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Original Article

Assessment of Metabolic Dysfunction-associated Steatotic Liver Disease and Liver Fibrosis: A Cross-sectional Study in Asymptomatic Individuals in Greater Vancouver

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Abstract

Background and Aims: Metabolic dysfunction-associated steatotic liver disease (MASLD) is a leading cause of hepatic fibrosis, yet its prevalence in asymptomatic populations remains unclear. This study aimed to assess the prevalence of steatosis and significant fibrosis in asymptomatic individuals without known liver disease in the Greater Vancouver Area. ***Methods:*** Interested individuals voluntarily registered online via the Canadian Liver Foundation website or by telephone. Inclusion criteria included age ≥ 19 years, no known liver disease, and low alcohol intake (<30 g/day for men, <20 g/day for women). Demographic and clinical data were collected, and all participants underwent transient elastography after a 3-h fast. The study aimed to collect 4,500 analyzable scans while reflecting the region's ethnic diversity. ***Results:*** A total of 4,193 participants were analyzed. The median age was 62 years, the median body mass index was 25.4, and 45% were male. Asian individuals comprised 42% of the cohort. Steatosis was present in 59.6% of participants, and 45.7% met diagnostic criteria for MASLD. Significant fibrosis (F2–F4) was found in 8.6%. Age, male sex, ethnicity, cardiac disease, diabetes, hypertension, and obesity were significantly associated with fibrosis. Logistic regression analysis confirmed age, weight, diabetes, dyslipidemia, hypertension, and obesity as independent predictors. ***Conclusions:*** A substantial proportion of asymptomatic individuals in Greater Vancouver have undetected MASLD and significant fibrosis. Early identification of high-risk groups may support broader implementation of transient elastography screening. This study provides one of the first North American population-based estimates of MASLD and fibrosis stratified by ethnicity, offering new insights into liver disease distribution among Caucasian, Chinese, and South Asian populations.

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Introduction

Metabolic dysfunction-associated steatotic liver disease (MASLD) is characterized by the accumulation of triglycerides in the liver, accounting for more than 5% of liver volume, without excessive alcohol consumption.[1–3] Globally, MASLD is now the most common cause of chronic liver disease[4,5] with an estimated prevalence ranging from 25%[3,6] to 38%.[7] Within Canada, the burden of liver disease is substantial, affecting an estimated one in ten individuals—more than three million Canadians.[8]

Multiple disease processes, including hypertension, type 2 diabetes mellitus, hyperlipidemia, and obesity, are strongly associated with MASLD.[5,6,9–13] For this reason, MASLD is considered the hepatic manifestation of metabolic syndrome.[6,9–11] Owing to the increased prevalence of these predisposing metabolic conditions,[12] MASLD has consequently emerged as a serious public health concern.

As a leading cause of liver transplantation, a diagnosis of MASLD necessitates long-term monitoring and lifestyle interventions for effective management. However, in many patients, MASLD diagnosis is often delayed,[14,15] as the condition is frequently asymptomatic in its early stages, allowing silent progression to more severe conditions such as metabolic dysfunction-associated steatohepatitis, cirrhosis, and hepatocellular carcinoma.[3,16,17] This insidious progression is particularly problematic when access to primary care is suboptimal. In Vancouver, where 18% of residents lack access to a family physician,[18] early identification of MASLD may help mitigate disease progression and improve patient outcomes.[9,16,17,19] Predictive models that integrate common risk factors, such as diabetes, obesity, and hypertension, could enable early identification of high-risk individuals and facilitate early intervention, thereby preventing MASLD progression.

This study examined how participant characteristics may predict the presence of MASLD and liver fibrosis in asymptomatic individuals. By identifying the burden of MASLD in

Keywords: Chronic liver disease; Metabolic risk factors; MASLD; FibroScan; Steatosis; Fibrosis; Cross-sectional study; Hypertension; Hyperlipidemia.

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at-risk, asymptomatic populations, health care practitioners may promote earlier detection of chronic liver disease, leading to timely interventions and improved outcomes.

Methods

Study design

We conducted a cross-sectional, population-based screening study in the Lower Mainland of British Columbia, Canada. In addition to data collected during this study, we utilized data from a previous study performed in 2022–2023,[20] which sought to evaluate the prevalence and severity of MASLD and liver fibrosis among asymptomatic individuals in the Greater Vancouver area. The data from this cohort were integrated into the 2024 dataset, ensuring consistency by harmonizing variables shared across all years. In the current study, additional data were collected on risk factors, including diabetes, hypertension, hyperlipidemia, alcohol consumption (categorized as none or ≥6 ounces (177.4 mL) of an alcoholic beverage per day), and body mass index (BMI).

