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Risk Factors for Liver Fibrosis in Patients with Type 2 Diabetes Mellitus and Metabolic Dysfunction–Associated Steatotic Liver Disease in Brazzaville

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Abstract

Introduction: Introduction: Type 2 diabetes mellitus is a factor in the initiation and progression of metabolic dysfunction-associated steatotic liver disease (MASLD) to its severe forms with hepatic fibrosis, with a risk of progression to cirrhosis and hepatocellular carcinoma. Hepatic fibrosis has prognostic value in patients with type 2 diabetes mellitus and MASLD. The aim of this study was to investigate the risk factors for hepatic fibrosis in subjects with type 2 diabetes and MASLD in Brazzaville. **Patients and Methods:** Cross-sectional study and analysis were conducted over 11 months at Brazzaville University Hospital and SUZA Clinic, including patients with type 2 diabetes and MASLD who underwent non-invasive evaluation of liver fibrosis by elastometry or Fibroscan®. **Results:** The study included 120 patients with a median age of 56 years (50.64), with a clear female predominance. The mean duration of diabetes mellitus was 6.1 ± 5 years. Patients were treated with oral antidiabetics (53.3%) and had a mean glycated hemoglobin of **8.75 ± 1.47%**. Liver fibrosis was present in 52 patients (43.3%). Advanced fibrosis was present in 32.6% of cases. Hepatic fibrosis was significantly associated with metabolic syndrome (p = 0.000), severe steatosis (p = 0.012), chronic complications of diabetes mellitus (p = 0.04), dyslipidemia (p = 0.000), and insulin therapy **(p = 0.0013)**. **Conclusion:** Hepatic fibrosis is common in patients with type 2 diabetes melli-

tus and MASLD. Its presence is statistically significantly related to metabolic syndrome, dyslipidemia, severe steatosis, insulin therapy, and chronic complications of diabetes mellitus.

Keywords

Type 2 Diabetes Mellitus, Metabolic Dysfunction-Associated Steatotic Liver Disease, Advanced Fibrosis, Brazzaville

1. Introduction

Type 2 diabetes mellitus (T2DM) is a major metabolic pathology whose prevalence is steadily increasing worldwide, particularly in developing countries [1]. It is often accompanied by metabolic comorbidities, including metabolic dysfunction-associated steatotic liver disease (MASLD), previously termed non-alcoholic fatty liver disease (NAFLD) [2]-[4].

MASLD is a hepatic manifestation of metabolic syndrome. It encompasses a spectrum of liver conditions ranging from simple steatosis to metabolic dysfunction-associated steatohepatitis (MASH, previously non-alcoholic steatohepatitis (NASH)), which can progress to fibrosis, cirrhosis, and even hepatocellular carcinoma. Liver fibrosis is considered the main prognostic determinant in these patients [5] [6].

The presence of MASLD in patients with type 2 diabetes mellitus is an aggravating factor for hepatic and cardiovascular prognosis. Due to its relationship with insulin resistance, type 2 diabetes mellitus is considered a factor in the initiation and progression of MASLD to its severe forms with fibrosis [3] [7].

Screening and assessment of liver fibrosis contribute to reducing morbidity and mortality from liver-related causes in patients with type 2 diabetes mellitus [3] [8]. However, not all patients with type 2 diabetes mellitus and MASLD develop significant fibrosis. It is, therefore, essential to identify the risk factors associated with the development of liver fibrosis in this population in order to better target screening and prevention strategies.

In Brazzaville, local data on the prevalence of liver fibrosis in this metabolic context are limited [9] [10]. The aim of this study is to investigate risk factors for liver fibrosis in patients with type 2 diabetes mellitus and MASLD in Brazzaville.

2. Patients and Methods

This was an analytical cross-sectional study conducted over a period of 11 months in the Department of Metabolic and Endocrine Diseases, in collaboration with the Department of Hepato-Gastro-Enterology of the University Hospital of Brazzaville and the Suza Medical Clinic, which has a Fibroscan.

