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Review

Transient Elastography for Liver Fibrosis and Steatosis in Inflammatory Bowel Disease: Current Evidence, Clinical Implications, and Future Directions

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Abstract

Inflammatory bowel disease (IBD) is frequently complicated by hepatobiliary conditions, including metabolic dysfunction-associated steatotic liver disease (MASLD), drug-induced liver injury, primary sclerosing cholangitis, and autoimmune liver disease. However, systematic liver assessment remains underused, and serum indices such as FIB-4 may be confounded by IBD-related thrombocytosis. Transient elastography (TE) quantifies liver stiffness measurement (LSM) for fibrosis and the controlled attenuation parameter (CAP) for steatosis through a rapid, noninvasive outpatient examination. This narrative review synthesizes evidence on the prevalence of fibrosis and steatosis, associated risk factors, the effects of IBD therapies, and the clinical utility of TE-based screening, drawing on original studies that used FibroScan®-based LSM and/or CAP in adult patients with IBD; studies using other elastography modalities were excluded. Across 13 studies involving more than 2,700 patients with IBD, the prevalence of significant fibrosis (F≥2) clustered around 4–12% (range, ~1–24%, depending on the threshold), while steatosis was detected in 29–46%. Anti-TNF therapy was associated with a lower prevalence of fibrosis, whereas cumulative corticosteroid exposure increased the risk of both fibrosis and steatosis. Two matched-control studies confirmed that IBD independently confers an increased hepatic risk: one reported steatosis in 42.0% versus 32.7% and advanced fibrosis in 9.5% versus 2.3% of controls (adjusted odds ratio [aOR] for steatosis, 1.99), while ELASTIBD reported significant fibrosis in 12.3% versus 4.2% (aOR, 3.31) and steatosis in 41.4% versus 28.3% (aOR, 2.51) compared with matched controls. TE therefore provides actionable information beyond standard liver function tests and should be integrated into IBD follow-up, particularly for patients with prolonged corticosteroid exposure, metabolic comorbidities, or longstanding disease, while standardization of cutoff values and longitudinal data remain research priorities.

Keywords: Controlled attenuation parameter, FibroScan, hepatic steatosis, inflammatory bowel disease, liver fibrosis, metabolic dysfunction-associated steatotic liver disease, transient elastography.

INTRODUCTION

Inflammatory bowel disease (IBD), comprising Crohn's disease (CD) and ulcerative colitis (UC), is a chronic, immune-mediated disorder of the gastrointestinal tract affecting an estimated 6.8 million individuals worldwide, with incidence rising particularly in newly industrialized regions.[1,2] Beyond intestinal inflammation, IBD is increasingly recognized as a systemic disease, with extraintestinal manifestations (EIMs) occurring in up to 50% of patients over the course of their illness.[3,4] Among these, hepatobiliary involvement is common and clinically underappreciated and includes metabolic dysfunction-associated steatotic liver disease (MASLD), primary sclerosing cholangitis (PSC), drug-induced liver injury (DILI), autoimmune hepatitis, and chronic viral hepatitis.[5]

The hepatic burden in IBD arises from several pathogenic pathways, including sustained systemic inflammation, gut microbiota dysbiosis driving aberrant gut-liver axis signaling, cumulative exposure to hepatotoxic drugs, and metabolic dysregulation that may paradoxically occur even in nutritionally compromised patients.[5,6] Despite this complexity, liver assessment in routine IBD care has historically relied on conventional liver function tests, which lack sensitivity for detecting early or subclinical fibrosis and steatosis.[7] Serum-based fibrosis indices such as FIB-4 and APRI are likewise unreliable in IBD because reactive thrombocytosis associated with chronic inflammation lowers their platelet-dependent scores and may mask significant fibrosis.[8] Liver biopsy, the reference standard for fibrosis staging, is invasive, subject to sampling error, and impractical for surveillance in an outpatient IBD population.[9]

Transient elastography, commercially implemented as FibroScan® (Echosens, Paris, France), addresses this diagnostic gap by providing simultaneous, noninvasive quantification of liver stiffness measurement (LSM) as a validated surrogate for fibrosis stage and the controlled attenuation parameter (CAP) as a quantitative index of hepatic steatosis.[10,11] The technique is rapid, reproducible, and well tolerated, making it particularly

suitable for integration into IBD outpatient workflows. Over the past decade, a growing body of evidence has evaluated transient elastography in IBD cohorts; however, important questions regarding optimal screening strategies, cutoff standardization, and the influence of IBD-specific therapies remain unresolved.

This narrative review synthesizes current evidence on the use of transient elastography in adult patients with IBD, examining the prevalence and determinants of liver fibrosis and steatosis, the impact of IBD therapies on hepatic outcomes, and the clinical scenarios in which TE-based assessment is most warranted. We also highlight key methodological limitations and priorities for future research.

Literature Search and Study Selection

This narrative review is based on a structured search of the PubMed/MEDLINE database from inception through 2025 using combinations of the terms "inflammatory bowel disease," "Crohn's disease," "ulcerative colitis," "transient elastography," "FibroScan," "vibration-controlled transient elastography," "liver stiffness," "controlled attenuation parameter," "liver fibrosis," and "hepatic steatosis." Original studies using FibroScan®-based LSM and/or CAP measurements in adult IBD populations were prioritized. Studies employing alternative elastography modalities, including acoustic radiation force impulse imaging, point or two-dimensional shear-wave elastography, and magnetic resonance elastography, were excluded to preserve methodological comparability. The reference lists of retrieved articles and relevant reviews were hand-searched to identify additional sources. Consistent with the narrative review design, no formal risk-of-bias assessment or quantitative meta-analysis was performed; the synthesis was qualitative and interpretive.

Burden Of Liver Disease In Inflammatory Bowel Disease

Hepatobiliary involvement is one of the most clinically significant extraintestinal manifestations of IBD. Although it has historically been estimated to affect approximately 5% of patients with IBD based on overt liver enzyme abnormalities or clinical presentation, this figure is widely considered an underestimate; subclinical hepatic involvement is considerably more prevalent when systematic noninvasive assessment is applied.[4,5] Liver enzyme elevations have been reported in up to 30% of patients with IBD at some point during their disease course; however, abnormal transaminase levels alone correlate poorly with the nature or severity of the underlying hepatic pathology.[7] The hepatobiliary complications of IBD vary widely, ranging from common and often silent conditions such as hepatic steatosis to rare but potentially fatal conditions such as cholangiocarcinoma arising from primary sclerosing cholangitis (PSC). These pathways converge on the liver through the mechanisms summarized in Figure 1.

Metabolic Dysfunction-Associated Steatotic Liver Disease

Hepatic steatosis, now classified under the umbrella of MASLD, is the most frequent hepatobiliary complication of IBD and accounts for the majority of abnormal liver findings in this population.[5,12] Reported prevalence rates vary considerably across studies, ranging from 1.5% to 55% in UC and from 1.5% to 39.5% in CD, owing to differences in diagnostic methods, population characteristics, and the extent of alcohol exclusion.[5] When quantitative noninvasive tools such as vibration-controlled transient elastography (VCTE) with the controlled attenuation parameter (CAP) are used, the prevalence of steatosis consistently ranges between 29% and 46%.[13–15]

The pathogenesis of MASLD in IBD is multifactorial and differs from that in the general population. In addition to conventional metabolic

