SAS Journal of Medicine
Abbreviated Key Title: SAS J Med
ISSN 2454-5112
Journal homepage: https://saspublishers.com

OPEN ACCESS

Hepato-Gastro-Enterology

The Role of Transient Elastography in the Non-Invasive Assessment of Portal Hypertension in Patients with Advanced Chronic Liver Disease

S. Zahraoui[1*], Y. Aroudam[1], M. Salihoun[1], F. Bouhamou[1], I. Serraj[1], M. Acharki[1], N. Kabbaj[1]

[1]Hepato-Gastro-Enterology Department "EFD-HGE", Ibn Sina University Hospital, Mohammed V University

DOI: https://doi.org/10.36347/sasjm.2025.v11i06.002 | **Received:** 21.04.2025 | **Accepted:** 26.05.2025 | **Published:** 03.06.2025

***Corresponding author:** S. Zahraoui
Hepato-Gastro-Enterology Department "EFD-HGE", Ibn Sina University Hospital, Mohammed V University

Abstract **Original Research Article**

Introduction: Portal hypertension (PH) is an inevitable consequence of cirrhosis. The Gold standard method for assessing PH are hepatic venous pressure gradient measurement and upper GI endoscopy. However, these methods are invasive, expensive and not widely available. Hepatic ultrasound elastography is a non-invasive alternative for assessing hepatic stiffness. The aim of the present study is to assess the correlation between elasticity values and the presence and severity of PH. ***Materials and Methods:*** This is a retrospective descriptive and analytical study carried out between 2020 and 2024, in the Gastroenterology department EFD-HGE of the CHU-IBN SINA in Rabat, including patients with chronic liver disease who had undergone liver stifness measurement (LSM) by FibroScan®, concluding to a severe fibrosis (F3-F4). All patients were evaluated clinically, biologically, radiologically and endoscopically for markers of PH. ***Results:*** 72 patients were included. The mean age was 55.6 years, with no gender predominance (sex ratio=1.05). The main etiology was chronic viral hepatitis C (33% of cases). Mean hepatic elasticity was 29.9kPa. Esophagogastroduodenoscopy EGD revealed esophageal varices (EVs) in 80.5% of cases. Mean elasticity was higher in the "presence of VO" group than in the "absence of VO" group, with a clinically significant difference. Similarly, mean elasticity was higher in the "EV grade ≥ II" group than in the "EV grade <II" group, with a clinically significant difference. The ROC curve was studied for the diagnosis of large EV (grade ≥ II). The area under the curve was 0.819. A threshold value of 20.5kPa was proposed for the diagnosis of large EVs, with a sensitivity of 82.6% and a specificity of 80.7%. ***Conclusion:*** These results indicate that FibroScan® is a reliable, non-invasive method that can be used to screen for and diagnose clinically significant PH.

Keywords: Portal hypertension (PH), Liver stiffness measurement (LSM), FibroScan®, Esophageal varices (EVs), Non-invasive assessment.

INTRODUCTION

Advanced liver fibrosis and cirrhosis, currently grouped under the term of "advanced chronic liver disease" (ACLD), are major causes of morbidity and mortality worldwide. PH is defined as an increase in the venous pressure gradient between the portal vein and the hepatic veins above the normal level (1-5 mm Hg). Clinically significant portal hypertension (CSPH) is defined by a hepatic venous pressure gradient (HVPG) ≥10 mm Hg and is independently associated with the development of severe complications, including ascites, gastroesophageal variceal bleeding, hepatic encephalopathy, hepato-renal syndrome and spontaneous bacterial peritonitis [1]. Gold standard methods for the assessment of PH and its complications include hepatic venous pressure gradient (HVPG) measurement and esophagogastroduodenoscopy (EGD); however, these methods are invasive, costly and not available in all centers. This is why non-invasive alternatives have been the subject of extensive research over the past 20 years. Controlled vibration transient elastography (FibroScan®), a new non-invasive technique that has emerged in recent years, is used to measure liver stiffness and, in the context of ACLD, to identify the presence of PH, its severity and the risk of complications. Our study aims to assess the correlation between liver elasticity values in patients with ACLD and markers of PH, particularly the presence of esophageal varices (EVs) on EGD, as well as the correlation between elasticity values and EVs grade. The secondary objective of our study is to determine an elasticity value that can predict CSPH represented by the presence of large VOs.

