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Correlation between Platelet Count and the Presence of Esophageal Varices on Upper Gastrointestinal Endoscopy in Cirrhotic Patients at the Souro Sanou University Hospital, Burkina Faso

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Abstract

Introduction: Esophageal varices (EV) are one of the consequences of portal hypertension during cirrhosis. Their rupture leads to upper gastrointestinal bleeding, which is dreaded by its high mortality. The diagnosis of these EVs is based on upper gastrointestinal endoscopy (UGIE), an invasive examination that is insufficiently accessible in our context. The objective of this work was to study the correlation between the platelet count and the presence of esophageal varices at upper gastrointestinal endoscopy in cirrhotic patients. **Methodology:** This was an analytical cross-sectional study, which took place from July 2019 to March 2022 in the hepato-gastroenterology (HGE) department of the Sourô Sanou University Hospital (CHU-SS). All patients aged 15 years or older, followed in the department for cirrhosis and in whom a blood count and upper digestive endoscopy were performed, were included. **Results:** 117 cirrhotic patients were included, with a mean age of 47.1 years +/− 13.6 years. The main causes were infections by the viruses of hepatitis B (61.45%) and C (20.51%) and by alcohol consumption (21.37%). Esophageal varices were found in 81 patients (69.8%). The platelet count appeared to be the only independent factor linked to the presence of EV (p = 0.005). Patients with platelet counts below 150,000/µl were 5.15 times more likely to have EV (OR = 5.15). The sensitivity, specificity, positive predictive value and negative predictive value of platelet counts below 150,000/µl were 77.7%, 74.3%, 87.5% and 59.1%,

respectively. **Conclusion:** Platelet count is a good predictor of EV in cirrhotic patients; and could, therefore, be an alternative that will help in the selection of patients for endoscopic screening of EV and thus allow early management of EV if necessary.

Keywords

Correlation, Platelet Count, Esophageal Varices, Upper Digestive Endoscopy, Cirrhosis, Burkina Faso

1. Introduction

Esophageal varices are one of the consequences of portal hypertension due to hepatic fibrosis during cirrhosis. They occupy a central place among the many complications associated with this fibrosing condition, due to their hemorrhagic potential which can be fatal [1] [2]. They are present at the time of diagnosis of cirrhosis in 50% of patients [3]. The prevalence of their rupture is approximately 30%. The resulting hemorrhage is the mode of revelation of cirrhosis in 10% of cases and is responsible for one third (1/3) of deaths in cirrhotic patients [4]. Mortality during the first episode of variceal bleeding is estimated at between 15 and 20%, but it is even higher in patients with Child-Pugh C cirrhosis, that is approximately one third of them [5] [6].

Primary prevention of bleeding risk is crucial for the survival of cirrhotic patients. It is based on their precise screening by upper gastrointestinal endoscopy [3], which, while costly, remains unpleasant for the patient when performed without anesthesia. The need for early, appropriate management has led to a series of conferences to place particular emphasis on non-invasive diagnostic methods. Thus, at the Baveno VI conference in 2015, the platelet count, accessible and commonly used [7]-[9] was proposed as a predictive indicator in the identification of patients at risk of esophageal varices. As a matter of fact, thrombocytopenia, often observed in cirrhotic patients, has been proposed as an indirect marker of portal hypertension, itself responsible for the formation of esophageal varices. According to the recommendations of the last Baveno VII conference in 2021 [9], clinically significant PH (HTP) is characterized by the presence of at least one of the following criteria: ascites including only radiological, collateral circulation, an elasticity measurement ≥ 25 kPa (excluding obesity), the presence of esophagogastric varices regardless of the grade and the Child-Pugh, or a PH (HTP) gradient ≥ 10mmHg. An elasticity measurement ≤ 15 kPa with a platelet count ≥ 150,000 mm3 permits to exclude clinically significant PH (HTP) [8]-[10]. In sub-Saharan Africa, digestive endoscopy remains financially and geographically inaccessible for the majority of populations and data are rare [11]. This study aims to study the correlation between the platelet count and the presence of EV detected during an upper digestive endoscopy in cirrhotic patients followed at the Sourô Sanou University Hospital in order

to have a simple and effective tool for screening esophageal varices in resource-limited contexts.

