

# Prevalence and Factors Associated with Hepatic Steatosis in Patients with Metabolic Syndrome in Cameroon: Cases of 4 Reference Hospitals

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**How to cite this paper:** Nga, W.T.B., Etoa, M.C., Marina, B.G., Bagnaka, S.A.F.E., Ndam, A.W.N., Malongue, A., Kowo, M., Tzeuton, C., Noah, D.N., Njoya, O., Andoulo, F.A. and Namme, L.H. (2023) Prevalence and Factors Associated with Hepatic Steatosis in Patients with Metabolic Syndrome in Cameroon: Cases of 4 Reference Hospitals. *Open Journal of Gastroenterology*, 13, 99-110.

<https://doi.org/10.4236/ojgas.2023.133011>

**Received:** January 26, 2023

**Accepted:** March 10, 2023

**Published:** March 13, 2023

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## Abstract

**Introduction:** Nonalcoholic fatty liver disease (NAFLD) is the leading cause of chronic liver disease worldwide and its prevalence increases with that of metabolic syndrome and its components. NAFLD is associated with complications such as cirrhosis and hepatocellular carcinoma. Diagnosis is mainly based on liver biopsy, but there are validated non-invasive methods. The purpose of the study was to assess the impact of metabolic steatopathy in patients with metabolic syndrome in Cameroon. **Methods:** This was a cross-sectional and analytical study conducted over a 6-month period from January 1<sup>st</sup>, 2019, to August 31<sup>st</sup>, 2022. Included were patients with metabolic syndrome who had consulted in endocrinology or gastroenterology at Yaoundé Central Hospital, Douala General Hospital and Douala Gyneco-obstetric and Pediatric Hospital. The diagnosis of NAFLD was made on abdominal ultrasound in front of a homogeneous or heterogeneous hyperechogenic aspect of the hepatic parenchyma compared to that of the right renal cortex called “brilliant liver” and fibrosis evaluated through non-invasive scores (Fib4 and NALFD Fibrosis score). Logistic regression by a uni- and multivariate analysis made it possible to search for the associated factors. **Results.** We included 133 pa-

tients. The female sex represented 64.7%. The mean age was  $55 \pm 9$  years. The prevalence of NAFLD was 48.9%. At the evaluation of fibrosis was significant according to FIB-4 and NAFLD fibrosis score respectively in 6.2% and 4.6% of cases. The independently associated factors were Triglyceridemia  $\leq 1.5$  g/l (OR = 0.33; 95% CI [0.11 - 0.95];  $p = 0.04$ ) and LDL hypercholesterolemia (OR = 2.94; 95% CI [1.07 - 8.11];  $p = 0.036$ ). **Conclusion:** NAFLD was present in almost half of patients with metabolic syndrome. We had very few patients with significant fibrosis, but it needs to be further evaluated. The associated factors are hypertriglyceridemia and LDL hypercholesterolemia.

## Keywords

Hepatic Steatosis, Metabolic Syndrome, Prevalence, Cameroon

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## 1. Introduction

Non-alcoholic or nonalcoholic fatty liver disease (NAFLD) is defined as excessive accumulation of fat in the form of triglycerides in hepatocytes, greater than 5% in histology, apart from any context of excessive alcohol consumption, steatogenic treatment and other causes of chronic liver disease [1] [2] [3] [4].

NAFLD is the leading cause of chronic liver disease worldwide [4] [5]. Global prevalence was estimated at 32.4% in 2022 [6]. There are geographical and ethnic variations in the prevalence of NAFLD [7] [8]. In the United States, prevalence increased from 15% to 25% in the adult population between 2005 and 2010 and varied with ethnic origin [8] [9] [10]. In Asia, it is estimated at 30% [11]. In France, according to data from the CONSTANCE cohort in 2020, there was a prevalence of 18.2% [12]. In South America, the prevalence of NAFLD was 35.7% [7]. In Africa, prevalence was estimated at 13.48% [13]. In Cameroon, Mawo *et al.* in 2020 found a prevalence of 56.1% in patients with type 2 diabetes [14]. In Côte d'Ivoire, Abodo *et al.* found a prevalence of 23% [15]. In Burkina Faso in 2021, Compaoré *et al.* found fatty liver disease in 71.13% of patients and was associated with metabolic syndrome in 30.38% of patients [16]. The average age of patients is between 50 and 60 years with a clear increase in the prevalence among the older patients and there is a male predominance [4] [6] [13] [17].

