Journal of Population Therapeutics & Clinical Pharmacology

RESEARCH ARTICLE
DOI:10.67145/7nhb3065

PLATELETCRIT VERSUS TRANSIENT ELASTOGRAPHY (FIBROSCAN) FOR ASSESSING THE SEVERITY OF LIVER FIBROSIS IN CHRONIC HEPATITIS C: A CROSS-SECTIONAL STUDY FROM A RURAL TERTIARY CARE CENTRE IN NORTH INDIA

Sushil Kumar[1*], Ranjit Kumar Nim[2]

[1*]Associate Professor, Department of Internal Medicine, Uttar Pradesh University of Medical Sciences (UPUMS), Saifai, Etawah, Uttar Pradesh, India, Email: sushilyadavcvts@gmail.com
[2]Assistant Professor, Department of Gastroenterology, GSVM Medical College, Kanpur, Uttar Pradesh, India

Abstract

Background: Staging of liver fibrosis is central to the management of chronic hepatitis C virus (HCV) infection. Transient elastography (FibroScan) is the preferred non-invasive tool, but its cost and limited availability restrict its use in rural India. Plateletcrit (PCT), the percentage of blood volume occupied by platelets, is reported automatically in every complete blood count and may serve as a low-cost surrogate marker.

Objective: To assess the correlation of plateletcrit with FibroScan-defined fibrosis stage and its diagnostic accuracy for advanced fibrosis (F3–F4) in patients with chronic hepatitis C.

Methods: This retrospective cross-sectional analytical study included 180 anti-HCV-positive adults evaluated at the Department of Internal Medicine, UPUMS, Saifai, between January 2024 and December 2025. Plateletcrit was compared across FibroScan stages (F0–F1, F2, F3, F4) using one-way ANOVA, and its correlation with fibrosis stage was assessed using Pearson's correlation. Receiver operating characteristic (ROC) analysis was used to determine the optimal plateletcrit cut-off for advanced fibrosis.

Results: Fibrosis stages were distributed as F0–F1 in 28%, F2 in 24%, F3 in 26% and F4 in 22% of patients. Mean plateletcrit declined progressively from 0.26 ± 0.05% in F0–F1 to 0.14 ± 0.02% in F4 ($p < 0.001$). Plateletcrit showed a significant inverse correlation with fibrosis stage ($r = -0.58$, $p < 0.001$). For advanced fibrosis, plateletcrit had an area under the ROC curve of 0.81; at a cut-off of < 0.18%, sensitivity was 78% and specificity 74%.

Conclusion: Plateletcrit falls progressively with increasing fibrosis and identifies advanced fibrosis with good accuracy. As a zero-cost parameter of the routine blood count, it may help triage HCV

patients for elastography or specialist referral in resource-limited settings, but it should complement rather than replace established tools until validated externally.

Keywords: Hepatitis C; Plateletcrit; Platelet indices; Transient elastography; Liver fibrosis; Non-invasive markers; Rural India

Introduction

Chronic hepatitis C virus (HCV) infection remains a major global cause of chronic liver disease. According to the World Health Organization's 2024 Global Hepatitis Report, an estimated 50 million people were living with hepatitis C in 2022, with nearly one million new infections that year, and India is one of six countries that together account for half of the global HCV burden.1,2 Without treatment, a substantial proportion of chronically infected individuals progress to fibrosis, cirrhosis, portal hypertension and hepatocellular carcinoma over two to three decades.1 India's National Viral Hepatitis Control Program (NVHCP), launched in 2018, offers free diagnosis and direct-acting antiviral (DAA) therapy through a network of Model Treatment Centres and Treatment Centres, many of which serve predominantly rural populations.3

Staging liver fibrosis remains essential even in the DAA era. The presence of advanced fibrosis (METAVIR F3) or cirrhosis (F4) determines the treatment regimen and duration, the need for surveillance for hepatocellular carcinoma and oesophageal varices, and the requirement for continued follow-up after sustained virological response.4–7 Liver biopsy, the historical reference standard, is invasive, carries a small but real risk of complications, is subject to sampling error, and is impractical for population-level programmes.6

Transient elastography (FibroScan) measures liver stiffness in kilopascals and has been validated against histology in chronic hepatitis C, with good accuracy for significant fibrosis and excellent accuracy for cirrhosis.8–10 Current EASL guidance endorses elastography as a first-line non-invasive test.6 However, the device is expensive, requires trained operators, and is concentrated in urban tertiary centres. In rural districts of Uttar Pradesh, many patients diagnosed through NVHCP screening camps have no ready access to elastography, which delays staging and can lead to under-recognition of cirrhosis.

