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Charactistics of Anemia in Cirrhotics in the Gastroenterology Department at Brazzaville University Hospital

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Abstract

Objective: Study of the frequency of anemia, its characteristics and its relationship with different epidemiological, diagnostic and prognostic parameters of cirrhosis. **Patients and methods:** This was a retrospective descriptive study from January 2016 to December 2019 in the Gastroenterology and Internal Medicine department at the Brazzaville University Hospital. It concerned all cirrhotics hospitalized during the study period and presenting anemia. The epidemiological, diagnostic, and prognostic data studied were collected from medical records. The characteristics of anemia on the blood count were defined based on WHO values. The information collected was entered and processed with Excel 8.0 and EPI data 3.1 software. The Pearson chi2 test at the significance level ≤ 5% was used to compare the results. **Results:** We collected 102 files of cirrhotic patients during the study period. There were 63 men and 39 women, a sex ratio of 1.6. Among these patients, 77 presented anemia, *i.e.*, a frequency of 75.5% with a male predominance (57.2%). Anemia increased without a significant relationship with age and was more severe in young female subjects (42.8%). The normochromic normocytic character was the most observed (52%). Anemia was more frequent in post-hepatitic cirrhosis, representing 67.5% of cases of anemia. It worsened during digestive bleeding, with the abundance of ascites and the severity of hepatic encephalopathy ($p > 0.05$). It was significantly associated with the drop in prothrombin level (63.6%), hypoalbuminemia (64.9%), and the Child-Pugh score ($p < 0.05$).

Keywords

Anemia, Cirrhosis, CHU, Brazzaville

1. Introduction

Cirrhosis is a serious condition that represents the final common stage of most chronic liver diseases, regardless of their cause [1]. Due to its increasing incidence, it poses a significant public health problem globally, especially in developing countries [2]. In Congo, its prevalence is estimated at 8.7% according to hospital data [3]. The treatment of cirrhosis depends on the clinical stage and the onset of complications, which influence the disease's prognosis. Among these complications, the occurrence of anemia is very common and plays a major role in the worsening of cirrhosis. Hospital studies conducted in France and Burkina Faso reported anemia rates of 54% and 74.5%, respectively [4] [5]. The type of anemia depends on its origin, which is multifactorial. The most well-known causes of anemia in cirrhosis include gastrointestinal bleeding, hemolysis, and hemodilution; however, it may also have inflammatory origins related to the presence of cytokines, deficiencies, or other causes [6]-[8]. However, identifying these etiologies remains difficult, especially in developing countries where technical resources are limited. In Congo, a recent study showed that gastrointestinal bleeding complicated by anemia was the second leading cause of death among cirrhotic patients, with a rate of 27.3% [3]. Proper interpretation of the blood count could point toward a group of etiologies and help avoid systematic and excessive prescriptions of iron. In 2007, Nacoulma *et al.* in Burkina Faso reported that normocytic normochromic anemia, followed by microcytic hypochromic anemia, were the most observed characteristics in a study on different types of anemia in cirrhosis [5].

Regarding its severity, the onset of anemia could alter the clinical, biological, and morphological parameters induced by cirrhosis, as well as its prognosis. In his study, Dénié *et al.* suggested that anemia could intensify the hemodynamic changes already present and could worsen portal hypertension and hepatocellular insufficiency, especially in women [4]. These studies indicate that anemia, sometimes underestimated, can play a major role in the worsening of cirrhosis.

To contribute to improving the care of cirrhotic patients in Congo, we conducted this study with the general objective of examining the epidemiological, diagnostic, and prognostic characteristics of cirrhotic patients with anemia, and more specifically to:

- Determine the frequency of anemia in cirrhotic patients;
- Identify the different types of anemia observed in cirrhotic patients;
- Assess the prognostic stage of patients presenting with anemia.

2. Population and Method

We conducted a descriptive, retrospective study from January 2016 to December 2019, covering a three-year period. The study was carried out at the University

Hospital Center (CHU) of Brazzaville, in the Gastroenterology and Internal Medicine departments. The general study population included all patients hospitalized for cirrhosis, with diagnoses based on liver characteristics, signs of hepatocellular insufficiency, and portal hypertension. Patients with anemia constituted the target population of our study. We included in the study any patient aged 18 or older with a medical file containing a blood count. The patients were collected from hospitalization registers, then we completed data in medical records.

