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Quantitative Assessment of Liver Fibrosis by Elastography in Patients with Chronic Liver Disease: A Cross-Sectional Study in Lomé (Togo)

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Abstract

Aim: To describe the two-dimensional elastographic profile according to the Shearwave (2D-SWE) technique in patients with chronic liver disease in Lomé. **Materials and method:** Cross-sectional, descriptive study conducted over seven month at the Autel d'Elie Clinic in Lomé, from January to August 2022, on adult patients with chronic liver disease who underwent abdominal ultrasound coupled with two-dimensional elastography. **Results:** The sample size was 54 patients. The mean age of the patients was 33 ± 12 years, with extremes of 18 and 66 years. Patients aged 30 years or less accounted for 48.1% (n = 26). All patients (n = 54) had at least one transaminase assay with a mean of 69.3 ± 78.3 IU/l (AST) and 59.3 ± 82.8 IU/l (ALT). There was no statistically significant association between the biological parameters and the presence of fibrosis. Viral liver disease was the main cause, accounting for 81.5% (n = 44) of cases, with no significant association with the degree of fibrosis. Ultrasound revealed a dysmorphic liver (57.4%; n = 31) and portal hypertension (18.5%, n = 10). Fibrosis stages F1, F2 and F4 accounted for (48.1%, n = 26), (24.1%, n = 13) and (13%, n = 7) of cases respectively. Liver dysmorphia was significantly associated with the presence of fibrosis (p = 0.012) and portal hypertension was significantly associated with the degree of fibrosis (p = 0.0063). **Conclusion:** Assessment of liver fibrosis in patients with chronic liver disease using 2D-SWE elastography is essential for patient follow-up.

Keywords

Hepatic Fibrosis, 2D-SWE Elastography, Chronic Liver Disease, Lomé

1. Introduction

Hepatic fibrosis, caused by chronic liver disease, is a major public health issue. It is a key factor in the progression of chronic liver disease and corresponds to the accumulation of normal extracellular matrix constituents in the liver, particularly collagens, which are the main constituents. It results from an imbalance between their synthesis, deposition and degradation [1], and if diagnosed late has the potential to progress to cirrhosis and hepatocellular carcinoma [2]. It is essential to detect the presence and extent of liver fibrosis as early as possible in order to prevent various complications. Until 2008, liver biopsy puncture (LBP) was the gold standard for diagnosing and assessing liver fibrosis [3]. However, LBP has limitations. Apart from its invasive nature and the risk of morbidity associated with biopsy, it is limited to a sample of the liver, ignoring the state of the rest of the parenchyma. Several non-invasive techniques have been developed for diagnosing liver fibrosis, including serum biomarkers for calculating fibrosis scores (Fibrotest R, Fibrometer R and Hepascore) and elastography techniques [4] [5]. Lawson-Ananissoh *et al.* in 2018 [6] conducted a study in which liver fibrosis was assessed in patients with viral hepatitis B using non-invasive methods such as APRI, Fibrosis-4 (FIB-4), fibrotest and fibroscan. Fibroscan has some limitations, in particular its use is limited to measuring hepatic elasticity, without any morphological analysis of the liver, and also obesity, depending on the patient's body mass index (BMI) and the type of probe used [7].

Two-dimensional elastography using the Shearwave technique (2D-SWE) is a new, innovative technique. Several studies around the world and in Africa have already shown very promising results in the assessment of liver fibrosis [7] [8] [9]. Screening for fibrosis in people with chronic liver disease is a major challenge. However, there are few data on ultrasound quantification of liver fibrosis in the African literature. This study aimed to describe the two-dimensional elastographic profile using the Shearwave (2D-SWE) technique for quantifying liver fibrosis in patients with chronic liver disease in Lomé, and more specifically to describe the clinico-biological and ultrasound profile of patients with chronic liver disease, to establish the staging according to the degree of fibrosis on elastography, to determine the aetiologies of chronic liver disease and then to determine the link between the clinico-biological and ultrasound signs and the degree of fibrosis.