2. Patients and Methods

2.1. Study Design, Population and Sampling

This was an analytical cross-sectional study, which took place from July 27, 2019 to March 31, 2022 in the hepato-gastroenterology (HGE) department of the Sourô Sanou University Hospital (CHU-SS). The study included all patients aged at least 15 years, hospitalized in the hepato-gastroenterology (HGE) department during the period and who had given verbal consent:

1) in whom the diagnosis of liver cirrhosis was made on the basis of clinical and paraclinical criteria (ultrasound and/or pulse elastometry).

2) with a blood count of less than one month and having undergone an upper gastrointestinal endoscopy (UGIE).

We carried out exhaustive consecutive recruitment of patients meeting the inclusion criteria, using a collection form.

2.2. Exclusion Criteria

This study excluded patients with coexisting cancers (leukaemia, multiple myeloma…), pregnancy, heavy alcohol use, autoimmune diseases such as systemic lupus erythematosus, certain treatments such as corticosteroids, immunosuppressive treatment or immunoglobulins.

2.3. Variables and Data Collection Technique

The collected data concerned patients' socio-demographic characteristics (age, sex, occupation, place of residence); clinical examination data (functional and physical signs); biological data (platelet count, transaminases, bilirubinemia, gamma-GT, prothrombin level, albuminemia, alpha-fetoprotein, blood urea and creatininemia); UGIE results (esophageal or gastric varices, portal hypertension gastropathy); liver ultrasound data (hepatic dysmorphia, dilatation of the liver). Most biological tests were performed at the CHU-SS laboratory. All UGIE examinations were performed in private clinics, as the digestive endoscopy unit at CHU-SS has not been operational since September 2017; the same applies to the performance of pulse elastometry. Liver ultrasound scans are either carried out at the CHU-SS imaging department or in private clinics in town due to the often long appointments offered by the imaging department.

A data collection form was drawn up and validated for data recording. The data collection forms were filled in by medical students in their doctoral year.

2.4. Data Processing and Analysis

Data were entered into EPI Data version 3.1 and analyzed using Stata version 13 software. Qualitative variables were described by their proportion and quantitative variables by their mean and standard deviation. The Chi2 test was used to compare

the proportions of variables, and the Student's t-test was used to compare the means of quantitative variables. Variables that were associated with EV at the p ≤ 20% threshold in bivariate analysis using the Chi2 test were retained for multivariate logistic regression. We then determined the positive predictive values (PPV) and negative predictive values (NPV) of platelet count for the presence of EV. The significance level (p-value) was 5% for all analyses.

2.5. Operational Definitions

In the absence of an anatomopathological study, which remains the gold standard for the diagnosis of cirrhosis, the diagnosis of cirrhosis in this study was made on clinical, biological, elastometric, endoscopic and ultrasonographic grounds.

1. Clinical evidence: painless, firm hepatomegaly with smooth or granular anterior surface and sharp lower border; associated or not with signs of portal hypertension (collateral venous circulation, splenomegaly), with or without ascites and/or hepatocellular insufficiency (asthenia, jaundice, stellate angiomas, hepatic encephalopathy).

2. Biological evidence:

1) Signs of hepatocellular insufficiency: hypo albuminemia (< 35 g/l), a lowered prothrombin rate (PT< 70%), elevated total bilirubinemia > 20 µmmol/l.

2) Hematological signs in particular an anemia with hemoglobin < 10g/dl; and thrombocytopenia with platelet count ≤ 150000/µl.

3. Ultrasound evidence: normal-sized, atrophic or hypertrophic liver, with irregular contours and granular or nodular parenchyma, with or without signs of portal hypertension (increased portal vein diameter, presence of portocaval shunts, splenomegaly, ascites).