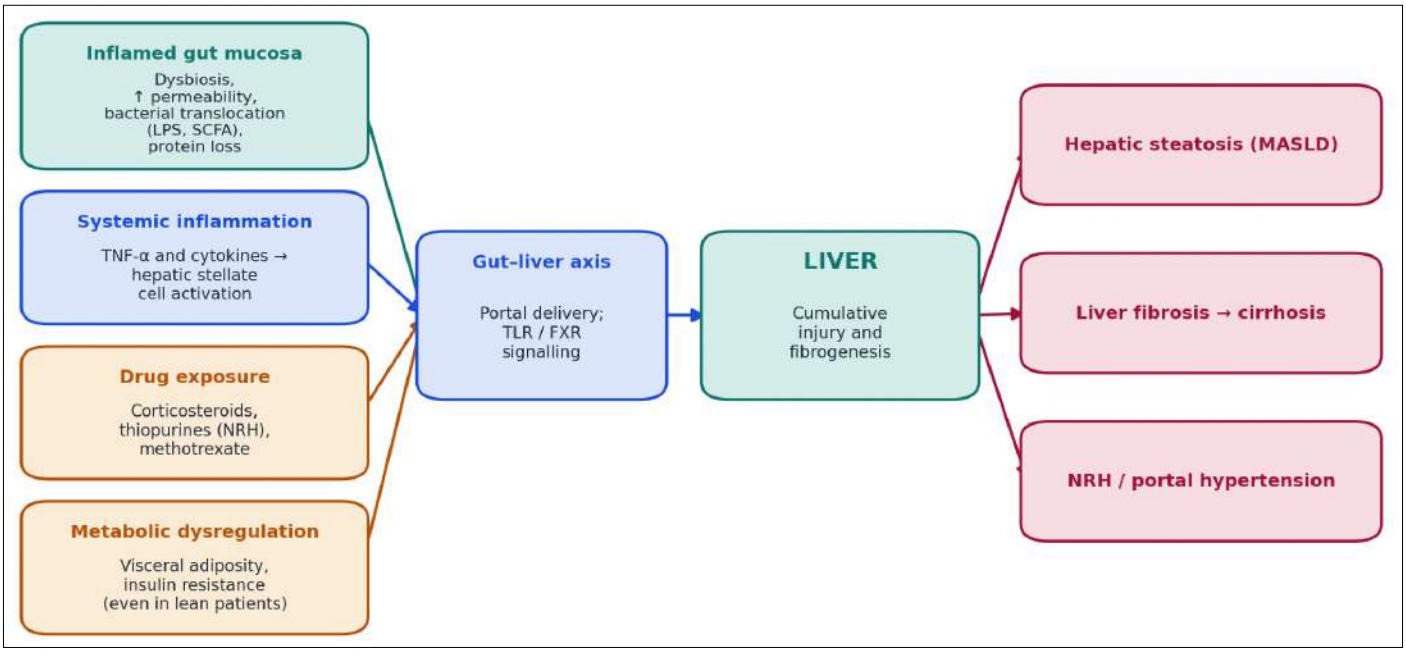

Figure 1. Pathogenesis of hepatic injury in inflammatory bowel disease. Intestinal, systemic inflammatory, iatrogenic, and metabolic pathways converge on the liver through the gut-liver axis, resulting in hepatic steatosis, fibrosis, and nodular regenerative hyperplasia/portal hypertension. FXR, farnesoid X receptor; LPS, lipopolysaccharide; MASLD, metabolic dysfunction-associated steatotic liver disease; NRH, nodular regenerative hyperplasia; SCFA, short-chain fatty acids; TLR, toll-like receptor; TNF, tumor necrosis factor.

risk factors, several IBD-specific contributors are well recognized, including sustained systemic inflammation, gut-liver axis dysregulation, malnutrition, and the metabolic effects of IBD therapies.[6,16] (Figure 1) These IBD-specific mechanisms, including the role of corticosteroids, are examined in detail in Metabolic Risk Factors: Shared and IBD-Specific.

Primary Sclerosing Cholangitis

PSC is the most IBD-specific hepatobiliary manifestation, occurring in approximately 4–5% of patients with IBD overall, with a marked predominance in UC.[17,18] Conversely, 70–80% of patients diagnosed with PSC have concurrent IBD, making IBD the strongest clinical association with this cholestatic disease.[17] PSC follows a progressive course characterized by biliary inflammation and fibrosis, ultimately leading to end-stage liver disease, and is associated with a substantially increased risk of cholangiocarcinoma and colorectal carcinoma.[19,20] Diagnosis is based on characteristic magnetic resonance cholangiopancreatography (MRCP) findings after exclusion of secondary causes; liver biopsy retains diagnostic utility primarily in small-duct PSC.[18] Although transient elastography is not a diagnostic modality for PSC, it has emerging value in monitoring fibrosis progression in patients with PSC-IBD and identifying those at risk of hepatic decompensation.[18]

Drug-Induced Liver Injury

IBD pharmacotherapy carries hepatotoxic potential and therefore warrants ongoing vigilance. Thiopurines, including azathioprine and 6-mercaptopurine, are associated with a range of hepatic adverse effects, including transaminase elevation, nodular regenerative hyperplasia (NRH), and noncirrhotic portal hypertension, which may remain clinically silent until esophageal varices develop because platelet counts are often preserved or even elevated in patients with IBD.[21] Methotrexate carries a cumulative dose-related risk of liver fibrosis, although this risk appears substantially lower in patients with IBD than in those with psoriasis or rheumatoid arthritis.[22,23] Repeated courses of corticosteroids promote visceral adiposity and hepatic fat accumulation, contributing to the burden of MASLD independently of disease activity.[13] In contrast, anti-TNF agents have been associated with a lower prevalence of fibrosis in multiple observational IBD cohorts, and experimental data suggest potential hepatoprotective effects through suppression of inflammatory cytokines implicated in hepatic stellate cell activation.[15,24]

Autoimmune Liver Diseases and Viral Hepatitis

Autoimmune hepatitis (AIH) and primary biliary cholangitis (PBC) occur more frequently in patients with IBD than in the general population, and AIH–PSC overlap syndrome presents a particularly challenging diagnostic and therapeutic scenario.[7,25] Vigilance for HBV reactivation is essential in patients initiating immunosuppressive therapy; screening for HBsAg and anti-HBc before the initiation of biologics or immunomodulators, followed by antiviral prophylaxis when indicated, is mandated by current guidelines.[26] Chronic HCV coinfection, although less specifically associated with IBD, also influences hepatic outcomes and treatment decisions, particularly with respect to the risk of thiopurine- and methotrexate-related hepatotoxicity.

Taken together, this diverse and overlapping spectrum of hepatic pathology, occurring in a population characterized by chronic systemic inflammation, complex polypharmacy, and metabolic dysregulation, provides a strong rationale for systematic noninvasive liver assessment as an integral component of IBD care.

Principles of Transient Elastography

Technical Basis and Liver Stiffness Measurement

FibroScan® is an ultrasound-based technique that quantifies hepatic mechanical properties through the propagation of a low-frequency shear wave generated by a vibrating probe placed intercostally over the right hepatic lobe.[10] The velocity of shear wave propagation, which is directly proportional to tissue stiffness, is measured by pulse-echo ultrasound and converted into a liver stiffness measurement (LSM), expressed in kilopascals (kPa). Each examination requires a minimum of 10 valid acquisitions, with reliability defined by a success rate ≥60% and an interquartile range-to-median ratio (IQR/M) ≤30%.[27,28] The examination samples a hepatic volume approximately 100-fold greater than that of a conventional biopsy core, substantially reducing the sampling error inherent in histological staging.[9]

LSM correlates with the METAVIR fibrosis staging system, with manufacturer-recommended and widely adopted thresholds for significant fibrosis (F≥2) ranging from 7.0 to 8.0 kPa, advanced fibrosis (F≥3) at approximately 8.7–9.5 kPa, and cirrhosis (F4) at ≥12.5 kPa, depending on the underlying etiology and probe type (Table 1).[27,29] The M probe is recommended for patients with a skin-to-liver capsule distance ≤25 mm, whereas the XL probe is indicated for patients with obesity and yields LSM values approximately 1.0–1.5 kPa lower for equivalent fibrosis stages.[30]

