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Hepatic Steatosis in the Internal Medicine and Medical Emergency Department of the Sylvanus Olympio University Hospital Center

Kodjo Agbeko Djagadou[1*], Lihanimpo Djalogue[2], Koffi Klouvi[1], Mohaman Awalou Djibril[1]

[1]Department of Internal Medicine, Faculty of Health Sciences, University of Lomé, Lomé, Togo
[2]Department of Internal Medicine, Faculty of Health Sciences, University of Kara, Kara, Togo
Email: *djagkodj11@gmail.com

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Abstract

Objective: Describe the etiological factors of hepatic steatosis in the Internal Medicine and Medical Emergencies department of the Sylvanus Olympio University Hospital of Lomé. **Framework and Method:** Cross-sectional and retrospective study carried out over a period of 5 years from February 1, 2021 to February 28, 2024 (4 years). All patients aged over 18 years with a hyperechoic appearance of the hepatic parenchymal structure on abdominal ultrasound suggestive of focal or diffuse steatosis of the liver were included. **Results:** The prevalence of steatosis was 17.21%. The average age of our patients was 42.91 ± 11.71 years. We noted a sex ratio (M/F) of 1.21. Diffuse abdominal pain was the main reason for consultation (38.60%). Hepatomegaly was found on abdominal ultrasound in 28.16% of cases. Two main groups of etiological factors were noted. These included metabolic factors (alcoholism, obesity, type 2 diabetes and high blood pressure (hypertension)) and infectious factors (HIV, HBV, HCV). **Conclusion:** Hepatic steatosis is a chronic pathology associated with etiological factors. It can progress to steatohepatitis, cirrhosis or even liver cancer.

Keywords

Hepatic Steatosis, Etiological Factors, CHU-SO, Togo

1. Introduction

Hepatic steatosis (HS) is a histological lesion of the liver linked to the overload or accumulation of fats, mainly triglycerides, in the cytoplasm of hepatocytes [1]. This disease emerged from the industrial revolution, which caused profound changes in our diet and led to a sedentary lifestyle [2]. HS, therefore has the same

roots as obesity and type II diabetes and is currently considered the hepatic complication of metabolic syndrome [3]. It is a silent disease whose dominant isolated form is indolent but requires treatment to avoid complications [2]. Although this lesion is benign in some cases, in other cases it can be associated with inflammatory lesions of the liver parenchyma called steatohepatitis. Indeed, at the time of diagnosis of steatosis, 30 to 40% of patients have significant fibrosis [2]. In developed countries, studies have shown that undiagnosed steatohepatitis is the most common cause of cirrhosis [3] and other consequences of steatosis were portal hypertension (PH) and hepatocellular carcinoma (HCC) [2]-[4].

The prevalence of HS is estimated between 20% and 30% in developed countries [2]. In Africa, several studies have been carried out with varying prevalences. In Nigeria, the prevalence of HS has been estimated at 9% in a diabetic population [5]. In Burundi, a study carried out on HS during metabolic syndrome in black African adults found a prevalence of 37.2% [6]. HS is a chronic liver disease whose prevalence is variably estimated in developing countries where the discovery is often incidental on abdominal ultrasound. Its histological confirmation after liver biopsy (PBH), an examination which is rarely performed due to the often limited technical capacity in public health structures in developing countries and the advanced stage of the patients. with bleeding disorders.

In Togo, we have little data on HS. The diagnosis of HS is limited to ultrasound results and most practitioners easily relate it to weight gain without carrying out etiological research and medical follow-up. This work was initiated in the Internal Medicine Department of Medical Emergencies of the University Hospital Center (CHU) Sylvanus Olympio (SO) with the general objective of updating data on the etiological profile of fatty liver disease. The specific objectives of this study were:

- Estimate the hospital prevalence of fatty liver disease.
- Describe their clinical, paraclinical, therapeutic and evolutionary aspects.
- Describe the etiological factors associated with hepatic steatosis.

2. Study Framework and Method

2.1. Study Framework

The Internal Medicine and Medical Emergencies department of the CHU-SO of Lomé served as the setting for our study.