We included patients at least 18 years of age, with type 2 diabetes mellitus diagnosed at least one year previously, no risky alcohol consumption, no known

liver disease, no steatogenic or hepatotoxic therapies, with hepatic steatosis detected on abdominal ultrasound or Fibroscan, and in whom fibrosis had been identified and quantified by elastometry or Fibroscan. Free and informed consent to participate in the study was required for each participant.

Data were collected using a data collection form, which collated information from the clinical examination, diabetic follow-up, and FibroScan evaluation. Presence of metabolic syndrome was defined in accordance with the guidelines of the International Diabetes Federation (Table 1).

Table 1. Definition of metabolic syndrome [11].

Critera	Definition of Metabolic Syndrome According to IDF/AHA/NHLBI (2009)
	Presence of three of the following five criteria:
Elevated Waist Circumference	≥ 94 cm for men and ≥ 80 cm for women (for people of Sub-Saharan Africa)
Elevated triglycerides	≥150 mg/dL (1.7 mmol/L) or ongoing treatment for hypertriglyceridemia.
Reduced HDL-C (high-density lipoprotein cholesterol)	< 40 mg/dL (1.0 mmol/L) for men; < 50 mg/dL (1.3 mmol/L) for women
Elevated blood pressure	Systolic ≥ 130 and/or diastolic ≥ 85 mm Hg, or ongoing treatment for high blood pressure
Elevated fasting glucose	≥ 100 mg/dL or ongoing treatment for diabetes mellitus

IDF = International Diabetes Federation; AHA = American Heart Association; NHLBI = National Heart, Lung, and Blood Institute.

Interviews with patients included in the study were conducted in French (the official language), Lingala, or Kituba (national languages), depending on the patient's preference.

Non-invasive quantification of hepatic steatosis and fibrosis was performed using an Echosens FibroScan at Suza Clinic, the only center in the city equipped with this imaging technology.

In order to minimize errors and optimize result quality, the FibroScan M probe was used for normal-weight patients, and the XL probe for overweight or obese patients. A total of 10 valid measurements were required for each patient.

Thanks to the CAP (controlled attenuation parameter) function incorporated in the FibroScan device, steatosis was quantified.

Table 2. Interpretation of the results of liver steatosis quantification by elastometry [12].

Liver steatosis quantification	CAP
No steatosis	<215 decibels per meter (dB/m)
mild steatosis	215 - 252 dB/m
moderate steatosis	>252 dB/m
severe steatosis	>296 dB/m

CAP measures the attenuation of the ultrasound signal emitted by elastometry, enabling steatosis to be detected and quantified in decibels (dB). Interpretation of the results of non-invasive quantification of hepatic steatosis is shown in Table 2.

Results of the non-invasive assessment of liver fibrosis were interpreted in accordance with the recommendations for the non-invasive diagnosis and monitoring of chronic liver diseases of the French Association for the Study of the Liver [13].

Data were entered using Microsoft Excel 2016 and then exported to Statistical Package for the Social Sciences (SPSS) IBM version 25 for analysis. Nominal and ordinal categorical variables were expressed as frequencies and percentages, while continuous and discrete numerical variables were expressed as mean ± standard deviation or median with interquartile ranges.

Contingency tables were established to compare qualitative variables.

Qualitative variables were compared using Pearson's chi-square test or Fisher's exact test for small sample sizes. Means and medians were compared using Student's t-test and the Mann-Whitney test, respectively, depending on normality or non-normality.

Contingency tables were created to compare qualitative variables. Comparison of categorical variables was performed using Pearson's chi-square test. Fisher's exact test was used for small sample sizes.

Means and medians were compared using Student's t-test and the Mann-Whitney test, respectively, depending on normality or non-normal distribution.

Univariate analysis consisted of correlating the variable of interest (liver fibrosis) with other explanatory variables to study the strength of the association between the variable of interest and the explanatory variable. Odds ratio (OR) with its 95% confidence interval (CI) was estimated at a significance threshold of < 5%. As the variable of interest was binary, we performed a bivariate logistic regression to eliminate confounding factors.