MATERIALS AND METHODS

This is a retrospective descriptive and analytical study conducted between January 2020 and October 2024, at the Gastroenterology department EFD-HGE of the IBN SINA University Hospital in Rabat, including patients with chronic liver disease of various etiologies who had liver stifness measurement (LSM) by FibroScan® concluding to a severe fibrosis (F3-F4). All patients were evaluated clinically, biologically, radiologically and endoscopically for markers of PH. Patients without complete lab tests, abdominal imaging or upper gastrointestinal endoscopy were excluded. Data were collected from patient files. They mainly included the following elements: gender, age, BMI, pathological history, etiology of liver disease, abdominal imaging data, biological work-up (platelets, albumin, prothrombin time, AST, ALT, bilirubin), EGD data, and elasticity and CAP values.

The data collected was entered into an Excel file. Statistical analysis was then carried out using JAMOVI 2.2.5 software and the Mann-Whitney statistical test.

RESULTS

Of a total of 680 patients who had LSM by FibroScan® in our department during the study period, 161 patients had high elasticity, indicating severe fibrosis (F3) or cirrhosis (F4), i.e. a prevalence of 23.7%. Only 72 patients (10.6%) were finally included in the study, after exclusion of incomplete files, particularly patients for whom the results of endoscopic exploration were not available. Average age was 55.6 years, with extremes ranging from 24 to 85 years. There was no gender predominance: 37 men (51.4%) and 35 women (48.6%), with a M/F sex ratio of 1.05 (Tables 1). The main etiology in our patients was chronic viral hepatitis C, present in 33% of cases (table 1). Comorbidities were present in 51.4% of our patients, dominated by arterial hypertension in 27.7% of cases, followed by diabetes in 20.8% of cases, and haemopathies (sickle cell disease, thalassaemia, multiple myeloma) in 9.72% of cases.

Table 1: Epidemiological data

Total number of patients who had LSM		690
Number of patients with severe fibrosis (%)		161 (23,7%)
Number of patients included in the study (%)		72 (10,6%)
Average age		55,6 years
Number of males		37 (51,4%)
Number of females		35 (48,6%)
Sex Ratio M/F		1,05
Etiologies	Viral hepatitis C (VHC)	24 (33%)
	Uknown	20 (27,7%)
	Viral hepatitis B (VHB)	11 (15,27%)
	Primary biliary cholangitis	4 (5,55%)
	Auto-immune hepatitis	3 (4,16%)
	Vascular hepatic disease	3 (4,16%)
	Primary sclerosing cholangitis	2 (2,77%)
	Metabolic dysfunction associated steatohepatitis	1 (1,38%)
	Alcohol related liver cirrhosis	1 (1,38%)
	Coinfection VHB and VHC	1 (1,38%)
	Caroli disease	1 (1,38%)

The clinical examination revealed a palpable splenomegaly in 31 cases (43%), abdominal collateral venous circulation in 26 cases (36.11%), sharp lower liver edge in 20 cases (27.7%), jaundice in 3 cases (4.16%), spider angiomas in 3 cases (4.16%). Clinical ascites was not present in any of our patients. It is worth noting that 12 patients (16.66%) were already on diuretics for well-controlled ascitic decompensation.

Biological parameter values were studied and expressed as a mean with quartiles at 25% (Q1) and 75% (Q3) of our sample. The data are summarized in the table 2.

All our patients underwent abdominal imaging: abdominal ultrasound coupled with Doppler or abdominal CT or abdominal MRI. Abdominal imaging abnormalities are summarized in the figure 1.

Elasticity results are expressed as a median with quartiles at 25% (Q1) and 75% (Q3) of our sample. They are summarized in the table 3. Based on the FibroScan® interpretation guide by Echosens [2], 4 patients (5.55%) were classified as F3, and 68 patients (94.44%) were classified as F4 (Figure 2).

