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"THE ROLE OF STATIN THERAPY IN PATIENTS WITH NON-ALCOHOLIC FATTY LIVER DISEASE AND ATHEROSCLEROSIS: A COMPREHENSIVE REVIEW"

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Abstract: Non-alcoholic fatty liver disease has emerged as a major global public health concern, affecting approximately one-third of the adult population worldwide. Increasing evidence indicates that NAFLD is not only a liver-specific condition but also a multisystem disease closely linked to Atherosclerosis and cardiovascular morbidity. Cardiovascular disease remains the leading cause of mortality among patients with NAFLD. This review aims to evaluate the role of statin therapy in patients with NAFLD and concomitant atherosclerosis, focusing on its efficacy, safety, and impact on cardiovascular outcomes. A narrative review of current literature was conducted, including observational studies, randomized clinical trials, and international guideline recommendations addressing statin use in NAFLD and cardiovascular disease. Accumulating data suggest that statins significantly reduce cardiovascular risk in patients with NAFLD by improving lipid profiles, stabilizing atherosclerotic plaques, and exerting anti-inflammatory effects. Importantly, statins have been shown to be safe in most patients with NAFLD, with no increased risk of clinically significant hepatotoxicity. Emerging evidence also indicates potential beneficial effects on liver enzymes and histological parameters. Statin therapy plays a pivotal role in the management of patients with NAFLD and atherosclerosis, offering dual benefits by reducing cardiovascular risk without adversely affecting liver function. Despite strong evidence, statins remain underutilized in this population, highlighting the need for increased awareness and adherence to clinical guidelines.

Introduction

The 2017 Global Burden of Disease Study reported approximately 2.14 million (2.06–2.30 million) deaths related to liver disease, representing an 11.5% increase compared to 2012. Most of these deaths were due to liver cancer and cirrhosis. Although chronic viral hepatitis remains the leading global cause of liver-related mortality, evidence shows that Non-alcoholic fatty liver disease is the fastest-growing contributor to both mortality and morbidity. In 2016, it was estimated that more than 64 million people in the United States had NAFLD, with annual direct healthcare costs of around $103 billion (approximately $1,613 per patient). In four European countries—France, Germany, Italy, and the United Kingdom—about 52 million individuals were affected, with total annual costs reaching €35 billion (€354 to €1,163 per patient). The highest costs were observed among individuals aged 45–65 years, a working-age group that also bears a significant economic burden from cardiovascular diseases. Cardiovascular diseases (CVDs), including ischemic heart disease and stroke, are the most common non-communicable diseases worldwide, causing an estimated 17.8 million deaths in 2017. More than three-quarters of these deaths occurred in low- and middle-income countries. Globally, CVD-related mortality

increased by nearly 21% between 2007 and 2017 and was generally higher in men than in women across most age groups, except among those aged 85 years and older, where women had higher mortality rates.

NAFLD affects at least 25–30% of adults in high-income countries and up to 70–90% of individuals with obesity or type 2 diabetes. Beyond liver-related complications, NAFLD significantly contributes to diseases in other organs. It is strongly associated with an increased risk of developing cardiovascular disease, type 2 diabetes, and chronic kidney disease, with the stage of liver fibrosis being the most important disease-specific risk factor. This review article highlights the growing clinical evidence linking NAFLD to an increased risk of cardiovascular disease, explains the underlying pathophysiological mechanisms connecting these conditions, and summarizes pharmacological treatments for NAFLD that may either reduce or increase cardiovascular risk.

Pathophysiology

The pathogenesis of NAFLD involves insulin resistance, lipid accumulation, oxidative stress, and inflammation. Atherosclerosis is driven by endothelial dysfunction, lipid deposition, and inflammatory processes. These overlapping mechanisms explain the strong association between NAFLD and cardiovascular diseases. Role of Statins - Statins inhibit HMG-CoA reductase, reducing cholesterol synthesis and improving lipid profiles. In patients with NAFLD, statins not only reduce cardiovascular risk but may also improve liver enzyme levels and hepatic inflammation. Clinical Benefits- Studies have shown that statin therapy significantly reduces the risk of cardiovascular events in patients with NAFLD. Additionally, statins may contribute to stabilization of atherosclerotic plaques and reduction of systemic inflammation. Safety of Statins in NAFLD - Historically, concerns existed regarding hepatotoxicity of statins. However, current evidence suggests that statins are safe in patients with NAFLD and mild to moderate liver enzyme elevations. Routine monitoring is recommended. Discussion -The benefits of statin therapy outweigh potential risks in patients with NAFLD and atherosclerosis. Clinical guidelines increasingly support the use of statins in these patients.

Conclusions

This review supports the notion that CVD is the leading cause of death in NAFLD patients and that NAFLD is closely associated with an increased risk of CVD events and other cardiac complications (ie, cardiomyopathy, cardiac valvular calcification and arrhythmias) independent of traditional cardiovascular risk factors and metabolic syndrome features. Although further research is needed to draw a definitive conclusion, these observations raise the possibility that NAFLD, especially its more advanced forms, is directly involved in the pathogenesis of CVD. Recent evidence discussed here suggests that this process is mediated not only via the atherogenic dyslipidaemia occurring with features of the metabolic syndrome and NAFLD, but also through the systemic release of multiple proinflammatory and proatherogenic mediators from both the steatotic and inflamed/fibrotic liver and the intestine via changes in gut microbiota. The existing evidence to date reinforces the notion that NAFLD is a multisystem disease affecting many extrahepatic organ systems, including the cardiovascular system. Thus, we believe that a purely ‘liver-centric’ approach to NAFLD is not sufficient and treatment of this burdensome liver disease needs to shift to a more patient-centred, multidisciplinary team-based approach. Since more patients with NAFLD will die from CVD than from the consequences of their liver disease, we strongly believe that a careful assessment of the 10-year CVD risk is

mandatory in all persons with NAFLD, together with early and aggressive treatment of all coexisting cardiometabolic risk factors.

References

1. Chalasani N, et al. The diagnosis and management of NAFLD. Hepatology.
2. Stone NJ, et al. ACC/AHA guideline on cholesterol treatment. Circulation.
3. Athyros VG, et al. Statins and NAFLD. Lancet.
4. Targher G, et al. NAFLD and cardiovascular disease. Journal of Hepatology.
5. Younossi ZM, et al. Global epidemiology of NAFLD. Hepatology.