3. Results

During the study period, we enrolled 120 patients with a median age of 56 years (50, 64). The study population comprised 75 women (62.5%) and 45 men (37.5%), giving a sex ratio of 0.6:1. Patients were employed in the public sector (56%), unemployed (27%), or working in the informal sector (11%). The mean duration of diabetes mellitus was 6.1 ± 5 years. Patients had poor glycemic control, with a mean HbA1C of 8.75 ± 1.47%. The drug therapy followed by patients is shown in Table 3.

Diabetes mellitus was associated with high blood pressure (76.7%), dyslipidemia (51.7%), obesity (35%), and metabolic syndrome (47.1%).

Liver biology revealed cytolysis below 5 times normal in 20 patients (16.7% of cases). The ratio of Alanine Aminotransferase (ALT) to Aspartate Aminotransferase (ASAT) was greater than 1 in 15 cases.

Results of liver steatosis quantification by elastometry using CAP software are

shown in Figure 1.

Table 3. Drug treatment of patients—oral and injectable non-insulin antidiabetics.

Drug treatment	number of patients	%
Non-insulin oral and injectable antidiabetics	64	53.3
exclusive insulin	22	18.3
Non-insulin oral and injectable antidiabetics + insulin	34	28.4
Total	120	100

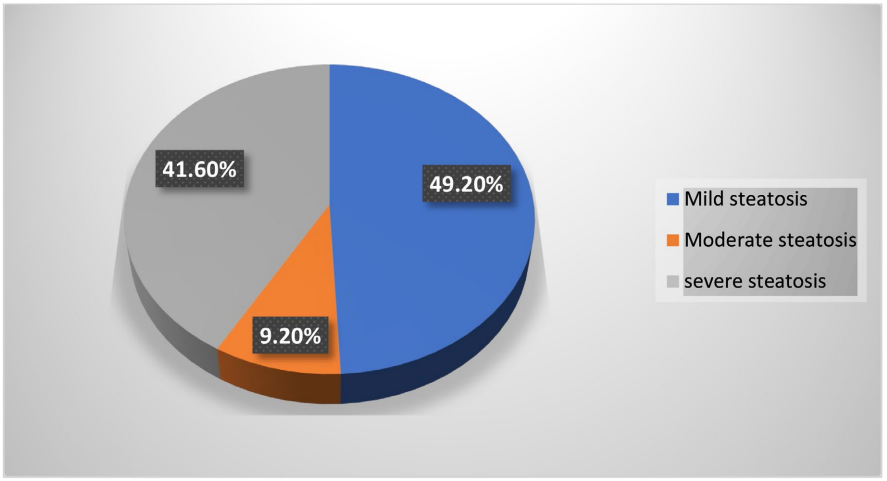

Figure 1. Quantification of hepatic steatosis by FibroScan using the CAP function.

Results of the analysis of risk factors associated with liver fibrosis are shown in Table 3.

Table 4. Risk factors for hepatic fibrosis.

Rick Factor	Liver fibrosis	No liver Fibrosis	p	Odds ratio (OR)	CI 95%
Age > 50 years	41	52	0.75		
Chronic Complications of Diabetes	32	29	0.040	2.15	1.03 - 4.49
Insulin therapy	31	25	0.013	2.54	1.21 - 5.33
High blood pressure	43	49	0.17		
Dyslipidemia	36	25	0.000	4.42	2.00 - 9.74
Chronic kidney failure	26	20	0.02	2.40	1.30 - 5.09
Obesity	22	20	0.15		
Metabolic Syndrome	43	24	0.000	8.76	3.66 - 20.99
Hemoglobin A1C >8%	38	43	0.25		
Severe steatosis	43	42	0.012	2.96	1.24 - 7.05
Abdominal obesity	50	63	0.34		

4. Discussion

Our study shows a high frequency of liver fibrosis in a population of patients with type 2 diabetes mellitus and MASLD, which has clinical and prognostic implications. These findings help to confirm the close and complex relationship between type 2 diabetes mellitus and MASLD [5].

