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Review

Integrating the Care of Metabolic Dysfunction–associated Steatotic Liver Disease Into Cardiac Rehabilitation: A Multisystem Approach

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ABSTRACT

Metabolic dysfunction–associated steatotic liver disease (MASLD) is the most prevalent chronic liver disease worldwide, affecting 1 in 3 adults and driven by the rising incidence of obesity and type 2 diabetes. MASLD is also a significant contributor to cardiovascular morbidity and mortality. Cardiovascular disease remains the leading cause of death in people with MASLD, often preceding liver complications. In this review, we outline the epidemiology and shared pathophysiologic mechanisms of MASLD with cardiovascular disease. Furthermore, we present a compelling case for integrating cardiac rehabilitation (CR) into MASLD care to address the shared metabolic and inflammatory drivers of both conditions. Evidence from recent clinical trials and guidelines supports the need for holistic, multidisciplinary strategies, including exercise, diet, and pharmacotherapy. CR is no longer solely a post–cardiac event intervention, but an opportunity for proactive cardiometabolic risk reduction in MASLD patients, especially those with fibrosis or type 2 diabetes. Incorporating MASLD into CR programs can facilitate early identification of high-risk individuals and deliver integrated care targeting both liver and cardiovascular outcomes. Cardiologists, hepatologists, and primary care providers must recognize MASLD as a cardiometabolic risk enhancer and consider CR referral, even

RÉSUMÉ

La stéatose hépatique associée à une dysfonction métabolique (MASLD) est la maladie hépatique chronique la plus répandue dans le monde, touchant 1 adulte sur 3, et est favorisée par l'incidence croissante de l'obésité et du diabète de type 2. La MASLD contribue également de manière significative à la morbidité et à la mortalité cardiovasculaires. Chez les personnes atteintes de MASLD, les maladies cardiovasculaires représentent d'ailleurs la principale cause de décès, précédant souvent les complications hépatiques. Dans cet article, nous décrivons l'épidémiologie et les mécanismes physiopathologiques communs de la MASLD et des maladies cardiovasculaires. En outre, nous présentons des arguments solides en faveur de l'intégration de la réadaptation cardiaque (RC) dans la prise en charge de la MASLD, afin de cibler les facteurs métaboliques et inflammatoires communs aux deux pathologies. Les résultats d'essais cliniques récents et les recommandations des lignes directrices soutiennent la nécessité de stratégies holistiques et multidisciplinaires, incluant l'activité physique, l'alimentation et la pharmacothérapie. La RC ne doit plus être considérée uniquement comme une intervention post-événement cardiaque, mais comme une opportunité de réduction proactive du risque cardiométabolique chez les patients atteints de MASLD, en particulier ceux présentant

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Burden of MASLD and Link to Metabolic Dysfunction

Formerly known as nonalcoholic fatty liver disease (NAFLD), metabolic dysfunction–associated steatotic liver disease (MASLD) affects 32.4% of adults globally.[1] It is now the second-leading indication for liver transplantation worldwide and already the leading cause in women.[2] In Canada, MASLD is projected to impact 12 million people by 2030, including 600,000 with advanced fibrosis and 200,000 at risk of decompensated cirrhosis, hepatocellular carcinoma (HCC), or liver transplantation.[3] The reclassification from NAFLD to MASLD in 2023 acknowledges the central role of metabolic dysfunction.[4] Unlike NAFLD, MASLD no longer

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before overt cardiovascular events occur. CR may be a critical yet underutilized opportunity to modify cardiovascular- and liver-related outcomes in people with MASLD.

une fibrose ou un diabète de type 2. La prise en compte de la MASLD dans les programmes de RC peut faciliter l'identification précoce des personnes à haut risque et fournir des soins intégrés ciblant à la fois les résultats hépatiques et cardiovasculaires. Les cardiologues, les hépatologues et les prestataires de soins primaires doivent reconnaître la MASLD comme un facteur aggravant du risque cardiométabolique et envisager d'orienter les patients vers la RC avant même l'apparition d'événements cardiovasculaires manifestes. La RC pourrait représenter une opportunité cruciale mais sous-utilisée d'influencer l'évolution des complications cardiaques et hépatiques.