2.2. Study Method

This was a cross-sectional study with a retrospective collection of data over a period of four (04) years, going from February 1, 2021 to February 28, 2024. The duration of data collection was 4 months (from June 2024 to September 2024).

The study focused on all patients whose abdominal ultrasound noted a hyperechoic structure suggesting hepatic steatosis.

The collection was made from patient files kept in the department's archives. Our data was collated using a previously established and validated survey form. All files of patients aged over 16 years who presented on abdominal ultrasound

with a hyperechoic appearance of the liver parenchyma suggestive of focal or diffuse steatosis of the liver and data were included in the study.

Unusable files (poorly completed or incomplete files) found in the department's archives were not included in our study.

The data were collected using a previously established survey form, which included sociodemographic data, clinical data, paraclinical data, and data on progress and treatment.

2.3. Operational Definitions

- Chronic alcohol consumption was considered as such, as reported in the medical file.
- Traditional treatment was essentially based on herbal infusions.
- Mixed intoxication concerns the chronic consumption of alcohol, hepatotoxic drugs and traditional treatment.
- Metabolic syndrome was defined according to the guidelines of the International Diabetes Federation (IDF). As criteria, we retained abdominal obesity, hyperglycemia and high blood pressure or antihypertensive treatment.
- We considered the dosages of:
  - AST greater than 1.5 times normal o ALT greater than 1.5 times normal o GGT greater than 50 IU/1 o ALP greater than 150 IU/1 o BT greater than 2 times normal o Total cholesterol greater than 2g /l o Triglycerides greater than l.60g/l
  - TP less than 50% witnesses of hepatocellular insufficiency
- The ultrasound diagnosis of steatosis was defined by the hyperechogenicity of the liver parenchyma compared to that of the right renal cortex. The ultrasound scans carried out by an experienced radiologist were retained.

The SPHINX Plus2 v.4.5.0.19 software was used to use our data and analyze the results. The results obtained were compiled into simple frequency tables. Excel software allowed us to create the graphs.

Before data collection, we requested and obtained authorization from unit managers. The identity of the patients remained strictly confidential.

3. Results

We identified a total of eight hundred and two (1012) usable files including 174 with hepatic steatosis, *i.e.* a hospital prevalence of 17.21%.

3.1. Sociodemographic Data

The average age of our patients was 42.91 years ± 11.71 with extremes of 18 and 82 years. We noted a sex ratio (M/F) of 1.21.

3.2. Clinical Data

The main reasons for consultation or hospitalization were diffuse abdominal pain, deterioration in general condition, isolated liver abnormality; ascites respectively

in 38.60%; 16.30%; 23.20%; 16.40% of cases

Long-term medication, alcoholism, and obesity were the main antecedents (Table 1).

Table 1. Distribution of patients according to antecedents.

	Effective	Percentage
Alcoholism	76	43.67
HT	62	35.63
Long-term medication	89	51.14
Obesity	75	43.10
Diabetes	42	24.13
HIV infection	21	12.06
Smoking	10	5.74
Chronic carrier of HBV	17	9.77
Chronic HCV carrier	9	5.17
Asthma	2	1.14

General signs

Asthenia was found in 44.82% of cases (Table 2).

Table 2. Distribution of patients according to general signs.

	Effective	Frequency
Asthenia	78	44.82
Pallor	28	16.09
Fever	33	18.96
Jaundice	29	16.66

Physical signs

Abdominal obesity and hepatomegaly accounted for 31.03% and 28.16%, respectively (Table 3).

Table 3. Distribution of patients according to physical signs.

	Effective	Fréquency
Hepatomegaly	49	28.16
Ascites	44	25.28
IMO	42	24.13
Abdominal obesity	54	31.03
Collateral venous circulation	15	8.62
Splenomegaly	19	10.91

3.3. Paraclinical Data

Glycemic disorders

Hyperglycemia was found in 46 (26.41%) patients. Glycated hemoglobin was measured in 42 patients and was elevated in 24 patients, with extremes of 7.1% and 14.20%.

Hepatic biological abnormalities

Liver failure was found in 59.1% of patients (Table 4).

Table 4. Distribution of patients according to hepatic biological abnormalities.

