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Beyond the Gut: Metabolic Dysfunction-associated Steatotic Liver Disease in Inflammatory Bowel Disease

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Metabolic dysfunction-associated steatotic liver disease (MASLD) has emerged as a frequent and clinically meaningful comorbidity in inflammatory bowel disease (IBD). Once considered incidental, MASLD is increasingly recognized as a marker of systemic metabolic and inflammatory burden with important hepatic and extrahepatic consequences. Recent studies indicate that MASLD affects approximately 24–32% of patients with IBD, with similar prevalence in Crohn's disease and ulcerative colitis after adjustment for metabolic risk factors. Importantly, liver fibrosis, the key prognostic hallmark in chronic liver disease, is detected in a substantial subset of patients and appears to progress over time. The pathogenesis of MASLD in IBD reflects the convergence of classical metabolic dysfunction with IBD-specific factors, including chronic systemic inflammation, gut–liver axis alterations, and changes in body composition. Although IBD is not yet systematically included in MASLD screening recommendations, emerging epidemiological and longitudinal evidence supports adapting established metabolic screening pathways to the IBD population using a risk-stratified case- finding approach. Pragmatic two-step algorithms, employing simple serum-based scores followed by transient elastography in selected patients, offer scalable and implementation-ready strategies. Management of MASLD in patients with IBD should align with general population guidelines while accounting for disease-specific considerations. Lifestyle modification remains foundational, complemented by pharmacologic therapies in patients with fibrosis or high cardiometabolic risk. Integrating liver risk assessment into routine IBD care represents a critical step toward improving long-term hepatic and extrahepatic outcomes in this growing population.

Introduction

Inflammatory bowel disease (IBD) is a chronic immune-mediated condition with a steadily rising global burden, with Canada reporting among the highest prevalence worldwide.[1] Once considered as a disorder confined to the gastrointestinal tract, IBD is now recognized as a systemic disorder characterized by chronic inflammation and a growing burden of extraintestinal comorbidities that influence long-term outcomes.[2] As disease control and survival improve, metabolic health has emerged as a key determinant of morbidity in people living with IBD.[3-6] In parallel, metabolic dysfunction-associated steatotic liver disease (MASLD), formerly termed non-alcoholic fatty liver disease, has become the most prevalent chronic liver disease worldwide, affecting approximately one-third of adults.[7-9] In 2023, an international consensus redefined fatty liver disease under the umbrella term steatotic liver disease, within which MASLD represents the dominant subtype, characterized by hepatic steatosis in the presence of cardiometabolic risk factors.[8] This nomenclature shift reflects advances in understanding disease biology, removes stigmatizing terminology, and emphasizes the systemic, metabolism-driven nature of the condition. The convergence of IBD and MASLD is therefore increasingly relevant. Obesity, insulin resistance, type 2 diabetes (T2D), and dyslipidemia—once considered uncommon in IBD—are now increasingly prevalent as disease duration lengthens and treatment paradigms evolve.[3-6] In contemporary IBD cohorts, abnormal liver enzymes are observed in up to 30% of patients, with MASLD representing one of the most frequent underlying causes encountered in clinical practice.[10-12] Despite this, MASLD in IBD has often been regarded as a benign or incidental finding, overshadowed by concerns related to drug-induced liver injury or immune-mediated hepatobiliary disorders. Accumulating evidence challenges this perception. In patients with IBD, MASLD has been associated with liver fibrosis, cardiovascular disease, adverse hospitalization outcomes, and increased healthcare utilization.[13-15] This review synthesizes current evidence on the epidemiology, pathophysiology, natural history, and clinical implications of MASLD in IBD, with particular emphasis on longitudinal data and non-invasive diagnostic strategies that are reshaping care paradigms as we approach 2026 and beyond.