4. Elastometric evidence: a value greater than or equal to 15 kPa.

5. Endoscopic evidence: presence of esophageal and/or gastric varices and/or congestive gastropathy with a "mosaic" appearance of portal hypertension.

6. Positive predictive value (PPV) of platelet count: probability of EV when platelet count < 150,000/µl.

7. Negative predictive value (NPV) of platelet count: probability of no EV when platelet count >150000/µl.

2.6. Ethical Aspects

Prior verbal consent was obtained from patients wishing to participate in the study. For those who gave their consent, data collection was carried out while respecting patient anonymity and confidentiality. To ensure confidentiality during our study, only the medical record number was reported.

1. Results

During the study period, 1492 patients were hospitalized in the HGE department, of whom 117 (7.84%) were included in the study.

2.7. Socio-Demographic Characteristics

The mean age of the patients was 48.9 ± 13.8 years, with extremes of 19 and 82

years. The 46-55 age group was the most represented, with 35 patients (29.9%). Males predominated (74.4%), giving a sex ratio of 2.9. Occupationally, they were mainly farmers (53%) and housewives (15.4%). More than half the patients came from rural areas (59%). The socio-demographic characteristics of the patients are shown in Table 1.

Table 1. Socio-demographic characteristics of 117 cirrhotic patients.

Variables	Number	Percentages
Age groups		
[15 - 25]	6	5.1
[26 - 35]	15	12.8
[36 - 45]	24	20.5
[46 - 55]	35	29.9
[56 - 65]	25	21.4
[66 - 82]	12	10.3
Gender		
Men	87	74.4
Women	30	25.6
Occupations and activities		
Farmers	62	53.0
Housewives	18	15.4
Informal sector	16	13.7
Shopkeepers	11	9.4
Retired	6	5.1
Private sector	4	3.4
Place of residence		
Urban	69	59.0
Rural	44	37.6
Semi-urban	4	3.4

2.8. Distribution of Esophageal Varices (EV)

2.8.1. Esophageal Varices and Socio-Demographic Data

There was no statistically significant relationship between EVs and socio-demographic data (age, age groups, gender, place of residence).

Table 2 shows the distribution of cirrhotic patients with EVs according to socio-demographic data.

Table 2. Distribution of EVs according to socio-demographic data (N = 117).

Variables	EV present n (%)	EV absent n (%)	p-value
Age*	47.1 ± 13.63	52.57 ± 13.74	0.0502
Age groups			
15 - 25 ans	5 (6.17)	1 (2.94)	
26 - 35 ans	12 (14.81)	3 (8.82)	
36 - 45 ans	18 (22.22)	6 (17.65)	0.678
46 - 55 ans	25 (30.86)	10 (29.41)	
56 - 65 ans	15 (18.52)	9 (26.47)	
66 - 82 ans	6 (7.41)	5 (14.71)	
Gender			
Masculin	64 (74.42%)	22 (25.58)	0.104
Féminin	17 (56.67%)	13 (43.33)	
Place of residence			
Rural area	51 (75%)	17 (25%)	0.096
Urban area	26 (59.09%)	18 (49.91%)	

*expressed as mean ± standard deviation.

2.8.2. Esophageal Varices and Clinical Data

Among clinical signs, on bivariate analysis, upper gastrointestinal bleeding (p = 0.021) and hepatomegaly (p = 0.000) were statistically significantly associated with the presence of EVs. The distribution of EVs according to clinical data is given in **Table 3**.

Table 3. Distribution of EVs according to clinical data (N = 117).

