Section: Radiodiagnosis

Original Research Article

ROLE OF TWO-DIMENSIONAL SHEAR WAVE ELASTOGRAPHY IN THE ASSESSMENT OF LIVER FIBROSIS IN CHRONIC LIVER DISEASE WITH SERUM FIBROSIS INDICES CORRELATION

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Abstract

Background: The aim is to evaluate the role of two-dimensional shear wave elastography (SWE) in the assessment of liver fibrosis in chronic liver disease. **Materials and Methods:** This prospective study was conducted in the Department of Radiodiagnosis, B. R. D. Medical College, Gorakhpur, Uttar Pradesh, India, from June 2023 to May 2024. Institutional ethics committee approval was obtained a priori. Informed consent was taken from the recruited patients. The study prospectively enrolled 120 patients who fulfilled with certain criteria. **Result:** Evaluated the role of teo-dimensional shear wave in assessment of liver fibrosis in chronic liver disease and noted. **Conclusion:** Integrating SWE with biochemical markers, careful use of diagnostic cut-off points, and regular patient monitoring are crucial strategies for improving the assessment and management of liver fibrosis in chronic liver disease. If implemented effectively, these recommendations can lead to more accurate diagnoses, better patient outcomes, and more efficient use of healthcare resources.

INTRODUCTION

Liver fibrosis is a major public health concern and is an essential factor in the prognosis of chronic liver disease (CLD). CLD can have various causes, including alcoholic, infective (like chronic viral hepatitis, including hepatitis B & C virus), metabolic, autoimmune, toxic, genetic, and cholestatic factors. Regardless of the cause, the accumulation of extracellular matrix (ECM) leads to liver fibrosis, affecting the liver's structure and function and potentially progressing to cirrhosis and hepatocellular carcinoma (HCC).

Liver fibrosis is a progressive and complex disease that may be prevented from developing into end-stage decompensated cirrhosis with early therapeutic intervention, provided it is correctly identified and staged.[1] The goal of early liver fibrosis detection, precise staging, and reevaluation is to prevent the disease from progressing.

Currently, liver biopsy is the gold standard for liver fibrosis diagnosis and staging. Only 1/50000 portion of the liver parenchyma is evaluated by liver biopsies. It is invasive, causing pain, discomfort, and a risk of bleeding; prone to intra- and inter-observer variability (sample error); and has a small but noticeable complication rate.[2] Over the past several years, there has been an increase in the validation of non-invasive methods for the diagnosis and staging of liver fibrosis due to the invasive nature of liver biopsies. While a laboratory test known as the Alkaline Aminotransferase Platelet Ratio Index (APRI) has been shown to have some use.[3] Shear wave elastography (SWE) has the potential to be favored over transient elastography (TE) since the latter cannot simultaneously conduct a conventional ultrasound while the former relies on vibration to produce shear waves. However, TE has been verified in several investigations.[4] Shear wave elastography is being used more often for liver fibrosis diagnosis and staging. Since recurrent measurements may be

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taken in individuals with chronic, progressing liver disorders, the non-invasive approach is useful. It is susceptible to intra- and inter-observer variability. In patients with a high BMI, inaccurate results might be seen. Confounding variables including edema, inflammation, cholestasis, and congestion are a very real pitfall. All of these need to be considered in their context, and the findings must be interpreted using a multidisciplinary therapeutic approach.[5]

MATERIALS AND METHODS

This prospective study was conducted in the Department of Radiodiagnosis, B. R. D. Medical College, Gorakhpur, Uttar Pradesh, India, from June 2023 to May 2024. Institutional ethics committee approval was obtained a priori. Informed consent was taken from the recruited patients. The study prospectively enrolled 120 patients who fulfilled the following criteria: (1) they were at least 18 years old, both male and female; (2) they had a confirmed case of cirrhosis based on histopathology; (3) they were diagnosed with chronic liver disease (viral hepatitis B or C, sonographic features of chronic liver disease; or (4) they were suspected to have chronic liver disease due to risk factors such as alcohol consumption or abnormal liver function tests.

Exclusion Criteria -

1. Uncooperative patients/patients unable to hold their breath.
2. Lactating and pregnant females whatever the gestational age.
3. Patients with focal liver lesions.
4. Patients with gross/tense ascites.