Controlled Attenuation Parameter

The controlled attenuation parameter (CAP) is acquired simultaneously with LSM using the same ultrasonic signal and quantifies hepatic steatosis by measuring ultrasound attenuation caused by fat within the liver parenchyma, expressed in decibels per meter (dB/m).[11] The most widely applied CAP thresholds define steatosis grades as follows: S0 (no steatosis) below 248 dB/m, S1 (mild, >5% hepatocytes) at 248–267 dB/m, S2 (moderate, >33%) at 268–279 dB/m, and S3 (severe, >66%) at ≥280 dB/m.31 CAP is more sensitive than conventional B-mode ultrasonography for detecting mild steatosis, and because it is acquired simultaneously with LSM, it enables noninvasive assessment of both fibrosis and steatosis during a single outpatient visit.[28,32]

Spleen Stiffness Measurement

More recent generations of FibroScan® permit spleen stiffness measurement (SSM) using the same shear wave principle applied intercostally over the spleen. SSM correlates with portal pressure and has demonstrated value in predicting clinically significant portal hypertension and esophageal varices independently of LSM.[33,34] The Baveno VII consensus proposed SSM cutoffs of <21 kPa to rule out and >50 kPa to rule in clinically significant portal hypertension; however, these thresholds were derived primarily from patients with viral compensated advanced chronic liver disease, and their application to IBD remains an extrapolation pending dedicated validation.[35,36] In IBD, SSM is particularly relevant for detecting thiopurine-associated NRH and noncirrhotic portal hypertension, conditions in which liver architecture and LSM may remain entirely normal despite established portal hypertension; however, this association has so far been reported only in preliminary studies.[37]

Cutoff Heterogeneity and IBD-Specific Considerations

A major methodological challenge in interpreting transient elastography findings across IBD cohorts is the lack of consensus regarding LSM cutoff values, reflecting the absence of IBD-specific validation against liver biopsy as a reference standard in large, well-characterized

Table 1. Widely adopted transient elastography thresholds for fibrosis staging (LSM), steatosis grading (CAP), and portal hypertension assessment (SSM). Values reflect manufacturer-recommended and meta-analytic cutoffs for the M probe; XL-probe- and etiology-specific adjustments may apply. For MASLD-predominant disease, a pragmatic dual-cutoff approach is widely used: LSM <8 kPa rules out clinically significant fibrosis, LSM ≥12 kPa rules in advanced fibrosis, and values of 8–12 kPa are considered indeterminate and warrant confirmatory testing.38–40 SSM cutoffs (<21 kPa to rule out; >50 kPa to rule in) follow the Baveno VII consensus.[35,36]

Grade/stage	Cutoff value	Clinical interpretation
Liver stiffness measurement (LSM) – METAVIR fibrosis equivalents (M probe)		
Significant fibrosis (F≥2)	7.0–8.0 kPa	Threshold for hepatology referral and structured follow-up
Advanced fibrosis (F≥3)	8.7–9.5 kPa	Marked fibrosis; expedite etiologic work-up
Cirrhosis (F4)	≥12.5 kPa	Surveillance for portal hypertension and HCC is indicated
Controlled attenuation parameter (CAP) – steatosis grade		
S0 – no steatosis	<248 dB/m	Normal hepatic fat content
S1 – mild (>5%)	248–267 dB/m	Early steatosis; reinforce metabolic optimization
S2 – moderate (>33%)	268–279 dB/m	Moderate steatosis
S3 – severe (>66%)	≥280 dB/m	Severe steatosis; consider evaluation for steatohepatitis
Spleen stiffness measurement (SSM)		
Spleen stiffness (SSM)	>21 kPa (with LSM <7 kPa)	Disproportionate elevation suggests NRH/noncirrhotic portal hypertension, particularly in thiopurine-treated patients

CAP, controlled attenuation parameter; dB/m, decibels per meter; HCC, hepatocellular carcinoma; kPa, kilopascals; LSM, liver stiffness measurement; MASLD, metabolic dysfunction-associated steatotic liver disease; NRH, nodular regenerative hyperplasia; SSM, spleen stiffness measurement.

cohorts. For clinical decision-making, contemporary MASLD guidance favors a pragmatic dual-cutoff approach: an LSM below 8 kPa reliably excludes clinically significant fibrosis (rule-out), a value of 12 kPa or higher indicates a high probability of advanced fibrosis warranting hepatology referral (rule-in), and intermediate values of 8–12 kPa define an indeterminate zone requiring a second noninvasive test or, when needed, magnetic resonance elastography or liver biopsy.[38–40] Reported thresholds for significant fibrosis (F≥2) across IBD studies nevertheless range from 7.0 to 8.7 kPa for the M probe, and this heterogeneity directly limits cross-study comparisons and pooled prevalence estimates.[41,42] Because most IBD-associated liver disease is metabolic in origin, this framework provides a reasonable default for LSM interpretation in IBD pending IBD-specific validation.

Several IBD-specific physiological factors may confound LSM interpretation independently of fibrosis. Active intestinal inflammation with elevated CRP levels drives systemic inflammatory signaling that may transiently increase liver stiffness regardless of fibrosis stage.[43] Hepatic congestion associated with right-sided heart involvement, postprandial increases in hepatic blood flow, and cholestasis in patients with PSC can each elevate LSM without corresponding histological fibrosis.18 These confounders underscore the importance of performing TE in clinically stable, fasting patients and interpreting the results within the overall clinical context rather than in isolation. CAP values are similarly influenced by probe type, body mass index, and the presence of significant fibrosis, as higher fibrosis stages independently increase CAP readings, which is particularly relevant in patients with IBD and concurrent steatosis and fibrosis.[11,31]

Despite these limitations, transient elastography remains the most practical, reproducible, and evidence-supported noninvasive tool for the simultaneous assessment of hepatic fibrosis and steatosis in IBD outpatient settings.[8]

Evidence for Liver Fibrosis Assessment in IBD

Early Studies and the Pre-CAP Era

The systematic application of transient elastography in IBD populations began in the early 2010s, initially focusing on LSM as a surrogate for fibrosis without simultaneous quantification of steatosis. Barbero-Villares et al.[22] examined 46 methotrexate-treated patients with IBD and found that advanced fibrosis was uncommon despite cumulative exposure. Thin et al.[42] subsequently evaluated 110 patients with IBD alongside 55 non-IBD controls using an LSM threshold of ≥8 kPa for clinically significant liver disease, identifying elevated stiffness in only 6.4% of patients with IBD, with no significant difference from controls at the group level; within the IBD cohort, however, higher BMI and older age were independently associated with elevated LSM. Meijer et al.[41] contributed the first prospective two-center cohort, reporting significant fibrosis (F≥2) in 4% and severe fibrosis (F≥3) in <1% of 168 patients and noting that prior azathioprine exposure was associated with lower liver stiffness. The full body of included studies is summarized in Table 2.

The Landmark Cohort and CAP Integration

A major advance came from Saroli Palumbo et al.,[13] who screened 384 patients with IBD using VCTE with CAP as part of a routine outpatient program, representing the first large prospective cohort to quantify both fibrosis and steatosis. The reported prevalence of NAFLD (32.8%) and significant fibrosis (12.2%) was substantially higher than that observed in earlier TE-only studies, reflecting the greater sensitivity of CAP for detecting steatosis compared with clinical suspicion alone. Older age and higher BMI emerged as the primary independent predictors of both outcomes in logistic regression, establishing the metabolic phenotype as the dominant risk profile in IBD-associated liver disease. The study also linked NAFLD to extrahepatic comorbidities, including chronic kidney and cardiovascular disease, thereby broadening its clinical implications.

