

Prevalence of Liver Fibrosis Assessed by Transient Elastography in Patients with Type 2 Diabetes and MASLD at the Brazzaville University Hospital

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ABSTRACT

Introduction: Metabolic dysfunction-associated steatotic liver disease (MASLD) is the most prevalent chronic liver disease worldwide, with type 2 diabetes being one of the major risk factors for its progression to fibrosis. Understanding the prevalence of hepatic fibrosis in these patients living in Brazzaville is essential.

Aim: To assess the prevalence of hepatic fibrosis in patients with type 2 diabetes and MASLD.

Methods: We included 120 patients with type 2 diabetes and ultrasound confirmed hepatic steatosis. Transient elastography (Fibroscan) was used to evaluate hepatic fibrosis and its stage, with an elasticity > 7kPa indicating the presence of fibrosis. We analyzed the prevalence of fibrosis and identified associated factors.

Results: The median age was 56 years, with 77.5% of patients aged 50 or older, and 62.5% were female. Hepatic fibrosis was found in 43.3% of patients, with 14.2% presenting severe fibrosis. Severe hepatic steatosis was observed in 41.6% of participants. Factors associated with fibrosis included metabolic syndrome, dyslipidemia, insulin treatment and chronic kidney disease.

Conclusion: Hepatic fibrosis detected via elastography is common in type 2 diabetes patients with hepatic steatosis, highlighting the importance of its screening in routine clinical follow-up.

Keywords

Diabetes, MASLD, Liver fibrosis, CHUB, Brazzaville, Congo.

Introduction

Chronic liver diseases represent a major global health burden. In 2021, these conditions affected 1.7 billion people worldwide and accounted for 4% of global mortality from all causes [1,2]. Among

these disorders, metabolic dysfunction-associated steatotic liver disease (MASLD), formerly known as non alcoholic fatty liver disease (NAFLD), has emerged as the most prevalent chronic liver disease globally, affecting 1.3 billion people or 16.1% of the world population, in 2023 [3].

Because of the central pathophysiological role of insulin resistance,

type 2 diabetes represents is one of the main risk factors for the onset and progression of metabolic dysfunction-associated steatotic liver disease (MASLD). Consequently, the rising incidence of type 2 diabetes is closely correlated with the increasing prevalence of MASLD [4]. Epidemiological projections estimate that by 2030, type 2 diabetes will affect 7.7% of the global population, with approximately 60% of these patients with diabetes concurrently presenting with MASLD [5,6].

The prognosis and severity of MASLD depend on the development of hepatic fibrosis, which can progress to cirrhosis or hepatocellular carcinoma (HCC) [7]. In this regard, the annual incidence of primary liver cancer has been estimated to range between 0.8% and 3.9% in patients with metabolic dysfunction-associated steatohepatitis (MASH) [8]. Consequently, early screening and long-term monitoring of hepatic fibrosis are crucial in patients with type 2 diabetes, particularly in the presence of concurrent MASLD.

Although liver biopsy remains the gold standard for staging fibrosis, its invasive nature and the associated risk of iatrogenic complications limit its reproducibility in routine clinical practice. Conversely, transient elastography (Fibroscan), despite being an indirect measurement, offers a noninvasive alternative free of complications. Furthermore, it shows a better histological correlation with biopsy findings than noninvasive blood-based fibrosis scores [9].

To raise awareness among clinicians and encourage the incorporation of this screening into the management of type 2 diabetes in Brazzaville, we conducted this study. The aim of this work was to determine the prevalence of hepatic fibrosis, assessed by transient elastography in patients with type 2 diabetes presenting with hepatic steatosis at the University Hospital Center of Brazzaville (CHUB).

Methods

This was a cross-sectional (descriptive and analytical) investigation with a prospective data collection approach.

Data were collected from April 4 to October 3, 2024, spanning a period of six (06) months. Patients were recruited at the Brazzaville University Hospital Center (CHUB) within the department of metabolic and endocrine diseases during both inpatient and outpatient diabetes consultations.

The study population consisted of patients with type 2 diabetes mellitus.

Included in the study were patients aged 18 years and older who did not engage in risky alcohol consumption, did not have chronic liver disease, and were not taking hepatotoxic or steatogenic medications; these patients had been diagnosed with hepatic steatosis via liver ultrasound and had usable medical records. All included patients consented to participate in the study.

