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EDITORIAL

Addressing the alarming link between nonalcoholic fatty liver disease and cardiovascular mortality in men

Wen-Rui Hao, Chun-Han Cheng, Tzu-Hurng Cheng

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Abstract

This editorial discusses the key findings presented in Batta and Hatwal's recent paper titled "Excess cardiovascular mortality in men with non-alcoholic fatty liver disease: A cause for concern!", which was published in the *World Journal of Cardiology*. Their original article highlights a notable correlation between non-alcoholic fatty liver disease (NAFLD) and increased cardiovascular mortality risk in men. The present commentary explores the implications of their findings, discussing potential mechanisms, risk factors, and the urgent need for integrated clinical approaches to mitigate the dual burden of these diseases. Emphasis should be placed on the importance of early detection, lifestyle modifications, and interdisciplinary collaboration for improving patient outcomes. This editorial aims to highlight the broad implications of NAFLD for cardiovascular health and to advocate for increased awareness and proactive management strategies within the medical community.

Key Words: Non-alcoholic fatty liver disease; Cardiovascular mortality; Men's health; Integrated clinical approaches; Risk factors

Core Tip: Men with nonalcoholic fatty liver disease (NAFLD) have a significantly increased risk of cardiovascular mortality, which is a major health concern. Early detection and comprehensive management strategies targeting both NAFLD and cardiovascular risk factors are essential to mitigate excess mortality.

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INTRODUCTION

Nonalcoholic fatty liver disease (NAFLD) has emerged as a major global health concern, affecting a substantial portion of the population worldwide. This editorial focuses on a key aspect of NAFLD, namely its association with excess cardiovascular mortality in men. Recent epidemiological studies and clinical observations have underscored that men with NAFLD have notably higher risks of cardiovascular events and mortality compared with their counterparts without this condition[1]. The increasing prevalence of NAFLD parallels the worldwide increase in obesity and metabolic syndrome, highlighting the intricate relationship between liver health and cardiovascular outcomes[2]. Despite advancements in understanding the pathogenesis and systemic implications of NAFLD, effective strategies for managing cardiovascular risks in patients with NAFLD remain limited and under-researched[3]. This editorial synthesizes the available evidence, discusses the underlying mechanisms linking NAFLD to cardiovascular mortality in men, and proposes potential methods for enhancing clinical management and patient outcomes[1]. By addressing these topics comprehensively, we intend to stimulate further research and inform clinical guidelines, thereby helping to reduce the dual burden of NAFLD and cardiovascular disease.

EPIDEMIOLOGICAL EVIDENCE

Recent epidemiological studies have consistently highlighted a concerning association between NAFLD and increased cardiovascular mortality risk, particularly in men. This association has been identified in diverse populations and through various methodologies, reinforcing the robustness of the correlation despite variations in demographic profiles and risk factor distributions[1-5]. The prevalence of NAFLD has increased in tandem with obesity rates, underscoring the urgent need to address the cardiovascular implications of NAFLD. The synergy between NAFLD and cardiometabolic conditions such as type 2 diabetes mellitus complicates this landscape and worsens both cardiovascular and liver-related outcomes[4]. Studies have demonstrated that NAFLD is a significant predictor of cardiovascular mortality, with men exhibiting a higher risk than women; sex-specific factors should this be considered in clinical management[1,6]. In addition to cardiovascular mortality, disparities in the prevalence and effects of metabolic-dysfunction-associated steatotic liver disease (MASLD) are notable across socioeconomic strata. For instance, a study reported that MASLD was disproportionately prevalent in low-income and lower-middle-income countries, reflecting a global health burden that is compounded by inconsistent access to health-care resources and preventive measures[3]. Research has identified specific dietary patterns and metabolic dysfunctions that predispose individuals to MASLD. The findings emphasize the role of lifestyle factors, such as diet, in the progression of NAFLD and MASLD. For example, healthy eating patterns have been linked to a reduced risk of MASLD, suggesting that dietary modification is a viable preventive strategy[5]. Conversely, inadequate dietary interventions have been demonstrated to be ineffective in mitigating disease progression, a finding that underscores the importance of tailored nutritional strategies. The identification of novel therapeutic targets is crucial for managing NAFLD and its cardiovascular implications. Recent studies have highlighted various potential treatments, such as epigallocatechin gallate, which alleviates NAFLD by inhibiting dipeptidyl peptidase 4 (DPP-4) activity, and the Yanxiao Di'naer formula, which has achieved promising outcomes according to metabolomic and RNA sequencing results[7,8]. These interventions represent ongoing efforts to address the progression of NAFLD and its sequelae. Understanding the intricate pathophysiological mechanisms underlying NAFLD and MASLD is essential for designing targeted preventive strategies and treatment modalities. This comprehensive approach aims to reduce cardiovascular mortality and address the broader spectrum of metabolic and hepatic complications associated with NAFLD and MASLD, which are becoming more prevalent[9,10]. Overall, although the epidemiological evidence linking NAFLD to increased cardiovascular mortality in men is robust, further research is required to elucidate the full spectrum of implications and optimize the relevant management strategies.