Epidemiology of MASLD in IBD

Canada has one of the highest IBD prevalence rates globally, with 1 in 140 Canadians living with Crohn's disease or ulcerative colitis. Over 270,000 Canadians were affected in 2018, a number projected to exceed 400,000 by 2030.[1,16] As the IBD population grows, metabolic health

is worsening: obesity now affects 15% to 40% of patients,[3] and the incidence of T2D and dyslipidemia is higher than that observed in the general population.[4-6] Together with chronic intestinal inflammation and exposure to hepatotoxic therapies, these factors likely elevate the risk of steatotic liver disease. Globally, MASLD affects approximately 32% of adults and represents the most common chronic liver disease.[7-9] Its growing burden parallels the global epidemics of obesity and T2D, which are the primary drivers of hepatic steatosis and fibrosis progression. MASLD is a heterogeneous condition, with clinical presentation and prognosis shaped by metabolic risk, systemic inflammation, and extrahepatic disease states.[7-9] Within this context, MASLD is frequently observed among patients with IBD. Reported prevalence estimates typically range between 24% and 32%, though values vary widely (6.9%–53.8%) depending on diagnostic methods, study design, and population characteristics.[17] Studies relying on administrative codes or conventional ultrasonography consistently underestimate disease burden, whereas those using modern non-invasive tools—such as transient elastography or validated biomarker-based scores—report substantially higher prevalence estimates. A meta-analysis including more than 14,000 individuals from 18 countries reported a pooled MASLD prevalence of approximately 36% among patients with IBD, comparable to that observed in the general population.[18] These findings underscore the dominant role of metabolic dysfunction, while highlighting IBD as a population in which MASLD is frequently underrecognized. Prospective screening cohorts using transient elastography have provided important clarity into disease burden. In a Canadian cohort of 384 adults with IBD who underwent systematic transient elastography screening, MASLD was identified in 32.8% of patients despite a relatively young median age, and was accompanied by a substantial prevalence of significant liver fibrosis (12.2%).[19] From a global perspective, the intersection of IBD and MASLD is likely to intensify as IBD incidence rises in newly industrialized regions alongside rapid increases in obesity and T2D. These populations remain underrepresented in current studies, suggesting that the true global burden of MASLD in IBD is likely underestimated.

Pathophysiology: Why MASLD is More Complex in IBD

The pathogenesis of MASLD in IBD extends beyond classical metabolic dysfunction and reflects the convergence of metabolic, inflammatory, and intestine-derived mechanisms (**Figure 1**). While insulin resistance and adipose tissue dysfunction remain central drivers, IBD-specific factors may modify disease susceptibility and progression. Chronic systemic inflammation is a shared hallmark of both conditions. Persistent inflammatory signalling promotes hepatic lipid accumulation, oxidative stress, and fibrogenesis.[17,20] In IBD, recurrent disease flares may amplify these pathogenic pathways, although direct associations between disease activity indices and MASLD severity remain inconsistent. The gut–liver axis plays a pivotal role. Increased intestinal permeability, dysbiosis, and altered bile acid signalling enhance hepatic exposure to microbial products and pro-inflammatory mediators.[17] These mechanisms may be particularly relevant in Crohn's disease with small-bowel involvement, where transmural inflammation and mesenteric adipose tissue activation further contribute to metabolic dysregulation. Additional complexity is introduced by alterations in body composition. Sarcopenia is common in IBD, even among individuals with normal or elevated body mass index, contributing to the increasingly recognized phenotype of lean MASLD.[21,22] The impact of IBD therapies on MASLD development and progression remains incompletely defined. Corticosteroid exposure may promote visceral adiposity and insulin resistance, thereby amplifying hepatic steatosis and fibrotic risk, and is widely considered a modifiable contributor. Although methotrexate is not a primary driver of MASLD, it may increase susceptibility to liver injury in the presence of underlying steatosis, underscoring the importance of baseline liver risk stratification. Thiopurines are not associated with steatosis but may cause alternative hepatic complications, including cholestasis and nodular regenerative hyperplasia.[17,18] Biologic therapies, including anti-tumour necrosis factor (TNF) agents, vedolizumab, and ustekinumab, are generally considered metabolically neutral, while emerging data suggests that TNF inhibition may confer potential protective metabolic effects. Janus kinase inhibitors are associated with lipid profile