requires the exclusion of alcohol use or viral hepatitis. Instead, the diagnosis is based on the presence of 1 or more metabolic risk factor(s)—obesity, type 2 diabetes (T2D), dyslipidemia, or hypertension. This paradigm shift broadens the eligible population for diagnosis and intervention, encouraging earlier detection and enabling more inclusive care, even for patients with dual liver disease etiologies.[5] T2D and obesity are major drivers of the epidemic: up to 70% of individuals with T2D and 90% of those with obesity have MASLD.[5-7] The growing prevalence of MASLD reflects global increases in obesity, sedentary behavior, and poor diet. This redefinition also removes the stigma and diagnostic ambiguity of the "nonalcoholic" label, aligning MASLD more closely with a cardiometabolic risk framework. It reinforces the need for coordinated interdisciplinary care across hepatology, cardiology, endocrinology, and primary care. Importantly, MASLD often progresses silently, as many individuals remain undiagnosed until presenting with advanced liver disease or complications such as cirrhosis or HCC. From a public health perspective, MASLD represents a major and escalating economic burden. MASLD imposes substantial direct and indirect health care costs due to its association with T2D, cardiovascular events, and progressive liver disease. Direct costs include laboratory and imaging tests, noninvasive fibrosis assessments, liver biopsies, hospitalizations, and cancer therapies. Indirect costs, such as lost productivity and long-term disability, are also substantial.[8] In the United States alone, the total annual economic burden of MASLD is estimated to exceed $100 billion, with the majority related to early-stage disease due to its high prevalence and cardiometabolic complications.[8] In parallel, cardiovascular disease (CVD) costs the US health care system approximately $240 billion annually.[9] Given that MASLD and CVD frequently co-occur and share underlying pathophysiology (eg, insulin resistance, systemic inflammation, endothelial dysfunction), integrated prevention strategies—such as cardiac rehabilitation (CR) tailored to MASLD—could deliver high-impact, cost-effective interventions. The greatest health and economic impact is associated with advanced stages of liver disease. Global data show a 47% surge in mortality from liver cirrhosis from 1990 to 2017, with currently 2 million deaths annually worldwide, largely driven by MASLD.[1,10] Liver diseases now rank as the 11th leading cause of death and 15th leading cause of disability-associated life-years globally.[1] In the US, liver cirrhosis has risen from 14th to 8th among causes of years of life lost, positioned between diabetes (7th) and Alzheimer disease (9th), and surpassing colorectal (10th) and breast cancers (13th).[11] The economic burden is similarly striking: in 2016, liver disease costs were estimated at $32.5 billion.[12] A recent microsimulation study of 2.8 million individuals projected that MASLD prevalence in the US will increase from 33.7% in 2020 to 41.4% in 2050, affecting an estimated 122 million adults. Over the same period, cases of decompensated cirrhosis are expected to more than triple, whereas liver cancer incidence nearly doubles and liver transplant needs almost quadruple.[13] In Canada, MASLD mirrors this global trend and is projected to account for virtually all new cases of liver cirrhosis by 2040.[14] MASLD particularly affects socioeconomically disadvantaged populations, who face higher barriers to accessing preventive care, further amplifying health disparities.[15] As a systemic metabolic disorder with both hepatic and extrahepatic consequences, MASLD calls for urgent attention and integration into chronic disease management models.