UNDERLYING MECHANISMS

The pathophysiological mechanisms linking NAFLD to increased cardiovascular mortality are multifaceted and involve several interconnected pathways. Insulin resistance, a hallmark of NAFLD, not only promotes hepatic lipid accumulation

but also contributes significantly to systemic inflammation and dyslipidemia[1,4]. These metabolic disturbances foster a proatherogenic environment by impairing endothelial function and promoting plaque formation[2]. Chronic inflammation, another critical feature of NAFLD, exacerbates this process by perpetuating atherosclerosis through the secretion of proinflammatory cytokines[3]. Additionally, oxidative stress, which is intensified by the presence of hepatic steatosis, further amplifies cardiovascular risk by damaging endothelial cells and promoting vascular dysfunction[5]. An oxidative milieu not only accelerates atherogenesis but also exacerbates systemic inflammation, creating a vicious cycle that contributes to adverse cardiovascular outcomes[7]. In addition to these mechanisms, genetic factors play a crucial role in the pathophysiology of NAFLD and its cardiovascular implications. Variants in genes such as *PNPLA3*, *TM6SF2*, and *MBOAT7* have been linked to increased risks of hepatic steatosis and cardiovascular diseases[11]. These genetic predispositions can influence lipid metabolism, inflammatory responses, and overall disease progression. Additionally, dysregulation in lipid metabolism, a central feature of NAFLD, contributes significantly to cardiovascular risk. Elevated levels of triglycerides and altered lipoprotein profiles promote the formation of atherogenic lipoprotein particles, which contribute to atherosclerosis[12]. Furthermore, the interplay between NAFLD and the gut microbiota has emerged as a key area of research. Dysbiosis, which refers to an imbalance in gut microbial communities, can influence NAFLD progression by modulating systemic inflammation, insulin resistance, and lipid metabolism[13]. The cumulative effect of these interconnected pathways results in an interplay between NAFLD and cardiovascular morbidity, which necessitates comprehensive management strategies that address both liver and cardiovascular health[8]. Overall, understanding the underlying mechanisms is crucial for developing targeted therapies that mitigate the cardiovascular risk associated with NAFLD. Future research should continue to explore these pathways to identify novel therapeutic targets and interventions aimed at reducing the burden of cardiovascular mortality in individuals with NAFLD[9,10]. By addressing the metabolic dysregulation, genetic factors, inflammatory processes, and gut microbiota associated with both NAFLD and cardiovascular disease, clinicians can optimize patient outcomes and improve overall cardiovascular health in this high-risk population.