Serum-based scores such as the AST-to-platelet ratio index (APRI) and Fibrosis-4 (FIB-4) were developed to fill this gap and are recommended by WHO for resource-limited settings.7,11–13 Notably, the platelet count is a component of both scores, and a falling platelet count is one of the earliest and most consistent laboratory correlates of progressive fibrosis in HCV.14 Thrombocytopenia in chronic liver disease results from splenic sequestration secondary to portal hypertension, reduced hepatic synthesis of thrombopoietin, and, in HCV specifically, direct viral and immune-mediated suppression of platelet production.15–19

Modern automated haematology analysers also report platelet indices — mean platelet volume (MPV), platelet distribution width (PDW) and plateletcrit (PCT) — at no additional cost. Plateletcrit represents the volume fraction of blood occupied by platelets and is calculated as the product of platelet count and MPV. Because it integrates both the number and the size of circulating platelets, it has been proposed as a more complete measure of total platelet mass than the platelet count alone. Platelet indices have been shown to correlate with liver fibrosis in HCV-infected patients,20,21 but data on plateletcrit specifically are limited and conflicting, particularly across different aetiologies of liver disease.22,23 To our

knowledge, plateletcrit has not been evaluated against transient elastography in a rural Indian HCV population.

We therefore conducted this study to evaluate the relationship between plateletcrit and FibroScan-defined fibrosis stage in patients with chronic hepatitis C, and to determine its diagnostic accuracy for advanced fibrosis in a rural tertiary care setting.

Objectives

Primary objective: To compare plateletcrit with FibroScan-based fibrosis staging in patients with chronic hepatitis C.

Secondary objectives: (i) to determine the correlation between plateletcrit and fibrosis stage; (ii) to evaluate the diagnostic accuracy of plateletcrit for advanced fibrosis (F3–F4) using ROC analysis and identify an optimal cut-off.

Materials and Methods

Study design, setting and period

This was a retrospective cross-sectional analytical study conducted in the Department of Internal Medicine, Uttar Pradesh University of Medical Sciences (UPUMS), Saifai, Etawah — a tertiary care centre serving a predominantly rural catchment of western Uttar Pradesh. Records of patients evaluated between January 2024 and December 2025 were analysed.

Study population

A total of 180 patients with chronic hepatitis C who had both a complete blood count with platelet indices and transient elastography during the study period were included.

Inclusion criteria:

- Age 18–65 years
- Confirmed HCV infection (anti-HCV positive)

Exclusion criteria:

- Co-infection with hepatitis B virus or HIV
- Known haematological disorders
- Alcoholic liver disease

Data collection

Demographic details (age, gender) and duration of illness were recorded. Laboratory parameters included platelet count, plateletcrit and liver function tests. Plateletcrit (%) was obtained from the automated haematology analyser, which derives it as the product of platelet count and mean platelet volume. Liver stiffness was measured by transient elastography (FibroScan®, Echosens, Paris), and patients were categorised into METAVIR-equivalent fibrosis stages F0–F4. For analysis, F0 and F1 were combined, and advanced fibrosis was defined as F3–F4.

Statistical analysis

Data were analysed using IBM SPSS Statistics version 26. Continuous variables are expressed as mean ± standard deviation and categorical variables as percentages. Plateletcrit was compared across fibrosis stages using one-way analysis of variance (ANOVA). The correlation between plateletcrit and fibrosis stage was assessed using Pearson's correlation coefficient. Receiver operating characteristic (ROC) curve analysis was used to assess the ability of plateletcrit to identify advanced fibrosis (F3–F4) and to determine the optimal cut-off, with sensitivity and specificity calculated at that cut-off. A two-tailed $p < 0.05$ was considered statistically significant.