Pathophysiology and Cardiovascular Overlap

MASLD and CVD share overlapping and synergistic pathophysiologic mechanisms, including insulin resistance, chronic low-grade inflammation, atherogenic dyslipidemia, oxidative stress, and endothelial dysfunction (Fig. 1).[16,17] These shared drivers not only promote disease progression in each organ but also create a bidirectional relationship whereby liver dysfunction exacerbates cardiovascular risk, and vice versa.[18] Importantly, CVD is the leading cause of mortality in individuals with MASLD, often preceding liver-related complications such as cirrhosis or HCC.[19] It accounts for up to 40% of all-cause mortality in this population.[20] Recent large-scale cohort studies and meta-analyses showed that people with MASLD have a 1.5- to 2-fold increased risk of major adverse cardiovascular events, including myocardial infarction, stroke, and cardiovascular death, independent of traditional cardiovascular risk factors.[21,22] This elevated risk is observed across MASLD subtypes, including early-stage disease, and extends to individuals who are lean but metabolically unhealthy.[23] These findings emphasize the need to move beyond body mass index–based assessments and adopt a broader focus on cardiometabolic risk in both clinical and public health settings. This high cardiovascular burden—not liver-specific complications—is the primary driver of morbidity and mortality in MASLD, highlighting the critical role of cardiologists in its management. MASLD is now recognized as a multisystem condition that shares pathophysiologic mechanisms with atherosclerosis, including insulin resistance, chronic inflammation, endothelial dysfunction, and dyslipidemia. Integrating MASLD into

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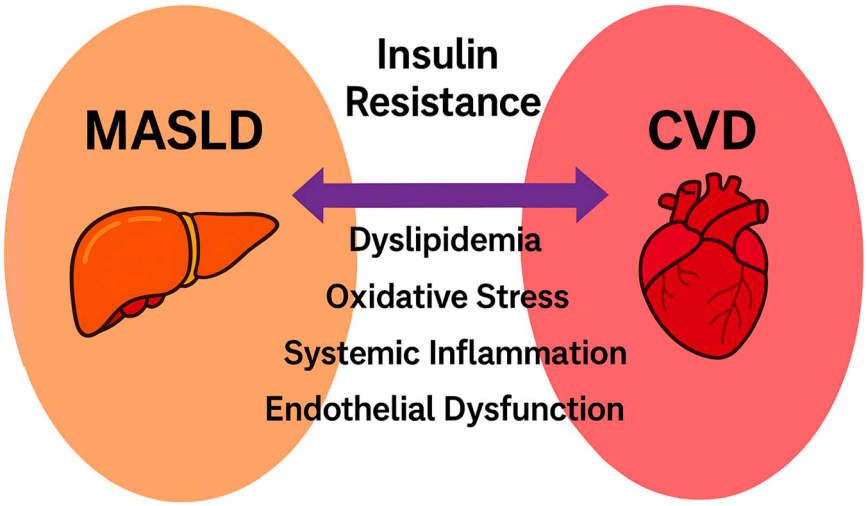

Figure 1. Shared pathophysiology between MASLD and CVD. CVD, cardiovascular disease; MASLD, metabolic dysfunction–associated steatotic liver disease.

cardiovascular prevention and rehabilitation frameworks presents a unique opportunity to address 2 interconnected conditions through synergistic interventions. Recent evidence further reinforces the concept of MASLD as a systemic disease with both hepatic and extrahepatic consequences.[16] The liver, as a central metabolic organ, regulates glucose and lipid metabolism. In MASLD, hepatic steatosis and lipotoxicity impair these regulatory functions, leading to the accumulation of triglycerides within hepatocytes and the release of atherogenic lipoproteins into the circulation. In addition, the liver becomes a source of pro-inflammatory cytokines (eg, tumor necrosis factor-α and interleukin-6) and reactive oxygen species, which contribute to systemic inflammation, vascular damage, and accelerated atherosclerosis.[24] Endothelial dysfunction—a hallmark of early CVD—is commonly observed in MASLD and is exacerbated by both hyperinsulinemia and oxidative stress. This dysfunction impairs nitric oxide bioavailability, leading to increased vascular tone, platelet aggregation, and ultimately plaque formation and instability.[17] In addition, MASLD is associated with coronary artery calcification, a validated surrogate of coronary atherosclerosis and predictor of major cardiovascular events. Multiple large cross-sectional and longitudinal studies have demonstrated that individuals with MASLD, especially those with more severe steatosis or fibrosis, have significantly higher prevalence of coronary artery calcification compared to those without MASLD, independent of traditional cardiovascular risk factors.[25] For instance, in the **M**ulti-**E**thnic **S**tudy of **A**therosclerosis (MESA), participants with hepatic steatosis had a 1.4-fold increased odds of having detectable coronary artery calcification, and those with both steatosis and elevated liver enzymes had an even greater risk.[26] Similar findings were reported in the Framingham Heart Study, which showed that fatty liver was associated with higher coronary artery calcification burden and carotid intima-media thickness after multivariate adjustment.[27] These findings support the hypothesis that MASLD contributes directly to coronary plaque formation through chronic hepatic inflammation, increased oxidative stress, and altered lipid metabolism. Therefore, coronary artery calcification may represent an important link between hepatic and cardiovascular risk in MASLD and could serve as a clinically useful imaging biomarker for stratifying risk in affected patients. Taken together, these processes create a high-risk phenotype in MASLD patients, especially those with additional cardiometabolic conditions such as T2D, hypertension, or chronic kidney disease. The strong interconnection between MASLD and CVD reinforces the need for early screening, risk stratification, and comprehensive management approaches that target both liver and heart health.[28]