All our patients underwent EGD under sedation to look for endoscopic signs of PH, prior to FibroScan®. EGD results are summarized in figure 3.

Table 2: Biological data of our serie

	Mean value (Q1-Q3)	Minimal value	Maximal value	Inter-quartile range
AST (UI/L)	60 (32-66)	16	136	34
ALT UI/L)	50,6 (25-51)	12	145	26
Bilirubin t mg/l	15,9 (8-17)	4	115	9
Albumin g/l	36,9 (35-40)	16	46	5
Platelets count $x10^3$/mm3	130 (63-161)	20	509	98
Prothrombin time	66,82% (56%-77%)	30%	100%	21%

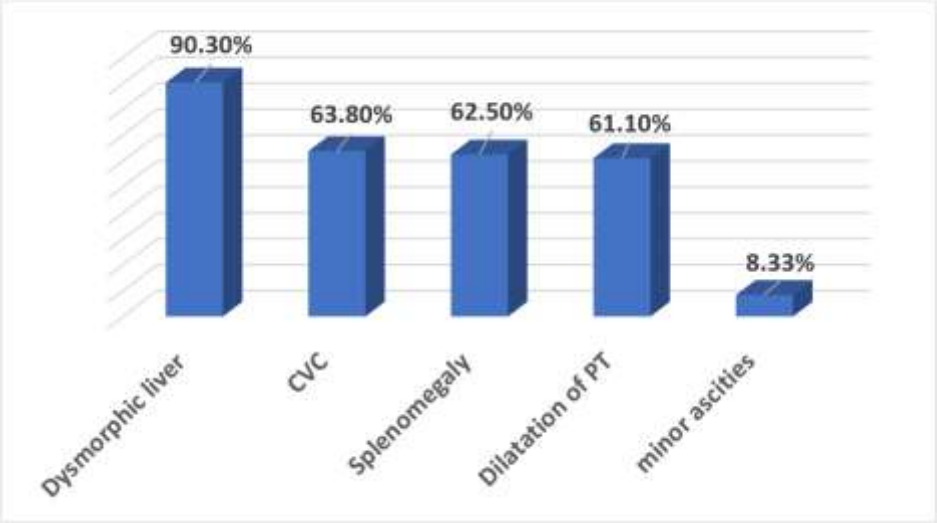

Figure 1: Abdominal imaging data

CVC: collateral venous circulation; PT: Portale trunk

Table 3: Elasticity results

Mean	Minimal	Maximal	Q1	Q3	Inter-quartile range
29,9kPa	11kPa	75kPa	18kPa	35kPa	17kPa