This study received ethical approval from the University of British Columbia Clinical Research Ethics Board and was conducted in compliance with the Declaration of Helsinki. Participants for the current study were enrolled between May 2024 and September 2024. Recruitment occurred via educational seminars, medical channels, and social media platforms facilitated by the Canadian Liver Foundation. Interested participants were interviewed by study coordinators to collect baseline demographic data, including age, sex, ethnic background, and family physician status (if applicable). Data were also collected on comorbid conditions, including diabetes, hypertension, hyperlipidemia, and cardiac disease. Furthermore, we collected data on pre-existing chronic liver diseases and patterns of alcohol consumption. All participants provided written informed consent, witnessed and documented using a standardized subject information and consent form. Each participant was assigned a unique, anonymized study identifier; all data were encrypted and password protected.

Participants were excluded in a stepwise fashion. First, individuals under the age of 19 were excluded. Next, those who reported alcohol consumption exceeding the MASLD diagnostic thresholds of >30 g/day in men (less than two beers/ten ounces of wine/two standard drinks of hard liquor) or >20 g/day in women were excluded. Participants with a history of non-MASLD chronic liver disease (n = 846) were then excluded. Finally, participants with an elastography interquartile range (IQR) percentage greater than 30% were excluded (n = 187). Thus, individuals with potential metabolic-alcohol-related liver disease or alcoholic liver disease were not included in the final analytic cohort.

Assessment of liver steatosis and fibrosis

Each participant underwent transient elastography (FibroScan) to assess liver stiffness (kPa) and controlled attenuation parameter (CAP) scores. Liver stiffness measurements (LSMs) were used to estimate fibrosis, while CAP scores served as indicators of hepatic steatosis. Unlike liver biopsies, which are invasive and carry a risk of complications,[9,21,22] FibroScan provides risk-free, accurate, painless, and immediate measurements, making it ideal for large-scale population screening.[9,23]

Data pre-processing steps

Data preprocessing began with merging liver scan datasets from the years 2022–2024. For each participant, only the first valid scan was included in the analysis. Individuals with pre-existing knowledge of underlying liver disease were excluded to minimize potential bias. Outliers were removed based on predetermined criteria, specifically scans with an elastography IQR greater than 30%. Additionally, three individuals with extreme and implausible values (e.g., CAP = 13, weight = 1 kg or 4.5 kg) were excluded.

To ensure consistency, each participant was assigned a unique identifier by combining the year of the scan with their original patient ID. Steatosis grades were categorized as follows: S0 for CAP < 238, S1 for 238 ≤ CAP < 260, S2 for 260 ≤ CAP < 290, and S3 for CAP ≥ 290. Similarly, fibrosis stages were defined based on LSM scores: F0–F1 for LSM < 7, F2 for 7 ≤ LSM < 10, F3 for 10 ≤ LSM < 14, and F4 for LSM ≥ 14. A binary fibrosis classification was also used, where F0–F1 indicated mild or no fibrosis, and F2–F4 indicated advanced fibrosis. The LSM thresholds for advanced fibrosis were selected based on prior validation studies,[24–26] including Wong *et al*. (2010),[27] which reported the highest AUROC values of 0.84 for significant fibrosis and 0.95 for cirrhosis, with optimal thresholds ranging from 8.7–9.6 kPa. While no universal consensus exists on exact cutoff values, these thresholds are pragmatic and are also recommended by EchoSens, the manufacturer of FibroScan, in their clinical guidance.[28]

Obesity classification was included using race-specific BMI thresholds.[29] Obesity was defined as a BMI of ≥30 for Caucasian participants, ≥23.9 for South Asian participants, ≥26.9 for Asian participants, and ≥28.1 for participants classified as "Other". The "Other" category encompassed a heterogeneous group, including individuals of Indigenous, Black/African heritage, and other unspecified or mixed racial identities. After applying these preprocessing steps, the final dataset included 4,193 participants.

Statistical analysis

Associations were examined using bivariate analysis of variance (ANOVA). Multivariate logistic regression was performed to calculate odds ratios and assess the significance of independent risk factors. All statistical analyses were conducted using R version 4.4.0. Statistical significance was defined as $p < 0.05$.