Screening and Risk Stratification

Early detection of MASLD-related fibrosis is essential to prevent irreversible liver damage and to reduce long-term complications such as cirrhosis, HCC, and liver failure. Given the asymptomatic nature of MASLD in its early stages, many individuals remain undiagnosed until advanced disease has developed. This highlights the importance of proactive screening strategies, particularly in high-risk populations such as those with obesity, T2D, metabolic syndrome, or persistently elevated liver enzymes.[29] Noninvasive tests have become central to MASLD screening pathways.[30] Among them, the fibrosis-4 (FIB-4) index and vibration-controlled transient elastography (VCTE), commonly known by the name of the device FibroScan (Echosens, Paris, France), are widely validated and recommended as first- and second-line tools, respectively. The FIB-4 score, which incorporates age, aspartate aminotransferase, alanine aminotransferase, and platelet count, is a simple, inexpensive, and readily accessible test that can be implemented in both primary care and specialty settings. It effectively stratifies individuals into low-, intermediate-, and high-risk categories for advanced fibrosis. In cases where FIB-4 is in the indeterminate or high-risk

range, VCTE provides additional information by measuring liver stiffness, which correlates with fibrosis stage (Fig. 2). FibroScan is noninvasive, rapid, and increasingly available in outpatient clinics and diabetes centers, allowing for real-time liver assessment and facilitating timely referral to hepatology when needed.[30,31] Noninvasive tools and a tiered diagnostic approach help detect fibrosis before irreversible damage occurs. This is a significant shift from earlier approaches that relied heavily on alanine aminotransferase and aspartate aminotransferase levels, which can remain normal in around 20% of patients with significant liver fibrosis. The **S**ystemic **Co**ronary **R**isk **E**valuation **2** (SCORE2) may be used to evaluate CVD risk in MASLD patients.[32] Peak oxygen consumption and cardiorespiratory fitness decline are also independently associated with liver fibrosis.[33] By targeting metabolic risk profiles rather than isolated lab abnormalities, this strategy allows for broader case detection and earlier intervention. Beyond FIB-4 and VCTE, serum biomarkers such as Pro-C3 and the enhanced liver fibrosis (ELF) score offer promising alternatives or adjuncts for fibrosis assessment. These serum-based tests can provide insights into active fibrogenesis, potentially identifying patients with progressive disease even before structural changes become evident. However, their current use is limited by cost, lack of widespread availability, and a need for further validation in diverse populations. Advanced imaging modalities, including magnetic resonance imaging—derived proton density fat fraction and magnetic resonance elastography, offer high sensitivity and specificity for quantifying hepatic fat and fibrosis. These tools are particularly useful in clinical trials and specialized centers but remain cost-prohibitive for routine screening and are generally reserved for complex or equivocal cases.[30] Given the spectrum of available diagnostic tools, a tiered screening approach is recommended. This begins with broad implementation of FIB-4 in at-risk populations, followed by selective use of second-line tests such as FibroScan, ELF, or magnetic resonance imaging—based methods, based on initial findings (Fig. 2). Such an approach balances accuracy, accessibility, and cost-effectiveness, making it feasible for both high-resource and resource-limited settings.[30,31] Crucially, non-hepatology providers, including family physicians, endocrinologists, cardiologists, and diabetes educators, must be empowered to integrate MASLD screening into routine clinical workflows. Awareness campaigns, decision-support tools, and clear referral pathways can help bridge the gap between risk identification and specialist evaluation. Expanding the use of noninvasive tests across primary care and chronic disease management programs will be key to reducing the burden of undiagnosed advanced fibrosis and improving long-term outcomes in MASLD.

