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Prevalence and Spectrum of Hepatic Fibrosis and Steatosis in Patients With Inflammatory Bowel Disease: A Cross-Sectional Study.

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Abstract

Background: Hepatic steatosis and fibrosis are increasingly recognized in inflammatory bowel disease, but their burden in Indian patients without established chronic liver disease is incompletely characterized. ***Objective:*** To estimate the prevalence and describe the spectrum of liver stiffness and steatosis detected by vibration-controlled transient elastography with controlled attenuation parameter in adults with inflammatory bowel disease. ***Methods:*** This cross-sectional study included 100 adults with confirmed inflammatory bowel disease at a tertiary referral center. Patients with known primary liver disease, cirrhosis, diabetes mellitus, severe heart failure, pregnancy were excluded. Liver stiffness measurement (LSM) and controlled attenuation parameter (CAP) were obtained using a FibroScan 502 Touch system after fasting. Study-defined liver stiffness strata were <6.5, 6.5-7.0, and >=7.1 kPa. Steatosis was defined by a controlled attenuation parameter >=248 dB/m and categorized as S1 (248-267), S2 (268-280), or S3 (>=281 dB/m). Prevalence estimates are reported with Wilson 95% confidence intervals. Agreement between ultrasonography and controlled attenuation parameter was assessed using Cohen kappa and exact McNemar testing. ***Results:*** The mean age was 36.21 ± 10.99 years; 55% were female, 79% had ulcerative colitis, and 21% had Crohn disease. Mean liver stiffness was 6.05 ± 0.80 kPa and mean controlled attenuation parameter was 216.04 ± 35.97 dB/m. Liver stiffness was 6.5-7.0 kPa in 20 patients and >=7.1 kPa in nine patients (9.0% (4.8%-16.2%)). Controlled attenuation parameter-defined steatosis was present in 22 patients (22.0% (15.0%-31.1%)): S1 in nine, S2 in six, and S3 in seven. Ultrasonography identified fatty liver in 29 patients (29.0% (21.0%-38.5%)). Agreement between ultrasonography and controlled attenuation parameter was 79%, with moderate concordance (kappa=0.451; 95% confidence interval 0.25-0.65). Liver stiffness and controlled attenuation parameter were not significantly correlated (Spearman rho=0.09; p=0.386). ***Conclusions:*** Subclinical abnormalities of liver stiffness and steatosis were frequent in this selected inflammatory bowel disease cohort despite exclusion of recognized primary liver disease and diabetes. Controlled attenuation parameter and ultrasonography showed only moderate agreement, and increased liver stiffness did not parallel steatosis. These non-invasive findings should be interpreted as risk stratification rather than histologic confirmation and require validation against standardized reference methods.

Keywords: controlled attenuation parameter; Crohn disease; hepatic fibrosis; hepatic steatosis; inflammatory bowel disease; transient elastography; ulcerative colitis.

INTRODUCTION

Inflammatory bowel disease (IBD), comprising ulcerative colitis and Crohn disease, is a chronic immune-mediated disorder whose incidence and prevalence are increasing rapidly in newly industrialized regions, including South Asia [1]. Although the gastrointestinal tract is the primary site of disease, extraintestinal complications contribute substantially to long-term morbidity. Hepatobiliary abnormalities are among the most clinically important and include primary sclerosing cholangitis, autoimmune and drug-induced liver injury, gallstone disease, portal venous thrombosis, hepatic steatosis, and progressive fibrosis [2].

Steatotic liver disease is now recognized as one of the most frequent hepatic comorbidities in IBD. Meta-analyses have estimated that approximately one-third of patients with IBD have imaging-defined hepatic steatosis, although prevalence

varies markedly with geography, diagnostic method, age, adiposity, and case selection [3,4]. The pathogenesis is likely multifactorial. Classical metabolic risk factors remain important, but chronic systemic inflammation, altered intestinal permeability, dysbiosis, nutritional disturbance, sarcopenia, corticosteroid exposure, and disease-related changes in body composition may contribute independently through the gut-liver axis [2-5]. Consequently, steatosis can occur in patients who are not obese and may be overlooked when screening is based only on body mass index or aminotransferase values.