Results

Participant characteristics

Baseline characteristics of study participants are displayed in Table 1. In total, 4,193 participants were enrolled in the study (females = 54.9%). The median age of the population was 62 years (IQR: 51–69). The median BMI was 25.4 kg/m^2 (IQR: 22.8–28.5), with over a third (34.7%) of the population classified as obese. A total of 41.9% of the population identified their ethnicity as Asian, 20.3% as South Asian, 32.8% as White, and 5.0% as belonging to other racial backgrounds. Diabetes was present in 12.1% of the population, hypertension was reported in 26.5%, and 28.8% had hyperlipidemia. Cardiac disease was present in 8.1%, and alcohol consumption exceeding six ounces (177.4 mL) of alcoholic beverages per day was reported by 13.6% of the population.

Steatosis and fibrosis prevalence

Over half of the population (59.6%) showed some degree of steatosis, with 15.5% having mild steatosis (S1), 17.6% having moderate steatosis (S2), and 26.6% having severe steatosis (S3) (Table 1). Advanced fibrosis (F2–F4) was present in 8.6% of the population (Table 1). Specifically, 6.6% had moderate fibrosis (F2), 1.4% had severe fibrosis (F3),

Table 1. Descriptive statistics of participants (n = 4,193)

Continuous variables	Median [IQR]
Age	62 [51–69]
Height	166 [160–173]
Weight	70.5 [60.3–83.5]
BMI	25.4 [22.8–28.5]

Categorical variables	n (%)
Gender	
Male	1,890 (45.08)
Female	2,303 (54.92)
Race	
Asian	1,756 (41.88)
Other	208 (4.96)
South Asian	852 (20.32)
White	1,377 (32.84)
Cardiac disease	
N	3,854 (91.92)
Y	339 (8.08)
Diabetes	
N	3,686 (87.91)
Y	507 (12.09)
Hyperlipidemia	
N	2,984 (71.17)
Y	1,209 (28.83)
Hypertension	
N	3,082 (73.50)
Y	1,111 (26.50)
Alcohol consumption	
N	3,623 (86.41)
Y	570 (13.59)
Steatosis degree	
S0	1,693 (40.38)
S1	649 (15.48)
S2	736 (17.55)
S3	1,115 (26.59)
Estimated fibrosis degree	
F0 to F1	3,832 (91.39)
F2	275 (6.56)
F3	60 (1.43)
F4	26 (0.62)
Estimated fibrosis stage	
F0 to F1	3,832 (91.39)
F2 to F4	361 (8.61)
Obesity	
Yes	1,455 (34.70)
No	2,738 (65.30)

BMI, body mass index; IQR, interquartile range.

and 0.6% had cirrhosis (F4). Most participants (91.4%) had no fibrosis or mild fibrosis (F0–F1) (Table 1). Of the 4,193 participants, 1,916 (45.7%) met the diagnostic criteria for MASLD, defined as the presence of liver steatosis (CAP > 238 dB/m) in conjunction with at least one cardiometabolic risk factor (obesity, hypertension, diabetes, or hyperlipidemia).

Additionally, a strong positive correlation was observed between the degree of steatosis and the presence of advanced fibrosis (Table 2). Among participants with S0 steatosis, only 3.9% exhibited advanced fibrosis, whereas in individuals with S3 steatosis, it was found in 17.8%. Among all subjects with advanced fibrosis (n = 361), 55.1% had S3 steatosis. A Chi-square test confirmed that this association was strongly statistically significant ($p < 0.001$).

Factors associated with increased risk of advanced fibrosis

Multiple risk factors were associated with advanced fibrosis (F2–F4). In bivariate ANOVA, participants with diabetes, hypertension, and obesity were more likely to have advanced fibrosis (F2-F4) compared to their counterparts (Table 3). Other variables, including gender, BMI, alcohol consumption, and cardiac disease, were not significantly associated with advanced fibrosis in bivariate ANOVA (Table 3). Although South Asians had slightly lower odds of advanced fibrosis compared to other groups (OR = 0.898), this difference was not statistically significant (Table 3).

Results of logistic regression demonstrated that obesity showed the strongest association with fibrosis (OR = 4.401, 95% CI: 3.476–5.601, $p < 0.001$) (Table 4). Age was also independently associated with higher odds of advanced fibrosis (OR = 1.013, 95% CI: 1.004–1.023, $p = 0.006$), as were diabetes (OR = 1.664, 95% CI: 1.231–2.229, $p < 0.001$) and hypertension (OR = 1.397, 95% CI: 1.080–1.804, $p = 0.011$) (Table 4). Conversely, hyperlipidemia was associated with lower odds of advanced fibrosis (OR = 0.627, 95% CI: 0.477–0.818, $p \leq 0.001$) (Table 4).