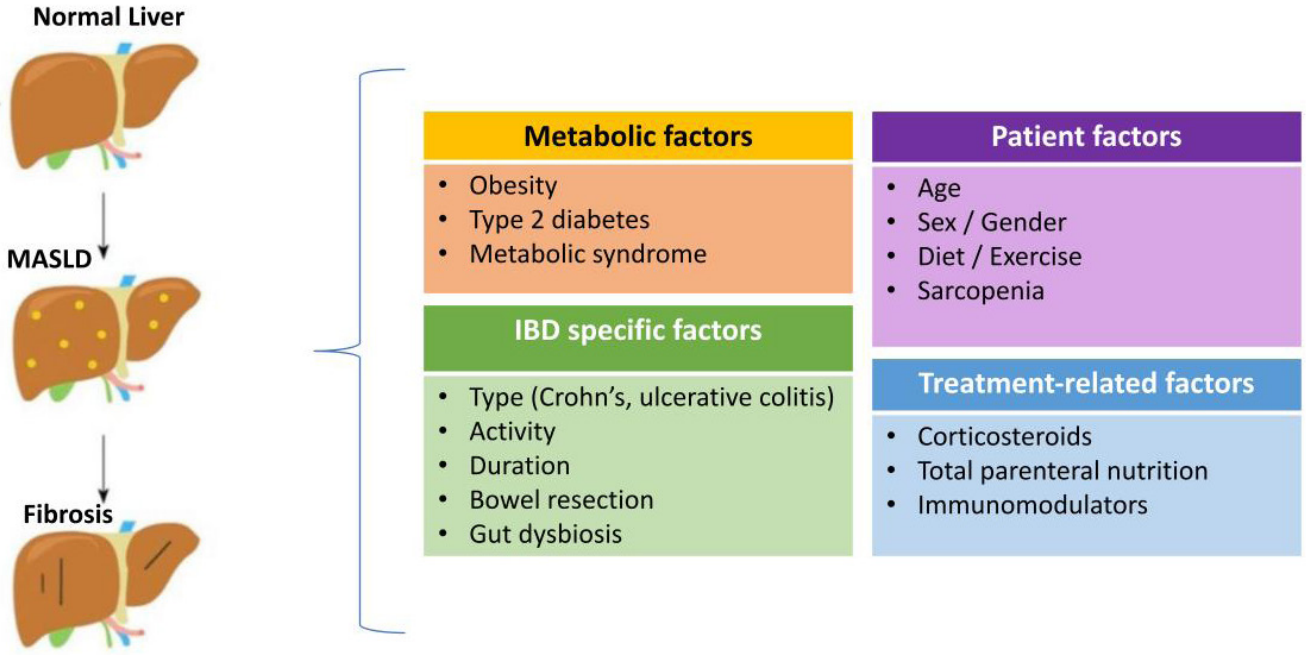

Figure 1. Factors implicated in the development of MASLD; *courtesy of Giada Sebastiani, MD, FAASLD.*

Abbreviations: MASLD: metabolic-dysfunction-associated steatotic liver disease

changes; however, their long-term hepatic impact remains unknown.[17]

Collectively, these mechanisms support the concept that MASLD in IBD is not a coincidental comorbidity, but rather the result of intersecting metabolic and inflammatory processes. Despite disease-specific pathogenic pathways, epidemiological studies consistently demonstrate similar MASLD prevalence in Crohn's disease and ulcerative colitis after adjustment for metabolic risk factors. Across IBD populations, older age, male sex, obesity, T2D, and dyslipidemia remain the most consistently reported associated factors associated with MASLD burden.[17-19]

