Alanin amino-transferase  
**ASAT:** Aspartate amino-transferase  
**EV:** Esophageal varices  
**GOV:** Gastro-oesophageal varices  
**IGV:** INTRA gastric varices  
**PH:** Portal Hypertension  
**PT:** Prothrombin time