Table 2. Original studies of transient elastography in adult IBD populations included in this review (n=13), presented in chronological order

Study	Country	Design	N (IBD)	Steatosis	Fibrosis (F≥2)	Key finding
Barbero-Villares[22]	Spain	Prospective, multicenter	46	—[a]	23.9%	Advanced fibrosis was uncommon in MTX-treated patients with IBD
Thin[42]	Australia	Case-control	110	—[a]	6.4%	Higher BMI and older age were associated with increased LSM; no significant difference versus controls
Meijer[41]	Netherlands	Prospective, two-center	168	NR	4%	Prior azathioprine exposure was associated with lower LSM
Saroli Palumbo[13]	Canada	Prospective	384	32.8%	12.2%	First large CAP-based cohort; age and BMI were the main predictors
Arieira[44]	Portugal	Cross-sectional	161	16.8–45.3%[b]	NR	CAP reclassified steatosis compared with clinical indices
Yen[46]	Taiwan	Retrospective	81	29.6%	1.2%	Lower prevalence was observed in an Asian cohort
van Lingen[43]	Netherlands	Prospective, longitudinal	112	40%	20%[c]	Active IBD was associated with progression of steatosis and fibrosis
Trifan[14]	Romania	Prospective	82	46.3%	NR	Highest reported steatosis prevalence; associated with metabolic factors
Veltkamp[45]	Germany	Retrospective	132	30.3%	8%	Fibrosis occurred predominantly in patients with Crohn's disease
Hsiao[47]	Taiwan	Retrospective	120	29.2%	Low	MAFLD was assessed in patients in clinical remission
Rodriguez-Duque[48]	Spain	Matched case-control	831	42.0%[d]	9.5%[c]	IBD was an independent predictor of MAFLD (aOR, 1.99)
Ferraz-Amaro[8]	Spain	Cross-sectional	197	NR	NR	FIB-4 did not correlate with elastography-defined fibrosis
ELASTIBD[15]	Türkiye	Matched case-control	358	41.4%[d]	12.3%[d]	Anti-TNF therapy was associated with lower fibrosis risk; corticosteroid courses were associated with increased fibrosis risk

aOR, adjusted odds ratio; BMI, body mass index; CAP, controlled attenuation parameter; CD, Crohn's disease; FIB-4, Fibrosis-4 Index; FLI, Fatty Liver Index; HSI, Hepatic Steatosis Index; IBD, inflammatory bowel disease; LSM, liver stiffness measurement; MAFLD, metabolic dysfunction-associated fatty liver disease; MTX, methotrexate; NR, not reported; TNF, tumor necrosis factor. [a]Steatosis was not quantified because the study predated CAP. [b]Arieira et al.[44] reported hepatic steatosis as a range across noninvasive methods (HSI, FLI, and CAP) rather than as a single CAP-based prevalence. [c]Reported using higher liver stiffness thresholds (van Lingen, LSM ≥7.3 kPa; Rodriguez-Duque, advanced fibrosis ≥9.7 kPa); fibrosis cutoffs differed across studies, limiting direct comparisons. [d]Prevalence in patients with IBD versus matched controls. Steatosis: Rodriguez-Duque, 42.0% vs 32.7%; ELASTIBD, 41.4% vs 28.3%. ELASTIBD significant fibrosis: 12.3% vs 4.2%. Definitions of steatosis and fibrosis varied across studies; these figures should therefore be interpreted in light of this heterogeneity.

Confirmatory Cohorts and Geographic Variation

Subsequent studies across multiple healthcare settings reinforced and further refined these findings. Arieira et al.[44] demonstrated that TE with CAP identified hepatic steatosis in a substantial proportion of patients with IBD and reclassified cases compared with clinical steatosis indices such as the HSI and FLI, highlighting the value of quantitative CAP-based assessment. van Lingen et al.[43] contributed longitudinal data showing that active IBD at baseline, defined by elevated CRP and fecal calprotectin levels, was independently associated with both increased liver fat content and fibrosis progression at 6–12 months of follow-up, providing the first prospective evidence linking IBD disease activity to hepatic outcomes over time. Trifan et al.[14] reported the highest steatosis prevalence in the literature, at 46.3% among 82 Romanian patients, with steatosis associated with older age, higher BMI, longer disease duration, and adverse metabolic markers, possibly reflecting the cohort's metabolic profile. A German retrospective single-center cohort of 132 patients further demonstrated that, although steatosis was similarly distributed between CD and UC, liver fibrosis occurred predominantly in patients with CD, suggesting a disease-specific fibrogenic pathway potentially related to transmural inflammation, intestinal failure, or nutritional compromise.[45] Two Taiwanese series, reporting NAFLD/MAFLD prevalences of 29.6% and 29.2%, showed that IBD-associated MASLD is not confined to Western populations, although absolute fibrosis rates were lower, possibly reflecting differences in metabolic background and IBD phenotype in Asian populations.[46,47] The reported prevalences of steatosis and significant fibrosis across these cohorts are compared in Figure 2.

Case-Control Evidence and the Independent Role of IBD

Cohort-only designs cannot determine whether the elevated prevalence observed in IBD reflects disease-specific mechanisms or shared metabolic risk factors. Two matched case-control studies have directly addressed this question. Rodriguez-Duque et al.[48] enrolled 831 consecutive patients with IBD alongside general-population controls at two university hospitals and found a MAFLD prevalence of 42.0% in the IBD group versus 32.7% in controls and an advanced fibrosis prevalence of 9.5% versus 2.3%, with IBD remaining an independent predictor of both outcomes after adjustment for BMI, diabetes, and other conventional metabolic covariates. Liver biopsy in a subset of 40 patients confirmed the elastography-based staging; patients with IBD-MAFLD also had a lower BMI and a lower prevalence of type 2 diabetes than severity-matched non-IBD MAFLD controls, reinforcing the concept that inflammation-driven pathways operate independently of metabolic adiposity.

The ELASTIBD study[15] individually matched 358 adult patients with IBD to 358 controls according to age, sex, BMI, and diabetes status, thereby controlling for the principal metabolic confounders. Despite this matching, patients with IBD had a higher prevalence of significant