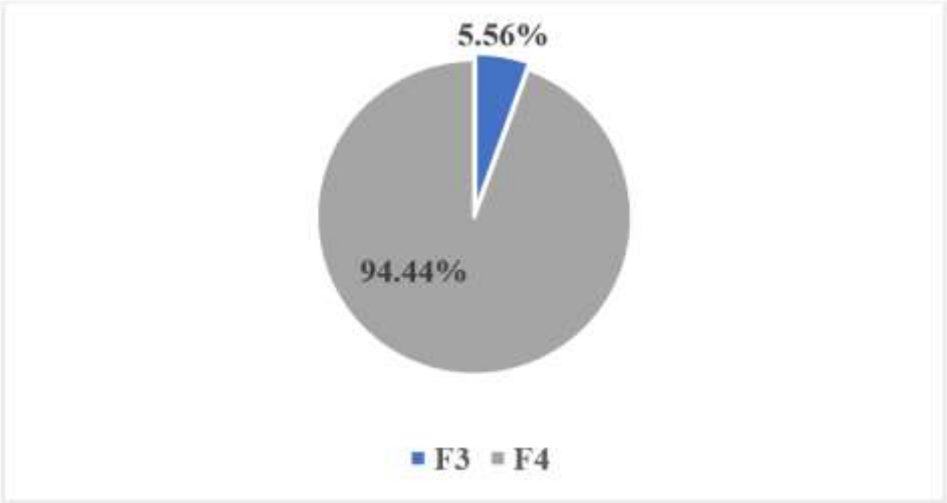

Figure 2: Elasticity values interpretation

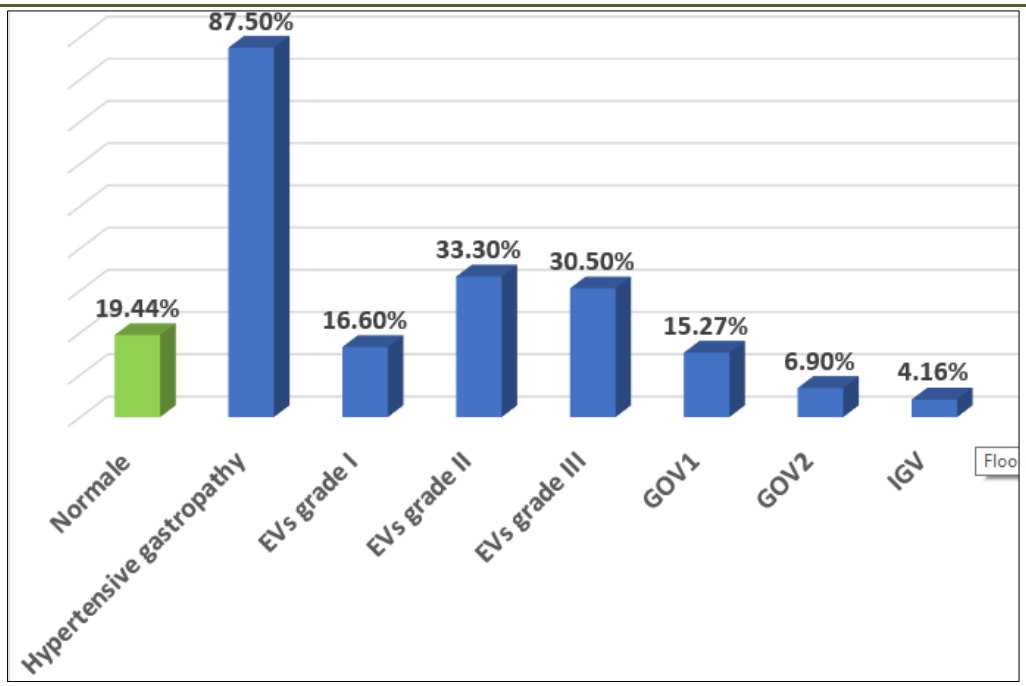

Figure 3: EGD results

EVs: Esophagal varices; GOV: Gastro-esophagal varices, IGV: intragastric varices

Elasticity values and presence of EVs:

The comparison of elasticity values between the "Absence of EVs" and "Presence of EVs" groups shows that the mean elasticity in the "Presence of EVs" group is higher than in the "Absence of EVs" group (32.9 kPa and 17.9 kPa, respectively), with a clinically significant difference ($p<0.001$) (Table 4).

Table 4: Comparison of elasticity values between "No EVs" and "Presence of EVs" groups

Elasticity (kPa)						
Groupe	**Number**	**Mean**	**Median**	**Standard deviation**	**Standard error**	***p***
Absence of EVs	14	17.9	18.4	3.06	0.819	
Presence de VO	58	32.9	28.1	18.0	2.37	**<0.001**

Elasticity values and grade of EVs:

Comparison of elasticity values between the "EVs grade < II" or "small EVs" group and the "EVs grade ≥ II" or "large EVs" group shows a higher mean elasticity in the "large EVs" group compared with the "small EVs" group (36.1 KPa and 19 KPa respectively) with a clinically significant difference between the 2 groups ($p<0.001$).

Table 5: Comparison of elasticity values between the "Small EVs" and "Large EVs" groups

Elasticity (Kpa)						
Groupe	**Number**	**Mean**	**Median**	**Standard deviation**	**Standard error**	***p***
Small EVs	26	19.0	18.4	4.50	0.883	
Large EVs	46	36.1	32.6	18.7	2.76	**< 0.001**

Elasticity and diagnosis of large EVs:

The ROC curve was studied for the diagnosis of large EVs "Grade ≥ II" by transient elastography (Figure 4). The area under the curve (AUC) was O.819.