Patients were gathered from hospitalization records, and we then completed the data using medical files. Patients with a previously confirmed diagnosis as the cause of anemia were not included. Age and sex were the epidemiological variables studied. Diagnostic variables focused on clinical aspects (hepatic encephalopathy, gastrointestinal bleeding), morphological aspects (esophageal varices, ascites), etiological factors (HBV, HCV, alcohol, diabetes), and biological factors (prothrombin rate, albumin) of cirrhosis. Blood count parameters (Hb, MCV, MCHC) were used to determine the frequency and characteristics of anemia.

The collected data were entered and processed using Excel 8.0 and EPI Data 3.1 software. Variables were described using statistical tables and charts. Pearson's chi-squared test, with a significance level of ≤5%, was used to compare results.

Operational Definitions

Based on WHO data, we defined the characteristics of anemia using patients' blood counts [9]. Anemia was defined as any hemoglobin (Hb) level ≤ 11 g/dL, regardless of sex. Anemia was classified as mild when Hb was between 10 and 11 g/dL, moderate when Hb was between 8 and 9 g/dL, and severe when Hb was below 8 g/dL. Anemia was considered normocytic when the mean corpuscular volume (MCV) was between 80 and 100 fL, microcytic when MCV was below 80 fL, and macrocytic when MCV was above 100 fL. The mean corpuscular hemoglobin concentration (MCHC) was considered normal when it was between 27 and 31 pg.

3. Results

3.1. Characteristics of the General Population

We collected 102 patient records for cirrhosis in the study. Male patients were predominantly represented, accounting for 61.7% (sex ratio: 1.6). The average age was 54.7 ± 16.3 years, ranging from 17 to 86 years. **Table 1** shows the distribution of the population according to age and sex.

Hepatic encephalopathy was observed in 62.7% of patients, with grade I predominating (38.3%). Ascites was present in 82.4% of patients, mainly grade III (45.1%).

Esophageal varices (EV) were found in 52% of patients (n = 53). Grade III EVs accounted for 35.2% of cases, followed by grade II (14.8%) and grade I (2%). Gastrointestinal bleeding was observed in 15.7% of cases (n = 16).

Biologically, patients had low prothrombin levels (TP) in 76.4% (n = 63), hypoalbuminemia in 68.6% (n = 68), and hyperbilirubinemia in 46.1% of cases (n = 47).

The main causes of cirrhosis were HBV (36.3%), HCV (30.4%), alcohol

(21.5%), and diabetes (5.9%).

The majority of patients, 52% (n = 53), were in stage C of the Child-Pugh classification, followed by Child-Pugh B score in 45% of cases (n = 46). The frequency of anemia was 75.5% (n = 77).

3.2. Characteristics of Patients with Anemia

The average age of patients with anemia was 53.9 ± 17.5 years (range: 17 - 86 years) compared to 56.8 ± 12.3 years (range: 29 - 78 years) for patients without anemia. The frequency of anemia increased with age but was more severe among younger subjects, with no statistically significant difference. Male patients were relatively more affected (57.2%). However, anemia was more severe in female patients, with an average Hb of 7.9 ± 1.8 g/dL compared to 8.2 ± 1.8 g/dL in male patients, with no significant difference (Table 1).

Table 1. Distribution of patients with anemia according to sex and age.

Variables	N = 77				P value
	Settings	n	%	Hemoglobin (g/dl)	
Age (years)	≤20	2	2.6	7.3	0.408
	[20 - 40]	19	24.6	7.6	
	[40 - 60]	28	36.4	7.7	
	>60	28	36.4	8.8	
Gender	Male	44	57.2	8.2	0.147
	Female	33	42.8	7.9	

In most cases (52%), it was a moderate, normocytic, normochromic anemia (mean Hb: 8.1 g/dL; range: 3.8 - 10.9 g/dL; mean MCV: 80.4 fL; mean MCHC: 27.8 pg). Table 2 shows the distribution of patients by anemia characteristics.

Table 2. Distribution of patients according to the characteristics of anemia.