2. Materials and Methods

This was a prospective descriptive cross-sectional study conducted over seven

months from 10 January 2022 to 10 August 2022. During this study, 54 patients with chronic liver disease underwent abdominal ultrasound coupled with two-dimensional elastography. The study population consisted of all patients aged 18 or over with chronic liver disease (liver disease that has been evolving for more than 6 months) of various aetiologies. We considered a patient with chronic viral liver disease to be any patient who had been infected with a hepatitis B or C virus for more than 6 months. The criteria for identifying patients with alcohol-related chronic liver disease were clinical-biological: a history of chronic alcohol consumption of more than 12 g/d in women and 24 g/d in men, elevated transaminases, particularly at the expense of ASAT (ASAT/ALAT > 1.5), increased gamma-glutamyltranferases (GGT) or macrocytosis on the blood count [10] [11]. Adult patients aged 18 and over who had given verbal or written consent and were being followed for chronic liver disease were included. Patients with a complication (hepatocellular carcinoma HCC), patients with a liver transplant and patients for whom elastography was difficult to perform (difficult apnoea) were not included.

For data collection, the medical records of the patients selected for the study were analysed based on a pre-established form. For each patient, the following parameters were studied: socio-demographic characteristics (age, sex), clinical-biological data (history, clinical presentation, biological data), ultrasound data (B-mode ultrasound and Doppler) and measurement of liver hardness using the STE method (elastography).

The morphological ultrasound examination and measurement of liver hardness were carried out on patients who had been fasting for at least 06 hours and were lying supine using a Mindray® "Resona 7" ultrasound scanner. This device uses the "Shear Wave 2D" technique with the "Sound Touch Elastography (STE)" module, which is a module integrated into the ultrasound scanner with exclusive "Ultrasound wide beam tracking Technology". The examination consisted of performing a B-mode morphological ultrasound scan with colour and pulsed Doppler study for each patient, followed by STE measurement of liver elasticity using a 5 MHz "SC5-1 U" curved probe. A 13 MHz linear probe was used to analyse liver contours.

In a supine patient, we considered low-grade ascites to be the accumulation of fluid in the Douglas's dead end; medium-grade ascites to be the presence of fluid around the liver, in the pelvis, in the paracolic gutters and Morison's recess; and high-grade ascites to be cases where the small intestines took on a polycyclic appearance, floating vertically in the fluid [12]. The diameter of the portal vein was considered normal when it was less than 13 mm and pathological when it was greater than 13 mm [13]. We considered any circulatory velocity in the portal vein ranging from 11 to 23 cm/s to be normal, and any mean portal velocity of less than 10 cm/s to be pathological, allowing us to confirm the existence of portal hypertension [14].

The protocol for carrying out the examination followed the steps below: B mode optimisation by positioning at 8 cm depth (**Figure 1**); the transducer

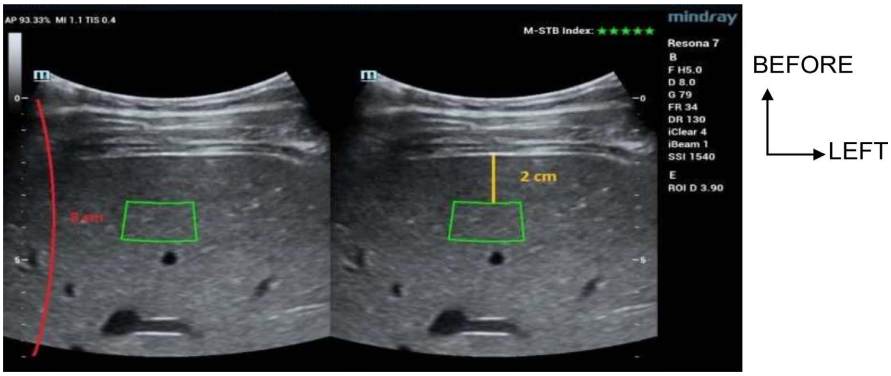


Figure 1. Inter-costal axial section with B-mode optimisation and distance of the ROI box from the liver capsule according to 2D-SWE.

perpendicular to the liver capsule; the upper edge of the SWE™ elastography "region of interest box" (ROI box) was placed 2 cm from the liver capsule (**Figure 1**); the size of the "ROI box" was between 2 and 3 cm wide and 1 to 2 cm deep (an area of liver parenchyma free of large vessels, focal lesions and distant from the gallbladder was chosen).