Fibrosis is the principal determinant of liver-related prognosis across chronic liver diseases, but liver biopsy is unsuitable for population screening because it is invasive, subject to sampling variability, and difficult to repeat. Vibration-controlled transient elastography (VCTE) provides a rapid, non-invasive liver stiffness measurement (LSM), while the controlled attenuation parameter (CAP) estimates ultrasound attenuation related to hepatic fat [6]. International guidelines support non-invasive tests for fibrosis risk stratification, but emphasize that thresholds depend on disease etiology, probe, measurement quality, inflammation, cholestasis, congestion, and the clinical context [6,7]. Importantly, IBD-specific histologically validated thresholds are not firmly established.

Published VCTE studies in IBD have produced heterogeneous estimates. Saroli Palumbo et al. reported CAP-defined steatosis in 32.8% and significant fibrosis in 12.2% of an IBD cohort [8]. A Romanian study reported higher rates of steatosis and elevated stiffness [9], whereas other cohorts have found clinically significant stiffness in approximately 6%-10% [10,11]. Differences in metabolic profile, exclusion criteria, disease activity, treatment exposure, and chosen cutoffs probably explain much of this variation. Indian data using simultaneous LSM and CAP remain limited.

The present study therefore evaluated adults with IBD attending a tertiary center after excluding recognized primary liver diseases, cirrhosis, diabetes, and significant alcohol-related or viral liver disease. The primary objective was to estimate the prevalence and spectrum of study-defined elevations in liver stiffness and CAP-defined steatosis. Secondary objectives were to examine the overlap between stiffness and steatosis and to assess concordance between CAP and conventional ultrasonography.

MATERIALS AND METHODS

Study design and setting

This was a single-center cross-sectional study conducted in the Department of Medical Gastroenterology, Gandhi Medical College and Gandhi Hospital, Secunderabad, Telangana, India, over a 24-month period (Jan 2023 – dec 2024).

Participants

Adults aged 18 years or older with a confirmed diagnosis of ulcerative colitis or Crohn disease were recruited from inpatient and outpatient services by convenience sampling. The diagnosis of IBD was documented in the clinical record.

Exclusion criteria were autoimmune hepatitis, primary sclerosing cholangitis, primary biliary cholangitis, viral hepatitis, alcohol-related liver disease, liver tumors, known cirrhosis, severe heart failure, diabetes mellitus, pregnancy, and refusal to provide informed consent. These exclusions were intended to reduce major non-IBD causes of altered liver stiffness or steatosis, but they also limit generalizability to an unselected IBD population.

Clinical, laboratory, and ultrasonographic assessment

Demographic variables, residence, anthropometry, IBD phenotype, disease duration, extraintestinal manifestations, C-reactive protein (CRP) status, and medication exposure were recorded using a standardized case record. Laboratory assessment included hemoglobin, aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, bilirubin, and lipid measurements. Because substantial missingness and source-data inconsistencies were identified for selected biochemical variables, the present primary analysis was restricted to variables that could be verified across all participant records.

Abdominal ultrasonography was performed to classify the liver as normal, grade I fatty liver, or grade II fatty liver. For the prespecified agreement analysis, grade I or II fatty liver was considered ultrasonographic steatosis.

Vibration-controlled transient elastography and controlled attenuation parameter

Liver stiffness and CAP were measured using a FibroScan 502 Touch system (Echosens, Paris, France) by two physicians who had each performed more than 300 examinations. Participants fasted for at least four hours and were examined in the standard supine position with the right arm maximally abducted. The probe was placed in an intercostal space over the right hepatic lobe, generally between the ninth and eleventh intercostal spaces. The M probe was used initially, with the XL probe selected when recommended by the device.

A valid examination required at least 10 valid measurements and an interquartile range-to-median ratio below 30% for LSM. LSM was expressed in kilopascals (kPa) and CAP in decibels per meter (dB/m). The dataset contained complete LSM and CAP results for all 100 participants, with no recorded failed examinations.