CLINICAL IMPLICATIONS AND CHALLENGES

Managing cardiovascular risk in patients with NAFLD or MASLD is challenging because of the unique metabolic and inflammatory profiles associated with liver fat accumulation, which traditional cardiovascular risk assessment tools often underestimate[1]. Comprehensive management strategies must integrate both liver health and cardiovascular risk factors, addressing the complex interaction between metabolic dysfunction and cardiovascular disease[2,4]. Type 2 diabetes significantly exacerbates outcomes related to cardiovascular health, liver disease, and cancer; personalized management strategies tailored to the multifaceted nature of MASLD are thus required[3,4]. Emerging treatment strategies for NAFLD and MASLD include both lifestyle interventions and pharmacological therapies. Lifestyle modifications, such as dietary changes and increased physical activity, are fundamental in managing these conditions. Specific dietary patterns, such as the Mediterranean diet, have shown promise in improving liver health and reducing cardiovascular risk[5]. Pharmacological interventions are evolving, with several novel agents under investigation. These include glucagon-like peptide-1 receptor agonists and sodium-glucose co-transporter-2 inhibitors, which have been demonstrated to have beneficial effects in terms of liver fat reduction and cardiovascular outcomes[9]. Recent advancements have also been made in targeting the specific molecular pathways involved in NAFLD and MASLD. For example, studies have indicated that drugs targeting the NLRP3 inflammasome and TLR4 pathways can reduce liver inflammation and fibrosis[14,15]. Additionally, agents such as epigallocatechin gallate have been effective in inhibiting DPP-4 activity and thereby ameliorating NAFLD[7]. Noninvasive biomarkers and imaging techniques are increasingly being used to assess disease progression and therapeutic response and enable the creation of precise and personalized treatment plans[16,17]. Recent research efforts have aimed to develop and validate specific treatment methods and interventions for improving cardiovascular health in patients with NAFLD. For example, observational and genetic studies have established an association between NAFLD and calcific aortic valve disease, suggesting that cardiovascular complications are a major concern for these patients[11]. Additionally, diagnostic biomarkers such as CXCL9, IL2RB, and SPP1 have been identified as potential indicators of comorbid atherosclerosis and nonalcoholic steatohepatitis, and these findings provide new avenues for early detection and targeted therapy[18]. Global health initiatives must be launched to provide equitable access to care and tailored interventions to mitigate the disparities in the prevalence and effects of MASLD in low-income and lower-middle-income countries[3,7]. Implementing comprehensive guidelines that integrate metabolic and liver-specific approaches is crucial for improving patient outcomes globally[19]. Overall, managing cardiovascular risk in patients with NAFLD or MASLD requires a paradigm shift toward integrated, personalized care strategies. These strategies should incorporate comprehensive risk assessment tools, novel therapeutic approaches, and global health initiatives to optimize patient outcomes and reduce the burden of cardiovascular disease in this vulnerable population. The transition from NAFLD to MASLD highlights the importance of adopting new terminologies and comprehensive management guidelines to effectively address the interplay between metabolic and liver diseases.

ONGOING RESEARCH AND POTENTIAL FUTURE STUDIES

Recent studies have begun to explore the complex relationship between NAFLD and cardiovascular diseases. Batta and Hatwal noted excessively high cardiovascular mortality in men with NAFLD and reported an urgent need to obtain primary data to validate their findings and examine the underlying mechanisms[1]. Similarly, Riley *et al*[4] discussed the

synergistic effect of type 2 diabetes and MASLD on various health outcomes, including those related to cardiovascular and liver diseases. Their findings highlight the importance of conducting longitudinal studies to elucidate various causal relationships and potential interventions. The shift from the term NAFLD to MASLD, as discussed by Malnick and Zamir [2], reflects a broader understanding of the disease's metabolic origins and the key implications for future research. This new terminology encourages a more comprehensive approach to studying the disease in which the metabolic dysfunctions involved are considered. Future studies should focus on examining the genetic and environmental factors contributing to MASLD and identifying potential therapeutic targets. Beygi *et al*[20] reviewed various strategies for managing MASLD, including medication therapy and nutritional interventions. They emphasized the need for clinical trials to test the efficacy of these treatments in diverse populations. Observational and genetic studies, such as those conducted by Hao *et al*[11], have revealed associations between NAFLD and other conditions such as calcific aortic valve disease, suggesting new avenues for research into shared pathophysiological pathways. Wu *et al*[18] identified potential diagnostic biomarkers of comorbid atherosclerosis and nonalcoholic steatohepatitis, and their results pave the way for the development of more precise diagnostic tools. Huang *et al*[5] identified a strong association between beneficial lifestyle modifications and reduced mortality risk in patients with NAFLD, underlining the relevance of lifestyle interventions in managing the disease. These findings should be validated in randomized controlled trials to establish causality and facilitate the development of optimal intervention strategies. The role of triglyceride–glucose indices in predicting mortality in patients with NAFLD, as highlighted by Chen *et al*[12], suggests that metabolic markers are crucial in risk stratification and management. Future research should validate these indices in larger cohorts and diverse populations. Studies on sex and race-ethnic disparities in MASLD, such as that conducted by Fu *et al*[6], are crucial in understanding the disease's epidemiology and developing targeted interventions. Given the aforementioned disparities, more inclusive research that considers genetic, social, and environmental factors affecting disease prevalence and progression is needed. Research on noninvasive biomarkers and their prognostic value, as reviewed by Amoroso *et al*[16], has demonstrated that these biomarkers have promise in predicting disease outcomes and tailoring treatment plans. These biomarkers should be validated in large, multicenter studies to verify their reliability and applicability in clinical practice. The exploration of novel therapeutic agents, such as those discussed by Yang *et al*[7] and Zheng *et al*[8], provides hope for new treatment options for NAFLD and MASLD. These studies underscore the relevance of ongoing research into the molecular mechanisms of the disease and the development of targeted therapies. Overall, this review of the literature has revealed key gaps and opportunities in NAFLD and MASLD research. Specifically, ongoing and future studies should focus on longitudinal data collection, genetic and environmental factors, the efficacy of various treatments, and the development of noninvasive diagnostic tools. Addressing these areas will yield new primary data, advance the understanding of the disease, and improve patient outcomes.