RESULTS

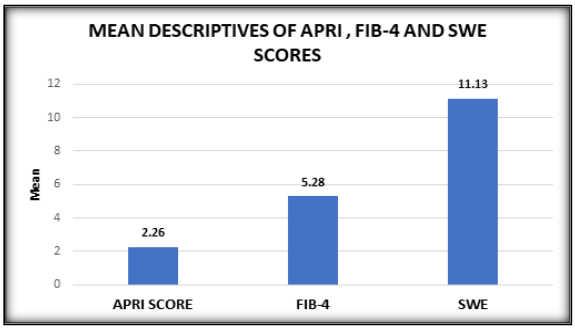

Figure 1: Mean descriptives of APRI, FIB-4 and SWE scores

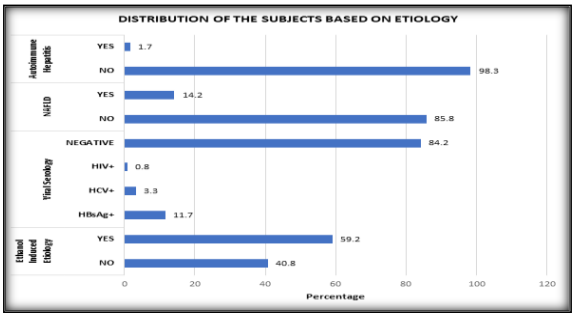

Figure 2: Distribution of subjects based on etiology

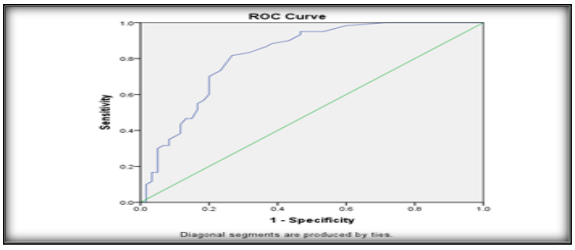

Figure 3: ROC curve to predict the severity of fibrosis with APRI scores

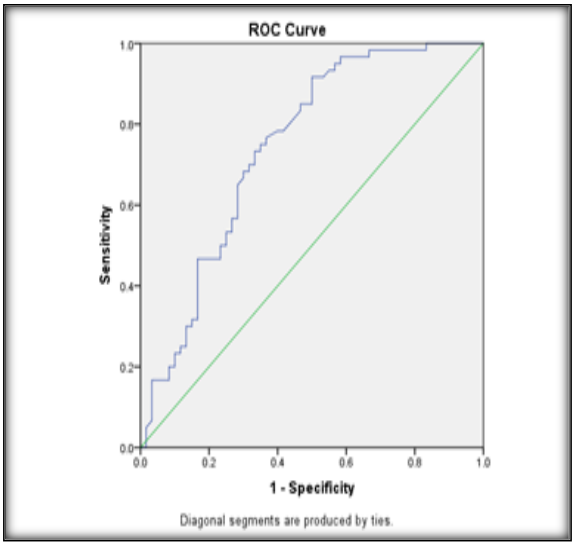

Figure 4: ROC curve to predict the severity of fibrosis with FIB 4 scores

Table 1: mean age distribution of the subjects.

	N	Minimum	Maximum	Mean	S. D
AGE	120	18	81	48.10	14.26

Inference: The mean age of patients was 48.10 ± 14.26 years.

Table 2: distribution of the subjects based on age groups

Age Groups	Frequency	Percent
18 to 30 yrs	16	13.3
31 to 40 yrs	25	20.8
41 to 50 yrs	25	20.8
51 to 60 yrs	33	27.5
61 to 70 yrs	15	12.5

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71 to 81 yrs	6	5.0
Total	120	100.0

Inference:16 patients (13.3%) belonged to the age group of 18 to 30 years, 25 patients (20.8%) belonged to the age group of 31 to 40 years, 25 patients (20.8%) belonged to the age group of 41 to 50 years, 33 patients (27.5%) belonged to the age group of 51 to 60 years, 15 patients (12.5%) belonged to the age group of 61 to 70 years and 6 patients (5%) belonged to the age group of 71 to 81 years.