Variables	Anemia (N = 77)		
	Settings	n	%
Severiity anemia	Light	12	15,6
	Moderate	34	44.2
	Severe	31	40.2
Type of anemia	Macrocytic	3	4
	Normocytic normochromic	40	52
	Normocytic hypochromic	4	5
	Microcytic normochromic	6	8
	Microcytic hypochromic	24	31

Diagnostically, hemoglobin concentration decreased with the presence of gastrointestinal bleeding, the stage of hepatic encephalopathy, and the abundance

of ascites. Anemia was significantly associated with decreased TP and albumin levels. It did not vary according to the grade of EV. Table 3 presents these results.

Table 3. Distribution of patients with anemia according to certain signs of cirrhosis.

Variables	N = 77				P value
	Settings	n	%	Hemoglobin (g/dl)	
Digestive hemorrhage	Present	14	18.2	7.4	1.000
	Absent	63	818	82	
Ascites	Grade 1	2	2.6	8.5	1.000
	Grade 2	23	49.3	7.6	
	Grade 3	38	29.9	6.7	
Encéphalopaty Hépatic	Stage 1	7	9.1	8.3	0.451
	Stage 2	14	18.2	7.9	
	Stage 3	29	37.6	7.7	
Prothrombin level	Normal	28	36.4	8.5	0.001
	Low	39	63.6	7.8	
Albumin	Normal	27	35.1	8.2	0.001
	Low	50	64.9	7.7	
Esophageal varices	Grade I	2	2.6	7.1	0.094
	Grade II	9	11.7	8.1	
	Grade III	31	40.3	8.4	

Anemia was more frequent in post-hepatic cirrhosis due to HBV and HCV, with 67.5% (n = 52/77), and was more severe in patients with HBV, though without significant difference. Table 4 shows the distribution of patients with anemia by etiology.

Table 4. Distribution of patients with anemia according to the etiology of cirrhosis.

Variables	N = 77				P Value
	Setting	n	%	Hb (g/dl)	
HBV	Yes	27	35.1	7.5	0.985
	No	44	57.1	8.3	
HVC	Yes	21	27.3	8.2	0.655
	No	52	67.5	7.9	
Alcohol	Yes	18	23.4	8.8	0.531
	No	58	75.3	7.8	
Diabetes	Yes	5	6.5	7.7	1.000
	No	71	92.2	8.1	
Others	Yes	6	7.8	8.6	-
	No	71	92.2	8.8	

Others: HVB + HVC (n = 4), HVB + Alcohol (n = 1), HVB + diabetes (n = 1).

Unlike patients without anemia, the average hemoglobin concentration significantly decreased with the Child-Pugh score in those with anemia (p = 0.002). Figure 1 illustrates the distribution of cirrhotic patients with anemia according to the Child-Pugh score.

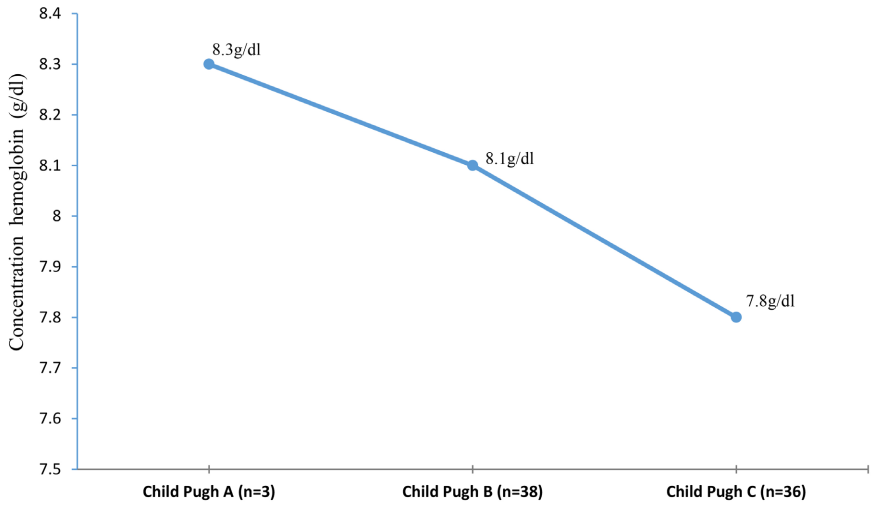

Figure 1. Distribution of patients with anemia according to the Child Pugh score.

3.3. Etiologies of Anemia

Information on meal composition and frequency was not mentioned in the records. Additionally, no iron profile was performed for any patient.