Diagnostic Considerations, Fibrosis Risk Stratification, and Disease Progression

Although MASLD is common in IBD, its natural history was long considered relatively benign. Early cross-sectional studies reported low prevalence of advanced fibrosis, reinforcing this perception. However, more recent longitudinal studies using contemporary non-invasive tools challenge this view. In cohorts systematically assessed with transient elastography, liver fibrosis has been detected in 12%–14% of unselected IBD patients—rates that appear higher than those reported in the general population.[19,23] Longitudinal follow-up with serial biomarkers further demonstrates that fibrosis progression is not uncommon, with reported rates of approximately 0.5 per 100 person-years, while steatosis progression may exceed 9 per 100 person-years.[24] These findings suggest that fibrosis risk in IBD has been underestimated due to short follow-up, inconsistent diagnostic approaches, and selection bias. Although fibrosis progression appears primarily driven by metabolic risk rather than IBD activity alone, a subset of patients develops fibrosis in the absence of overt MASLD, pointing to additional contributory factors, including inflammatory, nutritional, or treatment-related influences. MASLD in IBD is increasingly recognized as a marker of systemic risk. Cardiovascular disease is of particular concern, with higher rates of incident events observed among IBD patients with MASLD, especially in those with fibrosis, lean MASLD, or combined metabolic dysfunction.[13,14] MASLD has also been associated with worse hospitalization outcomes, including longer length of stay, increased complication rates, and greater healthcare

utilization, reinforcing its role as a marker of global metabolic and inflammatory burden.[15]

Current clinical practice guidelines increasingly recommend case-finding for MASLD-related advanced fibrosis in high-risk metabolic populations, particularly individuals with prediabetes or T2D, in whom fibrosis is the key determinant of hepatic and extrahepatic outcomes.[25,26] These guidelines endorse the Fibrosis-4 index (FIB-4) as a first-line triage tool, followed by second-line testing, most commonly transient elastography, for individuals with indeterminate or high-risk results. In these care pathways, pragmatic thresholds are typically applied: an FIB-4 score <1.3 indicates a low likelihood of advanced fibrosis and supports periodic reassessment and cardiometabolic risk management; values between 1.3 and 2.67 suggest indeterminate risk warranting further evaluation; and an FIB-4 score >2.67 supports referral for specialist assessment due to higher probability of advanced fibrosis. Although IBD is not yet systematically included in MASLD screening recommendations, accumulating epidemiological and natural-history data support adapting established metabolic screening pathways to IBD using a risk-stratified case-finding approach rather than universal screening. In clinical practice, this strategy may prioritize IBD patients with readily identifiable triggers, including: **a)** steatosis detected on imaging (ultrasound, computed tomography, or magnetic resonance imaging); **b)** persistently abnormal liver enzymes after exclusion of alternative etiologies and acute drug-induced liver injury; **c)** overweight or obesity, including central adiposity; **d)** prediabetes or T2D; and **e)** a high cardiometabolic risk profile, such as multiple metabolic comorbidities and increasing age. Operationally, an IBD-adapted algorithm can mirror the scalable two-step screening pathways already used in metabolic populations. This approach employs the FIB-4 index calculated from routinely available laboratory tests as a first-line assessment, followed by transient elastography (or an alternative validated second-line test where available) for patients with indeterminate or high FIB-4 scores or compelling clinical suspicion.[25,26] This strategy relies on widely accessible, low-cost tools that integrate seamlessly into gastroenterology workflows, while reserving elastography capacity and hepatology referral for those most likely to benefit. As the global metabolic disease burden rises in parallel with IBD incidence, such implementation-ready strategies will be essential to prevent delayed diagnosis of advanced fibrosis and to embed liver health within comprehensive IBD care.

Management and Treatment of MASLD in IBD

Management of MASLD in patients with IBD should align with principles established in the general population, while accounting for IBD-specific factors that may influence feasibility, adherence, and risk-benefit considerations. Current guidelines emphasize a stage-specific approach, with particular focus on patients with liver fibrosis, given its central role in determining liver-related and extrahepatic outcomes. Lifestyle modification remains the cornerstone of MASLD management.[25,26] Sustained weight loss is the most effective intervention for reducing hepatic steatosis and improving fibrosis, with evidence indicating that a ≥7%–10% reduction in body weight is associated with histological improvement and fibrosis regression. Dietary interventions should prioritize caloric reduction and cardiometabolic benefit rather than restrictive, disease-specific exclusion diets. A Mediterranean-style diet, rich in mono- and polyunsaturated fats, fibre, and plant-based foods, has been associated with improvements in hepatic steatosis and cardiovascular risk and is generally well tolerated in patients with stable IBD.[27] Regular aerobic and resistance exercise (targeting 150–200 minutes per week across 3–5 sessions) improves insulin sensitivity and reduces liver fat and should be encouraged even in the absence of substantial weight loss.[25,26] In IBD populations, particular attention is warranted for sarcopenia and altered body composition, as weight loss strategies that exacerbate muscle loss may worsen functional status and metabolic risk.[21,22] Multidisciplinary care involving dietitians familiar with both MASLD and IBD is therefore essential. Pharmacologic therapy should be considered in patients with MASLD who have moderate-to-advanced fibrosis, progressive disease despite lifestyle intervention, or high cardiometabolic risk. Among available options, glucagon-like peptide-1 receptor agonists (GLP-1 RAs) currently have the strongest evidence base. In 2025, Health Canada approved semaglutide for the treatment of metabolic dysfunction-associated steatohepatitis (MASH) with stage 2–3 fibrosis, based on results from the