After positioning the ROI Box, the patient was asked to maintain apnoea for a few seconds. Capture quality criteria were defined as follows: a 5-star Green Stars Motion Stability Index (**Figure 2**); a homogeneous green Reliability Map: RLB, indicating the quality of the shear waves for improved reproducibility in measuring elasticity (**Figure 2**); a Reliability Index (RLB index) > 90% (**Figure 2**). The 10 mm circular ROI was placed in the ROI box and several measurements per acquisition were taken (**Figure 3**).

According to the European Federation Societies for Ultrasound in Medicine and Biology (EFSUMB) [15], three measurements are sufficient for a reliable examination. However, we opted for 10 measurements to be as close as possible to the elastometry protocol.

The liver elasticity measurements were presented in the form of a ratio within which the most important quality factor for judging the reliability of the test was found. This factor is the ratio of the inter-quartile range to the median, which must be less than or equal to 30% (**Figure 4**).

Data were entered using CsPro version 7.1 software and statistical analyses were performed using the Statistical Package for Social Sciences (SPSS version 23). Variables were significant when p-value < 0.05. We then performed multivariate logistic regression to identify measures of association with a 95% confidence interval. Every precaution was taken to protect the confidentiality of personal information concerning the identifiable data involved in the research.

3. Results

The sample size was 54 patients.

The average age of patients was 33 ± 21, with a range of 18 and 66. Patients aged 30 and under accounted for 48%, 35% between 30 and 50 years and 17% were over 50 old.

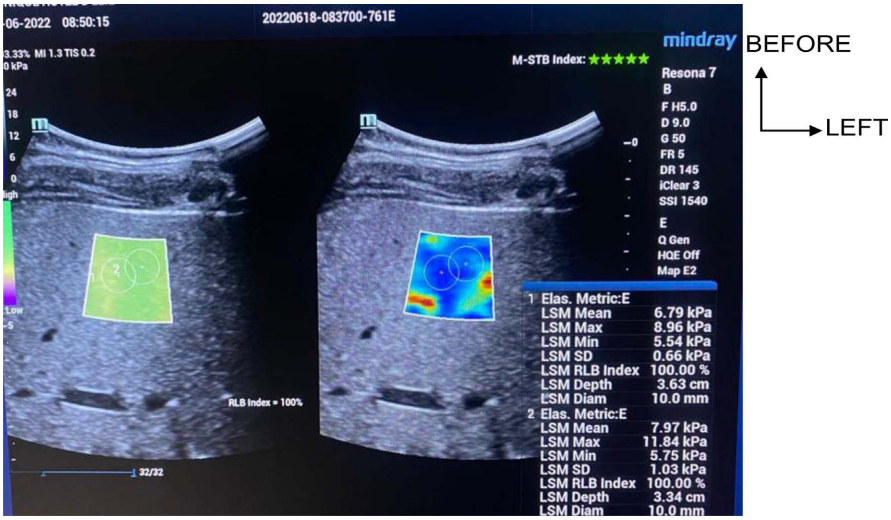


Figure 2. Axial inter-costal section showing the quality criteria for positioning the ROI Box according to 2D-SWE.