4. Discussion

4.1. Analysis of the Methodology

Our study aimed to determine the prevalence of anemia among cirrhotic patients hospitalized in the **GEMI** department at the University Hospital of Brazzaville, to identify its various characteristics, and to examine its relationship with different epidemiological, diagnostic, and prognostic parameters to improve patient care. In this context, the discussion focused primarily on two aspects.

In this study, we assessed the role of anemia in the complications of cirrhosis and investigated its association with various patient profiles. Patients were selected based on data gathered from medical records and operational definitions we chose for anemia and other variables.

4.2. Study Limitations

The main challenge was accessing medical records post-hospitalization. During the study period, 234 cases of cirrhosis were admitted to the Gastroenterology Department. Due to the high cost of certain tests and poor record-keeping, several files were unusable, and many patients were discharged against medical advice. These factors, along with a high rate of loss to follow-up, introduced selection bias, explaining the low sample size in our study. This also applied to reticulocyte

counts, initially planned for our study to explore normochromic normocytic anemias, and other tests like erythrocyte ferritin levels, soluble transferrin receptor, and inflammatory profiles (ESR, CRP, fibrinogen) for microcytic anemias. Dietary factors that could have contributed to the etiological research were not studied because they were not mentioned in the medical records.While nutritional surveys may be performed for certain patients with specific malnutrition issues, they are not done systematically. Similarly, iron profile tests and other explorations may be conducted for persistent anemias, but in cirrhotic patients, these are generally considered only for severe and persistent anemia in the absence of bleeding. In the context of this retrospective survey, information on dietary factors was not available.

The frequency of anemia in cirrhosis varies across studies. Our results align with those of Nalcoulma *et al.* in Burkina Faso, who reported a 74.5% rate in 2007 [5]. In 2011, Sawadogo in Burkina Faso and Doumbia in Mali reported rates of 54.9% and 44.9%, respectively [10] [11]. These differences could be attributed to various factors, including the causes of anemia, the severity of liver disease, and the operational definitions used in each study, as in the study by Boivin *et al.*, where the criterion chosen was the isotopic measurement of globular volume [12]. Maruyama *et al.* found 70%, defining normal Hb levels at 13 g/dl for men and 12 g/dl for women [6]. Eichner and Hilman reported 60%, considering anemia as a hematocrit below 38% in alcoholic patients [7]. Despite these differences, it can be concluded that the prevalence of anemia is high in cirrhosis.

The male predominance among study patients may explain the higher anemia frequency in this population. The severity of anemia in women, not described in other studies, could be linked to additional mechanisms not reported here, such as blood loss due to menstruation.

Regarding the increased frequency of anemia with age, our findings align with the literature, as tissue tolerance is lower in older individuals. Conversely, the severity of anemia in younger patients could be explained by its rapid onset or physical activity. Indeed, these conditions might account for the earlier and more severe presentation of anemia in younger patients compared to older ones [13].

The diagnostic approach to anemia in cirrhosis is not different from general practice. However, two factors complicate it: first, usual diagnostic tools lack specificity in cirrhosis; second, there is a degradation of some diagnostic markers due to the disease (ferritin, MCV, reticulocytes, haptoglobin, etc.) [8] [14] [15].

The approach to diagnosing anemia in cirrhosis is similar to that for other conditions but is complicated by the disease's multifactorial and often concurrent nature and the lack of specificity of usual biological tests [8]. In cirrhosis, several diagnostic markers deteriorate, including reticulocyte counts, giving a false impression of central anemia, increased ferritin levels, reduced hepatic synthesis of transferrin associated with increased transferrin saturation, and decreased plasma haptoglobin levels [14]-[18]. Similarly, the dilution and splenic sequestration phenomena associated with cirrhosis may produce or worsen false anemia.

However, an etiological search is essential as there is a correlation between liver disease severity, assessed by the Child-Pugh score, and anemia severity [6] [19]. The pathophysiology, informed by knowledge of red blood cells and erythroblast physiology, enables a logical diagnostic approach.