MASLD is a frequently encountered entity in patients with type 2 diabetes mellitus and is a common cause of elevated transaminases in these patients [2]. It is also associated with a substantial increase in cardiometabolic risk in people with type 2 diabetes mellitus [2] [14].

Type 2 diabetes mellitus may be favoured by intrahepatocyte lipid accumulation and, through chronic hyperglycemia and insulin resistance, may predispose to MASLD [3] [7].

Characteristics of the study population and diabetes mellitus-related data:

In our study, patients with type 2 diabetes mellitus and MASLD had a median age of 56 years, with a clear female predominance (62.5%). These results are in agreement with those of several African authors who reported a high frequency of MASLD in women due to nutritional, hormonal, and socio-economic factors [3] [15] [16].

The mean duration of progression of type 2 diabetes mellitus in our study was 6.1 ± 5 years. This reflects a long-standing disease and suggests prolonged exposure to other metabolic disorders, which may contribute to the progression of liver steatosis to a severe form with liver fibrosis.

In a prospective study of the prevalence of MASLD, advanced fibrosis, cirrhosis, and hepatocellular carcinoma in people with type 2 diabetes, Ajmera V *et al.* [17] in the USA reported that the presence and duration of type 2 diabetes mellitus are the main determinants of the progression of steatosis to hepatic fibrosis and the development of hepatocellular carcinoma.

Poor glycemic control, reflected in a mean glycated hemoglobin of 8.6 ± 1.8% observed in our study, reflects uncontrolled diabetes mellitus, further exposing patients to the risk of complications such as liver damage. This also reinforces the hypothesis that chronic hyperglycemia contributes to the progression of MASLD to more severe forms, such as liver fibrosis, by promoting oxidative stress, inflammation, and hepatic lipotoxicity [2] [3].

Our findings are similar to those reported by Halloul *et al.* [18], in Tunisia, who found unbalanced diabetes in 71% of patients with hepatic steatosis, with a mean glycated hemoglobin of 9.22 ± 2.22%. Our results differ, however, from those of Hickman IJ *et al.* [18] in Australia and Mandal A *et al.* [19] in the USA, who found good glycemic control in a population of patients with diabetes mellitus and MASLD, with mean HbA1C of 6.9% and 7 ± 2.4%, respectively.

Frequency and severity of liver fibrosis:

Type 2 diabetes mellitus is also one of the most important risk factors for the rapid progression from MASLD to MASH, advanced fibrosis, or cirrhosis [20] [21].

This finding was confirmed in our study, where hepatic fibrosis was found by elastometry in 43.3% of our patients. This result of our research work reflects the importance of this hepatic complication in the population of patients with diabetes mellitus and MASLD. This result is higher than the figures reported in some European series [22] [23] but is comparable to that reported in several other African studies carried out in a similar context [10] [16].

Of the patients with hepatic fibrosis detected by mechanical pulse elastometry or Fibroscan in our series, 32.6% had advanced hepatic fibrosis. This finding reflects the real risk of MASLD progressing to severe forms. Advanced hepatic fibrosis has prognostic value in patients with type 2 diabetes mellitus. It is associated with an increased risk of progression to cirrhosis and hepatocellular carcinoma, especially in the context of limited surveillance [2] [3] [16].

Risk factors associated with liver fibrosis:

- **Age and duration of diabetes:**

In our study, Type 2 diabetic patients with hepatic fibrosis were not significantly older and did not have a longer duration of progression than those without hepatic fibrosis.

Our results are similar to those of Eloillaf *et al.* [24] in Tunisia, who, like us, noted the absence of any significant relationship between age and hepatic fibrosis. They did, however, note the existence of a significant relationship between the length of diabetes mellitus and significant fibrosis.

Gonzales *et al.* [23], in France, reported that age over 50 years was a risk factor significantly associated with hepatic fibrosis.

- **Metabolic syndrome:**

Metabolic syndrome presents favourable grounds for the development and progression of hepatic steatosis to hepatic fibrosis. With its combination of abdominal obesity, insulin resistance, dyslipidemia, and arterial hypertension, metabolic syndrome contributes to the development of MASLD, a substrate of liver fibrosis. It reflects ongoing activation of hepatic inflammatory and fibrogenic pathways [25] [26].