phase 3 ESSENCE trial.[28] Beyond liver-specific benefits, GLP-1 RAs reduce major adverse cardiovascular events and improve glycemic control, positioning them as dual-purpose agents in MASLD management. Although data in IBD-specific populations remain limited, available evidence does not suggest increased risk of IBD disease activity. Nonetheless, careful monitoring during treatment initiation is advised, particularly in patients with active disease or prominent gastrointestinal symptoms.

MASLD is a strong marker of systemic cardiometabolic risk, and cardiovascular disease remains the leading cause of mortality in this population.[26] Accordingly, aggressive management of cardiovascular risk factors is a core component of MASLD care in patients with IBD. Statins are safe and recommended in patients with MASLD, including those with compensated liver cirrhosis, and should not be withheld because of mild liver enzyme elevations.[26] Statin therapy reduces cardiovascular events and may also confer modest hepatic benefit. Blood pressure control, glycemic optimization, and smoking cessation should likewise be addressed systematically as part of an integrated care strategy.

Importantly, MASLD management should not be siloed from IBD care. Treatment decisions should be integrated into routine gastroenterology follow-up, with close coordination among IBD specialists, hepatologists, endocrinologists, and primary care providers. Minimizing cumulative corticosteroid exposure, optimizing IBD control, and selecting therapies that do not exacerbate metabolic risk are key complementary strategies. As pharmacologic options for MASH continue to evolve, patients with IBD and MASLD—particularly those with fibrosis—represent a priority group in whom early identification and treatment may substantially alter long-term hepatic and extrahepatic outcomes.

Clinical Implications and Future Directions

The recognition of MASLD as a common and clinically meaningful comorbidity in IBD has important implications for clinical practice (**Figure 2**). Universal screening is unlikely to be feasible or cost-effective. Instead, a targeted, risk-stratified approach focused on patients with metabolic risk factors, persistently abnormal liver enzymes, or incidental findings of steatosis on imaging is warranted. In this context, non-invasive tools, including serum-based scores and transient elastography, provide practical and scalable options for liver risk assessment within IBD clinics. MASLD management should be integrated into routine IBD care rather than treated as a parallel condition. Lifestyle interventions, optimization of metabolic risk factors, and minimization of corticosteroid exposure remain foundational. Key research priorities include defining IBD phenotypes at highest risk for fibrosis progression, clarifying interactions between IBD therapies and liver outcomes, and evaluating whether MASLD-targeted interventions improve hepatic and extrahepatic outcomes. As the global prevalence of both IBD and metabolic disease continue to rise, MASLD will increasingly shape long-term outcomes in this population. Recognizing and addressing MASLD as a common comorbidity represents a critical step toward more comprehensive, patient-centred IBD care.