The predominant normochromic normocytic character found in our study is consistent with Nacoulma *et al.* [5]. Normocytic anemia in cirrhotic patients can have various causes, including hemorrhagic anemia, dilutional (false) anemia, hemolytic anemia, and marrow insufficiency. The term "normocytic" here should be understood in a broad sense, encompassing slightly macrocytic anemias, especially after ruling out vitamin deficiencies, since macrocytosis may be simply an expression of the alcohol-related macrocytosis often seen in cirrhotics [9] [12] [13] [16]. In cirrhotics, a normocytic anemia, especially if it presents acutely or with poor clinical tolerance, should raise suspicion of gastrointestinal bleeding, even if reticulocyte counts are not significantly elevated [8]. However, normocytic anemia in cirrhosis is often false anemia due to hemodilution, linked to plasma volume expansion [12] [20] [21]. This dilution can be exacerbated by overhydration, as seen in alcohol withdrawal. This contributes to what is commonly referred to as the "simple and common anemia of cirrhosis" [22]. In the absence of gastrointestinal bleeding, red cell morphological abnormalities, signs of hemolysis, or iron/vitamin deficiency, this diagnosis may be retained for moderate anemia, with hemoglobin levels above 10 g/dl in women and 11 g/dl in men.

The origin of normocytic anemia may also be hemolytic. Hemolysis is a major determinant of anemia in cirrhosis, as shown by studies measuring erythrocyte half-life isotopically in patients with shortened erythrocyte lifespans, indicating pathological hemolysis [20]. Hemolytic anemia, in its simplest form, reflects hypersplenism. Splenic sequestration and the impact of splenomegaly contribute to this often-latent hemolysis.

Bone marrow failure may be a cause of normocytic anemia seen in most patients. Bone marrow failure is almost constant in cirrhotic anemia due to the direct toxicity of alcohol or its metabolites, or medications. It may also be a consequence of partial iron or vitamin deficiencies (nutritional anemias). This marrow insufficiency, more often relative than absolute, becomes a cofactor of anemia in cases of hemolysis or mild bleeding [12] [22]. Anemia results because inefficient erythropoiesis fails to compensate for losses. For over a decade, erythropoietin secretion in cirrhotics has been a research focus, though results are highly controversial. Some studies report higher EPO levels in cirrhotics than in non-anemic controls, while others find lower EPO levels in cirrhotics than in anemic controls [23] [24]. A blood smear would have allowed us to specify possible marrow involvement after ruling out other causes [8].

Microcytic anemias are also frequently observed in cirrhosis, often as microcytic hypochromic anemias, as seen in our study and in previous ones [22] [25] [26]. The most common cause of these anemias is iron deficiency, which must be distinguished from inflammatory anemia, especially if there are clinical and

biological signs of inflammation (elevated ESR, CRP, low transferrin). Iron deficiency anemia is a significant factor in cirrhotic anemias, raising questions about the role of portal hypertension; portal hypertension gastropathy (telangiectasias, lesions similar to angiodysplasia, and colonic varices) is common in cirrhotic patients and may cause slow gastrointestinal bleeding [26]. However, Calès *et al.* found no significant relationship between large esophageal varices and hemoglobin levels after adjusting for the Child-Pugh score. Similarly, there is no significant relationship between serum iron and these endoscopic signs of portal hypertension [25]. Erythrocyte ferritin measurement, a marker less affected by liver disease than serum ferritin, would have enabled diagnosis [27].

Inflammatory microcytic anemia may also occur in cirrhosis, although it is less frequently discussed. Its impact is likely underestimated and should be more accurately assessed, especially considering the current focus on inflammatory processes in alcoholic cirrhosis [22].

Normocytic hypochromic and microcytic normochromic ane-mias are less frequent. Nalcoulma et al. and Doumbia observed these in 11.7% and 10% of cases, respectively [5] [11], which is close to our cumulative frequency of 13% (10/77). These unusual hematologic features, difficult to categorize, may result from multiple anemia-inducing factors, such as malnutrition due to vitamin and trace element deficiencies and intestinal parasitic infections.

Regarding macrocytic anemia, Nalcoulma *et al.* and Doumbia reported it in 25% and 5% of cases, respectively [5] [11]. Our results are lower than those of Dénié *et al.* in France, who found a rate of 48% [4]. This type of anemia, commonly seen in alcoholic cirrhosis, is much more prevalent in Europe, where alcoholism is the main etiology of cirrhosis. Chronic alcoholism often leads to macrocytosis with increased gamma-glutamyl transferase levels [5]. Macrocytosis regression following alcohol cessation supports the diagnosis [6]. Folate or vitamin B12 deficiencies are the other main causes of macrocytic anemia, especially in cases of moderate macrocytosis, with a mean cell volume between 95 and 105 fl [15].