Operational definitions and outcomes

Because no universally accepted IBD-specific VCTE thresholds define histologic fibrosis, results were reported primarily as continuous LSM and as study-defined strata. LSM <6.5 kPa was considered lower stiffness, 6.5-7.0 kPa an intermediate elevation, and >=7.1 kPa a higher stiffness category suggestive of clinically relevant fibrosis risk. These categories were derived from the study protocol and published IBD VCTE literature [8-10] and should not be interpreted as biopsy-confirmed METAVIR stages.

CAP <248 dB/m was classified as S0. CAP 248-267 dB/m was classified as S1, 268-280 dB/m as S2, and >=281 dB/m as S3, consistent with commonly used VCTE thresholds while acknowledging imperfect accuracy across etiologies [7,13,14]. The primary outcomes were the prevalence of LSM >=7.1 kPa and CAP-defined steatosis (CAP >=248 dB/m). Secondary outcomes included the full distributions of LSM and CAP strata, the coexistence of elevated LSM and CAP, and agreement between CAP and ultrasonography.

Statistical analysis

Participant-level data were reanalyzed for this manuscript. Continuous variables are presented as mean ± standard deviation and median with interquartile range; categorical variables are presented as number and percentage. Prevalence estimates are accompanied by two-sided Wilson 95% confidence intervals. The association between ordinal ultrasonographic grade and CAP was assessed using Spearman rank correlation and the Kruskal-Wallis test; epsilon-squared was reported as the effect size. Agreement between binary ultrasonographic steatosis and CAP >=248 dB/m was assessed using overall percentage agreement, Cohen kappa with a 95% confidence interval, and the exact McNemar test. The relationship between continuous LSM and CAP was evaluated using Spearman correlation. All tests were two-sided, and $p<0.05$ was considered statistically significant. No imputation was performed, and no multivariable prediction model was fitted because the primary aim was descriptive and the higher-stiffness group contained only nine events.

Analyses were performed using Python version 3.13.5 (Python Software Foundation, Wilmington, Delaware, United States), pandas version 2.2.3, SciPy version 1.17.0, statsmodels version 0.14.6, and scikit-learn version 1.8.0.

Ethical considerations

The parent study was approved by the Institutional Ethics Committee of Gandhi Medical College. Written informed consent was obtained before participation. The present analysis used de-identified data and introduced no additional intervention.

RESULTS

All 100 enrolled participants had complete LSM and CAP measurements and were included in the analysis. The mean age was 36.21 ± 10.99 years, 55 participants were female, and 57 lived in urban areas. Ulcerative colitis accounted for 79 cases and Crohn disease for 21. The mean body mass index was 21.25 ± 1.96 kg/m², and 92 participants were in the normal body mass index category reported in the parent dataset. Baseline characteristics are summarized in Table 1.

Table 1. Baseline characteristics of the study cohort

Characteristic	Overall cohort (n=100)
Age, years	36.21 ± 10.99; median 36.0 (26.8-45.2)
Female sex	55 (55.0)
Urban residence	57 (57.0)
Body mass index, kg/m²	21.25 ± 1.96; median 21.1 (20.0-22.3)
Disease duration, months	11.99 ± 8.90; median 8.0 (7.0-14.0)
Ulcerative colitis	79 (79.0)
Crohn disease	21 (21.0)
C-reactive protein positive	35 (35.0)
Current or prior corticosteroid exposure	48 (48.0)
5-aminosalicylic acid exposure	84 (84.0)
Anti-tumor necrosis factor exposure	16 (16.0)
Hemoglobin, g/dL	10.82 ± 0.93
Aspartate aminotransferase, U/L	22.39 ± 4.21
Alanine aminotransferase, U/L	22.05 ± 6.40

Values are mean ± standard deviation, median (interquartile range), or number (percentage), as indicated. Body mass index was calculated as weight in kilograms divided by height in meters squared. Exposure variables are those recorded in the parent dataset; cumulative dose and duration were unavailable.

The mean LSM was 6.05 ± 0.80 kPa, with a median of 6.1 kPa (interquartile range 5.6-6.5) and a range of 3.8-7.8 kPa. Seventy-one participants had LSM <6.5 kPa, 20 had LSM 6.5-7.0 kPa, and nine had LSM >=7.1 kPa. Thus, the prevalence of the higher study-defined stiffness category was 9.0% (4.8%-16.2%). The mean CAP was 216.04 ± 35.97 dB/m, with a median of 206 dB/m (interquartile range 189-236). CAP-defined steatosis was present in 22 participants (22.0% (15.0%-31.1%)), comprising nine S1, six S2, and seven S3 cases. The complete spectrum is shown in Table 2.