	Effective	Percentage
Hepatocellular insufficiency	27	15.51
Cytolysis + Cholestasis	67	38.50
Isolated cytolysis	16	09.19

Lipid abnormalities

All patients had completed the lipid profile, as shown in Table 5.

Table 5. Distribution of patients according to lipid abnormalities.

	Effective	Percentage
Mixed hypercholesterolemia	39	32.65
Pure hypercholesterolemia	32	28.58
Hype Pure hypertriglyceridemia	21	14.29

Serum protein electrophoresis (EPP)

One hundred and forty-four (104) patients had completed the EPP as shown in Table 6.

Table 6. Distribution of patients according to EPP result.

	Effectif	Percentage
Hypergammaglobulinemia	53	30.45
Hypergammaglobulinemia + Beta-gamma block	37	21.26
Normal	17	9.77

Alpha-feto-protein (AFP) assay

The AFP assay was carried out in 99 patients with a significantly high level (greater than 400 ng/ml) in 3 cases (3.03%).

Serologies carried out

- Retroviral HIV serology was positive in 25 (14.36%) patients
- HIV/HBV co-infection was found in 8 (4.59%) patients.
- HBsAg was positive in 22 (12.64%) patients
- Anti-HCV Ab was positive in 9 (5.17%) patients.

Abdominal ultrasound data

Hepatomegaly and splenomegaly were represented in 48.27% and 12.64% of cases respectively (Table 7).

Table 7. Distribution of patients according to ultrasound lesions.

	Effective	Percentage
Hepatomegaly	84	48.27
Liver atrophy	4	2.29
Splenomegaly	22	12.64
Ascites	49	28.16
Gallstone lithiasis	16	9.19

Etiological factors

HIV infection represented 26.43% of cases (Table 8).

Table 8. Distribution of patients according to probable etiologies of HS.

	Effective	Percentage
HIV infection	46	26.43
HT	62	35.63
Diabetes	42	24.13
Metabolic syndrome	18	10.34
Long-term medication		9.52
Alcohol	75	43.01
Obesity	75	43.01
Chronic HBV infection	22	12.64
Chronic HCV infection	18	10.34
HIV-HBV co-infection	8	4.59

$P = 0.0576$.

3.4. Therapeutic Data

Only 32.65% of patients had received statin treatment (Table 9).

Table 9. Treatment received.

	Effective	Percentage
Antiretrovirals	46	26.43
Antihypertensives	62	35.63
Insulin/ADO	42	24.13
Statin	39	32.65
Tenofovir	22	12.64
Sofosbuvir + daclasvir	18	10.34
Attempting alcohol withdrawal	75	43.01
Weight reduction	75	43.01

EVOLUTION

Of the 174 patients, we recorded 16 deaths (9.19%) as shown in Table 10.

Table 10. Distribution of patients according to probable causes of death.

	Effective	Percentage
HIV-related encephalopathy	5	2.87
Diabetic ketoacidosis	7	4.02
Stroke*	2	1.14
Hepatic encephalopathy	2	1.14

4. Discussion

4.1. Strength and Weakness of Our Study

Our workforce was reduced due to the non-use of certain files; similarly, PBH was very rarely performed in patients presenting with HS in the Internal Medicine department to allow a definitive diagnosis. This could constitute a weakness of the study. However, our study has the merit of highlighting HS and its etiologies in a context of limited resources.

4.2. Epidemiological Data

The hospital prevalence was 17.01%. This prevalence is higher than that of Sidibe *et al*. in Mali [7] and Nomura *et al*. in Japan [8], who found that 4.97% and 14% of HS were confirmed on histology. The prevalence of Hepatic Steatosis in our study was probably overestimated because it was discovered incidentally on abdominal ultrasound.

The average age of our patients was 42.91 years ± 11.71 with extremes of 18 and 82 years. Sidibe *et al*. in Mali found an average age of 41.6 ± 14.6 years [7]. HS is an uncommon condition before the age of 20 and is most often diagnosed after the age of 40 [2]-[9].

We also noted male dominance, a sex ratio of 1.21, contrary to certain data in the literature [6]-[10]. Indeed, the westernization of eating habits and a sedentary lifestyle seem to predominate among men, facilitating obesity, especially abdominal obesity, which could lead to fatty liver disease.