The following were excluded from the study: pregnant women, patients with type 1 diabetes, those with HIV infection, those receiving corticosteroid therapy, those with unusable medical records, and those who did not provide consent for the study.

All patients admitted as inpatients or seen as outpatients during the study period who met the inclusion criteria constituted our study population.

Data were collected using a structured questionnaire, which included:

- Sociodemographic data : age, sex
- Clinical data : blood pressure, weight, height, abdominal circumference, duration of diabetes, presence or absence of acute decompensation of diabetes, comorbidities associated with diabetes, as well as the presence of microvascular and macrovascular complications.
- Therapeutic data : use of antidiabetic medication and type of medication taken
- Biological data : blood glucose levels, glycated hemoglobin transaminases, creatinine levels, triglycerides, total cholesterol, HDL cholesterol, and LDL cholesterol
- Morphological data:
 - Presence of hepatic steatosis on ultrasound, stage of steatosis evaluated by the controlled attenuation parameter (CAP) of the Fibroscan.
 - Presence of hepatic fibrosis and its stage, assessed by Fibroscan. Patients with a liver stiffness measurement exceeding 7kPa on the transient elastography (Fibroscan) were considered to have hepatic fibrosis. A liver stiffness greater than 9.6 kPa indicated severe fibrosis, while a stiffness measurement between 7 and 9.5 kPa indicated mild to moderate fibrosis.

Statistical analysis

Data were entered using Microsoft Excel 2016 and then exported to IBM SPSS Statistics version 25 for analysis. Categorical variables were expressed as frequencies and percentages, while quantitative variables were presented as mean $\pm$ standard deviation or median with interquartiles ranges.

The Pearson Chi-square test or Fisher's exact test was utilized for the comparison of categorical variables. A bivariate analysis was conducted to identify determinants associated with the occurrence of hepatic fibrosis. The significance level was set at $p < 0.05$.

Results

Of the 177 patients identified, 120 met our selection criteria and were included in the study.

Demographic Characteristics

The median age was 56 years (50, 64). Of the 120 patients, 93 were at least 50 years old (77.5%) and 27 were younger than 50 (22.5%). There were 75 women (62.5%) and 45 men (37.5%), for a sex ratio of 0.6.

Clinical Characteristics

The average duration of type 2 diabetes mellitus was 6.1 ± 5 years.

Metabolic syndrome was found in 67 patients, representing 55.8% of cases.

Angiopathic complications were observed in 61 patients, representing 50.8% of our study population (see Table 1). These included microangiopathic complications in 58 patients (95.1% of angiopathic complications and 48/3% of the overall study population) and macroangiopathic complications in 3 patients (4.9% of angiopathic complications and 2.5% of the overall study population).

Table 1: Diabetic microangiopathic lesions.

Microangiopathy	N	%
Retinopathy	13	22.4
Nephropathy	31	53.4
Neuropathy	14	24.2
Total	58	100

Abdominal obesity was the most common comorbidity (113 patients, 94.2%), followed by hypertension (92 patients, 76.7%).

Among dyslipidemias, hypercholesterolemia was the most common (34 patients, 55.7%), followed by hypertriglyceridemia (17 patients, 27.9%) and mixed dyslipidemias (10 patients, 16.4%). The frequencies of the various comorbidities are shown in Table 2.

Table 2 : Diabetes related comorbidities.

Comorbidity	Numbers	Frequencies
Hypertension	92	76.7
Obesity	42	35
Grade I	18	42.9
Grade II	11	26.2
Grade III	13	30.9
Abdominal obesity	113	94.2
Dyslipidemia	61	51.7

Liver Lesions
Hepatic Steatosis

The most common stage of steatosis according to the CAP Fibroscan was mild steatosis (59 patients, 49.2%). The distribution of patients according to the different stages of steatosis is shown in the table below.

Table 3: Stages of liver steatosis.