Lifestyle Interventions: Evidence and Guidelines

CR is a multidisciplinary, evidence-based intervention with well-established benefits in reducing cardiovascular morbidity and mortality.[34] CR programs are ideally positioned to deliver lifestyle modifications that are central to MASLD treatment and offer proven benefits for both liver and cardiovascular health (Table 1). Sustained weight loss of 7%-10% improves steatohepatitis and may even reverse fibrosis, while concurrently reducing cardiometabolic risk factors such as hypertension, dyslipidemia, and insulin resistance.[30,35] Exercise, particularly aerobic and resistance training, has direct hepatic and cardiovascular benefits. Aerobic activity reduces hepatic fat and systemic inflammation and enhances cardiorespiratory fitness, a key predictor of cardiovascular morbidity and mortality.[33,36] Resistance training improves insulin sensitivity, preserves lean mass, and shows excellent adherence, especially in low-fitness individuals.[37] A 2025 systematic review of randomized controlled trials confirmed that resistance training alone improves liver enzymes and insulin sensitivity while strengthening cardiovascular resilience.[37] The Mediterranean diet, rich in monounsaturated fats, antioxidants, and fibers, improves hepatic steatosis, insulin resistance, systemic inflammation, and lipid profiles, collectively reducing atherosclerotic risk.[38-40] When combined with caloric restriction and physical activity, its effects are amplified. A recent randomized controlled trial showed that patients following a low-glycemic Mediterranean diet alongside aerobic or high-intensity interval training experience reductions in liver fat, waist circumference, cortisol levels, and improvements in emotional well-being and quality of life—factors known to influence cardiovascular outcomes.[41] Although the cardioprotective role of the Mediterranean diet is well recognized, emerging evidence highlights the detrimental impact of unhealthy dietary patterns. Diets high in ultraprocessed foods, red and processed meats, saturated fats, and added sugars significantly increase MASLD risk. A mechanistic study demonstrated that diets rich in refined carbohydrates and saturated fats worsened MASLD by promoting abdominal obesity, insulin resistance, and hepatic inflammation, independent of total caloric intake.[42] The Golestan Cohort Study reported that higher red meat consumption was independently associated with incident MASLD (hazard ratio 1.24, 95% confidence interval 1.11-1.39), even after adjusting for physical activity, energy intake, body mass index, and socioeconomic status.[43] Likewise, a meta-analysis of $>$ 50,000 participants found that regular consumption of fructose-sweetened beverages was associated with significantly higher risk of hepatic steatosis (odds ratio 1.53, 95% confidence interval 1.34-1.75), supporting the role of fructose-induced lipogenesis in MASLD pathogenesis.[44] Collectively, these findings establish a strong rationale for implementing Mediterranean-style nutrition and physical activity as both primary and secondary prevention strategies for MASLD in cardiometabolic populations. These interventions address shared mechanisms between MASLD and CVD, including insulin resistance, oxidative stress, dyslipidemia, and endothelial dysfunction, and have been shown to reduce the risk of myocardial infarction, stroke, arrhythmias, and heart failure.[24,36] Even substituting sedentary time with light physical activity lowers cardiovascular risk, including in genetically predisposed individuals.[45] Crucially, lasting clinical impact requires structured behavioral support. Health coaching, motivational interviewing, and cognitive-behavioral therapy empower patients to make sustainable lifestyle changes,[46] whereas group-based programs and peer support further enhance adherence and engagement. Ultimately, lifestyle interventions for MASLD are high-impact, low-risk strategies that improve both hepatic and cardiovascular outcomes. A