Based on the ROC curve, different threshold values of hepatic elasticity were studied in order to propose a threshold that would maximize the sensitivity and specificity of transient elastography for the diagnosis of large EVs. This threshold value was 20.5 Kpa, with a sensitivity of 82.6% and a specificity of 80.7%.

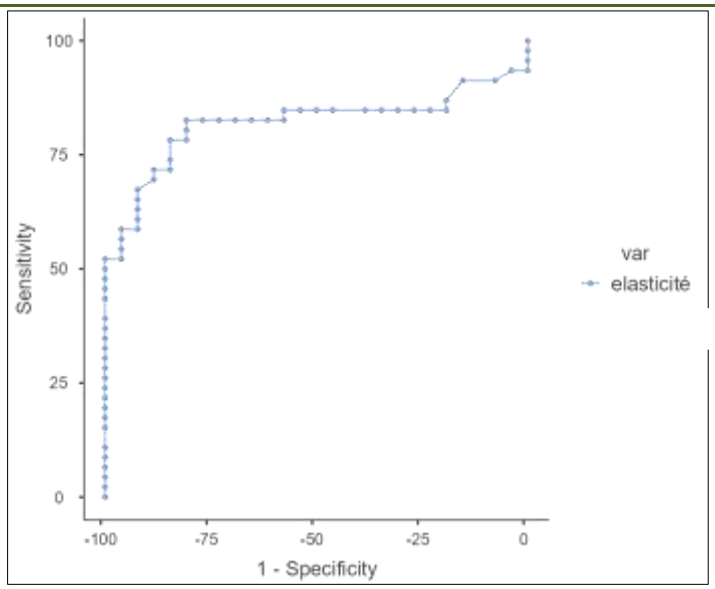

Figure 4: The ROC curve

DISCUSSION

Portal hypertension (PH) is a common clinical syndrome. It is hemodynamically defined by an increase in the venous pressure gradient across the liver, calculated from its entry through the portal vein to its exit through the hepatic veins [3]. Increased resistance to portal blood flow is the initial factor leading to increased portal pressure. This resistance can be located anywhere in the hepatic circulation, i.e. pre-hepatically, intra-hepatically or post-hepatically. In the Western world, around 90% of PH cases are due to advanced chronic liver disease (ACLD), which causes structural damage through fibrogenesis, parenchymal extinction and regeneration. PH then develops at the intrahepatic sinusoidal site [3]. The detection for PH is important because of its complications, which include upper GI hemorrhage due to ruptured gastroesophageal varices, ascites, spontaneous bacterial peritonitis, hepatorenal syndrome and hepatic encephalopathy [4]. The gold standard method for the detection of intra-hepatic portal hypertension is the hepatic venous pressure gradient measurement (HVPG). This method measures sinusoidal pressure levels by calculating the difference between wedjed and free hepatic venous pressure [3,5]. An HVPG of up to 5 mmHg is considered normal; Subclinical PH is defined by an HVPG of 6–9 mmHg; and an HVPGP of 10 mmHg represents the CSPH threshold [5]. When HVPG reaches 10 mmHg or above, PH can become symptomatic as patients can develop gastroesophageal varices and hyperdynamic circulation, increasing their risk of clinical decompensation. Therefore, patients with compensated ACLD should be screened for the presence of CSPH [6]. EGD is the best method to determine the presence of oesophageal and gastric varices, and allows the identification of additional signs used to stratify bleeding risk (size of varices; presence of red colour signs and wale marks) [7].

HVPG measurement and endoscopy are the backbone for the assessment of PH in ACLD. However, they are invasive and may lead to complications; in addition, a specialised clinical setting and specific expertise are required to carry out these tests, limiting their availability [8].