Table 2. Prevalence and spectrum of study-defined liver stiffness and controlled attenuation parameter categories

Non-invasive liver phenotype	Number (%)	95% confidence interval
LSM <6.5 kPa	71 (71.0)	71.0% (61.5%-79.0%)
LSM 6.5-7.0 kPa	20 (20.0)	20.0% (13.3%-28.9%)
LSM >=7.1 kPa	9 (9.0)	9.0% (4.8%-16.2%)
CAP S0 (<248 dB/m)	78 (78.0)	78.0% (68.9%-85.0%)
CAP S1 (248-267 dB/m)	9 (9.0)	9.0% (4.8%-16.2%)
CAP S2 (268-280 dB/m)	6 (6.0)	6.0% (2.8%-12.5%)
CAP S3 (>=281 dB/m)	7 (7.0)	7.0% (3.4%-13.7%)
Any CAP-defined steatosis (>=248 dB/m)	22 (22.0)	22.0% (15.0%-31.1%)
Both LSM >=6.5 kPa and CAP >=248 dB/m	7 (7.0)	7.0% (3.4%-13.7%)
Either LSM >=6.5 kPa or CAP >=248 dB/m	44 (44.0)	44.0% (34.7%-53.8%)

CAP, controlled attenuation parameter; LSM, liver stiffness measurement. Confidence intervals are two-sided Wilson 95% confidence intervals. LSM categories are operational study strata and must not be interpreted as biopsy-confirmed METAVIR fibrosis stages. CAP S0-S3 categories were defined using the thresholds shown in the table.

Only seven participants had both LSM >=6.5 kPa and CAP-defined steatosis, whereas 44 had either finding. Notably, none of the nine participants with LSM >=7.1 kPa had CAP >=248 dB/m. The cross-classification of LSM and CAP strata is presented in Table 3. Continuous LSM and CAP were not significantly correlated (Spearman rho=0.088; p=0.386), supporting the interpretation that increased stiffness and steatosis represented partially distinct non-invasive phenotypes in this cohort.

Table 3. Cross-classification of liver stiffness and controlled attenuation parameter strata

Liver stiffness stratum	CAP S0	CAP S1	CAP S2	CAP S3	Row total
<6.5 kPa	56	4	4	7	71
6.5-7.0 kPa	13	5	2	0	20
>=7.1 kPa	9	0	0	0	9
Column total	78	9	6	7	100

Values are participant counts. CAP, controlled attenuation parameter. The limited overlap between increased LSM and CAP-defined steatosis demonstrates that the two measurements characterized different non-invasive liver phenotypes in this cohort.

Ultrasonography was normal in 71 participants, showed grade I fatty liver in 20, and grade II fatty liver in nine. Mean CAP increased across ultrasonographic grades from 204.6 dB/m in normal examinations to 235.3 dB/m in grade I and 263.6 dB/m in grade II fatty liver (Kruskal-Wallis H=23.64, df=2, p<0.001; epsilon-squared=0.223). Ultrasonographic grade correlated moderately with CAP (Spearman rho=0.481; p<0.001). CAP values by ultrasound category are shown in Table 4.

Table 4. Controlled attenuation parameter according to ultrasonographic category

Ultrasonographic category	n	CAP, mean ± SD	CAP, median (IQR)	CAP-defined steatosis, n (%)
Normal	71	204.6 ± 28.5	198.0 (186.0-223.0)	7 (9.9)
Grade I fatty liver	20	235.3 ± 36.5	237.5 (198.0-254.2)	8 (40.0)
Grade II fatty liver	9	263.6 ± 33.5	282.0 (268.0-282.0)	7 (77.8)
Total	100	216.0 ± 36.0	206.0 (189.0-236.0)	22 (22.0)

CAP, controlled attenuation parameter; IQR, interquartile range; SD, standard deviation. Across the three ultrasonographic categories, Kruskal-Wallis H=23.64, degrees of freedom=2, p<0.001, epsilon-squared=0.223. Spearman correlation between ordinal ultrasound grade and CAP was rho=0.481, p<0.001.