New biological tools provide relevant insights for diagnosing anemia in cirrhosis, particularly of alcoholic origin. For instance, analyzing erythrocyte morphology on blood smears shows promise for diagnosing hemolytic anemias [6] [7] [15]. The protein profile, particularly the albumin–transferrin dissociation, soluble transferrin receptor levels, orosomucoid–haptoglobin dissociation, and iron reserves measured on Perls-stained bone marrow smears, are uncommon in clinical practice but could contribute to characterizing and investigating the etiology of anemia in cirrhotic patients, especially in alcoholic cirrhosis [28]-[30].

Data regarding anemia's impact on hepatic encephalopathy align with literature findings that gastrointestinal hemorrhage complicated by anemia is a risk factor for the occurrence and worsening of hepatic encephalopathy [31] [32].

Aside from platelet counts, the prothrombin level is one of the most commonly prescribed tests for detecting hemorrhagic disease, whether acquired or congenital. In cirrhosis (an acquired condition), hepatocellular insufficiency results in a coagulopathy with signs that depend on the severity of liver damage, regardless of

its cause. This leads to a decrease in PT, which may be accompanied by anemia [33].

As for the association between anemia and hypoalbuminemia, it could be linked to malnutrition, a frequent complication of cirrhosis, or to a pseudo-hypoalbuminemia related to hemodilution. The same goes for the relationship between anemia and ascites grade, which could suggest a false anemia due to hemodilution.

The effects of anemia on the splanchnic circulatory changes of cirrhosis are not precisely known; only preliminary findings suggest that anemia may worsen the hyperdynamic circulation characteristic of the disease [34]. In the study by Calès *et al.*, a significant relationship was indeed found between the size of esophageal varices and hemoglobin reduction; however, this relationship disappeared after adjusting for the Child-Pugh score [8] [25]. Conversely, gastric antral vascular ectasia or "watermelon stomach" in its most advanced form is less controversial and is well recognized as a cause of iron deficiency anemia [8] [35].

The prognostic significance of anemia in cirrhosis varies widely, ranging from a mere epiphenomenon to severe anemia that can threaten life, particularly when caused by gastrointestinal bleeding. Our findings support existing literature, which confirms a correlation between the severity of liver disease, as measured by the Child-Pugh score, and the depth of anemia [4] [11].

5. Conclusion

The frequency of anemia in cirrhotic patients is high in our study. However, diagnosing it in cirrhotics is sometimes challenging due to the lack of specificity of common simple biological tools and the multiplicity and complexity of often interwoven pathophysiological mechanisms.

Conflicts of Interest

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

References

[1] Trinchet, J. and Grando-Lemaire, V. (2004) Diagnostic de cirrhose. *Gastroentérologie Clinique et Biologique*, **28**, 121-128. https://doi.org/10.1016/s0399-8320(04)94996-2

[2] Mouri, S. and Thabout, D. (2016) Suivre un patient cirrhotique en 2016: Quelles complications chercher? Comment les dépister et quels sont les grands principes de prise en charge? *Hépato-Gastro & Oncologie Digestive*, **23**, 710-720.

[3] Mimiesse, J.F., Marlyse, N.M., Clausina, A.A., Arnaud, M.O., Ollandzobo Lucie, A., Bossali, F., *et al.* (2020) Evolutionary and Pronostic Aspects of Cirrhosis at the University Hospital of Brazzaville (Congo). *Open Journal of Gastroenterology*, **10**, 23-29. https://doi.org/10.4236/ojgas.2020.101003

[4] Denié, C., Poynard, T., Gadano, A., Vachiery, F., Soupison, T., Elman, A., *et al.* (1997) Influence de l'anémie sur les modifications hémodynamiques des malades atteints de cirrhose. *Gastroentérologie Clinique et Biologique*, **21**, 29-33.

[5] Nacoulma, E.W.C., Zongo, S., Drabo, Y.J. and Bougouma, A. (2007) Les différents

types d'anémie au cours des cirrhoses au Centre Hospitalier Universitaire Yalgado Ouedraogo de Ouagadougou (Burkina Faso). *Cahiers Santé*, **17**, 87-91.