Discussion

The findings from this cross-sectional study highlight the significant prevalence of MASLD and liver fibrosis in an asymptomatic population within the Greater Vancouver area. More than half of the study population (59.6%) exhibited some degree of hepatic steatosis, with severe steatosis (S3) present in over a quarter of participants (26.6%). These findings highlight that hepatic steatosis is a highly prevalent condition, even among individuals who are asymptomatic. Furthermore, an estimated 8.6% of the study population were at increased risk of advanced fibrosis (F2–F4) based on transient elastography, with 6.6% at risk for moderate fibrosis (F2), 1.4% for severe fibrosis (F3), and 0.6% for cirrhosis (F4). The majority (91.4%) had readings suggestive of no or only mild fibrosis (F0–F1). These findings reflect a potentially high burden of steatotic liver disease and estimated fibrosis risk, underscoring the insidious progression of MASLD toward more severe liver outcomes. Our findings complement those of Zhu *et al*.,[20] which also emphasized the substantial public health burden of MASLD and fibrosis.

It is important to note that LSM derived from transient elastography reflects an estimate of risk for advanced fibrosis, rather than a definitive histological diagnosis. Accordingly, all fibrosis-related outcomes reported in this study refer to estimated risk based on non-invasive elastography. However, prior studies have demonstrated that higher liver stiffness measurements are independently associated with an increased risk of liver-related events, including hepatic de-

Table 2. Relationship between steatosis degree and advanced fibrosis (stage F2–F4)

Steatosis degree	No advanced fibrosis (n = 3,832)	Advanced fibrosis (n = 361)	% with advanced fibrosis
S0 (<238 dB/m)	1,627 (42.5%)	66 (18.3%)	3.9%
S1 (238–259 dB/m)	617 (16.1%)	32 (8.9%)	4.9%
S2 (260–289 dB/m)	672 (17.5%)	64 (17.7%)	8.7%
S3 (≥290 dB/m)	916 (23.9%)	199 (55.1%)	17.8%

compensation and hepatocellular carcinoma.[30,31] Thus, despite limitations in sensitivity and specificity, VCTE remains a clinically valuable tool for identifying individuals at increased risk of adverse liver outcomes.

In the current study, obesity emerged as the most significant metabolic risk factor in predicting advanced liver fibrosis. Obese participants had 2.46 times higher odds of advanced fibrosis compared to non-obese individuals. Given

Table 3. Bivariate analysis of variance (n = 4,193)

		Estimated risk of fibrosis Stage		*p*-value
		F0-F1 (n = 3,832)	F2-F4 (n = 361)	
Continuous variables				
Age, median [IQR]	62 [51–69]	62 [51, 69]	63 [54, 69]	0.002
Height, median [IQR]	166 [160–173]	165.1 [160, 173]	168 [160.1, 177.8]	0.180
Weight, median [IQR]	70.5 [60.3–83.5]	69.6 [59.8, 82.0]	85.0 [70.0, 99.0]	0.049
BMI, median [IQR]	25.4 [22.8–28.5]	25.1 [22.6, 28.1]	29.6 [25.6, 33.9]	0.386
Categorical variables				
Gender, n (%)				<0.001
Male	1,890 (45.08)	1,695 (44.2)	195 (54)	
Female	2,303 (54.92)	2,137 (55.8)	166 (46)	
Race, n (%)				<0.001
Asian	1,756 (41.88)	1,655 (43.2)	101 (28)	
Other	208 (4.96)	189 (5)	19 (5.3)	
South Asian	852 (20.32)	760 (19.8)	92 (25.5)	
White	1,377 (32.84)	1,228 (32)	149 (41.3)	
Cardiac disease, n (%)				0.022
N	3,854 (91.92)	3,534 (92.2)	320 (88.6)	
Y	339 (8.08)	298 (7.8)	41 (11.4)	
Diabetes, n (%)				<0.001
N	3,686 (87.91)	3,401 (88.8)	285 (78.9)	
Y	507 (12.09)	431 (11.2)	76 (21.1)	
Hyperlipidemia, n (%)				0.423
N	2,984 (71.17)	2,720 (71)	264 (73.1)	
Y	1,209 (28.83)	1,112 (29)	97 (26.9)	
Hypertension, n (%)				<0.001
N	3,082 (73.50)	2,862 (74.7)	220 (60.9)	
Y	1,111 (26.50)	970 (25.3)	141 (39.1)	
Alcohol Consumption, n (%)				0.819
N	3,623 (86.41)	3,313 (86.5)	310 (85.9)	
Y	570 (13.59)	519 (13.5)	51 (14.1)	
Obesity, n (%)				<0.001
Yes	1,455 (34.70)	1,213 (31.7)	242 (67)	
No	2,738 (65.30)	2,619 (68.3)	119 (33)	