Table 3: distribution of the subjects based on gender

Gender	Frequency	Percent
Females	29	24.2
Males	91	75.8
Total	120	100.0

Table 4: mean descriptives

	N	Minimum	Maximum	Mean	S. D
S. BILIRUBIN	120	.16	39.47	5.70	7.50
S. ALBUMIN	120	1.8	4.9	2.79	0.53
INR	120	1.00	4.10	1.61	0.47
AST (IU/L)	120	15.0	390.0	109.99	70.30
ALT (IU/L)	120	11.0	238.0	73.65	48.32

Inference: The mean serum albumin was 2.79 ± 0.53, mean INR was 1.61 ± 0.47, mean AST was 109.99 ± 70.30, mean ALT was 73.65 ± 48.32.

Table 5: Mean Descriptives of APRI Score and Fib-4

	N	Minimum	Maximum	Mean	S. D
APRI SCORE	120	.4	10.2	2.26	1.64
FIB-4	120	.51	17.02	5.28	3.07

Inference: The mean APRI score was 2.26 ± 1.64 and mean FIB-4 was 5.28 ± 3.07.
[Table 5] revealed that in a cohort of 120 subjects, the APRI Score was found to have a mean of 2.26 with a standard deviation of 1.64, indicating considerable variability among the participants. Similarly, the FIB-4 index, with a mean of 5.28 and a standard deviation of 3.07, reflects the variability in this measure across the group. These metrics underscore the reliability of the findings, suggesting that the variations in APRI and FIB-4 scores are meaningful and could be indicative of underlying clinical conditions that warrant further exploration.

Table 6: distribution of the subjects based on etiology

		Frequency	Percent
Ethanol Induced Etiology	NO	49	40.8
	YES	71	59.2
Viral Serology	HBsAg+	14	11.7
	HCV+	4	3.3
	HIV+	1	0.8
	NEGATIVE	101	84.2
NAFLD	NO	103	85.8
	YES	17	14.2
Autoimmune Hepatitis	NO	118	98.3
	YES	2	1.7

Table 7: mean scores of fibrosis stage

	N	Minimum	Maximum	Mean	S. D
Fibrosis Stage	120	3.4	22.4	11.13	4.29

Inference: The mean fibrosis stage score was 11.13 ± 4.29.

Table 8: distribution of the subjects based on fibrosis stage

Fibrosis Stage	Frequency	Percent
No Fibrosis(F0)	21	17.5
Normal / Mild (F0-F1)	39	32.5
Mild-Moderate(F2-F3)	32	26.7
Moderate-Severe (F3-F4)	28	23.3
Total	120	100.0

Inference: 21 patients (17.5%) had F0 stage, 39 patients (32.5%) had normal/mild (F0-F1) stage, 32 patients (26.7%) had mild-moderate stage (F2-F3) and 28 patients (23.3%) had moderate-severe (F3-F4) stage.

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Table 9: ROC curve to predict the severity of fibrosis with APRI scores

Area Under the Curve					
Test Result Variable	**Area**	**Std. Error**	**p value**	**Asymptotic 95% Confidence Interval**	
				Lower Bound	**Upper Bound**
APRI Score	.820	.039	.001*	.743	.896

*significant

Inference: The area under the curve for APRI Score is 0.820 and is statistically significant (p=0.001). The best cut off to predict severity of fibrosis would be 1.75 with 81.7% sensitivity and 73.3 % specificity.

Table 10: ROC curve to predict the severity of fibrosis with FIB 4 scores

Area Under the Curve					
Test Result Variable	**Area**	**Std. Error**	**p value**	**Asymptotic 95% Confidence Interval**	
				Lower Bound	**Upper Bound**
FIB 4 Score	.744	.045	.001*	.655	.833

*significant

Inference: The area under the curve for FIB 4 Score is 0.744 and is statistically significant (p=0.001). The best cut off to predict severity of fibrosis would be 3.82 with 80% sensitivity and 56.7% specificity.