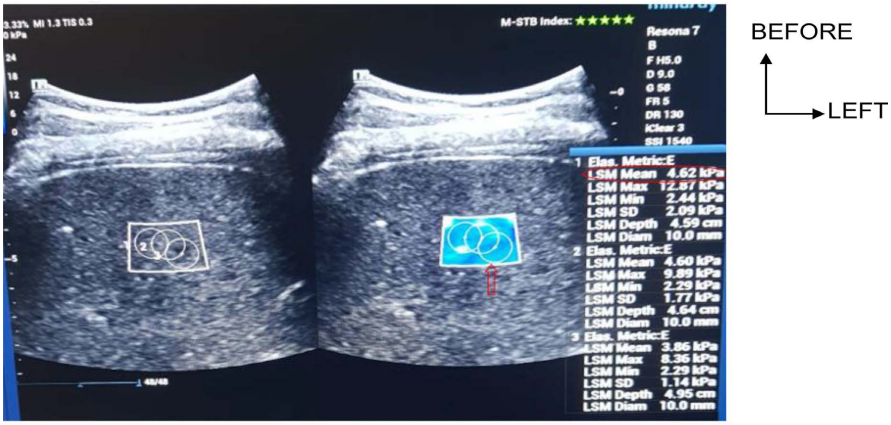
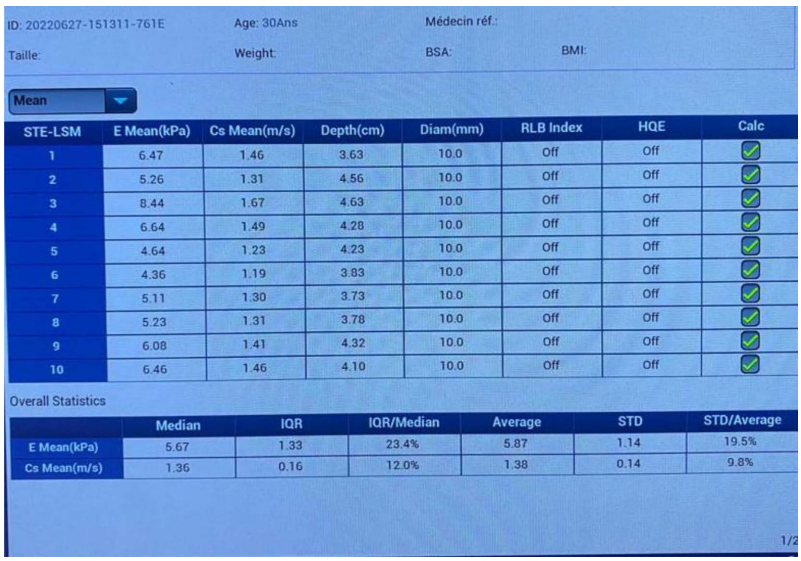


Figure 3. Inter-costal axial section with 2D-SWE measurement of liver elasticity.



ID: 20220627-151311-761E Age: 30Ans Médecin réf.:

Taille: Weight: BSA: BMI:

Mean

STE-LSM	E Mean(kPa)	Cs Mean(m/s)	Depth(cm)	Diam(mm)	RLB Index	HQE	Calc
1	6.47	1.46	3.63	10.0	Off	Off	✓
2	5.26	1.31	4.56	10.0	Off	Off	✓
3	8.44	1.67	4.63	10.0	Off	Off	✓
4	6.64	1.49	4.28	10.0	Off	Off	✓
5	4.64	1.23	4.23	10.0	Off	Off	✓
6	4.36	1.19	3.83	10.0	Off	Off	✓
7	5.11	1.30	3.73	10.0	Off	Off	✓
8	5.23	1.31	3.78	10.0	Off	Off	✓
9	6.08	1.41	4.32	10.0	Off	Off	✓
10	6.46	1.46	4.10	10.0	Off	Off	✓

Overall Statistics

	Median	IQR	IQR/Median	Average	STD	STD/Average
E Mean(kPa)	5.67	1.33	23.4%	5.87	1.14	19.5%
Cs Mean(m/s)	1.36	0.16	12.0%	1.38	0.14	9.8%

1/2

Figure 4. Final report of 2D-SWE liver elasticity measurements.