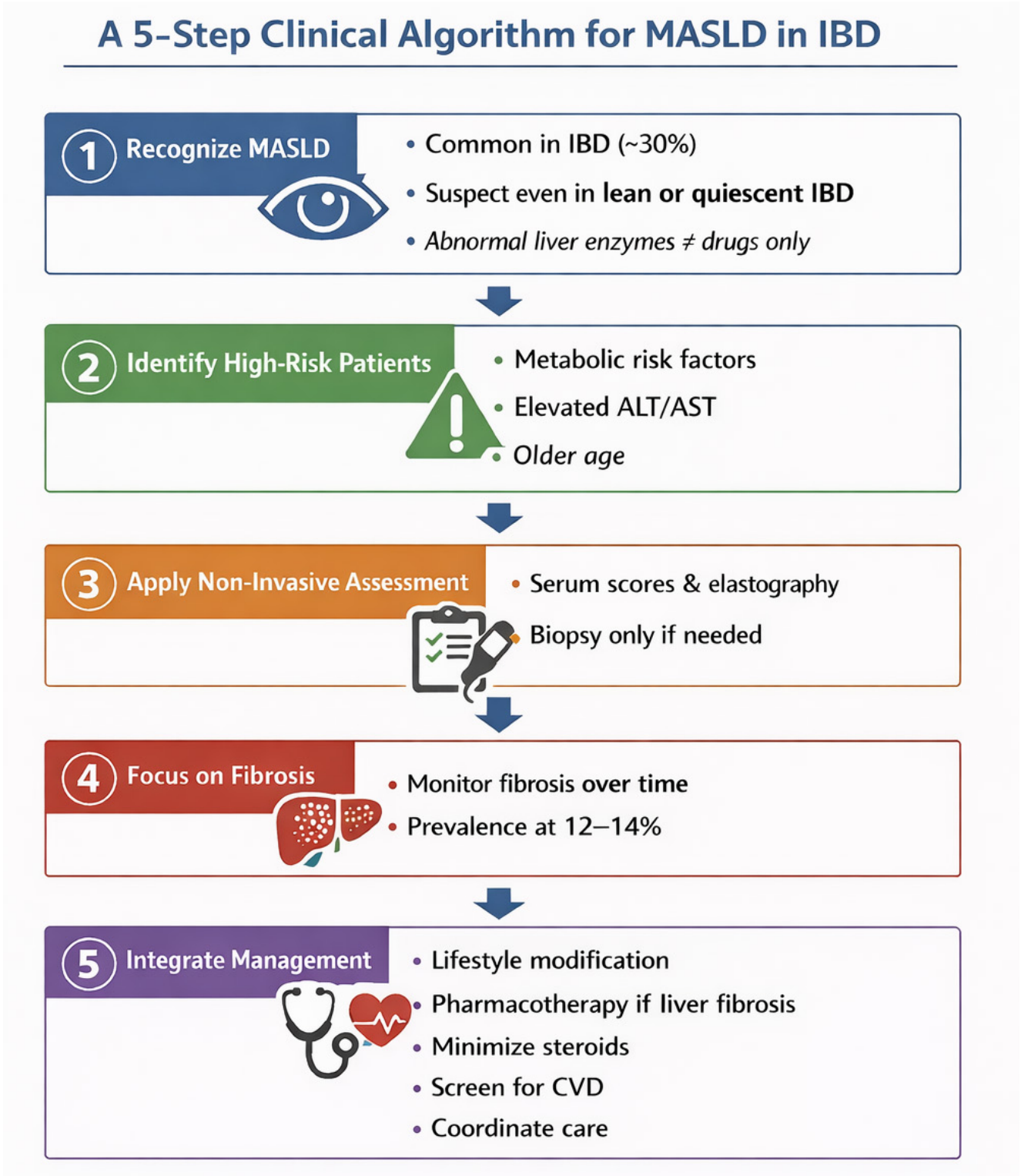

Figure 2. Five-step clinical algorithm for MASLD in IBD; *courtesy of Giada Sebastiani, MD, FAASLD.*

Abbreviations: CVD: cardiovascular disease; **ALT:** alanine aminotransferase; **AST:** aspartate aminotransferase; **IBD:** inflammatory bowel disease; **MASLD:** metabolic-dysfunction-associated steatotic liver disease