[6] Maruyama, S., Hirayama, C., Yamamoto, S., Koda, M., Udagawa, A., Kadowaki, Y., *et al.* (2001) Red Blood Cell Status in Alcoholic and Non-Alcoholic Liver Disease. *Journal of Laboratory and Clinical Medicine*, **138**, 332-337. https://doi.org/10.1067/mlc.2001.119106

[7] Eichner, E.R. and Hillman, R.S. (1971) The Evolution of Anemia in Alcoholic Patients. *The American Journal of Medicine*, **50**, 218-232. https://doi.org/10.1016/0002-9343(71)90151-3

[8] Bladé, J.-S., Desramé, J., Corberand, D., Lecoules, S., Blondon, H., Carmoi, T., *et al.* (2007) Diagnostic des anémies au cours des cirrhoses alcooliques. *La Revue de Médecine Interne*, **28**, 756-765. https://doi.org/10.1016/j.revmed.2007.05.005

[9] Organisation mondiale de la Santé (2011) Concentrations en hémoglobine permettant de diagnostiquer l'anémie et d'en évaluer la sévérité. Organisation mondiale de la Santé. https://apps.who.int/iris/handle/10665/85841

[10] Sawadogo, W.A. (2012) Aspects épidémiologiques, cliniques, paracliniques, étiologiques et évolutifs de la cirrhose du foie au Centre Hospitalier Universitaire Sourô Sanou de Bobo Dioulasso. Université de Ouagadougou UFR/SDS. http://www.beep.ird.fr/collect/uouaga/index/assoc/M13092.dir/M13092.pdf

[11] Doumbia, K., Sow, H., Sanogo, S.D., Dicko, M.Y., Tounkara, M.S., Coulibaly, I., *et al.* (2021) Caractéristiques et valeurs prono-stiques de l'anémie chez le cirrhotique. *Health Sciences and Disease*, **22**, 20-23.

[12] Boivin, P., Hartmann, L. and Fauvert, R. (1961) L'anémie des cirrhosis: Fréquence et mécanismes. *Nouvelle Revue Française d'hématologie*, **1**, 3-18.

[13] Delabesse, E., Corre, J., Ysebaert, L., Laharraque, P. and Laurent, G. (2010) Anémie. Dans Polycopié Sémiologie hématologie cellulaire. Faculté de médecine Toulouse-Rangueil, 15-25.

[14] Delmotte, V., Foidart, P., Voeght, A., Lejeune, M. and Delwaide, J. (2019) Diagnostic différentiel de l'anémie dans la cirrhose éthylique. *Revue Médicale de Liège*, **74**, 527-534.

[15] Héry, L., Courtine, E., Iquel, S., Brugneaux, J., Schoenwald, M., Bouibede, F., *et al.* (2016) Anémie hémolytique chez le cirrhotique: Le frottis sanguin est toujours aussi utile. https://inserm.hal.science/inserm-01378104v1/document

[16] Savage, D. and Lindenbaum, J. (1986) Anemia in Alcoholics. *Medicine*, **65**, 322-338. https://doi.org/10.1097/00005792-198609000-00005

[17] Mehta, A.B. and McIntyre, N. (1998) Haematological Disorders in Liver Disease. *Forum* (*Genova*), **8**, 8-25.

[18] Jurczyk, K., Wawrzynowicz-Syczewska, M., Boron-Kaczmarska, A. and Sych, Z. (2001) Serum Iron Parameters in Patients with Alcoholic and Chronic Cirrhosis and Hepatitis. *Medical Science Monitor*, **7**, 962-965.

[19] Sztern, B., Lustman, F., Dufrane, J.P. and de Saint-Georges, A. (1988) Anemia and Cirrhosis. *Revue Medicale de Bruxelles*, **9**, 233-237.

[20] Sheehy, T.W. and Berman, A. (1960) The Anemia of Cirrhosis. *Journal of Laboratory and Clinical Medicine*, **56**, 72-82.

[21] Boivin, P. (1978) Les anémies des alcooliques. *Revue du Praticien*, **28**, 4427-4435.