Table 11: association of apri score and fibrosis stage

APRI SCORE		**Fibrosis stage**			**Total**
		F0-F1	**F2-F3**	**F3-F4**	
<1.0	Count	11	1	0	12
	%	28.2%	3.1%	0.0%	12.1%
1 to 1.49	Count	11	5	1	17
	%	28.2%	15.6%	3.6%	17.2%
≥1.5	Count	17	26	27	70
	%	43.6%	81.3%	96.4%	70.7%
Total	Count	39	32	28	99
	%	100.0%	100.0%	100.0%	100.0%
Chi-square value- 26.85					
p value- 0.001*					

*significant

Inference: 12 patients (12.1%) had a APRI score of 0 to 0.9, of which. 28.2% had F0-F1 stage and 3.1% had F2-F3 stage. 17 patients (17.2%) had a APRI score of 1 to 1.49, of which. 28.2% had F0-F1 stage, 15.6% had F2-F3 stage and 3.6% had F3-F4 stage. 70 patients (70.7%) had a APRI score of >1.5, of which, 43.6% had F0-F1 stage, 81.3% had F2-F3 stage and 96.4% had F3-F4 stage. The association of APRI score and fibrosis stage was statistically significant. (p=0.001)

Table 12: association of fib 4 score and fibrosis stage

FIB 4		**Fibrosis stage**			**Total**
		F0-F1	**F2-F3**	**F3-F4**	
< 1.45	Count	3	0	0	3
	%	7.7%	0.0%	0.0%	3.0%
1.45 to 3.25	Count	11	3	0	14
	%	28.2%	9.4%	0.0%	14.1%
>3.25	Count	25	29	28	82
	%	64.1%	90.6%	100.0%	82.8%
Total	Count	39	32	28	99
	%	100.0%	100.0%	100.0%	100.0%
Chi-square value- 17.42					
p value- 0.002*					

*significant

Inference: 3 patients (3%) had a FIB 4 score of <1.45, all of which belonged to F0-F1 stage. 14 patients (14.1%) had a FIB 4 score of 1.45 to 3.25, of which, 11 patients (28.2%) belonged to F0-F1 stage, 3 patients (9.4%) belonged to F2-F3 stage. 82 patients (82.8%) had a FIB 4 score of >3.25, of which, 25 (64.1%) belonged to F0-F1 stage, 29 patients (90.6%) belonged to F2-F3 stage and 28 patients belonged to F3-F4 stage. The association of FIB 4 score and fibrosis stage was statistically significant. (p=0.002).

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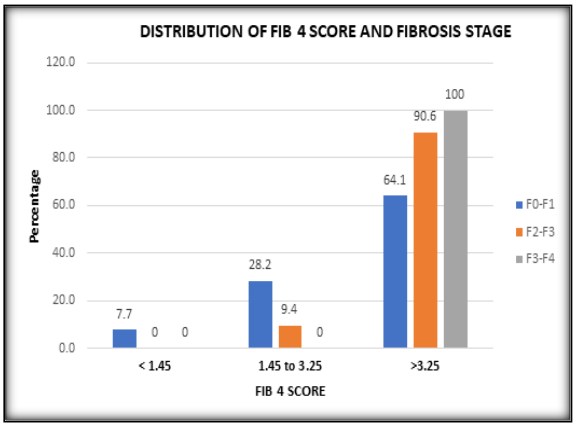

Figure 5: Association of FIB-4 scores and fibrosis stage

DISCUSSION

The prognosis and treatment of chronic liver disease are contingent upon the degree of fibrosis.6 The gold standard for diagnosing liver fibrosis is a liver biopsy. The development of noninvasive technologies for estimating liver fibrosis, such as serum fibrosis indexes and SWE, has led to a decline in liver biopsies in recent years. Liver fibrosis is assessed noninvasively using serum fibrosis indices, such as APRI and FIB-4.[7]

Age and gender: Aging is linked to several alterations in liver cells, including hepatic sinusoidal endothelial cells, as well as a progressive modification of the structure and function of the liver.8 In the present study, the mean age of patients was 48.10 ± 14.26 years. Most patients (27.5%) belonged to the age group of 51 to 60 years. There were 29 females (24.2%) and 91 males (75.8%). Samir AE et al.'s results, which included patients with a mean age of 47 years ± 13.02 and a male preponderance, were consistent with our findings.[8,9] Bellamkonda S. et al. reported that the patients had a male predominance and an average age of 46.9 years.[10]

Laboratory investigations: Evaluation of the risk of cirrhosis and its treatment both depend on the diagnosis and stage of liver fibrosis. According to Park DW et al, the average bilirubin was 0.8 (0.6–1.4), the average AST was 68 (36–191), the average ALT was 62 (38–232), and the average platelet count was 216 (159–256).16 In the present study, the mean serum bilirubin was 5.70 ± 7.50, mean serum albumin was 2.79 ± 0.53, mean INR was 1.61 ± 0.47, mean AST was 109.99 ± 70.30, mean ALT was 73.65 ± 48.32.