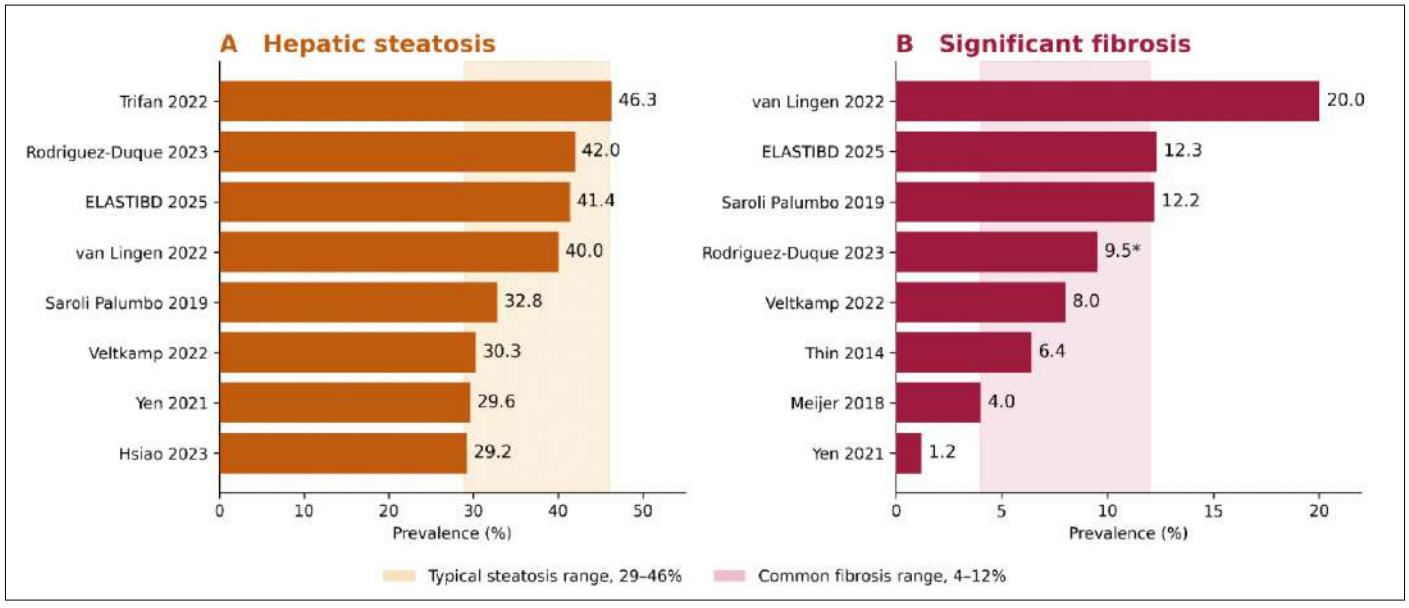

Figure 2. Reported prevalence of hepatic steatosis (panel A) and significant fibrosis (panel B) assessed by transient elastography across IBD cohorts. Shaded bands indicate the typical prevalence ranges referenced in the text (steatosis, 29–46%; fibrosis, 4–12%). Thresholds varied across studies (CAP cutoffs commonly 248–280 dB/m; LSM cutoffs for significant fibrosis, 7.0–8.0 kPa); the asterisk denotes advanced fibrosis (LSM ≥9.7 kPa) in the Rodriguez-Duque study.[48] Studies that did not report prevalence for a given parameter are omitted from the corresponding panel.

fibrosis (F≥2; 12.3% vs 4.2%) and hepatic steatosis (41.4% vs 28.3%) than controls. Because ELASTIBD excluded patients with other identifiable liver diseases, including PSC, viral hepatitis, autoimmune liver disease, and significant alcohol use, this excess could not be attributed to concurrent hepatic conditions and instead reflected an IBD-associated, predominantly metabolic and inflammatory hepatic burden, strengthening the inference of an independent IBD effect. Among patients with IBD, anti-TNF therapy was independently associated with 61% lower odds of fibrosis (aOR, 0.39; 95% CI, 0.16–0.95), whereas each corticosteroid course was associated with 41% higher odds of fibrosis (aOR, 1.41; 95% CI, 1.04–1.92). These findings provide the strongest available evidence that IBD itself, through mechanisms beyond conventional metabolic risk, contributes to hepatic fibrogenesis and that IBD-specific therapies may materially modify this risk.

Synthesis: Consistent Risk Factors Across the Literature

Despite methodological heterogeneity across studies, several risk factors for liver fibrosis and steatosis in IBD emerge consistently (Table 3). Older age and higher BMI are the most reproducibly identified predictors across virtually all cohorts.[13–15,45] Corticosteroid exposure, assessed as both cumulative dose and number of courses, is consistently associated with a higher risk of steatosis and fibrosis.[15,43] In contrast, anti-TNF therapy appears to be associated with a lower prevalence of fibrosis in multiple observational datasets.[15,24,48] Longer IBD duration correlates with steatosis in CD cohorts, whereas disease activity scores have shown an inverse association with steatosis.[15,45] Despite its validation in general MASLD populations, the FIB-4 index does not reliably correlate with elastography-defined fibrosis in patients with IBD, limiting its utility as a fibrosis-screening surrogate in this specific context.[8] A likely explanation is that both FIB-4 and APRI incorporate platelet count into their calculations; therefore, reactive thrombocytosis driven by chronic intestinal inflammation in IBD can spuriously lower these scores and mask significant fibrosis. The same inflammation-driven platelet elevation may also obscure the thrombocytopenia that would otherwise signal hypersplenism due to thiopurine-associated nodular regenerative hyperplasia and noncirrhotic portal hypertension, further favoring elastography, including spleen stiffness measurement, over platelet-based indices in this population.

Steatosis and MASLD in Inflammatory Bowel Disease

Evolving Nomenclature and Diagnostic Reclassification

The terminology for hepatic steatosis has changed considerably over the period covered by the IBD elastography literature. The transition from nonalcoholic fatty liver disease (NAFLD) to metabolic dysfunction-associated fatty liver disease (MAFLD) in 2020 and subsequently to MASLD in 2023 reflects a shift toward a positive, criteria-based diagnosis rather than one based on exclusion.[49,50] These nomenclature changes are not merely semantic: MAFLD and MASLD criteria allow concurrent alcohol use and secondary causes of steatosis to coexist with the diagnosis and reclassify patients based on the presence of at least one metabolic risk factor alongside hepatic steatosis. In IBD cohorts, this reclassification meaningfully affects prevalence estimates; not all TE-defined steatosis meets MASLD criteria, and not all patients meeting MASLD criteria have CAP-confirmed steatosis, creating important challenges when comparing studies published under different nomenclature frameworks.[50] Throughout this review, MASLD is used as the contemporary umbrella term, whereas the original terminology (NAFLD or MAFLD) is retained when reporting the findings of individual studies as originally defined by their authors.

CAP-Based Prevalence and Grade Distribution

Across IBD cohorts employing VCTE with CAP, the prevalence of hepatic steatosis ranges from 29% to 46%, with most affected patients exhibiting mild to moderate steatosis (S1–S2) rather than severe steatosis (S3).[13–15,44] The ELASTIBD study, which applied a CAP threshold of 248 dB/m in a matched-control design, found steatosis in 41.4%

Table 3. Determinants of hepatic fibrosis and steatosis in IBD and the directional effects of IBD-specific therapies. The aOR values shown for corticosteroid exposure and anti-TNF therapy are derived from the ELASTIBD matched-control cohort

Factor	Effect on hepatic outcomes	Evidence/mechanism
Older age	↑ fibrosis and steatosis	Most reproducible predictor across cohorts
Higher BMI	↑ fibrosis and steatosis	Dominant metabolic driver; effect persists after matching
Cumulative corticosteroid exposure	↑ fibrosis and steatosis	Visceral adiposity and insulin resistance; most modifiable factor (ELASTIBD aOR, 1.41 per course)
Anti-TNF therapy	↓ fibrosis	Suppression of TNF-α-driven hepatic stellate cell activation (ELASTIBD aOR, 0.39)
Thiopurines (azathioprine)	↑ NRH/portal hypertension; ↑ CAP at higher doses	Dose-related; may increase SSM despite normal LSM
Methotrexate	Minimal fibrosis risk in IBD	Lower risk than in psoriasis or rheumatoid arthritis
Longer disease duration	↑ steatosis, particularly in CD	Cumulative inflammatory burden mediated through the gut-liver axis
Active luminal disease (high activity)	Inverse association with steatosis	Nutritional depletion and lower visceral fat in active disease
Gut dysbiosis/increased permeability	↑ steatosis independent of adiposity	LPS and SCFA translocation; may contribute to lean MASLD

aOR, adjusted odds ratio; BMI, body mass index; CAP, controlled attenuation parameter; CD, Crohn's disease; IBD, inflammatory bowel disease; LPS, lipopolysaccharide; LSM, liver stiffness measurement; MASLD, metabolic dysfunction-associated steatotic liver disease; NRH, nodular regenerative hyperplasia; SCFA, short-chain fatty acids; SSM, spleen stiffness measurement; TNF, tumor necrosis factor.

of patients with IBD compared with 28.3% of individually matched controls, yielding an adjusted odds ratio of 2.51 (95% CI, 1.66–3.79) and confirming that IBD independently confers excess steatosis risk beyond shared metabolic determinants.[15] This finding is consistent with that of Rodriguez-Duque et al.,[48] who reported a higher prevalence of MAFLD in patients with IBD than in general-population controls after adjustment for metabolic factors. ELASTIBD also showed that steatosis and fibrosis frequently coexist in IBD, with elevated CAP scores independently associated with higher LSM values, suggesting that progressive steatohepatitis may be an underrecognized driver of fibrogenesis in this population.[15]

Metabolic Risk Factors: Shared and IBD-Specific

Conventional metabolic risk factors, including older age, higher BMI, increased waist circumference, type 2 diabetes, arterial hypertension, dyslipidemia, and elevated fasting glucose, are consistent predictors of steatosis in IBD across geographic regions and study designs.[13–15,44,45] However, these shared risk factors do not fully account for the excess burden of steatosis observed in patients with IBD compared with matched controls. IBD-specific contributors appear to operate through at least three partially overlapping mechanisms.