NAFLD risk factors are well identified in the literature. These include high-calorie diets, physical inactivity, genetic factors [18], abdominal obesity [6] [19] [20] [21] [22] [23] and viral hepatitis [24].

NAFLD evolves in a dysmetabolic and insulin-resistance context [23] [25]. Insulin resistance observed during type 2 diabetes and present in cases of abdominal obesity leads to lipolysis and neoglucogenesis with release of free fatty acids, which go directly to the liver and constitute 60% of triglycerides. Oxidative stress and mitochondrial cytopathy are thought to play an essential role in the development of steatohepatitis lesions [25].

NAFLD encompasses a broad spectrum of liver lesions ranging from simple

steatosis to steatohepatitis (NASH), which is associated with the risk of progression to fibrosis, cirrhosis, and hepatocellular carcinoma [26] [27]. In France, about 220,000 patients with metabolic steatopathy have advanced pre-cirrhotic fibrosis or cirrhosis [12]. The diagnosis of HEPATIC steatosis and the evaluation of liver fibrosis is an important parameter in the follow-up of patients with NAFLD, to prevent the occurrence of complications. The gold standard for diagnosis of fibrosis remains liver biopsy. However, several non-invasive methods have been developed, including impulse elastometry, which is a reliable method but not always accessible in our context, and clinico-biological scores such as the FIB-4 index and the NAFLD Fibrosis Score [28] [29]. In particular, they were used in a study conducted by Bekolo *et al.* in Cameroon to evaluate fibrosis in patients with viral hepatitis C and metabolic syndrome with good negative predictive values [30]. The diagnosis of steatosis can be made through abdominal ultrasound [29].

In the context of a steady increase in obesity and type 2 diabetes cases, sedentary lifestyles are a factor in the development of NAFLD, the purpose of this study was to investigate the prevalence and factors associated with metabolic steatopathy in patients with metabolic syndrome.

## 2. Patients and Methods

This was a cross-sectional study from January 1<sup>st</sup>, 2019, to August 31<sup>st</sup>, 2022. It had three health facilities in the cities of Douala and Yaoundé, namely the General Hospitals of Douala and Yaoundé, and the Central Hospital of Yaoundé. These are first class hospitals each with specialized services (Endocrinology and Cardiology) where patients with metabolic syndrome are treated.

We included all patients over the age of 18 with metabolic syndrome, followed in endocrinology and/or cardiology, and having performed at least one abdominal ultrasound needed for the diagnosis of hepatic steatosis. The Metabolic Syndrome was defined according to the 2009 IDF harmonization consensus which takes into account the mandatory presence of abdominal obesity (BMI greater than or equal to 30 kg/m<sup>2</sup> or a waist circumference greater than 94 cm for men/greater than 80 cm for women); and 2 of the 4 following criteria, blood pressure greater than or equal to 130/85 mmHg or antihypertensive treatment, triglycerides greater than or equal to 1.5 g/l, HDL-cholesterol less than 0.4 g/l in humans or less than 0.5 g/l in women or specific ongoing treatment, fasting blood glucose greater than or equal to 1 g/l or ongoing diabetes treatment [18]. Excluded patients who were those with incomplete records, those with another cause of chronic hepatopathy (viral hepatitis B and C, autoimmune hepatitis, overload disease), taking steatogenic medications or drinking risky alcohol. The incomplete records were those not having the age and sex of the patient, the results of abdominal ultrasound and some biological examinations, namely the assay of transaminases, platelet levels.