In contrast to liver biopsy, APRI SCORE, and FIB-4 scores are the two noninvasive techniques that may accurately detect advanced fibrosis and cirrhosis in CHC patients.[11] The research found that the average APRI score was 2.26 ± 1.64 and the average FIB-4 score was 5.28 ± 3.07. APRI Score has an area under the curve of 0.820, which is statistically significant (p=0.001). 1.75 would be the optimal cut-off point with 81.7% sensitivity and 73.3% specificity for predicting the degree of fibrosis. For the FIB 4 score, the area under the curve is 0.744, which is statistically significant (p=0.001). 3.82 would be the ideal cut-off point with 80% sensitivity and 56.7% specificity for predicting the degree of fibrosis. These findings were consistent with those of another research conducted by Rungta S et al., which found that the FIB-4 score was 2.26 and the median APRI score was 1.10 with 95% CI (0.09–27.86).[12] APRI may be an effective method for fibrosis assessment without a liver biopsy since the literature-validated cut-offs for substantial fibrosis were 0.5 with 77%–86% sensitivity and 49%–65% specificity and 1.5 with 32%–47% sensitivity and 89%–94% specificity. In 2003, Wai et al. created the APRI formula, which employed AST and platelet counts to assess liver fibrosis.[13]

Ethanol-induced etiology: Alcohol is a hepatotoxin that damages the liver by inducing oxidative stress and an inflammatory response via ethanol metabolism.22 71 individuals (59.2%) had an ethanol-induced etiology.

Viral serology: Four patients (3.3%) were HCV+, one patient (0.8%) was HIV+, and fourteen patients (11.7%) were HBsAg+ in the current research. Similarly, hepatitis B was shown to be the most common viral infection in another research conducted by Sande JA et al., affecting 30 patients (23.4%). The next highest percentage of patients had hepatitis C (13.2%) and HIV (18.1%) respectively. Hepatitis C has the most gradual and long-term clinical course, which is probably why it has greater levels of quantifiable liver fibrosis measured.[14]The mean SWE measurement in the current research was 11.53 ± 4.35 in patients with an ethanol-induced etiology, 13.48 ± 3.94 in HBsAg+ patients, 11.63 ± 2.39 in HCV+ patients, 12.30 ± 0.00 in HIV+ patients, 11.69 ± 4.21 in NAFLD patients, and 13.25 ± 3.46 in patients with autoimmune hepatitis.

NAFLD: NAFLD describes liver steatosis when it occurs without the presence of recognized lipid-accumulating factors in hepatocytes, such as alcoholism or steatogenic medication usage.[15] Non-alcoholic fatty liver disease was detected in 17 individuals (14.2%) in the current investigation.

Autoimmune hepatitis: The interaction of a genetic predisposition, an environmental trigger, and a malfunctioning native immune system is assumed to be the process for the development of autoimmune hepatitis, leading to persistent inflammation of hepatocytes and consequent fibrosis of the liver.16 Two individuals (1.7%) in the current research had autoimmune hepatitis.

USG findings: Often, an ultrasound is the first imaging test ordered when evaluating a patient for liver disease. According to USG results, there were nodular hepatic surfaces in 25 patients (20.8%), course echotextures in 80 patients (66.7%), abnormal portal flow and diameter in 21 patients (17.5%), hepatic steatosis in 57 patients (47.5%), mild ascites in 33 patients (27.5%), mild-moderate ascites in 8 patients (6.7%), and moderate ascites in 2 patients (1.7%). Mean SWE measurements were as follows:

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9.89 ± 3.43 for patients with nodular hepatic surface; 12.67 ± 4.08 for course echotexture; 16.50 ± 3.18 for patients with abnormal portal flow and diameter; 10.34 ± 4.29 for patients with hepatic steatosis; 11.44 ± 4.44 for mild ascites; 17.19 ± 3.74 for mild-moderate ascites; and 18.00 ± 3.39 for patients with moderate ascites.