Binary ultrasonography and CAP classifications agreed in 79 of 100 participants. Fifteen were positive by both methods, 64 were negative by both, 14 had ultrasonographic fatty liver with CAP <248 dB/m, and seven had CAP-defined steatosis with normal ultrasonography (Table 5). Cohen kappa was 0.451 (95% confidence interval 0.254-0.647; p<0.001), indicating moderate agreement. The exact McNemar test did not demonstrate significant directional discordance (p=0.189).

Table 5. Agreement between ultrasonography and controlled attenuation parameter for hepatic steatosis

Ultrasonography	CAP <248 dB/m	CAP >=248 dB/m	Row total
Normal	64	7	71
Fatty liver	14	15	29
Column total	78	22	100

CAP-defined steatosis was CAP >=248 dB/m. Overall agreement was 79.0%. Cohen kappa=0.451 (95% confidence interval 0.254-0.647; p<0.001). Exact McNemar p=0.189. Ultrasonography and CAP are both non-invasive tests; neither was treated as a definitive histologic reference standard.

DISCUSSION

This cross-sectional study provides a simultaneous description of liver stiffness and hepatic steatosis in a selected Indian IBD cohort without known primary liver disease, cirrhosis, or diabetes. Three findings are central. First, CAP-defined steatosis was present in 22% of participants, including 7% with the highest CAP category. Second, 9% had LSM >=7.1 kPa, while a further 20% had an intermediate elevation of 6.5-7.0 kPa. Third, stiffness and steatosis overlapped in only a minority and were not significantly correlated. These observations emphasize that the hepatic spectrum in IBD is heterogeneous and cannot be represented adequately by ultrasonography or aminotransferases alone.

The 22% prevalence of CAP-defined steatosis was lower than the pooled prevalence reported in several IBD meta-analyses, which has generally been around 30%-33% [3,4]. Saroli Palumbo et al. found CAP-defined steatosis in 32.8% of 384 patients [8], and Trifan et al. reported 46.3% among 82 patients [9]. The lower estimate in our cohort is biologically plausible because diabetes was excluded, the mean body mass index was low, and only 2% of the source cohort was classified as overweight. Differences in age, ethnicity, metabolic phenotype, CAP threshold, probe selection, and referral patterns may also contribute. Nevertheless, a prevalence of 22% in a predominantly normal-weight and non-diabetic cohort supports the concept that hepatic steatosis in IBD is not solely an expression of conventional obesity-related metabolic disease.

The study-defined higher stiffness category was present in 9%. This estimate is broadly comparable with the 6.4% prevalence of LSM >=8 kPa reported by Thin et al. [10] and the 12.2% prevalence of significant fibrosis reported by Saroli Palumbo et al. [8], but lower than estimates from some more metabolically enriched cohorts [9]. Direct comparison must be cautious because thresholds, probes, fasting conditions, disease activity, and exclusion criteria differed. The maximum LSM in our dataset was 7.8 kPa, and no participant had known cirrhosis. Thus, the findings predominantly represent mild or early stiffness elevation rather than advanced chronic liver disease.

An important observation was the weak relationship between LSM and CAP. None of the nine participants with LSM >=7.1 kPa met the CAP >=248 dB/m threshold. This does not establish non-steatotic fibrosis because VCTE is not histology and mild inflammation, cholestasis, congestion, technical factors, or measurement variability can influence stiffness [6,7]. Conversely, CAP quantifies ultrasound attenuation and does not establish steatohepatitis or fibrosis. The discordance illustrates why LSM and CAP should be considered complementary rather than interchangeable signals.

Ultrasonography detected fatty liver in 29%, compared with 22% by CAP. CAP rose progressively across ultrasound grades and the methods agreed in 79%, but Cohen kappa indicated only moderate concordance. Conventional ultrasound is operator dependent and has limited sensitivity for mild steatosis, while CAP performance varies with body habitus, probe, fasting, and the chosen threshold [13,14]. Fourteen patients were ultrasound-positive but CAP-negative and seven showed the reverse pattern. In the absence of magnetic resonance proton density fat fraction or biopsy, these discordances cannot be labelled false-positive or false-negative; the appropriate conclusion is that the modalities classify a clinically meaningful subset differently.