The data collected were socio-demographic data (age, sex), comorbidities

(high blood pressure, type 2 diabetes, sedentary lifestyle, occasional alcohol use, smoking, herbal medicine), clinical parameters (weight, height, waist circumference, BMI, blood pressure, fasting blood glucose), biological data (ASAT, ALAT, lipid profile, GGT, prothrombin levels, albuminemia), ultrasound data (liver size, echostructure), evaluation of fibrosis by non-invasive methods (FIB-4, NAFLD Fibrosis Score, impulsive elastometry).

Age gathered is the age of the patient during the study period. The listed co-morbidities of high blood pressure, diabetes are those found in the medical record of each patient and whose diagnosis was made by a specialist doctor (cardiologist and endocrinologist). Regarding alcohol consumption and smoking, that's the information that's in each patient's file. Clinical and para-clinical data were obtained from the patient's last visit during the study period. The NAFLD diagnosis was made in front of an ultrasound image suggestive of fatty liver disease, namely hyperechogenicity of the hepatic parenchyma compared to that of the right kidney in a diffuse or asymmetrical manner. Fibrosis was evaluated by non-invasive scores of NAFLD Fibrosis Score, FIB-4, and impulsive elastometry, which were correlated with the METAVIR score. Fibrosis was considered non-significant if <F2 and significant if F2. Although the diagnosis of cirrhosis is initially histological, we evoked it in front of a METAVIR F4 score and/or an ultrasound image showing a heteronodular liver with a granite appearance and irregular contours.

## 2.1. Operational Terms

- NAFLD Fibrosis Score =  $1.675 + 0.037 \text{ age (years)} + 0.094 \text{ BMI (kg/m}^2\text{)} + 1.13 \text{ fasting glucose or diabetes (yes = 1, no = 0)} + 0.99 \text{ AST/ALT ratio} + 0.013 \text{ platelets (10}^9\text{/L)} + 0.66 \text{ albumin (g/dL)}$ . Interpretation: Score < -1.455: prediction of significant fibrosis absence (F0 - F2); score > -1.455 and 0.675: undetermined score; score > 0.675: prediction of significant fibrosis (F3 - F4).
- FIB-4 = (ASAT age)/(ALAT platelets). Interpretation 1.45 (F0 - F2); >4 (F3 - F4).
- Impulsive elastometry: Interpretation: <7 kPa = absence of fibrosis or minimal fibrosis F0 - F1, 7 - 10 kPa = Moderate fibrosis (F2), 10 - 14 kPa = severe fibrosis (F3 - F4), 14 kPa = cirrhosis.
- Obesity: grade I (BMI between 30 - 35 Kg/m<sup>2</sup>), grade II (BMI between 35 - 40 Kg/m<sup>2</sup>).
- Morbid obesity: BMI ≥ 40 Kg/m<sup>2</sup>.

## 2.2. Statistical Analysis

The data were stored on an electronic sheet designed using Epi data version 4.6 (Epi data Association, Odense, Denmark). The analyses were performed by IBM-SPSS version 26.0 software for Windows (IBM, Chicago, USA). Qualitative variables were presented as percentages, proportions, and/or frequencies. The quantitative variables were represented by their mean and median. To compare

the different frequencies, we used the Chi Square test. The student test was used to compare averages. For the statistical analyses, we considered an error threshold of 5%. Mean values were expressed with their 95% confidence intervals. Property values  $p < 0.05$  were considered statistically significant. The uni- and multivariate regressions made it possible to look for the associated factors depending on whether they are protective or of exposure.

### 2.3. Ethical Considerations

A recruitment authorization has been obtained from the ethics committee of each of the health facilities where the study took place, to allow the collection of our data. The anonymity and confidentiality of each patient's data was respected.