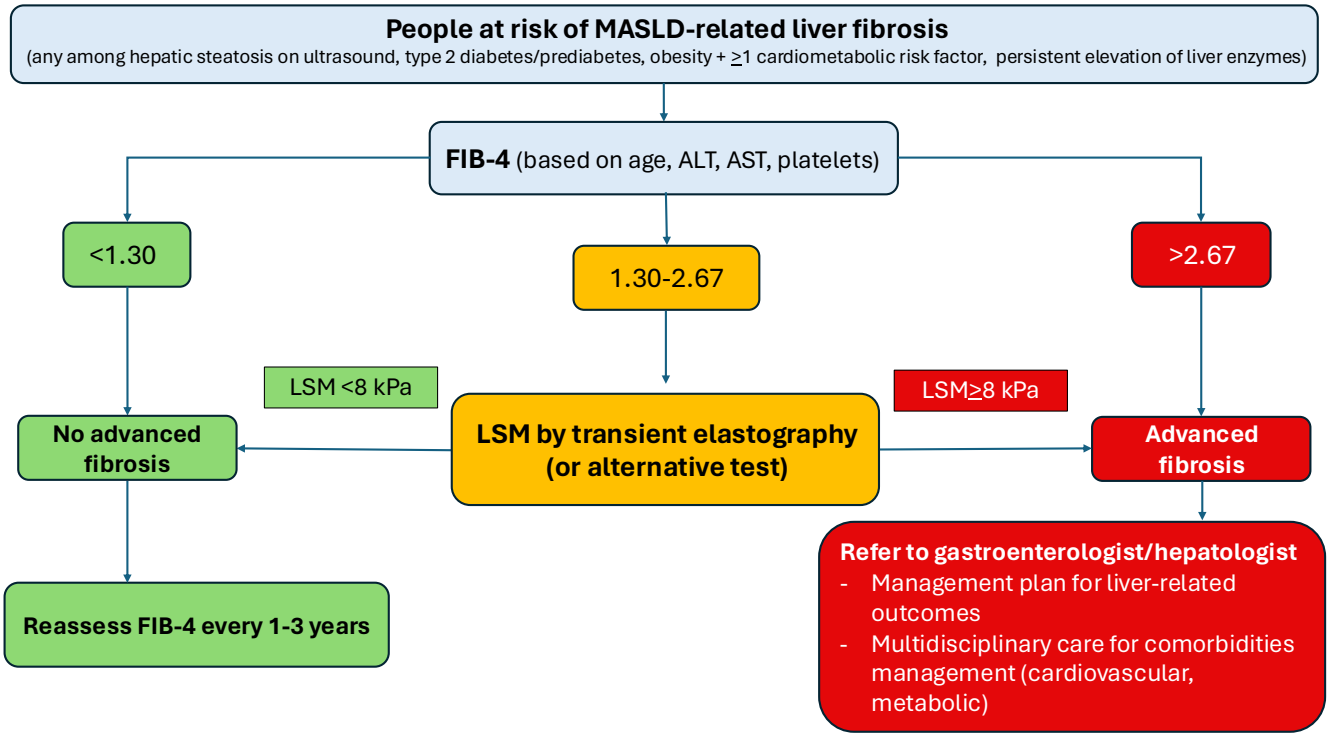

Figure 2. Tiered strategy to screen for MASLD-related liver fibrosis in at-risk populations. Alternative test: enhanced liver fibrosis (ELF) test or shear wave elastography. ALT, alanine aminotransferase; AST, aspartate aminotransferase; FIB-4, fibrosis-4; LSM, liver stiffness measurement.

multidisciplinary, patient-centered approach that integrates nutrition, exercise, and psychosocial support offers the best chance of long-term success, and CR represents an ideal platform for their implementation.