Stades	Numbers (N)	Frequencies (%)
Mild steatosis	59	49.2
Moderate steatosis	11	9.2
Sévère steatosis	50	41.6
Total	120	100

Hepatic fibrosis assessed by Fibroscan (pulsed elastography)
Hepatic fibrosis was identified in 52 patients (43.3%). Of these 52 patients, 17 (32.6% of patients with fibrosis) had liver stiffness measurements greater than 9.6 kPa, corresponding to severe fibrosis, while 35 (67.3%) demonstrated liver stiffness measurements between 7 and 9.5 kPa, corresponding to mild to

moderate fibrosis. The distribution of patients according to the presence and stage of liver fibrosis as determined by transient elastography (Fibroscan) is shown in Table 4.

Table 4: Distribution by Presence and Stage of Liver Fibrosis.

Hepatic fibrosis	Numbers (N)	Frequencies (%)
Patients without fibrosis	68	56.7
Patients with fibrosis	52	43.3
Mild and moderate fibrosis	35	29.2
Severe fibrosis	17	14.2

Risk Factors for Hepatic Fibrosis

The presence of dyslipidemia, severe steatosis, and metabolic syndrome were among the risk factors identified in our study population. The table below presents these various risk factors.

Table 5: Main risk factors of hepatic fibrosis.

	With fibrosis		Without fibrosis		OR	95% CI	P
	n	%	n	%			
Age (years)					1.14	0.48 – 2.73	0.75
>50 (n=93)	41	44.1	52	55.9			
≤50 (n=27)	11	40.7	16	59.3			
Insulin therapy					2.54	1.21 – 5.33	0.013
Yes (N=56)	31	55.4	25	44.6			
No (N=64)	21	32.8	43	87.2			
HBP					1.85	0.75 – 4.52	0.17
Yes (n=92)	43	46.7	49	53.3			
No (n=28)	09	32.2	19	67.8			
Dyslipidemia					4.42	2.00 – 9.74	0.00
Yes (n=61)	36	59	25	40.9			
No (n=59)	16	27.1	43	72.9			
CKD					2.4	1.13 – 5.09	0.02
Yes (n=46)	26	56.5	20	43.5			
No (n=74)	26	35.2	48	64.9			
Obesity					1.54	0.71 – 3.31	0.26
Yes (n=42)	22	52.4	20	47.6			
No (n=72)	30	41.7	42	58.3			
Metabolic syndrome					8.76	3.66 – 20.9	0.000
Yes (n=67)	43	64.2	24	35.8			
No (n=53)	28	35	52	65			
Diabetes control					1.57	0.31 – 3.46	0.25
Yes (n=81)	38	46.9	43	53.1			
No (n=39)	14	35.9	25	64.1			
Abdominal obesity					1.98	0.36 – 10.66	0.42
Yes (n=113)	50	44.2	63	55.7			
No (n=7)	02	28.6	05	71.4			

N: numbers; HBP: high blood pressure; CKD: chronic kidney disease; OR: odd ratio.

Discussion

Our study assessed liver fibrosis in 120 patients with type 2 diabetes and hepatic steatosis. The prevalence of liver fibrosis as measured by transient elastography (Fibroscan) was found to be

43.3%, including 14.2% with severe forms.

In comparison to patients without fibrosis, those with fibrosis exhibited a significant higher prevalence of metabolic syndrome (OR 8.76), dyslipidemia (OR : 4.42), chronic kidney disease (OR 2.40) and reliance on insulin therapy (OR 2.54).

In the literature, the prevalence of liver fibrosis among patients with type 2 diabetes exhibits substantial heterogeneity across cohorts. For instance, a study conducted in India by Gupta et al. highlighted a prevalence rate of 60% within this population [10]. In the United States, Lomonaco et al. reported a prevalence of 21% in a cohort of 561 patients [11]. Data from a Romanian study indicated a prevalence rate of 18.8% [12].

In sub-Saharan Africa, the rise of non-communicable disease driven by the epidemiological transition encompasses metabolic dysfunction-associated steatotic liver disease (MASLD). Its prevalence in the region is estimated at 29.21% in the general population and reaches 37% among patients with type 2 diabetes. [13] Within this specific cohort, a Ghanaian study reported a liver fibrosis prevalence of 11.6% [14]. However, a prevalence comparable to that observed in our study was found in a study conducted among non-type 2 diabetic patients with MASLD in Abidjan (Ivory Coast). In that study, 48.7% of patients had significant fibrosis [15].

