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Association between MASLD and risk of ASCVD in IBD patients: A systematic review and Meta - Analysis

Adinda Ayu Dyah Rahadini[1*], Adinda Rahadina[2], Budi Widodo[3]

[1]Internal Medicine, Faculty of Medicine, Universitas Airlangga, Surabaya, East Java, 60115, Indonesia
[2]Department of Obstetrics and Gynecology, Faculty of Medicine, Universitas Airlangga, Surabaya, East Java, 60115, Indonesia
[3]Division of Gastroentero-Hepatology, Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga, Surabaya, East Java, 60115, Indonesia

Abstract

Inflammatory Bowel Disease (IBD) is now recognized beyond intestinal inflammation, as a condition involving systemic cardiometabolic dysfunction. Metabolic Dysfunction–Associated Steatotic Liver Disease (MASLD) is common among people with IBD and independently raises the risk of cardiovascular complications. Yet, the exact increase in Atherosclerotic Cardiovascular Disease (ASCVD) risk within this overlapping patient group remains unclear. Adhering to PRISMA 2020 guidelines, we performed systematic searches of PubMed and ProQuest until September 2025, looking for cohort studies reporting adjusted hazard ratios (HRs) relating MASLD/NAFLD to ASCVD events in adults diagnosed with IBD. The quality of the studies was assessed using the Newcastle-Ottawa Scale, and the results were aggregated by a random-effects meta-analysis of log-hazard ratios (log-HRs). Out of 69 records evaluated, two cohort studies involving 526,839 participants were included. The occurrence of MASLD in IBD patients was linked to a markedly increased risk of ASCVD (combined HR = 1.54; 95% CI 1.23–1.94). These results were consistent regardless of study location or MASLD definition. Due to the small number of studies, assessment for publication bias was limited. MASLD considerably increases cardiovascular risk in individuals with IBD, underscoring the importance of integrated cardiometabolic monitoring and prevention in this group.

Keywords: Inflammatory bowel disease, Metabolic Dysfunction–Associated steatotic liver disease, MASLD, Cardiovascular disease, ASCVD

Introduction

IBD affects millions worldwide including approximately 2.4 to 3.1 million Americans [1]. In addition to prevalent cardiovascular risk factors, Inflammatory Bowel Disease (IBD) is associated with heightened risks of ischemic heart disease and stroke—with reported elevations of 24% and 19%, respectively [2,3]. Active disease phases appear especially risky, with flares correlating to short-term increases in major cardiovascular events [4,28]. Simultaneously, MASLD (formerly NAFLD) affects about 30% of adults globally and is the predominant cause of cardiovascular mortality in these patients [5,6]. Within IBD populations, MASLD prevalence is estimated at around 32%, often exceeding rates within the general population [7]. Given the overlap of these risk factors, it is crucial to clarify the extent to which combined MASLD and IBD elevate actual cardiovascular events. This study synthesizes adjusted hazard ratios from cohort studies to focus on incident cardiovascular events in IBD patients with vs. without MASLD/NAFLD. Concentrating solely on observed clinical events (rather than predictive scores) streamlines risk assessment relevance.

Material and Methods

Study selection: This review complied with PRISMA 2020 criteria. Two independent reviewers screened abstracts, titles, and full texts from PubMed and ProQuest, resolving disagreements through consensus. Search strategies incorporated medical subject headings and keywords relating to IBD, MASLD/NAFLD, and cardiovascular diseases (see Table 1). Only English-language cohort studies reporting adjusted HRs of ASCVD outcomes in adult IBD patients were eligible. Exclusions included non-cohort designs, incomplete data retrieval, conference abstracts, letters, reviews, and non-English publications.

Table 1. Search strategy

Search	Query	Results
PubMed		
	("Inflammatory Bowel Diseases"[Mesh] OR IBD OR "Crohn Disease"[Mesh] OR Crohn* OR "Colitis, Ulcerative"[Mesh] OR ulcerative colitis)	33
	AND	

Correspondence: Adinda Ayu Dyah Rahadini, Internal Medicine, Faculty of Medicine, Universitas Airlangga, Surabaya, East Java, 60115, Indonesia, **e-mail:** rahadiniadinda@gmail.com, **Received:** December 6, 2025 **Accepted:** January 30, 2026