Pharmacotherapy Synergies

Pharmacologic therapies traditionally used in cardiometabolic care are now emerging as important tools in the management of MASLD, offering dual benefits for both liver and heart health.[47,48] Although lifestyle modification remains the first-line treatment, pharmacologic agents are increasingly being integrated into MASLD care, particularly for patients with more advanced disease or multiple comorbidities. Among these, glucagon-like receptor agonists and coagonists, such as semaglutide, have shown substantial promise.[49] In the **E**ffect of **S**emiglutide in **S**ubj**e**cts with **N**on-cirrhotic Non-alcoholic **St**eatohepatitis (ESSENCE) phase 3 trial, semaglutide demonstrated significant efficacy in achieving resolution of metabolic dysfunction–associated steatohepatitis (MASH) and improvement in fibrosis.[50] Positive findings have also been reported in phase 2 trials of tirzepatide and survodutide for the treatment of MASH.[51,52] Furthermore, the **Se**magl**u**tide **Effec**ts on Heart Disease and Stroke in Patients with Overweight and Obesity (SELECT) trial demonstrated a significant reduction in major adverse cardiovascular events in people with established CVD and obesity, even in the absence of diabetes.[49] Mechanistically, these drugs improve insulin secretion, suppress glucagon, reduce *de novo* lipogenesis in the liver, lower systemic inflammation, and enhance endothelial function—addressing key drivers of both MASLD progression and atherosclerosis. Another notable agent is resmetirom, a selective thyroid hormone receptor beta agonist, which has demonstrated histologic efficacy in MASH with fibrosis and is currently the only pharmacotherapy for this condition approved by the US Food and Drug Administration.[53] Its liver-selective activity enhances hepatic fat oxidation and reduces lipid accumulation, without systemic thyromimetic effects. Additional classes under investigation include sodium-glucose cotransporter-2 (SGLT2) inhibitors (eg, empagliflozin and

Table 1. Components of a multidisciplinary MASLD-cardiac rehabilitation program

Component	Description
Medical assessment	Hepatology and cardiology evaluation, risk stratification for liver fibrosis and CVD
Lifestyle counseling	Nutrition, weight loss; alcohol and smoking cessation
Exercise training	Aerobic and resistance training; high-intensity interval training where appropriate
Pharmacologic review	Optimization of antihypertension, lipid-lowering, antidiabetic agents; weight management, and MASLD therapies
Psychosocial support	Mental health screening, behavioral coaching
Patient education	Disease awareness, self-management skills, goals setting
Care coordination	Integration with primary care, endocrinology, and long-term hepatology follow-up where appropriate

CVD, cardiovascular disease; MASLD, metabolic dysfunction–associated steatotic liver disease.

canagliflozin), triple incretin agonists (eg, retatrutide), and peroxisome proliferator–activated receptor agonists (eg, pioglitazone and lanifibranor).[54] Although SGLT2 inhibitors are less studied in MASLD-specific trials, they have demonstrated benefits in reducing visceral adiposity, insulin resistance, and systemic inflammation.[55] These effects, combined with cardiorenal protection and reduced heart failure hospitalizations, make them highly relevant for MASLD patients with overlapping cardiometabolic disease. For patients with T2D and MASLD, pioglitazone remains a recommended agent due to its anti-inflammatory and insulin-sensitizing properties, despite concerns about weight gain. Its effect on liver histology has been well documented, particularly in slowing fibrosis progression.[56] Emerging therapies such as fibroblast growth factor 21 analogs and peroxisome proliferator–activated receptor-α/δ agonists aim to target multiple metabolic pathways simultaneously, reflecting the heterogeneous nature of MASLD.[57,58] As these agents progress through clinical trials, treatment is likely to become more phenotype-driven, allowing clinicians to tailor therapy to the patient's metabolic profile, degree of liver involvement, and cardiovascular risk. Anti-inflammatory and antidiabetic pathways converge in their effect on hepatic and cardiovascular endpoints. In summary, the convergence of cardiometabolic and hepatologic pharmacotherapy represents a major advance in MASLD care. Agents such as glucagon-like receptor agonists and SGLT2 inhibitors herald a new era in which targeting shared pathophysiologic mechanisms can simultaneously reduce cardiovascular events and halt liver disease progression—offering a comprehensive strategy for improving outcomes in this high-risk population.