FUTURE DIRECTIONS AND RESEARCH NEEDS:

Future research should prioritize elucidating the precise mechanisms through which NAFLD predisposes individuals to increased cardiovascular mortality risk, focusing on sex-specific disparities and tailored management strategies. The synergistic effect of type 2 diabetes and MASLD on cardiovascular outcomes underscores the need for comprehensive studies examining the interplay of these diseases and their shared pathophysiological pathways[2,4]. Longitudinal investigations are crucial for validating risk stratification models and therapeutic interventions aimed at mitigating cardiovascular morbidity and mortality in patients with NAFLD or MASLD[3]. Collaborative efforts among hepatologists, cardiologists, and primary care providers are essential for optimizing patient care and mitigating the increasing burden of NAFLD-related cardiovascular complications[1,10]. Furthermore, disparities in MASLD and associated cardiometabolic conditions in the diverse global context must be addressed through systematic analyses and tailored public health interventions[3,19]. Innovative approaches integrating metabolomics with RNA sequencing can provide deeper insights into the efficacy and underlying mechanisms of potential therapeutic agents for treating NAFLD and its cardiovascular consequences[8,15]. Efforts to characterize dietary patterns and their effects on MASLD underscore the potential of dietary interventions as a primary prevention strategy[5]. Additionally, increased understanding of the role of gut–liver axis interactions and mast cell involvement in liver disease progression has led to the identification of various novel therapeutic targets[13]. Insights from genetic studies underscore the relevance of identifying genetic determinants influencing susceptibility to NAFLD and cardiovascular risk in patients with coronary heart disease[21]. Overall, future research should focus on integrating multidisciplinary approaches to elucidate the mechanisms linking NAFLD to cardiovascular mortality, validate risk assessment tools, and develop effective therapeutic strategies tailored to individual patients' needs.

CONCLUSION

In conclusion, the strong correlation between NAFLD and increased cardiovascular mortality risk in men highlights a crucial intersection between liver health and cardiovascular outcomes. Epidemiological findings consistently indicate an increased cardiovascular risk in individuals with NAFLD due to shared metabolic dysregulation and inflammatory pathways[1,4]. To effectively address these connected health challenges, concerted efforts are required to enhance early detection, refine risk assessment tools, and implement integrated management strategies targeting both liver-specific pathology and cardiovascular risk factors. Future research endeavors should prioritize elucidation of the mechanistic underpinnings of NAFLD-associated cardiovascular mortality, focusing on sex-specific disparities and personalized

therapeutic approaches[3,8]. Collaborations across medical disciplines are essential for optimizing clinical pathways and improving outcomes for high-risk patients. Through bridging of the gaps in understanding and clinical practice, the dual burden of NAFLD and cardiovascular disease can be mitigated and the health trajectory of affected individuals enhanced [2,19].

FOOTNOTES

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