## 3. Results

We collected 419 records from patients with metabolic syndrome, of which 133 were included in our study, for an inclusion rate of 31.7%. The average age was  $55 \pm 9$  years and the median  $57 \pm 12$  year, with a minimum age of 19 and a maximum age of 85. The study population consisted of 64.7% ( $n = 86$ ) female patients, a sex ratio of 0.55 (**Table 1**). Hypertension and type 2 diabetes were found in 46.6% (62 patients) and 42.1% (56 patients) respectively (**Table 1**). There was a sedentary lifestyle in 75.3% or 100 patients and occasional alcohol use in 40.6% or 54 patients. Clinically, 57.1% had grade I obesity, 28.6% grade II obesity and 14.3% morbid obesity (**Table 1**). Biologically, hyperglycemia, hypertriglyceridemia, total hypercholesterolemia, elevated HDL-cholesterolemia, and elevated LDL-cholesterolemia were present in 51.1%, 63.9%, 55.6%, 60.2% and

**Table 1.** General characteristics of population.

|                                    | N (%)      | Mean $\pm$ SD |
|------------------------------------|------------|---------------|
| <b>Mean Age (years)</b>            |            | $55 \pm 9$    |
| <b>Sex</b>                         |            |               |
| Female                             | 86 (64.7)  |               |
| Male                               | 47 (35.3)  |               |
| <b>Comorbidities and lifestyle</b> |            |               |
| Diabetes                           | 56 (46.6)  |               |
| Hypertension                       | 62 (42.1)  |               |
| Sedentarity                        | 100 (75.3) |               |
| Occasional alcohol consumption     | 54 (40.6)  |               |
| <b>Obesity</b>                     |            |               |
| Grade I                            | 76 (57.1)  |               |
| Grade II                           | 38 (28.6)  |               |
| Morbid obesity                     | 19 (14.3)  |               |

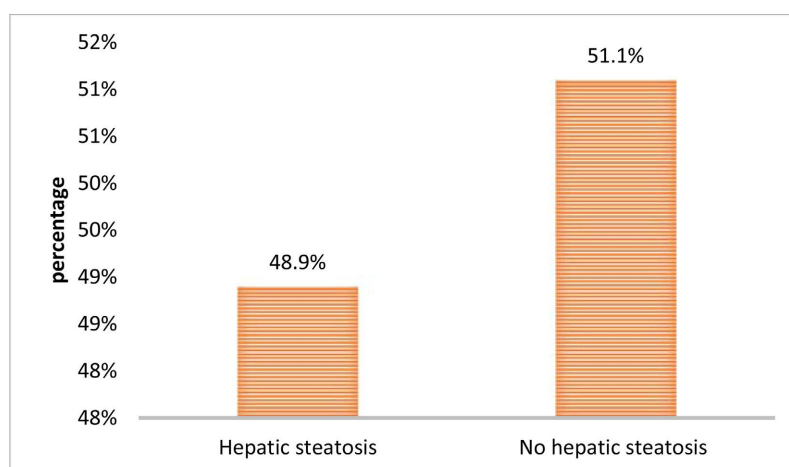
**Table 2.** Biological characteristics of population.