[22] Arteel, G., Marsano, L., Mendez, C., Bentley, F. and McClain, C.J. (2003) Advances in Alcoholic Liver Disease. *Best Practice & Research Clinical Gastroenterology*, **17**, 625-647. https://doi.org/10.1016/s1521-6918(03)00053-2

[23] Alcindor, T. and Bridges, K.R. (2002) Sideroblastic Anaemias. *British Journal of Haematology*, **116**, 733-743. https://doi.org/10.1046/j.0007-1048.2002.03378.x

[24] Tacke, F., Schöffski, P., Luedde, T., Meier, P.N., Ganser, A., Manns, M.P., *et al.* (2004) Analysis of Factors Contributing to Higher Erythropoietin Levels in Patients with Chronic Liver Disease. *Scandinavian Journal of Gastroenterology*, **39**, 259-266. https://doi.org/10.1080/00365520310008340

[25] Calès, P., Cohen, P., Vinel, J.P., Caucanas, J.P. and Pascal, J.P. (1990) Existe-t-il une relation entre les signes endoscopiques gastro-œsophagiens et la présence d'une anémie chez le malade cirrhotique? *Gastroentérologie Clinique et Biologique*, **14**, 1028-1029.

[26] Tam, T., Ng, W. and Lee, S. (1995) Colonic Mucosal Changes in Patients with Liver Cirrhosis. *Gastrointestinal Endoscopy*, **42**, 408-412. https://doi.org/10.1016/s0016-5107(95)70040-4

[27] Isa, L., Jean, G., Silvani, A., Arosio, P. and Taccagni, G.L. (1988) Evaluation of Iron Stores in Patients with Alcoholic Liver Disease: Role of Red Cell Ferritin. *Acta Haematologica*, **80**, 85-88. https://doi.org/10.1159/000205608

[28] Ferguson, B.J., Skikne, B.S., Simpson, K.M., Baynes, R.D. and Cook, J.D. (1992) Serum Transferrin Receptor Distinguishes the Anemia of Chronic Disease from Iron Deficiency Anemia. *Journal of Laboratory and Clinical Medicine*, **119**, 385-390.

[29] Cacoub, P., Thiolières, J.M., Alexandre, J.A., Foglietti, M.J., Giraudet, P. and Godeau, P. (1996) Profil protéique et carence martiale: Intérêt de l'étude du couple albumine-transferrine. *La Revue de Médecine Interne*, **17**, 627-634. https://doi.org/10.1016/0248-8663(96)87148-7

[30] Perier, C., Chamson, A., Engler, R. and Frey, J. (1983) Evolutionary Changes in Acute-Phase Proteins in Alcoholic Hepatocellular Diseases. *Clinical Chemistry*, **29**, 45-47. https://doi.org/10.1093/clinchem/29.1.45

[31] Berlioux, P. and Bureau, C. (2014) Encéphalopathie hépatique au cours de la cirrhose. Association Française de Formation Médicale Continue en Hépato-Gastro-Entérologie.

[32] Ferenci, P., Lockwood, A., Mullen, K., Tarter, R., Weissenborn, K. and Blei, A.T. (2002) Hepatic Encephalopathy—Definition, Nomenclature, Diagnosis, and Quantification: Final Report of the Working Party at the 11th World Congresses of Gastroenterology, Vienna, 1998. *Hepatology*, **35**, 716-721. https://doi.org/10.1053/jhep.2002.31250

[33] Société Française d'Hématologie (SFH) (2011) Trouble de l'hémostase et de la coagulation. Dans Université Médicale Virtuelle Francophone (UMVF). http://campus.cerimes.Fr/hematologie/enseignement/hematologie_339/site/html/auteurs.html

[34] Ciera, I., Piqué, J.M., Elizalde, I., Feu, F., Garcia-Pagan, J.C., Golden, E., *et al.* (1995) Chronic Anemia Worsens the Hyperdynamic Circulation and Increases the Risk of Gastrointestinal Bleeding in Patients with Cirrhosis. *Gastroentérology*, **108**, 1050-1053.

[35] Bellaiche, G., Nouts, A., Brassier, D., Ley, G. and Slama, J.L. (1998) Ectasies vasculaires antrales diffuses responsables d'une anémie ferriprive sévère chez un malade atteint de cirrhose. Succès du traitement par anastomose porto cave chirurgicale. *Gastroentérologie Clinique et Biologique*, **22**, 832-834.