Variables	EV present n (%)	EV absent n (%)	p-value
Upper gastrointestinal bleeding	26 (86.67)	4 (13.33)	0.021
Splenomegaly	14 (77.78)	4 (22.22)	0.576
Ascites	56 (71.79)	22 (28.21)	0.517
Enlarged abdomen	57 (69.51)	25 (30.49)	1.000
Jaundice	33 (66)	17 (34)	0.541
Abdominal pain	24 (34.29)	46 (65.71)	0.404
Edema of the lower limbs	37 (63.79)	21 (36.21)	0.226
Collateral venous circulation	12 (54.55)	10 (45.45)	86.67
Hepatomegaly	23 (53.06)	26 (46.94)	0.000

2.8.3. Esophageal Varices and Biological Data

On bivariate analysis, only platelet count had a statistically significant association with the presence of EV (p = 0.000). Among patients with platelet levels ≤ 150000/µl, 63 (87.5%) had esophageal varices and 9 (12.5%) did not. Among pa-

tients with platelet counts above 150000/µl, 18 (40.9%) had esophageal varices and 26 (59.1%) did not. The distribution of esophageal varices according to biological data is shown in Table 4.

Table 4. Distribution of EVs according to biological data (N = 117).

Variables	Norms	EV present		EV absent		p-value
		n	%	n	%	
ASAT	High rate	57	68.7	26	31.3	0.763
	Normal rate	09	64.3	05	35.7	
ALAT	High rate	40	66.7	20	33.3	0.824
	Normal rate	27	71.0	11	28.9	
GGT	High rate	42	67.7	20	32.3	1.000
	Normal rate	08	72.7	03	27.3	
Total bilirubin	High rate	33	68.7	15	31.2	0.805
	Normal rate	19	65.5	10	34.5	
Prothrombin level	Low rate	44	66.7	22	33.3	1.000
	Normal	15	65.2	08	34.9	
White blood cell count	High rate	22	61.1	14	38.9	0.193
	Normal rate	59	73.7	21	26.2	
Platelet count	Low	63	87.5	09	12.5	0.0001
	Normal	18	40.9	26	59.1	
Alpha-fetoprotein	High rate	30	81.1	07	18.9	0.132
	Normal rate	26	65.0	14	35.0	
Albuminemia	High rate	28	65.1	15	34.9	0.471
	Normal rate	08	80.0	02	20.0	
Blood urea	High rate	14	82.3	03	17.6	0.240
	Normal rate	39	63.9	22	36.1	
Blood creatinine	High rate	13	68.4	06	31.6	1.000
	Normal rate	60	69.8	26	30.2	

2.9. Measuring the Association between Platelet Count and Esophageal Varices

On multivariate analysis, only platelet count retained a statistically significant association (p = 0.005). Platelet count, therefore, emerged as the only independent factor associated with the presence of esophageal varices. The measure of the association between platelet count and the presence of EV is given in Table 5 below.

Table 5. Measure of the association between platelet count and the presence of EV.

Esophageal varices	Adjusted OR	IC (95%)	P value
Platelet count			

Continued

>150000	1	-	
≤150000	5.15	1.64 to 16.14	0.005
Hepatomegaly			
Yes	1	-	
No	2.04	0.63 to 6.85	0.223
HDH*			
Yes	1	-	
No	0.44	0.12 to 1.60	0.219

*Upper gastrointestinal bleeding.

2.10. Negative and Positive Predictive Values of Platelet Count for the Presence of Esophageal Varices

Thrombocytes had a good value for predicting the presence of EVs because 77.7% of patients with varices had a platelet count ≤ 150000/µl (Se = 77.7%) and among patients with a platelet count ≤ 150000/µl, 87.50% had EVs (PPV = 87.50%). The positive predictive value (PPV) of the platelet count indicates the probability that a thrombocytopenia indicates that the patient is really at risk of developing oesophageal varices. The negative predictive value (NPV) indicates the probability that thrombocytopenia indicates the absence of oesophageal varices in the patient. The PPV and NPV of thrombocytes to predict the presence of esophageal varices (EV+) or their absence (EV-) are represented in Table 6.

Table 6. Distribution of esophageal varices according to platelet count.