Results

Distribution of fibrosis stages

Of the 180 patients, 28% had no or mild fibrosis (F0–F1), 24% had significant fibrosis (F2), 26% had advanced fibrosis (F3) and 22% had cirrhosis (F4). Thus, nearly half of the cohort (48%) had advanced fibrosis or cirrhosis at evaluation (Table 1, Figure 1).

Table 1. Distribution of FibroScan fibrosis stages (N = 180)

Fibrosis stage	Description	Patients (%)
F0–F1	No / mild fibrosis	28
F2	Significant fibrosis	24
F3	Advanced fibrosis	26
F4	Cirrhosis	22
Total		100

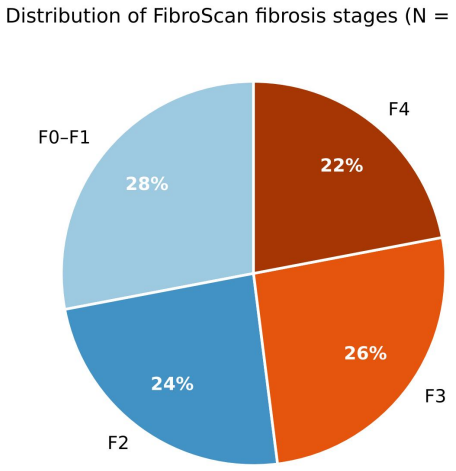


Figure 1. Distribution of patients by FibroScan fibrosis stage.

Plateletcrit across fibrosis stages

Mean plateletcrit fell progressively with each increase in fibrosis stage, from 0.26 ± 0.05% in F0–F1 to 0.21 ± 0.04% in F2, 0.17 ± 0.03% in F3 and 0.14 ± 0.02% in F4. The difference across stages was statistically significant (one-way ANOVA, $p < 0.001$; Table 2, Figure 2). Mean plateletcrit in F3 and F4 lay below the optimal cut-off of 0.18%, whereas that in F0–F1 and F2 lay above it.

Table 2. Plateletcrit according to fibrosis stage

Fibrosis stage	Plateletcrit (%), mean ± SD	p-value
F0–F1	0.26 ± 0.05	
F2	0.21 ± 0.04	< 0.001*
F3	0.17 ± 0.03	
F4	0.14 ± 0.02	

*One-way ANOVA across the four groups. SD, standard deviation.

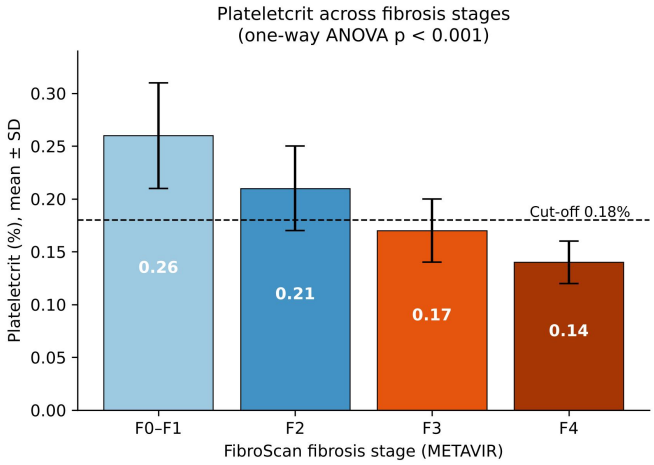


Figure 2. Mean plateletcrit (± SD) by fibrosis stage. The dashed line marks the optimal cut-off of 0.18% for advanced fibrosis.

Correlation analysis

Plateletcrit showed a significant inverse correlation with fibrosis severity ($r = -0.58$, $p < 0.001$), indicating that lower plateletcrit values were associated with more advanced fibrosis on FibroScan.