First, corticosteroid exposure promotes hepatic fat accumulation through visceral adiposity, hyperinsulinemia, and direct glucocorticoid receptor-mediated lipogenic signaling in hepatocytes.[16] The cumulative number of corticosteroid courses has been identified as an independent predictor of steatosis in multiple cohorts, and the temporal relationship between corticosteroid administration and CAP elevation is supported by longitudinal data showing that active IBD, which is often treated with corticosteroids, is associated with progression of steatosis during follow-up.[15,43]

Second, IBD disease activity exerts paradoxical, bidirectional effects on steatosis risk. In active Crohn's disease, high CDAI scores, reflecting nutritional depletion, weight loss, and negative energy balance, are inversely associated with steatosis prevalence because underweight patients with active luminal disease have less visceral fat.[15] Conversely, remission, by restoring nutritional status and promoting weight regain, sometimes accelerated by corticosteroid treatment, may increase the risk of MASLD. At the population level, however, longer disease duration and cumulative inflammatory burden appear to promote steatosis through systemic and gut-liver axis mechanisms independently of BMI.[14,45]

Third, gut microbiota dysbiosis and increased intestinal permeability, both characteristic of IBD, facilitate the translocation of bacterial products, including lipopolysaccharides and short-chain fatty acids, into the portal circulation, promoting hepatic inflammatory signaling, de novo lipogenesis, and impaired fatty acid oxidation through toll-like receptor and farnesoid X receptor pathways.[6,24] This gut-liver axis dysregulation operates independently of adiposity and may explain the occurrence of MASLD in lean patients with IBD, a subgroup in whom conventional metabolic risk factors are absent yet hepatic steatosis and even fibrosis may still be prevalent.[16,48]

IBD Therapy and Steatosis: Risk and Protection

The relationship between IBD pharmacotherapy and hepatic steatosis remains incompletely understood. Corticosteroids consistently emerge as risk factors for steatosis. Although azathioprine is primarily associated with fibrosis risk through NRH, some cohorts have also shown a positive correlation between higher azathioprine doses and CAP scores; however, whether azathioprine directly promotes steatosis or is preferentially used in patients with metabolic comorbidities remains uncertain.[15,21]

Anti-TNF therapy has attracted interest as a potential modifier of steatosis. Experimental models demonstrate that TNF-α inhibition reduces hepatic lipid accumulation and improves insulin sensitivity, while preliminary clinical data suggest a lower prevalence of MASLD among patients with IBD receiving anti-TNF agents.[24,48] Vedolizumab, a gut-selective anti-integrin agent, has shown a tentative inverse association with steatosis in observational data, possibly reflecting reduced intestinal mucosal inflammation and consequent attenuation of gut-liver axis dysregulation; however, the number of patients in available studies is insufficient to draw firm conclusions.[15] Whether biologic therapies with favorable metabolic or anti-inflammatory profiles can meaning-

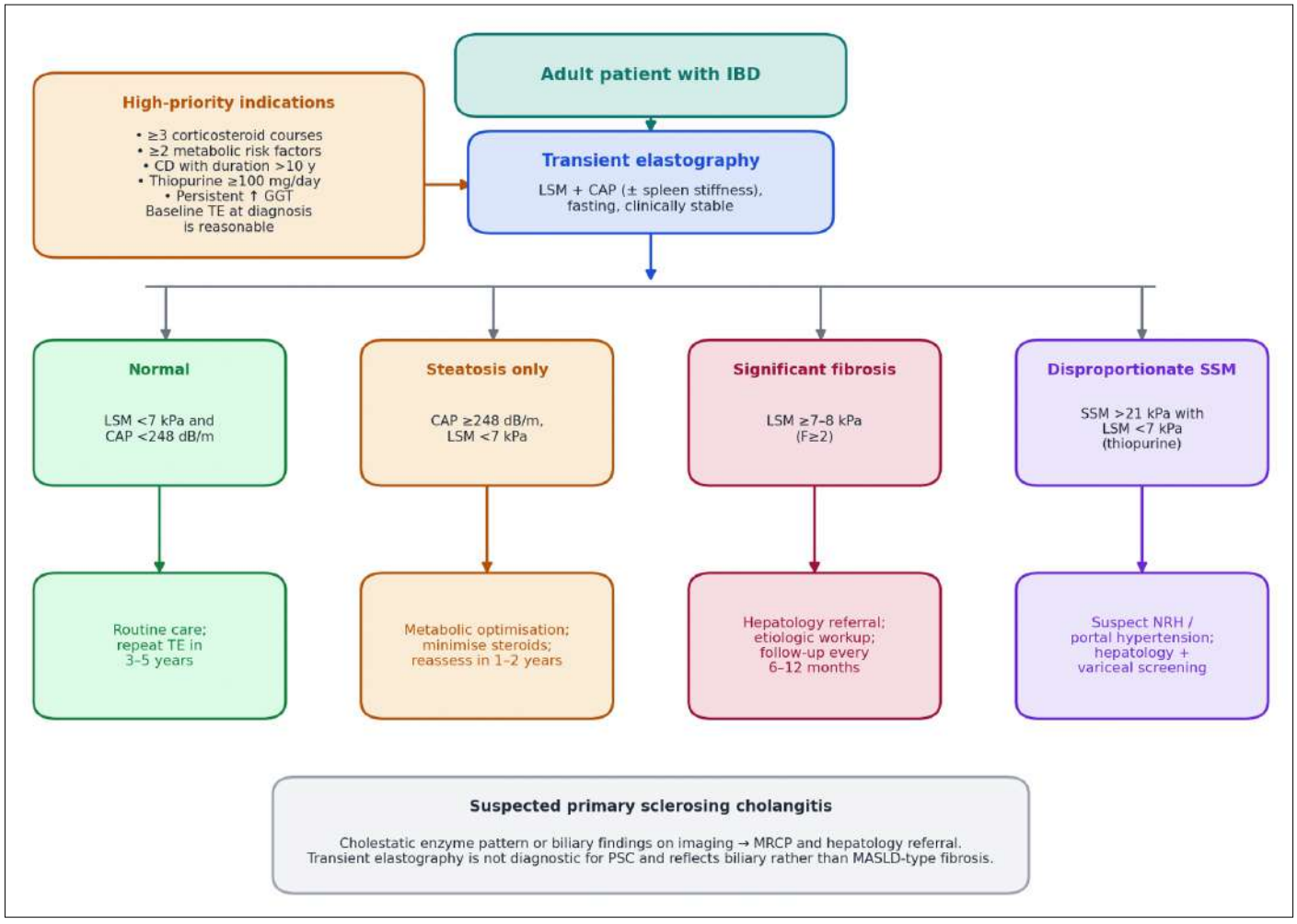

Figure 3. Proposed transient elastography screening and referral algorithm in IBD. After identification of high-priority indications, combined LSM and CAP (±SSM) assessment in a fasting, clinically stable patient stratifies management into four pathways. CAP, controlled attenuation parameter; CD, Crohn's disease; GGT, gamma-glutamyl transferase; LSM, liver stiffness measurement; MASLD, metabolic dysfunction-associated steatotic liver disease; MRCP, magnetic resonance cholangiopancreatography; NRH, nodular regenerative hyperplasia; SSM, spleen stiffness measurement.

fully reduce the burden of MASLD in IBD remains an important unanswered question.