	EV present		EV absent		Se	Sp	PPV	NPV
Variables	n	%	n	%	%	%	%	%
Platelet ≤ 150000/µl	63	87.5	9	12.5	77.77	74.28	87.50	59.09
Platelet > 150000/µl	18	40.90	26	59.09	74.28	77.77	59.09	87.50

*Se (sensitivity), Sp (specificity).

3. Discussion

In our study, the mean age of patients in this series was 48.9 ± 13.8 years. The 46-55 age group was the most represented, with 35 patients (29.9%). In Western countries [12] [13], the mean age of cirrhotic patients reported in studies is higher (> 55 years) than that reported (45-50 years) in most studies carried out in Africa. Thus, we have 48 - 49.5 in the Maghreb [14] [15], 44 - 50 in Central Africa [16] or 48.5 in Sub-Saharan Africa [11] [17]-[19]. Younger African patients are more often infected with hepatitis B and C viruses at birth or in childhood, and are therefore at greater risk of developing chronic liver disease.

A predominance of males (74.4%) was observed, giving a sex ratio of 2.9. This male predominance is also reported in most African studies [11] [16] [18] [20]. Professionally, the patients were mainly farmers (53%) and housewives (15.4%). More than half the patients came from rural areas (59%). The rural origin of the majority of patients also explains the predominance of farmers and housewives, where the main activity is farming, with the support of women. In rural areas, men probably have more economic power to facilitate access to care. Men are also more exposed than women to certain risk factors, such as risky sexual behavior, alcohol and injecting drug use. Financial and geographical difficulties in accessing health care facilities and specialists could explain the delay in seeking care, and hence in diagnosis at the cirrhosis stage.

The prevalence of esophageal varices, PH (HTP) gastropathy and gastric varices was 69.8%, 37.9% and 0.88% respectively. EV and PH (HTP) gastropathy were found in 82.7% and 40% of patients who underwent endoscopy in Somé's study [19]. The selection criteria in their study differed from ours. In their study, all cirrhotics were included, whether or not they had undergone fibroscopy, whereas in ours, the absence of upper gastrointestinal endoscopy was an exclusion criterion. Bouhlel *et al.*, who did not separate esophageal varices from gastric varices, reported a higher rate of esophageal varices at 82.4% [15].

Concerning factors associated with the presence of EVs, age and gender had no influence on the development of esophageal varices. These results are in line with those reported by Doffou *et al.* [11]. The absence of a relationship between the duration of symptomatology and the presence of EVs may be linked to the fact that the aim of this study was to identify factors correlated with the presence of EVs and not with their severity. In bivariate analysis, upper upper gastrointestinal bleeding ($p = 0.021$), hepatomegaly ($p = 0.000$) and platelet count ($p = 0.000$) were found to be factors associated with the occurrence of EV. Upper gastrointestinal bleeding (UGIB) was present in 86.7% of patients with esophageal varices.

But UGIB was not an independent predictive factor of the presence of EV ($p = 0.219$ on multivariate analysis). A variceal rupture in HTP/PH may also be due to gastric varices and hypertensive gastropathy. There are also other causes of bleeding in cirrhotics, such as other acute lesions of the gastric or duodenal mucosa, peptic ulcer disease or Mallory-Weiss syndrome. In our study, 53.1% of patients with EV had hepatomegaly. Hepatomegaly was not an independent predictor of the presence of EV. The presentation of cirrhosis, whether atrophic or hypertrophic, had no influence on the occurrence of EV. Compensated cirrhosis being often an asymptomatic pathology, the lack of correlation between clinical and endoscopic signs of portal hypertension raises the problem of discovery at the stage of complications, and the need for effective means of early detection of esophageal varices. Biologically, although our results show a predominance of AST, there was no relationship between cytolysis and EV. Doffou *et al.* [11] had comparable results with ASAT ($p = 0.264$). In fact, although cytolysis is part of the diagnosis of cirrhosis, it is not related to its severity. Prothrombin levels were low in 66.7% of