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References

1. Kaplan GG, Bernstein CN, Coward S, Bitton A, Murthy SK, Nguyen GC, et al. The impact of inflammatory bowel disease in Canada 2018: epidemiology. J Can Assoc Gastroenterol. 2019;2(Suppl 1):S6–S16. doi:10.1093/jcag/gwy054
2. Bernstein CN, Benchimol EI, Bitton A, Murthy SK, Nguyen GC, Lee K, et al. The Impact of inflammatory bowel disease in Canada 2018: extra-intestinal diseases in IBD. J Can Assoc Gastroenterol. 2019;2(Suppl 1):S73–S80. doi:10.1093/jcag/gwy053
3. Singh S, Dulai PS, Zarrinpar A, Ramamoorthy S, Sandborn WJ. Obesity in IBD: epidemiology, pathogenesis, disease course and treatment outcomes. Nat Rev Gastroenterol Hepatol. 2017;14(2):110–121. doi:10.1038/nrgastro.2016.181
4. Sang MM, Sun ZL, Wu TZ. Inflammatory bowel disease and diabetes: is there a link between them? World J Diabetes. 2022;13(2):126–128. doi:10.4239/wjd.v13.i2.126
5. Jess T, Jensen BW, Andersson M, Villumsen M, Allin KH. Inflammatory bowel diseases increase risk of type 2 diabetes in a nationwide cohort study. Clin Gastroenterol Hepatol. 2020;18(4):881–888.e881. doi:10.1016/j.cgh.2019.07.052
6. Koutroumpakis E, Ramos-Rivers C, Regueiro M, Hashash JG, Barrie A, Swoger J, et al. Association between long-term lipid profiles and disease severity in a large cohort of patients with inflammatory bowel disease. Dig Dis Sci. 2016;61(3):865–871. doi:10.1007/s10620-015-3932-1
7. Beaton MD. Current treatment options for nonalcoholic fatty liver disease and nonalcoholic steatohepatitis. Can J Gastroenterol. 2012;26(6):353–357. doi:10.1155/2012/725468
8. Rinella ME, Lazarus JV, Ratziu V, Francque SM, Sanyal AJ, Kanwal F, et al. A multi-society Delphi consensus statement on new fatty liver disease nomenclature. Hepatology. 2023;78(6):1966-1986. doi:10.1097/HEP.0000000000000520
9. Riazi K, Azhari H, Charette JH, Underwood FE, King JA, Afshar EE, et al. The prevalence and incidence of NAFLD worldwide: a systematic review and meta-analysis. Lancet Gastroenterol Hepatol. 2022;7(9):851–861. doi:10.1016/S2468-1253(22)00165-0
10. Cappello M, Randazzo C, Bravata I, Licata A, Peralta S, Craxi A, et al. Liver function test abnormalities in patients with inflammatory bowel diseases: a hospital-based survey. Clin Med Insights Gastroenterol. 2014;7:25–31. doi:10.4137/CGast.S13125
11. Patel P, Dalal S. Hepatic manifestations of inflammatory bowel disease. Clin Liver Dis (Hoboken). 2021;17(4):292–296. doi:10.1002/cld.1046
12. Roman AL, Munoz F. Comorbidity in inflammatory bowel disease. World J Gastroenterol. 2011;17(22):2723–2733. doi:10.3748/wjg.v17.i22.2723
13. Kablawi D, Aljohani F, Palumbo CS, Restellini S, Bitton A, Wild G, et al. Nonalcoholic fatty liver disease increases cardiovascular risk in inflammatory bowel diseases. Crohns Colitis 360. 2023;5(1):otad004. doi:10.1093/crocol/otad004
14. Zhang Q, Xu F, Liu S, Zhu S, Zhang S, Wu J, et al. Long-term risk of cardiovascular disease associated with MASLD and different cardiometabolic risk factors in IBD patients: a prospective cohort study. Liver Int. 2024;44(9):2315–2328. doi:10.1111/liv.15999
15. Noorian S, Jeon Y, Nguyen MT, Sauk J, Limketkai BN. The Impact of NAFLD on hospitalization outcomes in patients with inflammatory bowel diseases: nationwide analysis. Inflamm Bowel Dis. 2022;28(6):878–887. doi:10.1093/ibd/izab199
16. Jairath V, Afif W, Bressler B, Pope JE, Selchen D, Targownik LE, et al. Practical guidance for managing patients with moderate-to-severe ulcerative colitis using small molecule therapies. J Can Assoc Gastroenterol. 2024;7(4):282–289. doi:10.1093/jcag/gwae013
17. Grueso Navarro E, Lucendo AJ. Metabolic dysfunction-associated steatotic liver disease in inflammatory bowel disease: prevalence, risk factors, pathophysiological pathways and clinical consequences. Expert Rev Clin Immunol. 2025;21(7):875–891. doi:10.1080/1744666X.2025.2514605