	("Fatty Liver"[Mesh] OR NAFLD OR MASLD OR MAFLD OR "nonalcoholic fatty liver" OR "metabolic dysfunction-associated steatotic liver disease")	
	AND	
	(ASCVD OR "atherosclerotic cardiovascular" OR "cardiovascular disease" OR "ischemic heart" OR myocardial OR stroke OR cerebrovascular OR "peripheral arterial" OR "coronary artery" OR "intima media" OR plaque OR "risk estimator" OR "Pooled Cohort" OR "QRISK" OR "Framingham")	
ProQuest		
	(TI,AB,SU("inflammatory bowel disease" OR IBD OR Crohn* OR "ulcerative colitis"))	34
	AND	
	(TI,AB,SU(NAFLD OR MASLD OR MAFLD OR "nonalcoholic fatty liver" OR "metabolic dysfunction-associated steatotic liver" OR "fatty liver"))	
	AND	
	(TI,AB,SU(ASCVD OR atheroscler* OR "cardiovascular disease*" OR "ischemic heart disease" OR "coronary disease" OR "myocardial infarction" OR stroke OR "cerebrovascular disease" OR "peripheral arterial disease" OR "heart failure" OR "risk estimator" OR "pooled cohort"))	

Criteria for eligibility

The following criteria were used to include studies: (1) the research was a cohort study; (2) the study explored the relationship between the risk of ASCVD and the presence of NAFLD/MASLD in people with Crohn's disease or ulcerative colitis; (3) ASCVD was reported as Hazard Ratios (HRs), if applicable. (1) Brief communications, letters to the editor, conference abstracts, reviews, or case reports; (2) unretrievable data; and (3) non-English papers were among the eliminated literatures.

Evaluation of bias risk

The Newcastle-Ottawa Scale (NOS) was employed to assess bias risk.

Data extraction

A predefined form was employed by two investigators to conduct independent data extraction from each study, including author, year, country, follow-up year, exposure ascertainment, outcome definitions, covariate adjustment, and adjusted hazard ratios with 95% confidence intervals.

Statistical analysis

A meta-analysis of log-transformed hazard ratios was conducted utilizing fixed or random effects models. Heterogeneity was evaluated using I^2 statistics.

Results

Following the removal of duplicates and the assessment of titles and abstracts for pertinence, 63 out of the 69 initially identified documents were removed. Upon careful examination of the remaining four articles, two studies were selected for inclusion in the qualitative synthesis (see Fig. 1).

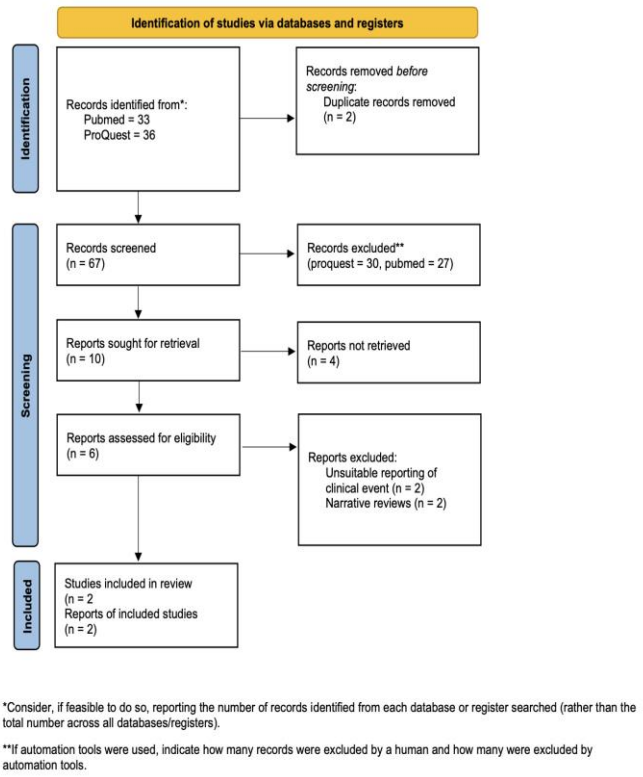

Fig 1. PRISMA Flow diagram

Table 2 Study characteristic

Author, year, country, sample size	IBD diagnosis criteria	NAFLD/MAFLD/MASLD diagnosis criteria	Outcome	Adjustment used
Jeon, 2021, United States, 522,635	ICD – coded diagnoses from Nationwide Readmission Database	NAFLD diagnostic criteria were ICD – coded using the Nationwide Readmission Database	CVD was defined as the occurrence of ischemic heart disease, myocardial infarction, cerebrovascular disease, and HF related admission and mortality	Age, sex, hypertension, diabetes, dyslipidemia, obesity, metabolic syndrome, smoking, Charlson comorbidity index, primary payer, region size, hospital size, and zip code income
Zhang, 2024, UK, 4,204	ICD – coded diagnoses from a UK Biobank, a large population – based prospective cohort	MASLD was defined as a concomitance of hepatic steatosis with at least one CMRF, as per AASLD-EASL-ALEH guidelines	Composite endpoint of ischemic heart disease, HF, as well as stroke.	Age, sex, Townsend deprivation index, education level, ethnicity, smoking status, weekly alcohol drinking, IPAQ, duration of IBD, and treatment status of IBD.