Diagnostic accuracy for advanced fibrosis

On ROC analysis, plateletcrit discriminated advanced fibrosis (F3–F4) from non-advanced fibrosis with an area under the curve (AUC) of 0.81, indicating good discriminative ability. The optimal cut-off was a plateletcrit below 0.18%, which yielded a sensitivity of 78% and a specificity of 74% (Table 3).

Table 3. Diagnostic performance of plateletcrit for advanced fibrosis (F3–F4)

Parameter	Value
Area under ROC curve (AUC)	0.81
Optimal cut-off	< 0.18%
Sensitivity	78%
Specificity	74%

Discussion

In this study of 180 patients with chronic hepatitis C from a rural tertiary care centre, plateletcrit declined in a stepwise manner with increasing FibroScan-defined fibrosis stage, correlated inversely with fibrosis severity, and identified advanced fibrosis with an AUC of 0.81. A plateletcrit below 0.18% detected approximately four of every five patients with F3–F4 fibrosis. These findings suggest that a parameter already available in every routine blood count carries useful information about fibrosis severity in HCV.

The decline in plateletcrit with advancing fibrosis is biologically plausible. As fibrosis progresses, rising portal pressure leads to congestive splenomegaly and sequestration of up to 90% of the circulating platelet pool in the spleen.17 At the same time, the fibrotic liver produces less thrombopoietin, the principal regulator of megakaryopoiesis, reducing platelet production.18 In HCV specifically, additional mechanisms — direct viral suppression of megakaryocytes and antiplatelet antibodies — contribute to thrombocytopenia.19 Because plateletcrit is the product of platelet count and platelet size, these processes lower it directly. Although MPV tends to rise in advanced fibrosis as the marrow releases larger, younger platelets,20,21 this compensatory increase is modest relative to the fall in platelet number, so the net effect is a reduction in total platelet mass, which is what plateletcrit measures.

Our results are consistent with the broader literature on platelets as fibrosis markers in HCV. Pohl et al. showed that platelet count and AST/ALT ratio predicted the degree of fibrosis in chronic hepatitis C,14 and the platelet count forms the denominator of both APRI and FIB-4, which are among the most widely validated non-invasive scores.11–13 Shao et al. studied 241 HCV-infected patients and found that platelet count correlated negatively, while MPV, PDW and the platelet large-cell ratio correlated positively, with both serum fibrosis markers and FibroScan measurements; platelet counts were significantly lower in advanced than in mild fibrosis.20 Purnak et al. similarly reported higher MPV in HCV patients with advanced fibrosis.21 Our finding of falling plateletcrit fits this pattern, since a fall in count outweighed any rise in size.

The direction of change in plateletcrit has not been uniform across studies, however. Michalak et al. found significantly lower plateletcrit in patients with alcohol-related cirrhosis than in controls,22 in agreement with our results. In contrast, Gadallah et al., in an Egyptian cohort, reported higher plateletcrit in cirrhotic patients than in controls and NAFLD patients, and found it to be an accurate discriminator of cirrhosis.23 Differences in aetiology, the inclusion of decompensated patients, the reference standard used (imaging versus elastography), analyser technology and sample handling — MPV, and hence plateletcrit, increases with time in EDTA — may all explain these discrepancies. This

heterogeneity underlines the need to establish population- and analyser-specific cut-offs before plateletcrit can be used in practice, and it is a reason our cut-off of 0.18% should be regarded as provisional.

The diagnostic accuracy observed here (AUC 0.81) is comparable to that reported for APRI in hepatitis C in meta-analysis,13 although lower than that generally reported for transient elastography in detecting cirrhosis.10 An important question, which our study was not designed to answer, is whether plateletcrit adds information beyond the platelet count itself or beyond APRI and FIB-4, which are equally cheap.

From a programmatic perspective, these findings are relevant to NVHCP treatment centres in rural India. Most patients identified at district level have a complete blood count at diagnosis, whereas elastography is available at only a few centres. A low plateletcrit could flag patients who should be prioritised for elastography, endoscopic variceal screening, hepatocellular carcinoma surveillance, or referral to a Model Treatment Centre, while a plateletcrit well above the cut-off could provide some reassurance. Used in this way — as a triage aid rather than a replacement for elastography — plateletcrit could help direct scarce diagnostic resources to the patients most likely to need them.