Clinical Significance of Steatosis in IBD

Beyond its hepatic consequences, MASLD in IBD carries prognostic implications for cardiovascular risk, IBD disease behavior, and long-term outcomes. Hepatic steatosis and liver fibrosis in patients with IBD have been associated with increased atherosclerotic cardiovascular disease risk, with patients with steatosis having significantly higher calculated ASCVD risk scores than those without steatosis.[51] The coexistence of steatohepatitis and progressive fibrosis raises concern about accelerated progression to cirrhosis in a population already burdened by chronic systemic inflammation.[12] Detection of steatosis by CAP therefore provides actionable information beyond fibrosis staging alone and supports its routine inclusion in TE-based hepatic assessment protocols for patients with IBD.

Clinical Use Cases: Who, When, and How Often

Rationale for a Structured Screening Approach

Standard IBD follow-up protocols have historically prioritized monitoring of intestinal disease, including endoscopic mucosal healing, fecal calprotectin, and imaging of transmural inflammation, while hepatic surveillance has remained largely opportunistic and predominantly triggered by abnormal liver function tests. This reactive approach has an important limitation: significant fibrosis (F≥2) and clinically relevant steatosis frequently occur in patients with IBD who have normal transaminase levels.[13,15,42] A structured, proactive TE-based approach is therefore warranted, and the existing evidence base is sufficient to define risk-stratified indications (Table 4).

High-Priority Screening Indications

The following profiles carry the highest probability of significant fibrosis or steatosis and warrant priority TE assessment; these indications, together with the proposed referral pathway, are summarized in the screening algorithm (Figure 3).

Prolonged or recurrent corticosteroid exposure is the most consistently identified modifiable risk factor for both fibrosis and steatosis across IBD cohorts.[43] Patients who have received three or more corticosteroid courses or required corticosteroids for more than three cumulative

months within the preceding two years represent a priority group in whom hepatic assessment should be considered standard of care rather than optional.

Metabolic comorbidities, including obesity (BMI ≥30 kg/m²), type 2 diabetes, hypertension, and dyslipidemia, substantially amplify IBD-associated hepatic risk. Because IBD confers excess risk independently of these factors, their coexistence further compounds this risk.[13,14] All patients with IBD who have two or more metabolic risk factors should undergo baseline TE assessment at diagnosis or at the time the metabolic comorbidity is recognized.

Long disease duration, particularly exceeding 10 years in CD, has been associated with steatosis in multiple cohorts, likely reflecting cumulative inflammatory burden, nutritional compromise, and prolonged exposure to pharmacotherapy.[5,14] Patients with longstanding disease who have never undergone noninvasive hepatic assessment represent a clinically underserved group.

Thiopurine use at azathioprine doses ≥100 mg/day or an equivalent dose is also relevant, given the documented association between higher thiopurine doses and both increased spleen stiffness and NRH risk.[21,37] In this subgroup, SSM in addition to LSM may provide added value by identifying portal hypertension in the presence of otherwise normal hepatic parenchyma, a pattern characteristic of thiopurine-associated NRH.[37]

Persistently abnormal liver enzyme levels, particularly GGT elevation, which shows the strongest correlation with both fibrosis and steatosis in IBD, should prompt formal TE assessment to characterize the underlying hepatic pathology beyond the enzyme pattern alone.[13,15]

Baseline Assessment at IBD Diagnosis

A reasonable case can be made for offering TE assessment at the time of IBD diagnosis, independent of risk factors, for two reasons. First, the prevalence of clinically significant hepatic findings in newly diagnosed patients with IBD is not negligible, and early identification may allow timely hepatology referral and inform treatment decisions, such as consideration of corticosteroid-sparing strategies in patients with established steatohepatitis. Second, a baseline measurement provides an essential comparator for monitoring hepatic progression or regression in response to IBD treatment, an area in which data remain limited because existing studies are predominantly cross-sectional. Where universal screening is not feasible, a risk factor-guided approach targeting the high-priority indications outlined above provides a pragmatic alternative. No prospective outcome or cost-effectiveness data currently support universal baseline screening; this proposal is based on biological and clinical rationale rather than high-level evidence.

Follow-Up Intervals and Monitoring

Evidence regarding optimal follow-up intervals for TE-based hepatic monitoring in IBD is limited, as available longitudinal data are confined to a single study with a 6–12-month follow-up period.[43] Based on this evidence and extrapolation from MASLD monitoring recommendations in the general population, the following pragmatic framework is proposed (Table 4): in patients with IBD who have normal baseline LSM and CAP values and no high-risk features, repeat TE every three to five years; in patients with baseline steatosis (CAP ≥248 dB/m) but no significant fibrosis (LSM <8 kPa), annual or biennial reassessment is warranted alongside corticosteroid minimization and metabolic optimi-

Table 4. Proposed risk-stratified framework for transient elastography-based hepatic screening and monitoring in IBD

Clinical scenario	Recommended transient elastography action
≥3 corticosteroid courses or >3 cumulative months of corticosteroid therapy within 2 years	Perform baseline TE; consider this part of standard care
≥2 metabolic risk factors (obesity, T2DM, hypertension, or dyslipidemia)	Perform baseline TE at diagnosis or when metabolic comorbidity is recognized
Long-standing disease, particularly CD >10 years	Perform baseline TE if hepatic assessment has not been performed previously
Thiopurine ≥100 mg/day (azathioprine equivalent)	Perform TE with SSM to screen for NRH/portal hypertension
Persistently abnormal liver enzyme levels, particularly elevated GGT	Perform TE to characterize the underlying hepatic pathology
Normal LSM and CAP with no high-risk features	Repeat TE every 3–5 years
Steatosis (CAP ≥248 dB/m) without fibrosis (LSM <8 kPa)	Reassess annually or biennially; minimize corticosteroid exposure and optimize metabolic risk factors
Elevated LSM: 8–12 kPa (indeterminate) or ≥12 kPa (advanced fibrosis likely)	For LSM 8–12 kPa, perform confirmatory testing or seek hepatology consultation; for LSM ≥12 kPa, refer to hepatology and repeat TE every 6–12 months
Severe steatosis (CAP ≥280 dB/m) ± elevated liver enzyme levels	Provide metabolic counseling; refer to hepatology if steatohepatitis is suspected
SSM >21 kPa with LSM <7 kPa in a thiopurine-treated patient	Suspect NRH; refer to hepatology and perform endoscopic screening for varices
Suspected PSC based on a cholestatic pattern or biliary imaging findings	Perform MRCP and refer to hepatology regardless of the TE result
New or escalating corticosteroid therapy in a patient with known steatosis or borderline LSM	Reassess within 6 months after completion of the corticosteroid course

CAP, controlled attenuation parameter; CD, Crohn's disease; GGT, gamma-glutamyl transferase; IBD, inflammatory bowel disease; LSM, liver stiffness measurement; MRCP, magnetic resonance cholangiopancreatography; NRH, nodular regenerative hyperplasia; PSC, primary sclerosing cholangitis; SSM, spleen stiffness measurement; T2DM, type 2 diabetes mellitus; TE, transient elastography.

zation; and in patients with significant fibrosis (F≥2, LSM ≥7–8 kPa), prompt hepatology referral is indicated, with TE reassessment every six to twelve months to monitor progression. Any new or escalating corticosteroid exposure in a patient with known hepatic steatosis or borderline LSM values should prompt reassessment within six months after completion of the corticosteroid course.