Cardiac Rehabilitation: An Untapped Opportunity

Traditionally designed for patients recovering from myocardial infarction or cardiac surgery, CR has expanded in scope to include individuals with metabolic syndrome, T2D, and other high-risk conditions, even in the absence of overt cardiovascular events. It may be time to extend this model to people with MASLD, given the strong pathophysiologic and clinical links between liver steatosis, metabolic dysfunction, and CVD. Structured CR programs typically include aerobic and resistance exercise, dietary counselling, smoking cessation, and psychosocial support—interventions that directly address both the hepatic and cardiovascular components of MASLD.[34] Studies have shown that CR participation can lead to improvements in hepatic steatosis, insulin sensitivity, waist circumference, and overall quality of life.[59,60] Importantly, MASLD should be recognized by cardiologists as not only a hepatic disorder but also as a cardiometabolic risk enhancer. This recognition should prompt early referral of MASLD patients—especially those with fibrosis, T2D, or established atherosclerotic disease—to CR programs. Integrating MASLD assessment into CR intake evaluations could identify previously unrecognized liver disease, offering a unique opportunity for early multidisciplinary intervention. These effects are particularly important given the limited pharmacologic options for early MASLD and the high prevalence of shared risk factors with CVD. By incorporating health coaching, motivational interviewing, and tailored exercise prescriptions, CR provides a supportive and individualized framework to help patients implement sustained lifestyle changes. Many MASLD patients struggle to initiate or maintain these changes independently; CR offers a supervised, goal-oriented environment that improves adherence and empowers patients through education and accountability. Crucially, CR programs are inherently designed for multidisciplinary collaboration, bringing together cardiologists, hepatologists, nutritionists, physiotherapists, psychologists, kinesiologists, and diabetes educators. This model is well-suited to the complex care needs of MASLD patients, who often fall between specialty silos. Moreover, regular monitoring of cardiometabolic parameters within CR settings may allow for early identification of MASLD progression and timely adjustments in care. Expanding access to CR for patients with MASLD represents an opportunity to leverage an existing, effective platform to deliver integrated metabolic care. CR is no longer solely a post–myocardial infarction tool, but a cornerstone of modern, holistic cardiometabolic management where MASLD should have a defined place. Embedding liver health into cardiometabolic rehabilitation aligns with a broader shift toward holistic, preventive models in chronic disease management.

Conclusions

MASLD is a growing global health challenge with profound implications for both liver and cardiovascular outcomes. Its close association with cardiometabolic dysfunction highlights the need for urgent, coordinated action that extends beyond hepatology. CR, a proven model for managing cardiovascular risk reduction, offers a promising yet underutilized setting to address the complexity of MASLD. Traditionally seen as a post–cardiac event intervention, CR should now be recognized as a proactive approach to addressing the shared pathophysiology of disease states such as MASLD and CVD. By integrating structured lifestyle modification, pharmacologic interventions, and patient education within a multidisciplinary framework, CR is uniquely positioned to meet the complex needs of individuals with MASLD. Importantly, cardiologists should begin to view MASLD not merely as a hepatic disorder, but as a cardiometabolic condition that significantly elevates cardiovascular risk. This evolving understanding should support a paradigm shift, prompting routine assessment for MASLD and early referral to CR, particularly in patients with metabolic dysfunction or liver fibrosis, even before cardiovascular events occur. Moving forward, the most effective approach to reducing morbidity and mortality will be a holistic strategy that bridges disciplines, leverages shared risk reduction, and empowers patients to participate actively in their care.

Ethics Statement

This article is an invited review article and does not report any original reseearch involving human participants. Therefore, ethical approval was not required.

Declaration of Generative AI and AI-Assisted Technologies in the Writing Process

During the preparation of this work the author(s) used ChatGPT-4o in order to review grammar, improve clarity and refine wording. After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the publication.

Patient Consent

We confirm that patient consent is not applicable as this is a review article; therefore, institutional review board approval was not required.

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