Strengths and limitations

The strengths of this study include a reasonably large sample from an under-studied rural population, the use of transient elastography as the reference standard, and the evaluation of a parameter that requires no additional cost or blood sample. Several limitations must also be acknowledged. First, this was a single-centre study, and the cut-off was derived and tested in the same population without internal (e.g., bootstrap) or external validation, which tends to overestimate accuracy. Second, fibrosis was staged by elastography rather than liver biopsy; liver stiffness can be raised by inflammation, congestion or cholestasis independent of fibrosis. Third, platelet indices vary between analysers and with the time from sampling to analysis, which limits the transferability of the cut-off. Fourth, we did not compare plateletcrit with platelet count, APRI or FIB-4 in the same patients. Fifth, its retrospective nature limited the availability of some baseline variables. Finally, the cross-sectional design does not allow assessment of whether plateletcrit tracks fibrosis regression after successful DAA therapy.

Future directions

Prospective multicentre studies across NVHCP sites, using standardised analysers and sample processing, are needed to validate the cut-off. Head-to-head comparison with APRI and FIB-4, evaluation of combined models incorporating plateletcrit, and longitudinal assessment of changes after sustained virological response would further define its role. Machine-learning models that combine routine blood-count indices with simple clinical variables may also improve risk stratification where elastography is unavailable.

Conclusion

Plateletcrit decreases progressively with increasing severity of liver fibrosis in chronic hepatitis C and identifies advanced fibrosis with good diagnostic accuracy. Because it is reported automatically and at no extra cost in every complete blood count, it may serve as a practical triage tool for prioritising elastography and specialist care in resource-limited rural settings. It should complement, not replace,

transient elastography and established serum scores until its value is confirmed in larger, externally validated studies.

Declarations

Ethics approval: The study was approved by the Institutional Ethics Committee, UPUMS, Saifai (Ref. No. _____, dated _____) and conducted in accordance with the Declaration of Helsinki and the ICMR National Ethical Guidelines (2017).24

Consent: The requirement for informed consent was waived by the Institutional Ethics Committee, as only anonymised retrospective data were used.

Conflicts of interest: The authors declare no conflicts of interest.

Funding: None.

Author contributions: SK: conceptualisation, data collection, analysis, drafting. RKN: methodology, interpretation, critical revision. Both authors approved the final manuscript.

Data availability: The anonymised dataset is available from the corresponding author on reasonable request.

References

1. World Health Organization. Global hepatitis report 2024: action for access in low- and middle-income countries. Geneva: WHO; 2024.
2. Polaris Observatory HCV Collaborators. Global change in hepatitis C virus prevalence and cascade of care since 2015: a modelling study. Lancet Gastroenterol Hepatol. 2022;7(5):396–415.
3. Ministry of Health and Family Welfare, Government of India. National Viral Hepatitis Control Program: operational guidelines. New Delhi: MoHFW; 2018.
4. Bedossa P, Poynard T. An algorithm for the grading of activity in chronic hepatitis C. The METAVIR Cooperative Study Group. Hepatology. 1996;24(2):289–93.
5. European Association for the Study of the Liver. EASL recommendations on treatment of hepatitis C: final update of the series. J Hepatol. 2020;73(5):1170–218.
6. European Association for the Study of the Liver. EASL Clinical Practice Guidelines on non-invasive tests for evaluation of liver disease severity and prognosis – 2021 update. J Hepatol. 2021;75(3):659–89.
7. World Health Organization. Guidelines for the care and treatment of persons diagnosed with chronic hepatitis C virus infection. Geneva: WHO; 2018.
8. Castéra L, Vergniol J, Foucher J, Le Bail B, Chanteloup E, Haaser M, et al. Prospective comparison of transient elastography, Fibrotest, APRI, and liver biopsy for the assessment of fibrosis in chronic hepatitis C. Gastroenterology. 2005;128(2):343–50.