One explanation for the variability in liver fibrosis prevalence rates across studies is the difference in liver stiffness thresholds (in kPa) chosen to define fibrosis. Indeed, diagnostic criteria varied between studies. Some authors included patients as soon as liver stiffness exceeded 7.0 kPa, whereas others applied stricter criteria, for instance setting the threshold at 7.8 kPa or higher.

We believe that further studies involving larger patient cohorts from sub-Saharan Africa, comparing histological results from liver biopsies with those from transient elastography, are essential to determine and validate the liver stiffness thresholds that correlate with clinically significant liver fibrosis in the African Black population.

Several factors account for the fibrosis observed in patients with type 2 diabetes. In these individuals, the onset of fibrosis follows chronic inflammation induced by hyperglycemia. Specifically, fatty acids, toxins and advanced glycation end products resulting from hyperglycemia activate the intrahepatic immune system, particularly Kupffer cells, leading to cell death [16]. This cellular stress is exacerbated by ballooned hepatocytes that secrete sonic hedgehog protein, which in turn stimulates the activation of liver stellate cells. Initially quiescent, liver stellate cells transform into myofibroblast-like cells upon activation, producing an excess of extracellular matrix and collagen [16]. This prolonged fibrogenesis leads to the progression of fibrosis characterized by the accumulation of scar tissue that is resistant to degradation [17].

As for the independent risk factors for liver fibrosis identified in our study, their impact is underpinned by interconnected biological

pathways from a pathophysiological perspective

Specifically, hypertriglyceridemia directly stimulates fibrogenesis by causing a massive influx of free fatty acids into hepatocytes which leads to severe oxidative stress and the release of inflammatory mediators. These mediators subsequently trigger the activation of liver stellate cells and initiate fibrogenesis [18-20]

Chronic kidney disease contribute to this process by exacerbating hepatic inflammation through the accumulation of uremic toxins and the concomitant activation of the renin-angiotensin system. This results in worsening liver damage [21].

Our results also reveal an association between fibrosis and treatment with exogenous insulin. This observation may be explained by the fact that diabetic patients requiring insulin often exhibit more pronounced insulin resistance [22,23]. This, in turn, increases hepatic triglyceride accumulation, promoting steatosis and its progression to fibrosis [24].

In the littérature, the conclusions of studies evaluating the influence of exogenous insulin on the progression of non-alcoholic steatohepatitis (NASH) are contradictory. While Goh et al. reported an association between insulin use and advanced fibrosis in patients with type 2 diabetes, Hamaguchi et al. conversely associated this treatment with histological improvement [25,26]. Furthermore, another study highlighted a link between insulin and the incidence of hepatocellular carcinoma (HCC), while metformin appeared to have a protective effect [27].

The major risk factor identified in our patients was metabolic syndrome. Data from the literature confirm that many of its components constitute a significant risk factor for liver fibrosis [28-36]. Consequently, the coexistence of these components in a single individual synergistically increases the risk of developing severe liver damage.

Our study does however, have certain limitations. One of these is that none of the patients underwent a liver biopsy. Histological analysis of liver biopsies is considered the gold standard for the diagnosis of liver fibrosis, and its implementation would have allowed for a more accurate diagnosis of this condition.

There was also a selection bias. In fact all the morphological and biological examinations were entirely the responsibility of the patients. Consequently, those without sufficient financial means to undergo the required tests, such as Fibroscan, were excluded from the study.

Finally the modest size of our study population does not allow us to extrapolate the conclusions of our work to the broader population of type 2 diabetic patients living in Brazzaville.

Conclusion

Hepatic fibrosis is prevalent among patients with type 2 diabetes presenting with hepatic steatosis in Brazzaville. The risk of

developing this complication is particularly high among those with dyslipidemia, chronic kidney disease, metabolic syndrome, as well as among those treated with insulin. Consequently, these patients should undergo noninvasive assessment for hepatic fibrosis during routine follow-up, ideally using transient elastography (Fibroscan). It is essential to raise awareness among the physicians managing these patients about the importance of involving hepatologists in their care, in order to optimize hepatic fibrosis screening and prevent progression to cirrhosis or primary liver cancer.

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