BMI, body mass index; IQR, interquartile range.

Table 4. Logistic regression analysis for risk of significant fibrosis (stage F0–F1 vs. F2–F4)

Variable	Odds ratio (OR)	95% CI (lower-upper)	*p*-value
Age	1.013	1.004–1.023	0.006
Gender			
Male	1.069	0.758–1.508	0.701
Race			
Other	1.012	0.582–1.758	0.966
South Asian	0.898	0.644–1.253	0.534
White	1.147	0.811–1.622	0.420
Cardiac disease			
Yes	1.285	0.874–1.851	0.188
Diabetes			
Yes	1.664	1.231–2.229	<0.001
Hyperlipidemia			
Yes	0.627	0.477–0.86	<0.001
Hypertension			
Yes	1.397	1.080–1.804	0.011
Alcohol consumption			
Yes	1.093	0.784–1.496	0.590
Obesity			
Yes	4.401	3.476–5.601	<0.001

CI, confidence interval.

the rising prevalence of obesity globally,[13,32] the incidence of steatotic liver disease is also expected to increase,[33] further emphasizing the need to address obesity to mitigate steatotic liver progression. Diabetes and hypertension were also independently associated with advanced fibrosis, supporting findings by van Son *et al*.,[9] which emphasize the role of metabolic conditions as major contributors to MASLD progression. Awareness of the prevalence of MASLD in asymptomatic populations may encourage early lifestyle interventions to halt disease progression. Interestingly, hyperlipidemia was associated with lower odds of advanced fibrosis. A potential explanation is that individuals with hyperlipidemia may be more likely to use statin therapy, which has been shown to have protective effects on the liver and is associated with a lower prevalence and reduced risk of progression of advanced liver fibrosis.[34,35] However, these findings warrant further investigation to determine whether this reflects a true protective effect or whether confounding variables in the study population may have influenced the results.

Findings from our study indicate that gender, race/ethnicity, BMI, alcohol consumption, and cardiac disease were not significantly associated with advanced fibrosis. This lack of association between alcohol consumption, cardiac disease, and MASLD differs from previous literature suggesting that these factors contribute significantly to MASLD progression.[36] A potential explanation is that individuals may underreport their true alcohol intake due to social desirability bias or concerns related to stigma.[37] Such underreporting could lead to an underestimation of alcohol's impact on MASLD progression.

Overall, the results of the study suggest that metabolic risk factors such as obesity, diabetes, and hypertension play a more dominant role in MASLD progression. These findings further support the need for targeted interventions that address modifiable metabolic risk factors to reduce the risk of advanced fibrosis and severe liver outcomes.

A notable strength of this study is the unique ethnic composition of the Greater Vancouver population. British Columbia is one of the few provinces in North America where Caucasians constitute less than 50% of the population,[38] enabling valid comparisons between major ethnic groups. To our knowledge, this is the first population-based study in North America with sufficient statistical power to compare the prevalence of MASLD and fibrosis among Caucasian, Chinese, and South Asian individuals in non-clinical or non-referral settings. While our findings did not demonstrate statistically significant differences in fibrosis prevalence by ethnicity, this null result is clinically meaningful. It suggests that metabolic risk factors may be stronger predictors of disease progression than ethnicity alone, indicating that screening and prevention strategies should prioritize these modifiable risk factors regardless of racial background. Another strength of this study is its large cohort of over 4,000 asymptomatic participants and the robust assessment of steatosis and fibrosis, which provided sufficient statistical power to detect trends in MASLD and liver fibrosis prevalence. The use of FibroScan, a non-invasive, accurate, and painless diagnostic tool, enabled reliable assessments while minimizing risks associated with liver biopsies. By focusing on asymptomatic individuals, the study demonstrates the silent progression of MASLD and identifies at-risk populations before the development of clinical symptoms. Notably, this study also builds upon previous research by incorporating a wider range of variables, including alcohol consumption, hypertension, hyperlipidemia, and diabetes, thereby broadening the scope of analysis and allowing for more in-depth assessment of various risk factors.