18. Zamani M, Alizadeh-Tabari S, Singh S, Loomba R. Meta-analysis: prevalence of, and risk factors for, non-alcoholic fatty liver disease in patients with inflammatory bowel disease. Aliment Pharmacol Ther. 2022;55(8):894–907. doi:10.1111/apt.16879
19. Saroli Palumbo C, Restellini S, Chao CY, Aruljothy A, Lemieux C, Wild G, et al. Screening for nonalcoholic fatty liver disease in inflammatory bowel diseases: a cohort study using transient elastography. Inflamm Bowel Dis. 2019;25(1):124–133. doi:10.1093/ibd/izy200
20. Chao CY, Battat R, Al Khoury A, Restellini S, Sebastiani G, Bessissow T. Co-existence of non-alcoholic fatty liver disease and inflammatory bowel disease: a review article. World J Gastroenterol. 2016;22(34):7727–7734. doi:10.3748/wjg.v22.i34.7727
21. Calvez V, Becherucci G, Covello C, Piccirilli G, Mignini I, Esposto G, et al. Navigating the intersection: sarcopenia and sarcopenic obesity in inflammatory bowel disease. Biomedicines. 2024;12(6):1218. doi:10.3390/biomedicines12061218
22. Martinez-Dominguez SJ, Garcia-Mateo S, Gargallo-Puyuelo CJ, Gallego-Llera B, Callau P, Mendi C, et al. Inflammatory bowel disease is an independent risk factor for metabolic dysfunction-associated steatotic liver disease in lean individuals. Inflamm Bowel Dis. 2024;30(8):1274–1283. doi:10.1093/ibd/izad175
23. Rodriguez-Duque JC, Calleja JL, Iruzubieta P, Hernandez-Conde M, Rivas-Rivas C, Vera MI, et al. Increased risk of MAFLD and liver fibrosis in inflammatory bowel disease independent of classic metabolic risk factors. Clin Gastroenterol Hepatol. 2023;21(2):406–414 e407. doi:10.1016/j.cgh.2022.01.039
24. Bessissow T, Le NH, Rollet K, Afif W, Bitton A, Sebastiani G. Incidence and predictors of nonalcoholic fatty liver disease by serum biomarkers in patients with inflammatory bowel disease. Inflamm Bowel Dis. 2016;22(8):1937–1944. doi:10.1097/MIB.0000000000000832
25. Diabetes Canada Clinical Practice Guidelines Expert Working Group; Kim J, Bajaj HS, Ramji A, Bemeur C, Sebastiani G. Diabetes and metabolic dysfunction-associated steatotic liver disease in adults: a clinical practice guideline. Can J Diabetes. 2025;49(4):222–236. doi:10.1016/j.jcjd.2025.04.003
26. Kanwal F, Neuschwander-Tetri BA, Loomba R, Rinella ME. Metabolic dysfunction-associated steatotic liver disease: update and impact of new nomenclature on the American Association for the Study of Liver Diseases practice guidance on nonalcoholic fatty liver disease. Hepatology. 2024;79(5):1212–1219. doi:10.1097/HEP.0000000000000670
27. Godny L, Dotan I. Is the Mediterranean diet in inflammatory bowel diseases ready for prime time? J Can Assoc Gastroenterol. 2023;7(1):97–103. doi:10.1093/jcag/gwad041
28. Sanyal AJ, Newsome PN, Kliers I, Ostergaard LH, Long MT, Kjaer MS, et al. Phase 3 trial of semaglutide in metabolic dysfunction-associated steatohepatitis. N Engl J Med. 2025;392(21):2089–2099. doi:10.1056/NEJMoa2413258