Men accounted for 51.9% (n = 28) versus 26 women (48.1%), with a sex ratio of 1.1. Table 1 shows all the characteristics of patients.

Patients were asymptomatic in 85.1% (n = 46) of cases (Table 2).

From a biological point of view, all patients (n = 54) had at least completed a transaminase assay (ASAT, ALAT) with an average of 69.3 ± 78.3 IU/l for ALAT and 59.3 ± 82.8 IU/l for ASAT. The results of the liver enzyme tests are shown in Table 3.

Forty-four patients (81.5%) were hepatitis B virus carriers with a mean viremia of 14072058.7 ± 69364860.9 copies/ml.

Sonographically, hepatic dysmorphia accounted for 57.4%, portal hypertension for 23% and hepatic steatosis for 7%. B-mode ultrasound data are shown in Table 4.

Hepatic fibrosis classified as F1 accounted for 48.1% and fibrosis classified as F2 was observed in 24.1% of cases (Figure 5).

The aetiological profile of chronic liver disease was marked by 81.5% (n = 44) of cases of viral origin, 14.8% (n = 8) of alcoholic origin and 3.7% (n = 2) of other causes.

There was no significant association between the different aetiologies found and the degree of fibrosis (Table 5).

Disruption of laboratory parameters was more pronounced in patients with advanced liver fibrosis, however, this difference was not statistically significant with $p > 0.05$ for all parameters. So there was no statistically significant association between the biological parameters and the development of fibrosis (Table 6).

Analysis of signs such as splenomegaly and ascites showed no significant association with the presence or absence of fibrosis.

The rate of patients with hepatic dysmorphia increased with advanced liver fibrosis while the rate of patients without hepatic dysmorphia gradually decreased in the more advanced stages of fibrosis. The presence of hepatic dysmorphia was statistically significantly associated with the presence of hepatic fibrosis ($p = 0.012$) (Table 7). The rate of patients with portal Hertension increased with advanced liver fibrosis while the rate of patients without portal hypertension gradually decreased in the more advanced stages of fibrosis. The presence of portal hypertension was statistically significantly associated with an increase in the degree of fibrosis ($p = 0.0063$) (Table 7).

4. Discussion

The study was single-center in that we only had one ultrasound machine with the 2D-SWE elastography for the entire country. This is the reason for the small sample size. Similarly, very few patients had the minimum laboratory profile. The lack of comparison to a baseline examination was also a limitation of the study. However, these results are informative about the quantification of liver fibrosis.

Table 1. Sociodemographic characteristics of patients.

	Value
Sample size	54
Age (Year)	
Average	33 ± 21
Range	18 - 66
<30	26/54
30 - 50	19/54
>60	09/54
Sexe	
Men	28/54
Women	26/54
Sex ratio	1.1

Table 2. Distribution of patients by clinic.

	Complement (n = 54)	Proportion (%)
Ascites	3	5.6
Splenomegaly	2	3.7
Jaundice	2	3.7
Collateral venous circulation	1	1.9
No clinical signs	46	85.1
Total	54	100

Table 3. Liver enzyme results.

	ASAT	ALAT	GGT	PAL
Average	69.3	59.3	132.7	261.8
Standard deviation	78.3	82.8	323.0	733.1
Minimum	15.0	8.0	12.0	28.0
Maximum	329.0	564.0	1616.0	4660.0

Normal values: ASAT & ALAT (10 - 50 IU/l); GGT (10 - 71 IU/l); PAL (30 - 150 IU/l).

Table 4. Distribution of patients according to ultrasound data.

