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EDITORIAL

Excess cardiovascular mortality in men with non-alcoholic fatty liver disease: A cause for concern!

Akash Batta, Juniali Hatwal

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Abstract

Non-alcoholic fatty liver disease (NAFLD) has emerged as the commonest cause of chronic liver disease worldwide in recent years. With time, our understanding of NAFLD has evolved from an isolated liver condition to a systemic disease with significant manifestations beyond the liver. Amongst them, cardiovascular diseases (CVDs) are the most important and clinically relevant. Recent research supports a strong independent link between NALFD and CVD beyond the shared risk factors and pathophysiology. Female sex hormones are well known to not only protect against CVD in pre-menopausal females, but also contribute to improved adipose tissue function and preventing its systemic deposition. Recent research highlights the increased risk of major adverse cardiovascular-cerebral events (MACCE) amongst male with NAFLD compared to females. Further, racial variation was observed in MACCE outcomes in NAFLD, with excess mortality in the Native Americans and Asian Pacific Islanders compared to the other races.

Key Words: Non-alcoholic fatty liver disease; Cardiovascular diseases; Male sex; Major adverse cardiovascular-cerebral events; Inflammation; Endothelial dysfunction

Core Tip: Non-alcoholic fatty liver disease (NAFLD) has emerged as the commonest cause of chronic liver disease worldwide in recent years. In recent years, our understanding of NAFLD has evolved from an isolated liver condition to a systemic disease with significant manifestations beyond the liver. Amongst them, cardiovascular diseases (CVDs) are the most important and clinically relevant. Recent research supports a strong independent link between NALFD and CVD beyond the shared risk factors and pathophysiology. The findings from translational research and recent clinical data support the heightened risk of major adverse cardiovascular-cerebral events (MACCE) amongst male with NAFLD compared to females. Further, there was racial variation in MACCE outcomes in NAFLD, with excess mortality in the Native Americans and Asian Pacific Islanders compared to the other races largely attributable to the increased comorbidity burden and unfavorable genetics.

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INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) has emerged as the commonest cause of chronic liver disease worldwide in recent years. The current estimates indicate that the prevalence is expected to rise logarithmically in the coming years especially so in the developing world[1,2]. Recent evidence has improved our understanding of the disease and appropriately it is now being viewed as truly a multisystem disease. Significant systemic implications of the disease necessitate a comprehensive multidisciplinary approach to improve outcomes[3,4]. Amongst the systemic manifestations of NAFLD, notable mentions include cardiovascular diseases (CVDs), chronic kidney disease, type 2 diabetes mellitus and certain malignancies[5]. Accordingly, it is imperative for clinicians taking care of patients with NAFLD to be aware of these associations and identify those at increased risk so that appropriate measures can be instituted in a timely manner.

PATHOPHYSIOLOGICAL BASIS OF INTERACTION BETWEEN NAFLD AND CVD

NAFLD is characterized by dysfunction of the adipose tissue and its deposition in visceral organs including both the liver and the heart[6,7]. This ectopic/visceral deposition of adipose tissue results in activation of inflammatory cytokines and oxidative stress which underlies a common theme between NAFLD and CVD[8]. Despite the overwhelming evidence supporting the excess CVD in NAFLD, the exact pathophysiological mechanisms involved have remained speculative. Arguably, the common lifestyle risk factors between the two diseases like cigarette smoking, poor nutritional habits, sedentary life style together with other triggers in the form of oxidate stress, alterations in gut microbiome, and inflammation culminate in obesity, hypertension, dyslipidaemia and diabetes which in turn are common grounds for both NAFLD and CVD[9,10]. Further, the most validated pathogenic mechanism in NAFLD which predispose to CVD are a combination of endothelial dysfunction, systemic inflammation, altered glucose metabolism and atherogenic dyslipidaemia which results in increased progression to microvascular dysfunction and macrovascular atherosclerotic CVD. The low-grade inflammation attributable largely to the unhealthy lifestyle habits and exposure to carbohydrate and fat rich diet results in altered endothelial function and increased predisposition for deposition of oxidized low-density lipoprotein into the visceral arterial bed. In particular, pro-inflammatory cytokines (IL-6, IL-17, IL-1B and TNF-a) and hepatokines (FGF-21 and fetuin-A) together with pro-coagulant cytokines (TGF-B, FVIII, FGF-21) remain central to the pathophysiology and contribute to enhanced systemic atherosclerosis and its deleterious effects[11,12]. Ultimately, this contributes to excess hypertension, coronary artery disease, heart failure (both reduced and with preserved ejection fraction) and cardiac arrythmias (most notably atrial fibrillation) which confers increased CVD related mortality in NAFLD patients. Even beyond the shared risk factors and pathophysiology, there is clear evidence supporting the independent CVD risk due to NAFLD[9]. Arguably most of the imminent guidelines recommend that the diagnosis of NAFLD should be followed by a careful and comprehensive cardiovascular risk assessment and evaluation for subclinical atherosclerosis where indicated[13]. To this end, there have been development of certain unique risk scores including Fibrosis-4 index, NAFLD activity and NAFLD fibrosis scores all of which may help in early identification of high-risk group who are at a heightened risk for developing CVD[12].