When to Refer to Hepatology

Clear thresholds for hepatology referral can be derived from existing evidence. In a fasting, clinically stable patient with IBD without acute hepatic congestion or significant cholestasis, LSM is best interpreted using a pragmatic dual-cutoff approach: values below 8 kPa make clinically significant fibrosis unlikely, values of 8–12 kPa are indeterminate and warrant a confirmatory noninvasive test or hepatology consultation, and values of 12 kPa or higher indicate probable advanced fibrosis and should prompt hepatological evaluation regardless of liver enzyme status.[29,38,42] Isolated CAP elevation without fibrosis warrants structured metabolic counseling within the IBD multidisciplinary team, with hepatology involvement when steatosis is severe (CAP ≥280 dB/m) or accompanied by elevated liver enzyme levels suggestive of steatohepatitis.[31] Spleen stiffness values that are disproportionately elevated relative to LSM, particularly SSM >21 kPa with LSM <7 kPa in a thiopurine-treated patient, should raise suspicion for NRH and prompt dedicated hepatological assessment, including upper gastrointestinal endoscopy for variceal screening.[34,35,37] Suspected PSC based on a cholestatic enzyme pattern, IBD disease characteristics, or incidental biliary findings on cross-sectional imaging warrants hepatology referral independently of TE findings because LSM in PSC reflects biliary fibrosis rather than MASLD-type hepatic fibrosis and requires disease-specific interpretation.[18]

Limitations and Research Gaps

Cutoff Heterogeneity and the Absence of IBD-Specific Validation

The most pervasive limitation is the absence of IBD-specific LSM cutoffs validated against liver biopsy in a sufficiently powered, well-characterized cohort. All thresholds currently applied in IBD studies are derived from general chronic liver disease populations, predominantly patients with viral hepatitis or NAFLD, and their diagnostic performance in IBD has not been formally established.[27,29] IBD-specific confounders, including active intestinal inflammation, thiopurine-associated NRH, and PSC-related biliary fibrosis, can each distort the relationship between LSM and fibrosis in ways that general-population cutoffs cannot fully accommodate.[21,43] A prospective IBD cohort with paired liver biopsy and TE measurements, although ethically challenging to design in a population without primary hepatological indications for biopsy, remains necessary for evidence-based threshold calibration.

Predominance of Cross-Sectional Designs

With the sole exception of van Lingen et al.,[43] every original TE study in IBD identified in this review employed a cross-sectional design. This limitation has three important consequences. First, the directionality of observed associations cannot be established from prevalence data alone; for example, it remains unclear whether corticosteroid exposure causes fibrosis or whether patients with more severe disease receive more corticosteroids and independently have worse hepatic outcomes. Second, the natural history of IBD-associated liver disease remains largely unknown: whether steatosis progresses to fibrosis, whether remission reverses hepatic abnormalities, and what proportion of patients with F≥2 fibrosis progress to cirrhosis remain unanswered because of the lack of longitudinal TE data. Third, the impact of therapeutic escalation, including biologic initiation, corticosteroid withdrawal, or metabolic interventions, on hepatic outcomes cannot be assessed without repeated measurements over time. Prospective longitudinal cohort studies with TE reassessment at predefined intervals, ideally linked to therapeutic milestones, represent the most pressing methodological need in this field.

Paucity of Matched Control Data

Most IBD elastography studies lack a control group, precluding quantification of the excess hepatic risk attributable to IBD itself beyond shared metabolic determinants. Of the 13 studies reviewed, only three included a non-IBD comparator group, and only two, Rodriguez-Duque et al.[48] and ELASTIBD,[15] used individual matching for key metabolic confounders. This gap has created uncertainty regarding whether the observed prevalence reflects a disease-specific burden or merely the metabolic profile of a chronically ill, often overweight, and frequently corticosteroid-treated population. The matched-control evidence currently available consistently suggests that IBD confers an independent hepatic risk; however, this conclusion rests on a limited evidence base and requires corroboration in larger, multicenter, prospectively enrolled cohorts.

Small Sample Sizes and Population Heterogeneity

Several cohorts in the existing literature are underpowered for subgroup analyses, particularly disease-specific comparisons between CD and UC, which may have distinct hepatic risk profiles because of differences in inflammatory mechanisms, nutritional consequences, and pharmacotherapy patterns.[41,45] Sample sizes ranging from 46 to 384 patients in the pre-ELASTIBD literature limit the precision of prevalence estimates and the statistical power available for multivariable adjustment.[13,22] Cohorts also differ in IBD phenotype, disease activity, medication exposure, geographic and ethnic background, and the stringency of exclusion criteria for alcohol use and other concurrent liver diseases, including viral hepatitis, PSC, and autoimmune liver disease, all of which contribute to the wide prevalence ranges reported for both fibrosis and steatosis and complicate meta-analytic pooling. In particular, cohorts that did not exclude concurrent liver disease may overestimate fibrosis prevalence, for example through occult PSC-related biliary fibrosis or viral hepatitis, whereas MAFLD-based definitions explicitly permit coexisting etiologies, introducing an additional source of between-study variation.

Absence of Pediatric Data and Lean IBD Subgroups

The entire TE-based literature on hepatic involvement in IBD has focused on adult populations, leaving pediatric and adolescent patients with IBD essentially unstudied using objective noninvasive assessment, despite their potentially distinct hepatic risk trajectory related to cumulative corticosteroid exposure, impaired nutritional growth, and long disease duration beginning at an early age.[4,5] Similarly, lean patients with IBD (BMI <25 kg/m²) and MASLD remain undercharacterized in the TE literature. In this subgroup, conventional metabolic risk stratification may fail and clinical suspicion may be particularly low, despite emerging evidence that IBD-specific mechanisms can drive hepatic steatosis and fibrosis independently of adiposity.[48]

Spleen Stiffness and Emerging Parameters

SSM in IBD remains at an early investigative stage, with available data limited to preliminary, single-center observations.[37] The clinical utility of SSM for identifying thiopurine-associated NRH and noncirrhotic portal hypertension in patients with IBD and normal LSM has been demonstrated at the case level but lacks prospective cohort val-

idation. Future studies incorporating SSM alongside LSM and CAP in longitudinal IBD cohorts, particularly among thiopurine-treated patients undergoing dose escalation, would substantially advance the field. Similarly, newer elastography-derived parameters, including the FibroScan-AST (FAST) score for identifying at-risk steatohepatitis, have not been evaluated in dedicated IBD cohorts and represent an additional investigative opportunity.[52] More broadly, transient elastography is increasingly being applied beyond hepatic fibrosis and steatosis to other organs and systemic complications relevant to IBD; liver and transplanted-kidney stiffness measurements have recently been used to characterize organ involvement in AA amyloidosis, a systemic sequela of chronic inflammatory disease, illustrating the broader potential of this technique.[53]

CONCLUSION

Transient elastography has emerged as the most practical and evidence-supported noninvasive tool for hepatic assessment in inflammatory bowel disease, enabling the simultaneous quantification of liver fibrosis, hepatic steatosis, and portal hypertension through spleen stiffness measurement. The evidence reviewed here indicates that patients with IBD carry a hepatic burden exceeding that predicted by conventional metabolic risk factors alone. Matched-control data establish IBD itself as an independent determinant of both fibrosis and steatosis, with corticosteroid exposure emerging as the most consistently modifiable risk factor and anti-TNF therapy as potentially hepatoprotective.

Despite this accumulating evidence, hepatic assessment remains systematically underused in IBD clinical practice. Standard liver function tests are insufficient for detecting the subclinical fibrosis and steatosis that TE can reliably identify, and the absence of structured TE protocols in IBD guidelines represents a gap between evidence and practice. Integration of FibroScan® into IBD outpatient workflows, stratified by corticosteroid exposure, metabolic comorbidity burden, disease duration, and pharmacotherapy profile, is both feasible and clinically warranted based on existing data.

Key research priorities include the development of IBD-specific, biopsy-validated LSM cutoffs; longitudinal evaluation of the natural history of IBD-associated liver disease and its response to therapy; and extension of TE-based assessment to pediatric and lean IBD subgroups in whom conventional risk stratification may fail. The role of spleen stiffness measurement in monitoring thiopurine-associated portal hypertension and the potential utility of newer composite elastography parameters, such as the FAST score, for identifying at-risk steatohepatitis in IBD cohorts represent additional areas warranting prospective evaluation.

As IBD management increasingly targets deep remission and long-term organ preservation beyond the intestine, the liver should be recognized as a target organ requiring systematic surveillance. Transient elastography, which is noninvasive, reproducible, and supported by a growing body of controlled evidence, provides a practical clinical tool to meet this need.

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