One key limitation of this study is potential selection bias,

as participants were recruited through public advertisement and volunteered to participate. This recruitment method may have led to the under- or over-representation of individuals with specific characteristics, such as heightened health awareness, greater perceived risk, or improved healthcare access. In some cases, participants may have been more likely to participate due to personal or familial experiences with liver disease, which could influence both participation and the accuracy of self-reported medical history. In addition, the self-reported nature of the data introduces potential recall and reporting bias, particularly for sensitive behaviors such as alcohol consumption. Furthermore, alcohol intake was categorized simply as none or ≥6 ounces (177.4 mL) of alcoholic beverages per day. This binary categorization may overlook lower levels of alcohol intake that could still negatively impact liver health. Another limitation is the lack of standardized, universally accepted cutoffs for LSM. For example, although we used a 10 kPa threshold to define advanced fibrosis based on prior validation studies and manufacturer guidance,[24,26–28,39,40] this does not align with the more conservative 12 kPa cutoff recently recommended by the American Association for the Study of Liver Diseases[41] and the European Association for the Study of the Liver.[42] Such variability in LSM thresholds may affect fibrosis prevalence estimates and complicate cross-study comparisons. Finally, the cross-sectional design of this study inherently limits its ability to establish causal relationships between identified risk factors and MASLD progression. Socioeconomic determinants of health—known to influence both MASLD risk and healthcare utilization[43–45]—were not included, and demographic information such as participants' income was not collected. Future studies would benefit from incorporating measures of socioeconomic status, neighborhood data, or healthcare access to enhance the representativeness and depth of analysis.

Conclusions

This study highlights the significant burden of MASLD and advanced fibrosis in an asymptomatic population within Greater Vancouver. More than half of the participants exhibited hepatic steatosis, demonstrating the high prevalence of this condition. Furthermore, 9% of the population had advanced fibrosis, underscoring the insidious progression of liver disease even in symptom-free individuals. In this study, obesity, diabetes, and hypertension emerged as significant risk factors for MASLD, reinforcing the strong association between metabolic dysfunction and MASLD development.[5,6,9–11,46] Interestingly, hyperlipidemia was associated with lower odds of advanced fibrosis, a finding that contrasts with the typical risk profile of metabolic conditions.[9,10,47] This unexpected association calls for additional research to determine the true relationship between hyperlipidemia and liver disease in asymptomatic individuals.

While MASLD has emerged as a critical global health issue, it remains largely underdiagnosed[15,16] and, therefore, untreated,[48] potentially leading to severe health consequences. These findings highlight the urgent need for public health initiatives to prioritize early detection, preventive care, and management strategies for MASLD and its predisposing risk factors. Targeted interventions focusing on metabolic health, lifestyle modification, and increased access to screening tools can help mitigate the prevalence of MASLD and prevent progression to severe liver disease. This study calls for future research to explore tailored strategies and policies that address the growing prevalence of MASLD and its associated comorbidities.

Funding

None to declare.

Conflict of interest

EMY was an investigator in clinical trials sponsored by Intercept Inc., Genfit Inc., Madrigal Inc., Allergan Inc., Pfizer Inc., and Novodisc Inc., and received an unrestricted grant from Paladin Laboratories. DC conducts metabolic dysfunction-associated steatohepatitis/metabolic dysfunction-associated steatotic liver disease industry trials for Merck Inc., 89Bio Inc., and Gilead Inc. DC also has a specialty pharmaceutical FibroScan collaboration with SRx Healthcare Inc. The other authors have no conflict of interests related to this publication.

Author contributions

Drafting and editing of the manuscript (NWT, DMPDF), data collection and analysis (NWT, TW, KL), graphical abstract design (NWT), study conception and design (EMY, PK, DC). All authors have approved the final version and publication of the manuscript.

Ethical statement

This study was conducted in accordance with the Declaration of Helsinki as revised in 2024. Ethical approval was obtained from the University of British Columbia Clinical Research Ethics Board (REB No. H21-00634). Written informed consent was obtained from all participants prior to enrollment, and all data were anonymized and securely stored. All procedures performed in the study adhered to established ethical guidelines.

Data sharing statement

Data from the current study are available upon reasonable request to the corresponding author.

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