Liver	**Complement**	**Proportion (%)**
Echogenicity of the liver in relation to the renal cortex (n = 54)		
Identical	51	94.4
More echogenic	3	5.6
Homogeneity of liver parenchyma (n = 54)		
Homogenous	44	81.5

Continued

Micronodular	6	11.1
Macronodular	4	7.4
Liver dysmorphia (n = 54)		
No	23	42.6
Yes	31	57.4
Atrophy of segment IV (n = 31)	16	51.6
Hypertrophy of segment I (n = 31)	7	22.6
Hypertrophy of the left liver (n = 31)	15	48.4
Portal hypertension (n = 54)		
Portal trunk size (mm)		
<13 (normal)	44	81.5
>13	10	18.5
Flow reversal	1	1.9
Average V < 11 cm/s (standard between 11 cm/s and 50 cm/s)	1	1.9
Ascites (n = 5)	5	9.2
Minimal abundance	2	40
Average abundance	2	40
Great abundance	1	20
Splenomegaly	6	11.1
Porto-systemic shunts (n = 4)	4	7.4
anterior (umbilical vein)	2	50
posterior (spleen + kidney)	1	25
Upper (œsophagus + stomach)	1	25

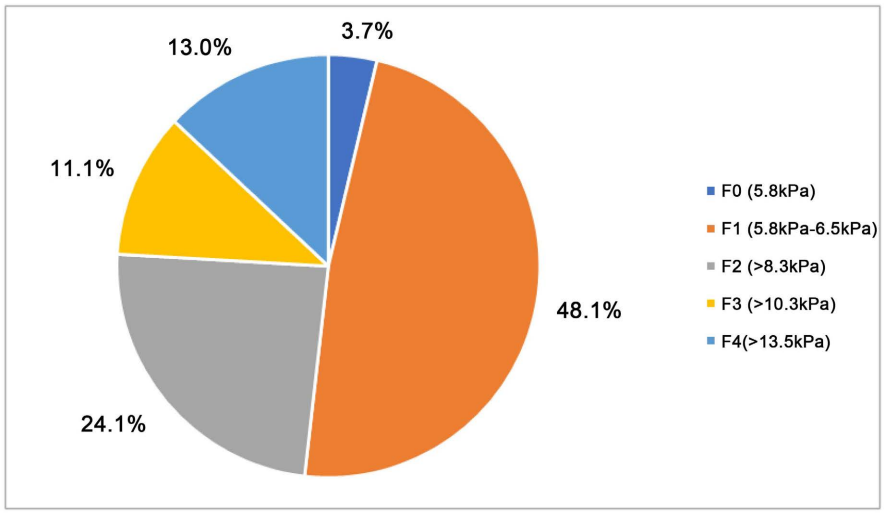


Figure 5. Distribution of patients according to degree of liver hardness by ETS.

Table 5. Association between aetiologies and degree of fibrosis.

Etiology	Degree of fibrosis					p-value
	F0 - F1 (28)	F2 (13)	F3 (6)	F4 (7)	Total (54)	
	n(%)	n(%)	n(%)	n(%)	n(%)	
						0.447
Viral	24 (85.7)	11 (84.6)	6 (100)	3 (42.9)	44 (81.5)	
Alcoholic	2 (7.1)	2 (15.4)	0 (0)	4 (57.1)	8 (14.8)	
Other	2 (7.1)	0 (0)	0 (0)	0 (0)	2 (3.7)	
Total	**28 (100)**	**13 (100)**	**6 (100)**	**7 (100)**	**54 (100)**	

Table 6. Association between biological parameters and degree of fibrosis.

Biology	Degree of fibrosis					p-value
	F0 - F1 (28)	F2 (13)	F3 (6)	F4 (7)	Total (54)	
	Avg* + Sd**	Avg + Sd	Avg + Sd	Avg + Sd	Avg + Sd	
ASAT	44.7 + 59.5	59.9 + 66.6	109 + 103.6	146.1 + 91.3	69.3 + 78.3	0.55
ALAT	33.1 + 22.3	99.8 + 159.3	72.5 + 45.3	79.3 + 29.9	59.3 + 82.8	0.87
GGT	39.1 + 44.2	42.9 + 25.3	133.5 + 201.2	569 + 670.5	132.7 + 323	1.69
PAL	119.5 + 82.8	142.3 + 183.3	1012.6 + 2039.1	270 + 130.6	261.8 + 733.1	0.11
BT	57.9 + 73.7	44.3 + 34.9	42.5 + 46	-	48.2 + 42.5	0.63
BC	-	35 + 39.7	-	-	25.6 + 32.4	1.05
TP	0.7 + 0.2	0.8 + 0.1	-	17.1 + 28.8	3.6 + 12	0.099
INR	1.5 + 1.3	1.2 + 0.1	-	1.6 + 0.3	1.4 + 0.9	0.58
Inserts	252000 + 67001.9	154600 + 67626.2	-	88000 + 73539.1	199882.4 + 86496.3	0.6