9. Ziol M, Handra-Luca A, Kettaneh A, Christidis C, Mal F, Kazemi F, et al. Noninvasive assessment of liver fibrosis by measurement of stiffness in patients with chronic hepatitis C. Hepatology. 2005;41(1):48–54.
10. Friedrich-Rust M, Ong MF, Martens S, Sarrazin C, Bojunga J, Zeuzem S, et al. Performance of transient elastography for the staging of liver fibrosis: a meta-analysis. Gastroenterology. 2008;134(4):960–74.
11. Wai CT, Greenson JK, Fontana RJ, Kalbfleisch JD, Marrero JA, Conjeevaram HS, et al. A simple noninvasive index can predict both significant fibrosis and cirrhosis in patients with chronic hepatitis C. Hepatology. 2003;38(2):518–26.
12. Sterling RK, Lissen E, Clumeck N, Sola R, Correa MC, Montaner J, et al. Development of a simple noninvasive index to predict significant fibrosis in patients with HIV/HCV coinfection. Hepatology. 2006;43(6):1317–25.
13. Lin ZH, Xin YN, Dong QJ, Wang Q, Jiang XJ, Zhan SH, et al. Performance of the aspartate aminotransferase-to-platelet ratio index for the staging of hepatitis C-related fibrosis: an updated meta-analysis. Hepatology. 2011;53(3):726–36.
14. Pohl A, Behling C, Oliver D, Kilani M, Monson P, Hassanein T. Serum aminotransferase levels and platelet counts as predictors of degree of fibrosis in chronic hepatitis C virus infection. Am J Gastroenterol. 2001;96(11):3142–6.
15. Afdhal N, McHutchison J, Brown R, Jacobson I, Manns M, Poordad F, et al. Thrombocytopenia associated with chronic liver disease. J Hepatol. 2008;48(6):1000–7.
16. Peck-Radosavljevic M. Thrombocytopenia in chronic liver disease. Liver Int. 2017;37(6):778–93.
17. Aster RH. Pooling of platelets in the spleen: role in the pathogenesis of "hypersplenic" thrombocytopenia. J Clin Invest. 1966;45(5):645–57.
18. Kawasaki T, Takeshita A, Souda K, Kobayashi Y, Kikuyama M, Suzuki F, et al. Serum thrombopoietin levels in patients with chronic hepatitis and liver cirrhosis. Am J Gastroenterol. 1999;94(7):1918–22.
19. Nagamine T, Ohtuka T, Takehara K, Arai T, Takagi H, Mori M. Thrombocytopenia associated with hepatitis C viral infection. J Hepatol. 1996;24(2):135–40.
20. Shao LN, Zhang ST, Wang N, Yu WJ, Chen M, Xiao N, et al. Platelet indices significantly correlate with liver fibrosis in HCV-infected patients. PLoS One. 2020;15(1):e0227544.
21. Purnak T, Olmez S, Torun S, Efe C, Sayilir A, Ozaslan E, et al. Mean platelet volume is increased in chronic hepatitis C patients with advanced fibrosis. Clin Res Hepatol Gastroenterol. 2013;37(1):41–6.
22. Michalak A, Cichoż-Lach H, Guz M, Kozicka J, Cybulski M, Jeleniewicz W, et al. Plateletcrit and mean platelet volume in the evaluation of alcoholic liver cirrhosis and nonalcoholic fatty liver disease patients. Biomed Res Int. 2021;2021:8867985.
23. Gadallah AA, Elshayeb EI, Montaser BA, Shaheen GA, Mostafa AE. Plateletcrit and mean platelet volume in the evaluation of liver cirrhosis and nonalcoholic fatty liver disease in Egyptian patients. Surg Gastroenterol Oncol. 2024;29(2):105. doi:10.21614/sgo-643
24. Indian Council of Medical Research. National ethical guidelines for biomedical and health research involving human participants. New Delhi: ICMR; 2017.

25. Boursier J, Zarski JP, de Ledinghen V, Rousselet MC, Sturm N, Lebail B, et al. Determination of reliability criteria for liver stiffness evaluation by transient elastography. Hepatology. 2013;57(3):1182–91.