Abbreviations: AASLD, American Association for the Study of Liver Diseases; ALEH, Latin American Association for the Study of the *Liver*; CVD, cardiovascular disease; CMRF, cardiometabolic risk factors; EASL, European Association for the Study of the *Liver*; HF, heart failure; IBD, inflammatory bowel disease; ICD, International Classification of Diseases; IPAQ, International Physical Activity Questionnaire; MAFLD, metabolic associated fatty liver disease; MASLD, metabolic dysfunction associated steatotic liver disease; NAFLD, non – alcoholic associated fatty liver disease.

Study characteristic

Table 2 provides an overview of the baseline features of the two studies. The combined research involved 526,839 patients from two different countries. The methods used to diagnose IBD and conditions such as steatosis, NAFLD, MAFLD, or MASLD differed between the studies, as shown in Table 2.

Table 3. Quality of included studies

Study	Selection	Comparability	Outcome/Exposure	NOS Score
Jeon, 2021	4	2	1	7
Zhang, 2024	4	2	3	9

Abbreviations: NOS, Newcastle-Ottawa scale

Quality of included studies

Table 3 illustrates the quality of the study.

Publication bias

The assessment of publication bias via funnel plot was not feasible due to the inclusion of fewer than 10 studies.

Association between ASCVD and MASLD in IBD patients

In patients with IBD, the presence of MASLD is linked to a higher risk of ASCVD, with a combined adjusted hazard ratio of 1.54 and a 95% confidence interval ranging from 1.23 to 1.94 (Fig. 2).

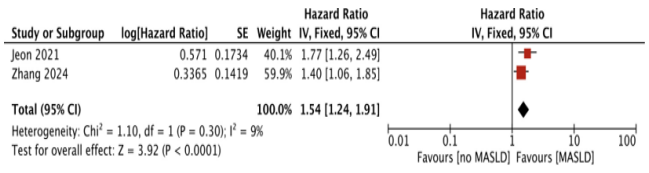

Fig. 2. ASCVD and MASLD in IBD patients

Discussion

In this HR-exclusive meta-analysis examining clinical cardiovascular events, our results demonstrate that the coexistence of NAFLD/MASLD in adults with IBD correlates with a notably elevated risk of composite cardiovascular disease compared to those with IBD alone (pooled HR 1.54, 95% CI 1.23–1.94). This level of risk is not only directionally aligned but appears to surpass the modest increase in cardiovascular risk associated with IBD when compared to population controls noted in previous studies. These earlier investigations include a meta-analysis highlighting an increased risk of ischemic heart disease and a nationwide cohort study showing heightened risks during active phases of IBD. Collectively, these findings underscore the additional cardiometabolic challenges posed when MASLD is present alongside IBD [2,8].

Biological mechanism

Early vascular damage is consistently associated with NAFLD/MASLD, as indicated by meta-analyses and aggregated data. These studies demonstrate an increased Carotid Intima-Media Thickness (cIMT), reduced flow-mediated dilation, and heightened arterial stiffness when compared to control groups. This suggests the presence of subclinical atherosclerosis and endothelial dysfunction [9–11]. Furthermore, longitudinal imaging studies indicate that acute Coronary Artery Calcification (CAC), which serves as a dependable marker for atherosclerosis, is associated with various hepatic steatosis phenotypes, such as MAFLD. Additionally, the regression of NAFLD has been linked to a decreased risk of CAC over an extended period [12–14]. Beyond the presence of subclinical disease, extensive meta-analyses indicate that Non-Alcoholic Fatty Liver Disease (NAFLD) is associated with an increased risk of developing heart failure. This finding reinforces the involvement of cardiac remodeling processes that are pertinent to clinical outcomes [15,16]. Ultimately, the risk of cardiovascular events seems to escalate with the progression of liver disease. Meta-analytic data indicate that both fatal and non-fatal cardiovascular disease risks rise in correlation with the severity of Non-Alcoholic Fatty Liver Disease (NAFLD) and the level of fibrosis [17].