The normal or near-normal mean aminotransferase values in this cohort should not be interpreted as excluding liver involvement. Steatotic liver disease and fibrosis can be present despite normal enzymes, and current non-invasive test

pathways are designed to identify risk before decompensated disease becomes clinically evident [6,7,15]. At the same time, universal VCTE screening of all patients with IBD is not yet mandated by a validated IBD-specific algorithm. ECCO guidance supports regular liver chemistry assessment and evaluation tailored to the pattern of abnormality and clinical risk [2]. The present data support a low threshold for non-invasive assessment in patients with persistent biochemical abnormalities, metabolic risk, long disease duration, hepatotoxic drug exposure, or abnormal liver imaging, while prospective studies should determine whether routine screening improves outcomes.

The study also highlights the need for terminology discipline. LSM categories should not be equated automatically with METAVIR fibrosis stages, particularly in IBD where disease-specific thresholds have not been robustly validated against biopsy. We therefore report 'study-defined stiffness strata' and 'higher LSM suggestive of fibrosis risk' rather than confirmed F1 or F2 fibrosis. Similarly, CAP-defined steatosis is a non-invasive phenotype and does not by itself establish metabolic dysfunction-associated steatotic liver disease, which requires appropriate clinical and metabolic characterization [7].

From a research perspective, the cohort offers a useful baseline for longitudinal work. Repeated VCTE could determine whether stiffness or CAP changes with disease control, corticosteroid withdrawal, biologic treatment, nutritional recovery, or emerging metabolic risk. Future studies should include validated IBD activity indices, cumulative medication exposure, waist circumference, fasting glucose or glycated hemoglobin, comprehensive lipid profiling, magnetic resonance-based fat quantification, and predefined referral for biopsy when clinically justified.

Strengths

The principal strengths were simultaneous measurement of LSM and CAP in all 100 participants, standardized fasting VCTE performed by experienced operators, complete primary-outcome data, and analysis of both prevalence and modality concordance. The exclusion of recognized primary liver disease and diabetes reduced major competing explanations for steatosis and stiffness, allowing assessment of subclinical hepatic phenotypes within IBD.

Limitations

The cross-sectional design precludes conclusions about causality, progression, or the temporal relationship between intestinal inflammation, treatment, and liver findings.

This was a convenience sample from a single tertiary center. Referral and selection bias may have enriched particular disease phenotypes, while exclusion of diabetes, primary sclerosing cholangitis, cirrhosis, and other liver diseases limits generalizability to routine IBD populations. There was no healthy or non-IBD control group, so the study cannot determine whether the observed prevalences exceed those in a demographically comparable background population.

VCTE and CAP were not validated against liver biopsy or magnetic resonance imaging. The thresholds were adopted from published non-IBD or mixed-etiology literature, and IBD-specific cutoffs remain uncertain. Inflammation, cholestasis, congestion, food intake, and technical factors may alter LSM independently of fibrosis. Ultrasonography was not interpreted centrally or blinded to clinical data, and the source record did not contain complete equipment and interobserver details. Ultrasound-CAP disagreement therefore cannot be resolved as diagnostic error by either modality.

CONCLUSION

In this selected tertiary-care cohort of adults with IBD and no recognized primary liver disease or diabetes, CAP-defined steatosis affected 22% and a study-defined higher LSM category affected 9%. Steatosis and stiffness showed limited overlap, and CAP and ultrasonography demonstrated only moderate agreement. These findings suggest that subclinical liver phenotypes are common and heterogeneous in IBD. VCTE with CAP may complement biochemical testing and ultrasonography for risk stratification, but the measurements should not be equated with histologic fibrosis or steatohepatitis without confirmatory evaluation. Prospective controlled studies using standardized IBD-specific thresholds and longitudinal outcomes are needed.

Declarations

Ethics approval and consent to participate: The study was approved by the Institutional Ethics Committee of Gandhi Medical College. Written informed consent was obtained from all participants.
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Conflicts of interest: The authors declare no conflicts of interest.
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