*Mean, **Standard deviation, GGT (Gamma-Glutamyltrasferase), PAL (Alkaline phosphatase), BT (Total bilirubin), BC (Conjugated bilirubin), PT (Prothrombin rate), INR (International normalised ratio).

Table 7. Association between ultrasound results and fibrosis.

Conclusion (n = 54)	Degree of fibrosis					p-value
	F0 - F1 (28)	F2 (13)	F3 (6)	F4 (7)	Total (54)	
	n(%)	n(%)	n(%)	n(%)	n(%)	
Liver dysmorphia						**0.012**
No	19 (67.9)	2 (15.4)	1 (16.6)	1 (14.2)	23 (42.6)	
Yes	9 (32.1)	11 (84.6)	5 (83.4)	6 (85.8)	31 (57.4)	
Portal hypertension						**0.0063**
No	28 (100)	10 (76.9)	3 (50)	3 (42.8)	44 (81.5)	
Yes	0 (0)	3 (23.1)	3 (50)	4 (57.1)	10 (18.5)	
Steatosis						0.82
No	27 (96.4)	13 (100)	5 (83.3)	6 (85.8)	51 (94.5)	
Yes	1 (3.6)	0 (0)	1 (16.6)	1 (14.2)	3 (5.5)	

In the study, the population was predominantly male, 28 men to 26 women, with a sex ratio of 1.08. Sehonou *et al.* [16] also reported a male predominance with a sex ratio of 2.25 in their 2010 series in Cotonou. The mean age was 33 years, with a median of 30 years and extremes of 18 and 66 years. The most common age group was under 30 (48.1%). Lawson-Ananisso *et al.* [6] reported 33.74 +/− 10.39 years and the most common age range was 30 to 40 years in their study. In contrast, earlier in 2010, Sehonou *et al.* [16] reported an average age of 49. The young age of the maximum number of patients in the Lomé studies could be explained by the high prevalence of viral hepatitis B among young people in Lomé [17].

The majority (85.1%, n = 46) of patients in the study were asymptomatic. Ascites was observed in 5.6% (n = 3) of cases, splenomegaly in 3.7% (n = 2), jaundice in 3.7% (n = 2) of cases and only 1.9% (n = 1) of patients had collateral venous circulation. Lawson-Ananissoh *et al.* [6] reported 4 cases (1.3%) of jaundice, while Sehonou *et al.* [16] reported 75% ascites and 71% jaundice.

The French National Authority for Health recommends that patients with chronic liver disease should have a minimum biological work-up [18]. In this study, all patients had at least been able to have a transaminase assay (ASAT, ALAT) with a mean of 69.3 ± 78.3 IU/l for ALAT and 59.3 ± 82.8 IU/l for ALAT. These levels are higher than those reported by Xiaoyu *et al.* [19] in 2020, with a mean level of 36 IU/l for AST with extremes of 25 and 47, 32 IU/l for ALT with extremes of 20.5 and 49, and 172.7 ± 71.1.