The statistically significant relationship found in our series between metabolic syndrome and liver fibrosis is in agreement with many authors in the literature [27] [28].

Marty *et al.* [22] in France reported that liver fibrosis in patients with type 2 diabetes mellitus was significantly associated with metabolic syndrome.

The strong relationship between metabolic syndrome and hepatic fibrosis highlights the need for systematic screening for hepatic impairment in patients with type 2 diabetes mellitus and metabolic syndrome.

- **Chronic complications of type 2 diabetes mellitus**

Several scientific studies have reported that MASLD is associated with an increased risk of cardiovascular events in patients with type 2 diabetes mellitus, independent of the presence of other cardiovascular risk factors. Also, the association of diabetes mellitus with NAFLD increases the risk of chronic complications

of type 2 diabetes mellitus [21] [29].

Our study demonstrated a statistically significant association between the presence of chronic diabetic complications and liver fibrosis. This result could be explained by prolonged exposure to hyperglycemia and systemic inflammation, which can affect hepatic microcirculation and promote fibrosis [30] [31].

In an analytical cross-sectional study carried out in the USA, Trivedi HD *et al.* [32] reported that the presence of chronic complications of type 2 diabetes was associated with the degree of liver fibrosis, independently of the level of glycemic control reflected by hemoglobin A1c levels.

This relationship was also studied by the team of Gonzales *et al.* [23] in France, who found a significant association among a history of diabetic foot disease, vascular complications, and liver fibrosis.

- **Dyslipidemia:**

Dyslipidemia is a major risk factor for cardiovascular disease. Its impact appears to be greater when associated with the presence of MASLD [33] [34].

Dyslipidemia, found in 51.7% of cases in our series, was noted as a risk factor significantly associated with hepatic fibrosis.

This finding may be explained by the central role of abnormal lipid metabolism in the development and progression of NAFLD. Indeed, the intrahepatic accumulation of triglycerides, associated with lipotoxicity, may favor the development of inflammatory lesions and promote progression to fibrosis [5] [33] [34].

- **Glycemic control and insulin therapy:**

Literature data show that MASLD, through intrahepatocyte accumulation of lipids, contributes to increased insulin resistance. As a result, insulin doses need to be increased to achieve better glycemic control in insulin-treated type 2 diabetic patients. Moderate weight gain frequently accompanies this therapeutic adaptation [29] [31].

In our study, insulin therapy was a risk factor significantly associated with hepatic fibrosis in patients with MASLD and hepatic fibrosis.

This finding could reflect the existence of poor metabolic control under oral antidiabetic therapy, requiring recourse to insulin therapy. Some scientific studies also show that exogenous insulin administration exacerbates hepatic lipogenesis in the absence of excellent metabolic control. This provides the basis for the development of MASLD, which can progress to liver fibrosis [31] [35].

The level of glycemic control was not statistically significantly associated with the presence of liver fibrosis in our series. This result is in line with the findings of Eloillaf *et al.* [24], who reported, in an analytical study devoted to the study of factors associated with significant fibrosis in patients with type 2 diabetes mellitus, the absence of a statistically significant relationship between the level of glycemic control and liver fibrosis.

5. Conclusions

Liver fibrosis represents a frequent and silent complication of non-alcoholic fatty

liver disease in diabetic patients. This study confirms a high frequency of hepatic fibrosis in type 2 diabetic patients in Brazzaville, with several identified risk factors, including metabolic syndrome, severe steatosis, dyslipidemia, chronic diabetic complications, and insulin therapy.

These results reinforce the importance of early, targeted screening for liver fibrosis in patients with type 2 diabetes mellitus and MASLD, with the aim of preventing progression to severe forms of liver disease. A multidisciplinary approach to the management of patients with type 2 diabetes mellitus and MASLD, incorporating non-invasive assessment of liver fibrosis, is essential.

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Conflicts of Interest

We have no conflict of interest.

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