|  | N (%)     | Mean ± SD   | Median ± IQR |
|--|-----------|-------------|--------------|
| <b>Glycemia (g/l) (n = 133)</b>          |           |             |              |
| <1                                       | 65 (48.9) |             |              |
| ≥1                                       | 68 (51.1) |             |              |
| <b>Total Cholesterol (g/l) (n = 133)</b> |           |             |              |
| >2                                       | 74 (55.6) | 1.9 ± 0.7   | 1.9 ± 0.8    |
| ≤2                                       | 59 (44.4) |             |              |
| <b>LDL cholesterol (g/l) (n = 82)</b>    |           |             |              |
| ≥1                                       | 42 (51.2) | 1.3 ± 0.8   | 1.2 ± 0.8    |
| <1                                       | 40 (48.8) |             |              |
| <b>HDL cholesterol (g/l) (n = 133)</b>   |           |             |              |
| ≤0.4                                     | 43 (39.8) | 0.4 ± 0.2   | 0.4 ± 0.2    |
| >0.4                                     | 80 (60.2) |             |              |
| <b>Triglycerides (g/l) (n = 133)</b>     |           |             |              |
| >1.5                                     | 85 (63.9) | 1.4 ± 0.6   | 1.3 ± 0.6    |
| ≤1.5                                     | 48 (36.1) |             |              |
| <b>ASAT (UI/l) (n = 65)</b>              |           |             |              |
| <2N                                      | 57 (87.7) | 43.3 ± 14.6 | 42.3 ± 13.6  |
| ≥2N                                      | 8 (12.3)  |             |              |
| <b>ALAT (UI/l) (n = 65)</b>              |           |             |              |
| <2N                                      | 55 (84.6) | 48.2 ± 11.8 | 46.2 ± 12.8  |
| ≥2N                                      | 10 (15.4) |             |              |

51.2% respectively (**Table 2**). Hepatic cytolysis greater than twice normal was present in 7.5% and 6% of patients on ALAT and ASAT, respectively. At imaging the prevalence of fatty liver disease was 48.9% or 65 patients (**Figure 1**). On abdominal ultrasound, 03 patients (2.6%) had a liver with irregular liver contours. There was significant fibrosis according to FB-4, NAFLD fibrosis score and impulsive elastometry in 6.2%, 4.6% and 1.5% of patients, respectively (**Table 3**). Six patients performed impulsive elastometry. The independent factors associated with fatty liver disease were Triglyceridemia  $\leq 1.5$  g/l (OR = 0.33; 95% CI [0.11 - 0.95]; p = 0.04); and LDL hypercholesterolemia (OR = 2.94; 95% CI [1.07 - 8.11]; p = 0.036) (**Table 4**).

#### 4. Discussion

The profile of patients included in the study is that of a female patient over the age of 50 with obesity and is like that found in some studies in Africa [31] [32]



**Figure 1.** Prevalence of hepatic steatosis.

**Table 3.** Evaluation of hepatic fibrosis.

|                                      | Hepatic steatosis |                  | p     | OR (IC à 95%)    |
|--------------------------------------|-------------------|------------------|-------|------------------|
|                                      | Yes<br>n = 65 (%) | No<br>n = 68 (%) |       |                  |
| <b>NAFLD fibrosis score (n = 13)</b> |                   |                  | 0.767 | 2 (0.07 - 5.15)  |
| ≤1.455 (F0 - F2)                     | 1 (1.5)           | 2 (2.9)          |       |                  |
| >0.675 (F3 - F4)                     | 3 (4.6)           | 2 (2.9)          |       |                  |
| Indeterminate interval               | 1 (1.5)           | 4 (5.9)          |       |                  |
| <b>FIB-4 (n = 65)</b>                |                   |                  | 0.896 | 1.05 (0.5 - 2.2) |
| 1.45 (No Fibrosis)                   | 7 (10.7)          | 12 (18.5)        |       |                  |
| >4 (presence of fibrosis)            | 4 (6.2)           | 3 (4.4)          |       |                  |
| Indeterminate interval               | 20 (30.8)         | 22 (32.4)        |       |                  |
| <b>Fibroscan (n = 6)</b>             |                   |                  | 0.600 | 3 (0.08 - 10.7)  |
| <10 kPa                              | 3 (4.6)           | 1 (1.5)          |       |                  |
| ≥10 kPa                              | 1 (1.5)           | 1 (1.5)          |       |                  |

**Table 4.** Factors associated to hepatic steatosis.

|                           | aOR   | CI 95%       | p     |
|---------------------------|-------|--------------|-------|
| Age > 57 years            | 3.027 | 0.90 - 10.12 | 0.072 |
| Hypertension              | 0.64  | 0.19 - 2.10  | 0.465 |
| LDL cholesterol ≥ 1 g/l   | 2.94  | 1.07 - 8.11  | 0.036 |
| HDL cholesterol ≤ 0.4 g/l | 1.08  | 0.38 - 3.03  | 0.88  |
| Triglyceridemia < 1.5 g/l | 0.33  | 0.11 - 0.95  | 0.04  |

[33] [34]. There is, however, a difference in age, particularly in the studies of Compaoré *et al.* and Ntagirabiri *et al.*, whose patients were younger [16] [34].