In this study, the majority (n = 26) had minimal hepatic fibrosis classified as F1 (n = 28). However, Xiaoyu *et al.* [19] in 2020 reported significant hepatic fibrosis in 130 of 161 cases of chronic hepatopathy linked to the hepatitis B virus, distributed as follows: Significant fibrosis F2 (n = 46), severe fibrosis F3 (n = 20) and cirrhosis (F4; n = 64). This difference in the degree of liver damage could be explained by the variable aetiologies of the liver diseases in our series. The different aetiologies of our patients were viral in 81.5% (n = 44), 60% of whom had viral hepatitis B (n = 30); alcohol-related in 14.8% (n = 8); and other in 3.7% (n = 2). However, there was no significant association between these aetiologies and the degree of fibrosis. A predominance of B viral aetiology in chronic liver disease was found, as was Sehonou *et al.* in 2010 [16] who reported 52 patients hospitalised for cirrhosis whose aetiologies were represented by B viral infection (30.8%, n = 16), C viral infection (5.8%, n = 3) and alcohol (13.3%, n = 7).

In the present study, 31 patients (57.4%) had hepatic dysmorphia and 10 patients (18.5%) had portal hypertension. There was a statistically significant association between the presence of hepatic dysmorphia (p = 0.012) and portal hypertension (p = 0.0063) and the degree of fibrosis, in particular the degree of fibrosis was higher in patients with hepatic dysmorphia. In early cirrhosis, the liver tends to be hypertrophied as a result of inflammation and oedema. Subsequently, parenchymal rarefaction develops, which is unevenly distributed. Atrophy predominates in segment IV and the right posterior segments. At the same time, the segments of the left lobe and especially segment I hypertrophy.

The mechanism of these size changes is thought to be due to changes in portal flow and therefore to the heterogeneous distribution of nutrients and trophic elements supplied by portal flow of splanchnic origin. However, these mechanisms remain incompletely understood [20].

Hepatic fibrosis is one of the factors influencing the prognosis of non-alcohol-related hepatic steatosis. In the study, only 3 patients (5.5%) had hepatic steatosis. We did not observe a statistically significant association between the presence of hepatic steatosis and the degree of fibrosis. This result is different from that of Jie Liu *et al.* [21] in 2022 where the median of liver hardness measurement was 8.8 kPa and 9.2 kPa in patients with moderate to severe steatosis, which was significantly higher than in patients with non-mild steatosis ($p < 0.05$). In their study, the presence of steatosis was an independent predictor of the degree of liver hardness ($p < 0.001$).

These results are superimposed on those of Xiaoyu Xie *et al.* [19] in 2020 who reported higher liver hardness values in patients with significant steatosis ($S \geq 10\%$) than their counterparts in fibrosis stages F0-2 (6.82 ± 1.57 vs 7.92 ± 1.99; $p = 0.010$) and F0-3 (7.18 ± 1.84 vs 8.25 ± 1.91; $p = 0.007$). In their study, false positive rates (FPR) in the diagnosis of advanced fibrosis (16.00% vs 37.04%, $p = 0.037$) were higher in patients with significant steatosis.

In 2022, Kumada *et al.* [22] and many other authors in the literature agreed that the presence of moderate to severe steatosis, detected by histology or other means, should be taken into account to avoid overestimating the measurement of liver hardness and thus improve its accuracy [23] [24] [25]. According to these authors, the use of 2D-SWE to measure liver elasticity overestimates the stage of fibrosis in patients with chronic liver disease and >10% hepatic steatosis.

5. Conclusion

This study shows that chronic liver disease is more prevalent in young males in Lomé, who are generally asymptomatic. Although asymptomatic, the biology of these subjects is marked by a disturbance in liver enzymes, and the aetiology is dominated by infection with the hepatitis B virus. However, these patients suffering from chronic liver disease are rarely investigated by elastography in Lomé. Morphologically, chronic liver disease in this series is often recognised on ultrasound by the heterogeneous appearance of the liver structure, liver dysmorphia and portal hypertension. Measurement of liver hardness by two-dimensional elastography is generally mild (F1). The degree of this hepatic hardness is not related to its aetiology or to liver enzyme disorders but, on the contrary, it is related to hepatic dysmorphia and increases with the presence of portal hypertension. These results underline the fact that the assessment of liver fibrosis in patients with chronic liver disease using this technique is essential for patient follow-up.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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