INFLUENCE OF SEX ON NAFLD RELATED CVD OUTCOMES

There is abundant evidence supporting the protective influence of female sex hormones when regards to initiation and progression of NAFLD. In general, NAFLD is diagnosed 5 years later in females compared to their male counterparts [14]. Further, it is well established that estrogen also has a positive effect on CVD profile in pre-menopausal women and after menopause, the risk for both NAFLD and CVD are comparable to men.

Consistently, most recent reports support the excess CVD related mortality in males with NAFLD compared to age and comorbidity matched females. The increased risk and accelerated progression of NAFLD resulting in heightened systematic inflammation is in part linked to the differences adipocyte metabolism in the male sex. In the reproductive years, female sex is largely protected from the adverse metabolism due to their preferential partitioning of free fatty acids towards the ketone body pathway as opposed to the very low-density lipoprotein-triacylglycerol pathway (more prevalent in males) which has a tendency for visceral deposition and initiation of inflammatory cascade. Further, sex-specific browning of white adipose tissue also adds to the protection against NAFLD in females[14-16]. Indeed, estrogen plays a crucial role in steatogenesis and lipidomics. Experimental rat models have consistently demonstrated estrogen deficiency results in reduced concentrations of peroxisome proliferator-activated receptor and upregulation of genes mediating endogenous synthesis of cholesterol and free fatty acids culminating in hepatic steatosis[17,18]. Post menopause, as the protective effect of estrogen weans, the risk of developing NAFLD becomes similar to males. In females, estrogen deficiency is often paralleled by development of cardio-metabolic risk factors including diabetes, dyslipidemia and obesity which often facilitates accelerated steatosis in the hepatocytes which may soon progress to fibroses and ultimately chronic liver disease[19,20].

IMPACT OF SEX AND RACE ON MAJOR ADVERSE CARDIOVASCULAR-CEREBRAL EVENTS IN NAFLD

In the recent retrospective analysis by Desai *et al*[21], adult hospitalizations for NAFLD were analyzed on the National Inpatient Sample (2019), in particular looking into the age, sex and racial determinants of major adverse cardiovascular-cerebral events (MACCE) amongst these patients. In their analysis of 409130 hospitalizations for NAFLD, these found out that females had a higher prevalence of obesity and uncomplicated diabetes compared to male gender. Likewise, male gender was associated with a greater prevalence of hypertension, dyslipidemia and complicated diabetes that their female counterparts. Overall, MACCE was strongly linked to advancing age ($P < 0.001$) which seems logical as with advancing age, the burden of atherosclerosis and its attendant complications are expected to increase. Notably, they also concluded that after adjusting for all variables, male sex was associated with higher odds of having MACCE, myocardial infarction and cardiac arrest compared to females. The higher CVD risk amongst males with NAFLD has been observed before and the usual reasons cited include the higher consumption of calorie-rich energy drinks and alcohol coupled with a greater likelihood of developing insulin resistance[22,23]. The index study together with prior research clearly indicates the greater degree of dysmetabolism and inflammation in males with NAFLD and makes a strong case for aggressive risk profile assessment and correction in this population.

Another observation from the study was the excess mortality in the Native Americans and Asian Pacific Islanders with NAFLD compared to the other races. Perhaps a higher prevalence of chronic diseases including diabetes, hypertension and dyslipidemia, a sedentary lifestyle and unfavorable genetic make-up are likely to be responsible for the same[24,25].

Despite the important and relevant conclusions from the index paper, one must also acknowledge the key limitations of the study which are likely to impact its generalizability to other parts to the world. Firstly, the retrospective design is inherently prone to numerous biases which could have altered the study results. Secondly, one must appreciate the fact that the index study only applies to NAFLD patients who required admissions. As such, the findings of this study do not apply to vast majority of NAFLD patients who are ambulatory and have not needed a hospitalization. Thirdly, the study solely looked in to the NAFLD patients across the United States and hence its validity needs to be established in other parts of the world.

CONCLUSION

NAFLD is a systemic disease with significant manifestations beyond the liver. Amongst them, CVDs are the most important and clinically relevant. Recent research supports a strong independent link between NALFD and CVD beyond the shared risk factors and pathophysiology. The findings from translational research and recent clinical data support the heightened risk of MACCE amongst male with NAFLD compared to females. Further, there was racial variation in MACCE outcomes in NAFLD, with excess mortality in the Native Americans and Asian Pacific Islanders compared to the other races.

FOOTNOTES

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