An important advance in the non-invasive assessment of PH has been the introduction of non-invasive LSM by transient elastography. In patients with ACLD, fibrosis is the major component of increased intrahepatic vascular resistance leading to PH. Therefore, LSM has been studied as a possible surrogate for PH [8]. The first study to assess the relationship between LSM and HVPG was published in 2006. It demonstrated a direct association between LSM (cut-off value 8.7 kPa) and the diagnosis of PH (defined as HVPG ≥6mmHG) in patients with post-viral C cirrhosis after liver transplantation, with an AUROC of 0.93 [9]. Several meta-analyses have confirmed the ability of LSM to diagnose CSPH. The most recent includes 26 studies involving a total of 4337 patients who underwent LSM transient elastography and HVPG measurement [10]. In this work, the AUROC was 0.91. The optimal threshold for the diagnosis of CSPH was 22.8 kPa, giving a sensitivity of 79% and a specificity of 88%.

The diagnostic accuracy of elastography in predicting the presence and size of EVs has been the subject of over 50 studies. LSM values are higher in patients with EVs and tend to be higher in patients with large EVs [8]. The French study by Kazeni *et al*, carried out in 2006 and including 172 patients with ACLD of various etiologies, was among the first to assess the contribution of FibroScan® in predicting the presence of EVs [11]. It showed a mean elasticity of 32.4kPa in the "Presence of EVs" group, compared with an elasticity of 18.2kPa in the "Absence of EVs" group, which is consistent with our results that found a mean elasticity of

32.9kPa in the "Presence of EVs" group compared with 17.9kPa in the "Absence of EVs" group with a clinically significant difference between the 2 groups (p<0.001). Another Egyptian study [12] assessing this correlation in patients with hepatitis C cirrhosis found a mean elasticity of 49.9kPa in the "presence of EVs" group versus a mean elasticity of 27kPa in the "absence of EVs" group, with a clinically significant difference between the 2 groups (p=0.001).

The study of Kazemi *et al* [11] showed that the value of elasticity increased with EVs size. Indeed, the mean elasticity in the "EVs grade < II" group was 21.7kPa, while in the "EVs grade ≥ II" group it was 43.2kPa, with a clinically significant difference between the 2 groups (p<0.001). The study of Saad *et al* [12] found similar results, with a mean elasticity of 38.4kPa in the "EVs grade < II" group versus a mean elasticity of 60.4kPa in the "EVs grade ≥ II" group, and a clinically significant difference (p=0.002). In our study, comparison of elasticity values between the "EVs grade < II" group and the "EVs grade ≥ II" group shows a higher mean elasticity in the 2nd group compared with the 1st group (36.1 kPa and 19kPa respectively) with a clinically significant difference between the 2 groups (p<0.001), which is in perfect agreement with the results of the two cited studies.

Several studies have investigated the diagnostic accuracy of hepatic elasticity in predicting the presence of large varices "EVs grade ≥ II". The table 6 summarizes the results of some of these studies, specifying the AUROC, sensitivity and specificity of the various proposed cut-offs. In our study, the AUC for the diagnosis of CSPH (the presence of large EVs) was 0.819. The optimal cut-off was 20.5 kPa for this purpose, with a sensitivity of 82,61% and specificity of 80,77%. Our results were similar to those reported in the literature, and the sensitivity of the threshold could be optimized if combined with other criteria such as platelet count. Based on these data, we can say that FibroScan® is discriminative for the detection of large EV (AUC = 0.819).

Table 6: Results of studies evaluating the performance of transient elastography for the presence of large esophageal varices

The study	Cut-off (kPa)	AUROC	Sensibility (%)	Specificity (%)
Kazemi *et al*, 2006 [11]	19	0,83	91	60
Bureau *et al*, 2008 [13]	29,3	0,762	81	61
Castéra *et al*, 2009 [14]	30,5	0,87	77	85
Wang *et al*, 2012 [15]	21	0,865	77	87
Bintintan *et al*, 2015 [16]	28,8	0,9	88	82
Hu *et al*., 2015 [17]	25,55	0,855	84	73
Omar *et al*, 2023 [18]	40,9	-	93	52
Our study, 2024	20,5	0.819	82.61	80,77

CONCLUSION

In summary, these results indicate that transient elastography FibroScan® is a reliable, non-invasive method to assess portal hypertension and can be used for the screening and diagnosis of clinically significant portal hypertension.

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