In primary care clinics, patients with an NFS < -1.455 may be safely followed up on. Trends in their fibrosis score should be monitored over time to see if they are progressing or stabilizing. The mean fibrosis stage score in the current research was 11.13 ± 4.29.

Fibrosis stage: Sixty-three (49.2%) and 61 (47.7%) of the patients in a Sande et al. research had a steatosis score of 0 and a histological fibrosis score of 0. Among the patients, 81 (63.3%) belonged to the F0-1 subgroup of the histological fibrosis score, whereas 47 (36.7%) were in the F2-F4 category.95 In the current research, the F0 stage was shown by 21 patients (17.5%), the normal/mild (F0-F1) stage by 39 patients (32.5%), the mild-moderate (F2-F3) stage by 32 patients (26.7%), and the moderate-severe (F3-F4) stage by 28 patients (23.3%).

This study's broader disease burden provides a more consistent picture of the pathophysiology of chronic liver fibrosis. In settings with restricted resources, the complementing usage of APRI score is very essential.

CONCLUSION

The number of liver biopsies conducted has decreased as a result of the increased use of SWE methods for the staging of fibrosis in patients with widespread liver disease. SWE approaches are quite beneficial for evaluating the clinical outcome of CLD patients, either by itself or in conjunction with other criteria in scores or algorithms. As the primary determinant in determining the course, length, and follow-up plan for a chronic hepatitis C infection, liver cirrhosis, the test should be able to distinguish between the greatest number of cirrhotic (F4) and considerable (F3) fibrosis cases from normal or early stages of fibrosis (F1 and F2). Good performance was also shown in identifying patients without liver fibrosis using the APRI and FIB-4 scores. FIB-4 has a cut-off of 3.82, 80% sensitivity, and 56.7% specificity, making it a dependable test for differentiating severe fibrosis and determining future therapy.

With 81.7% sensitivity and 73.3% specificity, ARFI may also be routinely used to assess the level of liver fibrosis for alcoholic liver disease, non-alcoholic fatty liver disease, and chronic hepatitis B and C. More extensive research is necessary because screening the population at risk is important due to the high frequency of CLD.

Based on the data analysis and correlation matrices, several recommendations can be made to enhance clinical practice and patient outcomes.

1. Integration of Shear Wave Elastography with Biochemical Markers: Given the moderate correlations between non-invasive biomarkers such as APRI, FIB-4, and the fibrosis score, it is recommended that these markers be used in conjunction with Shear Wave Elastography (SWE) rather than in isolation. While APRI and FIB-4 provide valuable insights into liver fibrosis, they capture different aspects of the disease. By combining these with SWE, which offers a direct and reliable measurement of liver stiffness, clinicians can achieve a more comprehensive and accurate assessment of fibrosis severity. This integrative approach should be adopted as a standard practice in hepatology clinics, particularly in settings where liver biopsy is not feasible.

2. Use of Multi-Marker Strategies in Resource-Limited Settings: In regions with limited access to advanced imaging technologies like SWE, APRI and FIB-4 scores can serve as effective, low-cost alternatives for fibrosis assessment. However, it is crucial to recognize their limitations, particularly regarding specificity and potential false positives. Clinicians in these settings should be trained to interpret these scores cautiously, considering other clinical parameters and possibly integrating them with other non-invasive tests such as ultrasound to improve diagnostic accuracy. This recommendation is particularly relevant in low-resource settings where liver biopsy and SWE are unavailable.

3. Regular Monitoring and Re-Evaluation: Given the progressive nature of chronic liver disease and the potential for rapid changes in fibrosis status, regular monitoring and re-evaluation of patients are essential. For patients with mild fibrosis or at-risk populations, routine assessments using SWE and biochemical markers can help detect early changes in fibrosis, allowing for timely intervention. This proactive approach can help prevent fibrosis progression to more severe stages, thereby improving patient outcomes and reducing the burden of liver-related complications.

In conclusion, integrating SWE with biochemical markers, careful use of diagnostic cut-off points, and regular patient monitoring are crucial strategies for improving the assessment and management of liver fibrosis in chronic liver disease. If implemented effectively, these recommendations can lead to more accurate diagnoses, better patient outcomes, and more efficient use of healthcare resources.

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