4.3. Clinical Data

The reasons for consultations of our patients were dominated by non-specific signs of HS (diffuse abdominal pain, AEG, liver function abnormalities). This calls on practitioners to consider the symptoms mentioned by the patient globally during their consultation and to screen for steatosis systematically because it is often discovered incidentally. The main antecedents were long-term medication, alcoholism and obesity. Obesity and alcoholism are known to be risk factors for the occurrence of HS. Concerning long-term medications, they can be a source of hepatic steatosis because certain medicinal molecules such as antiretrovirals and

certain antihypertensives cause dyslipidemia. The latter could lead to fatty liver.

Abdominal obesity, hepatomegaly and ascites were the predominant physical signs; If obesity was a cause of HS, hepatomegaly and ascites could be complications. Obesity is a pathology that is prevalent in sub-Saharan Africa associated with type 2 diabetes, the vascular risks of which are systemic and deleterious for the human body.

4.4. Paraclinical Data

Metabolic abnormalities consisting of glycemic imbalance in diabetics and dyslipidemia with mixed hypercholesterolemia were observed in our studies. These abnormalities are known to be proven risk factors for hepatic steatosis [11] [12]. Ouakaa-Kchaou *et al*. noted that the insulin resistance syndrome would likely be involved in both the genesis of steatosis and the progression of steatohepatitis [13]. They also found that a high level of insulin and glucose would lead to the initiation of fibrogenesis by stimulating the secretion of fibrinogenic growth factor by the stellate cells of the liver [14]. Dyslipidemia has been described by other authors as being able to lead to HS even outside of obesity. Although the liver's primary role is not to store lipids in humans, it is capable of doing so when there is an excess of lipids in the diet.

4.5. Etiological Data

We incriminated two main groups of etiological factors that were not statistically significant. These included metabolic factors (alcoholism, obesity, type 2 diabetes and high blood pressure (hypertension)) and infectious factors (HIV, HBV, HCV). Ntagirabiri *et al*. also noted metabolic factors such as obesity, hypertriglyceridemia and metabolic syndrome [8]. For Leclercq *et al*. in Belgium, steatosis was constantly associated with obesity, type II diabetes and dyslipidemia; however, the role of hypertension appears to be poorly understood in the genesis of hepatic steatosis [2]. The infectious factors raised were clearly associated with hepatic steatosis, according to the literature [8]. Their specific role and that of their treatment are clearly defined. In the literature, it is described a frequent high HS in patients whose ARV protocol contains non-nucleoside reverse transcriptase inhibitors (NRTI) such as AZT and d4T and protease inhibitors (PI), which can be responsible for clinical lipohypertrophy. In Togo, d4T was withdrawn from the treatment protocol for PLHIV because of adverse effects such as lipodystrophy and HS. These viral infections are on the rise in West Africa. Particular emphasis must be placed on their prevention through the intensification of media awareness and proximity campaigns.

4.6. Therapeutic and Evolutionary Data

In our study, all patients had received statin treatment combined with treatments for the etiological factor. We believe that dietary measures should be prescribed to patients upon discharge from hospitalization [2]. Indeed, according to Leclercq

et al., an average weight loss of approximately 1 kg/m^2 associated with an increase in exercise improves liver enzymes in obese or overweight patients with non-alcoholic HS [2]. In the event of HS of alcoholic or toxic origin, stopping alcohol, traditional treatments and hepatotoxic medications must be essential.

5. Conclusion

Our study carried out over a period of 4 years, noted that HS was present in patients hospitalized in the Internal Medicine and Medical Emergencies Department of the CHU-SO. Its discovery was fortuitous on abdominal ultrasound without histological confirmation. The two groups of etiological factors found without a statistically significant link were metabolic (alcoholism, obesity, type 2 diabetes and high blood pressure (hypertension)) and infectious (HIV, HBV, HCV). The absence of specific clinico-biological manifestations suggestive of hepatic steatosis makes ultrasound an essential tool for the exploration of HS in our settings. It is a potentially progressive pathology due to the subsequent occurrence of steatohepatitis. Prospective and analytical studies must be carried out in order to establish a list of all the etiological factors actually associated with HS in adults.

Conflicts of Interest

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

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