A framework that views early atherogenesis through the lens of systemic inflammation aligns with supporting vascular evidence in Inflammatory Bowel Disease (IBD). This evidence reveals issues such as endothelial dysfunction, increased arterial stiffness, and heightened Carotid Intima-Media Thickness (cIMT) [18]. Furthermore, phases of active Inflammatory Bowel Disease (IBD) are associated with immediate rises in the risk of myocardial infarction, stroke, and cardiovascular mortality, establishing a connection between the inflammatory load and clinical outcomes [8]. Common exposures related to Inflammatory Bowel Disease (IBD) have the potential to intensify cardiometabolic risks. Systemic glucocorticoids are linked to conditions such as hyperglycemia, hypertension, and negative metabolic consequences. Additionally, in patients with ulcerative colitis, the use of tofacitinib leads to slight but reversible elevations in both LDL Cholesterol (LDL-C) and HDL Cholesterol (HDL-C) [19,20]. The interplay among the gastrointestinal tract, liver, and circulatory systems might increase risk factors, as Trimethylamine-N-Oxide (TMAO), produced by the microbiome, serves as an indicator of potential severe adverse cardiovascular incidents in future cohort studies [21,22]. These interrelated mechanisms collectively establish a comprehensive framework for understanding the additive risk of Atherosclerotic Cardiovascular Disease (ASCVD) in individuals with both Inflammatory Bowel Disease (IBD) and Metabolic-Associated Fatty Liver Disease (MASLD). This underscores the necessity for increased focus on managing inflammation and assessing the severity of liver disease, particularly fibrosis, within cardiovascular risk evaluations [23].

Clinical implications

Given the increased risk associated with events, healthcare providers ought to conduct a comprehensive ASCVD risk evaluation for IBD patients diagnosed with NAFLD/MASLD. Furthermore, it is essential to proactively manage modifiable risk factors such as lipids, blood pressure, glycemic levels, and body weight utilizing established preventive strategies [5]. Statins are advised and considered safe for use throughout the spectrum of Non-Alcoholic Fatty Liver Disease (NAFLD), even in cases of compensated cirrhosis, and it is crucial to prevent therapeutic inertia concerning the reduction of LDL-C levels. Additionally, GLP-1 receptor agonists have been shown to reduce the incidence of serious cardiovascular events in individuals with type 2

diabetes or heightened cardiometabolic risk, as evidenced by liraglutide's results in the LEADER trial and semaglutide's findings in SUSTAIN-6 [24,25]. SGLT2 inhibitors significantly lower the OUTCOME for diabetes, and DAPA-HF/DELIVER for HFrEF/HFpEF), warranting their consideration when appropriate. Additionally, practical care strategies for NAFLD highlight the importance of stringent management of hypertension and blood sugar levels, consistent weight reduction, and effective lipid control [26].

IBD management must take cardiovascular risk into account, prioritizing the administration of the lowest effective doses of systemic steroids and minimizing the duration of treatment. When tofacitinib is utilized, it is crucial to monitor lipid levels, as both LDL-C and HDL-C frequently increase in patients with ulcerative colitis [27–29]. Cardiovascular incidents often occur in clusters during periods of active disease; therefore, it is crucial to enhance ASCVD monitoring while intensifying the management of risk factors during exacerbations and ongoing inflammation [8]. To assess liver involvement, utilize noninvasive methods like FIB-4 and elastography for staging fibrosis. Additionally, coordinate shared care with hepatology and cardiology as necessary when advanced fibrosis or cirrhosis is suspected. This approach will ensure that liver treatment aligns effectively with cardiovascular prevention strategies [5].

Strength and limitations

Strengths of this meta-analysis include a predefined focus solely on Hazard Ratios (HR) related to clinical events, as well as consistent findings across two separate cohorts. However, several limitations must be acknowledged: there is a limited pool of eligible HR-based studies (k=2), one of which is merely an abstract; the analysis depends on coded endpoints sourced from a claims database; and there is a possibility of residual confounding. With only two studies available, the precision of the results is constrained, and it is not feasible to adequately evaluate publication bias. Additionally, risk-score data in individuals with Inflammatory Bowel Disease (IBD) who also Have Non-Alcoholic Fatty Liver Disease (NAFLD)—such as increased likelihoods for intermediate/high categories of Atherosclerotic Cardiovascular Disease (ASCVD) risk—support these findings but cannot be directly combined with event-based HRs. Therefore, larger, rates of hospitalization due to heart failure and cardiovascular mortality across various phenotypes (such as demonstrated in EMPA-REG

adjudicated prospective cohorts that utilize current definitions of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD), while also assessing the severity of MASLD (particularly fibrosis), are essential for enhancing absolute risk assessment and informing prevention strategies.

Conclusions

IBD with concomitant NAFLD/MASLD is associated with increased hazard of clinical CVD. These results support routine ASCVD risk assessment and optimization in this overlap population.

Disclosures

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