A small number of patients had completed the morphological and/or biological assessments necessary for the diagnosis of metabolic liver disease. This contradicts the recommendations of learned societies that strongly advocate the diagnosis of metabolic steatosis in at-risk populations, namely diabetic patients, and those with metabolic syndrome [28] [29].

Almost half of the patients had fatty liver disease, which confirms the important place of hepatic steatosis in patients with metabolic syndrome as found in several studies worldwide, notably in Africa [14] [16] [20] [34] [35] [36] [37]. The prevalence found in our study is much higher than that of Ntagirabiri *et al.* in Burundi or Asabamaka *et al.* in Nigeria which had prevalences of 37.2% and 9% respectively [34] [35]. This difference is explained by the patient profile included in the study, notably that of Asabamaka *et al.*, who worked mainly in diabetic patients with a small proportion of patients with metabolic syndrome. Our prevalence is close to those of the Western series, whose up to 50% in patients with metabolic syndrome [19] [20]. Changes in eating habits, sedentary lifestyles, increased obesity, and metabolic diseases such as type 2 diabetes observed in countries with limited and intermediate resources strongly contribute to the increase in cases of fatty liver disease.

Hepatic fibrosis was very poorly evaluated. Although it is recommended, the evaluation of fibrosis in patients with metabolic syndrome is not a common practice in many practitioners even gastroenterologists and those despite the existence of non-invasive scores easily usable like FIB-4 or NAFLD fibrosis score. This assessment is more important because the progression to cirrhosis is much faster in case of fatty liver disease with the cancer risk known [19] [27] [28] [37] [38]. The prevalence of fibrosis ranged from 6.2% to 1.5% depending on the assessment method. However, the fact that it is underestimated makes this prevalence hard to interpret. Only three patients had a liver with irregular contours on the abdominal ultrasound.

The factors significantly associated with NAFLD were abnormalities in the lipid profile, namely hypertriglyceridemia and elevated LDL cholesterol. These are the factors traditionally found to be associated with the occurrence of NAFLD in patient with metabolic [4] [13] [35] [38] [39]. Dyslipidemia found in patients with metabolic syndrome especially those with obesity as is the case in our patients, results in accumulation of triglycerides in hepatocytes and contributes to changes in lipid metabolism in the liver [40].

The main limitation of the study is the lack of biological and morphological data necessary for the diagnosis of NAFLD in patients with metabolic syndrome. Only one third in patients could be included in the study as having performed at least one abdominal ultrasound for the diagnosis of fatty liver disease.

## 5. Conclusion

Patients with metabolic syndrome are female patients in the 5<sup>th</sup> decade. The prevalence of fatty liver disease was 49.8%. Fibrosis was assessed very little de-



spite the existence of non-invasive scores. Fibrosis when assessed was significant in 6.2% according to FIB4. The factors associated with fatty liver disease were hypertriglyceridemia and elevated LDL cholesterol.

### Contribution of the Authors

Data Collection: Marina Bidjogo Gwet and Winnie Bekolo; Writing and Corrections: Winnie Bekolo; Proofreading and Corrections: Bidjogo, Gwet Marina, Ndjitoyap Antonin, Kowo Mathurin, Eloumou Baganka Servais, Noah Noah Dominique, Ankouane Andoulo Firmin, Njoya Oudou.

### Conflicts of Interest

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

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