patients with EV (p = 1.000) and hypo-albuminemia in 65.1% of patients with EV (p = 0.471). Rye *et al*. [21] also found no relationship between albumin and EV (p > 0.05). Prothrombin levels and hypo-albuminemia are signs of hepatocellular insufficiency, but are not synonymous with the presence of portal hypertension. Alpha-fetoprotein was high in 81.08% of patients having EV with a p = 0.132. Although alphafetoprotein is used in our context as a complementary factor to imaging for the diagnosis of hepatocellular carcinoma (HCC), it is not sufficient to assess the relationship between EVs and HCC. In multivariate analysis, platelet count was the only factor associated with the presence of EVs in cirrhosis (p = 0.005). Patients with a platelet count less than or equal to 150,000/µl were 5.15 times more likely to have EVs (OR = 5.15) than patients with a platelet count greater than 150,000/µl. In line with the literature, several studies have also found a statistical association between low platelet levels and the presence of esophageal varices: De Mattos *et al*. [22] in Brazil (p < 0.05); Rye *et al*. [21] in the UK (p = 0.015); Nada *et al*. (p = 0.0011) [23] and Zoukal *et al*. [8] in Morocco (p < 0.001); Doffou *et al*. [11] in Ivory Coast (p < 0.0001); Moulion Tapouh *et al*. [24] in Cameroon (p = 0.003).

Several studies have also reported on the reliability of this test to assess its applicability as an independent non-invasive factor for predicting esophageal varices. In our study, a platelet count less than or equal to 150,000/µl had a good value for predicting the presence of EV (PPV = 87.50%) and was a good factor detecting 74.28% of patients with esophageal varices (Se = 74.28%), or a low false-positive rate (25.72%). A platelet count above 150,000/µl was a good test to exclude the presence of EV (NPV = 87.50%) and identified 77.77% of patients without EV, thus establishing a good link with a low false-negative rate (22.23%). Platelet counts above 150,000/µl had an NPV of 100% and 95% respectively in the studies by Doffou and Assi al [11] in Côte d'Ivoire. In all these studies, as in our own, a platelet count above 150,000/µl was a good factor for ruling out the presence of EV and thus avoiding the need for upper gastrointestinal fibroscopy. Nevertheless, the predictive values were higher than those reported in our series. This difference may be explained by the fact that our study focused on a general population of cirrhotics with or without varicose veins and without distinction of grade, unlike those studies which focused solely on the detection of large esophageal varices.

In these studies, platelet counts above 150000/µl also had good sensitivity and specificity for the detection of EV: respectively, 100 and 52.5% for Doffou *et al*. [11], and 95.8 and 54.3% for Assi *et al*. [25]. Bouazizi [26] and Petta [27] report a threshold platelet count of 106,000 and 110,000 elements/mm^3 respectively as a predictive factor for the occurrence of gastrointestinal haemorrhage due to rupture of an EV, with a sensitivity of 68% and specificity of 55% in Bouaziz's study. Despite some differences, these results highlight the good value of platelet count as a non-invasive test for the diagnosis of esophageal varices.

Our study nevertheless has a number of limitations. The retrospective nature of

our recruitment exposes us to the risk of selection and information bias. The relatively modest size of our sample reduces the statistical power of our analyses, and makes our results difficult to generalize. In our context, patients who are often poor rarely have complete records, and the archiving of records in the departments is not yet optimal.

4. Conclusion

This study demonstrated a correlation between platelet count and the existence of EVs in cirrhotic patients, and established that it is a good predictor of the existence of EVs in cirrhotic patients. In countries with limited resources, characterized by the absence of a cost-sharing system, platelet count would be an interesting alternative for screening for esophageal varices, and could help physicians to initiate appropriate primary prophylaxis. The results obtained could pave the way for prospective African work on larger samples, with more patients who have not yet reached an advanced stage of chronic liver disease, in order to test their applicability in our routine healthcare offer.

Conflict of Interest

The authors declare that they have no conflict of interest.

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