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A REVIEW ON CLINICAL PATTERN OF HYPERLIPIDEMIA IN CHRONIC KIDNEY DISEASE PATIENTS

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ABSTRACT

Chronic kidney disease (CKD) is a growing global health concern associated with a high risk of cardiovascular disease (CVD) and mortality. Dyslipidaemia is a common metabolic abnormality in CKD, characterised by elevated triglycerides, reduced high-density lipoprotein (HDL), increased small dense low-density lipoprotein (LDL), and elevated lipoprotein(a) levels. These lipid abnormalities contribute significantly to the development of atherosclerosis, inflammation, and oxidative stress, thereby accelerating cardiovascular complications in CKD patients. The pathogenesis involves impaired lipid metabolism, reduced lipoprotein clearance, altered enzyme activity, and changes in lipoprotein composition and function. Additionally, CKD alters the structure and function of HDL and LDL particles, increasing their atherogenic potential. Management strategies primarily include lipid-lowering therapies such as statins and fibrates, along with lifestyle modifications. However, the effectiveness of these treatments may vary depending on the stage of CKD. Early identification and appropriate management of dyslipidaemia are essential to reduce cardiovascular risk and improve clinical outcomes in CKD patients.

KEYWORDS: Chronic Kidney Diseases, Dyslipidaemia, Low Density Lipoprotein, Triglycerides.

INTRODUCTION

One of the most significant public health issues of our time is chronic kidney disease, which is becoming more common. The Kidney Disease Outcomes Quality Initiative (K/DOQI) defines chronic kidney disease as kidney damage or a reduced kidney glomerular filtration rate (GFR) of less than 60 mL/min/1.73 m^2 for at least three months. Cardiovascular disease (CVD) is more common in patients with chronic kidney disease. Patients with CKD are more likely to die from CVD than to develop end-stage renal disease (ESRD) and require renal replacement treatment. Compared with the general population, patients with CKD have an increased risk for developing CVD due to several risk factors often associated with CKD.[1] It is frequently noted that patients with chronic kidney disease have higher triglyceride levels, lower HDL-C levels, and higher concentrations of tiny dense low-density lipoprotein (LDL) particles. Additionally, renal failure results in raised Lp(a) levels as a result of decreased lipoprotein(a) [Lp(a)] catabolism, which raises LP(a) levels. These abnormalities in lipids increased the cardiovascular morbidity and death ratio. However, kidney transplantation or nephrosis remission can correct these acquired lipid abnormalities.[2] Patients with chronic kidney disease often exhibit high triglycerides (TG), low serum HDL-cholesterol (HDL-C), and an increased percentage of lipoprotein-a (Lp-a). However, individuals

undergoing maintenance haemodialysis (MHD) typically show reduced cholesterol levels. It is important to highlight that there is an inverse relationship between cholesterol levels and mortality in CKD patients. The association of hypercholesterolemia with reduced mortality, as well as lower total cholesterol levels correlating with increased mortality rates in MHD patients, may be associated with the triad of malnutrition, inflammation, and arteriosclerosis. According to patient histories, most individuals visiting the artificial kidney department (AKD) for haemodialysis at this tertiary care government hospital come from lower socioeconomic backgrounds. Additionally, there is a scarcity of studies examining serum lipid profiles in CKD patients.[3]

Patients with chronic kidney disease die primarily from cardiovascular disease and accelerated atherosclerosis. Oxidative stress, inflammation, and lipid abnormalities are the main causes of atherosclerosis, cardiovascular disease, and many other CKD complications. The purpose of this study is to give a summary of the lipid metabolism abnormalities linked to chronic kidney disease and how they contribute to the pathophysiology of atherosclerosis, inflammation, oxidative stress, reduced ability to exercise and wasting syndrome.[4]

PATHOGENESIS OF HYPERLIPIDEMIA IN CKD

Hypertriglyceridemia (HTG), decreased HDL-C, fluctuating LDL-C, increased non-HDL-C, increased small, dense LDL-C, and an increased apoB-to-apoA-I ratio are typical characteristics of the dyslipidaemia pattern observed in CKD. In CKD, elevations in Lp (a) are also typical. However, subgroups of people who express bigger Lp (a) isoforms typically have elevated Lp (a) levels.

Early stages of renal illness are associated with HTG, which has a complex aetiology that includes reduced lipoprotein lipase (LPL) activity leading to poor degradation of VLDL and chylomicrons. LPL inhibitors, such as apoC-III and pre-beta-HDL, are elevated as uraemia develops. There have also been reports of decreased expression of the apoA-I gene APOA1, the primary apolipoprotein of HDL, and a decrease in lecithin cholesterol ester transfer protein (LCAT), which is crucial for the maturation of HDL. Reverse cholesterol transport and anti-oxidation are two important HDL activities that are affected by these modifications in gene expression and protein availability.[5]

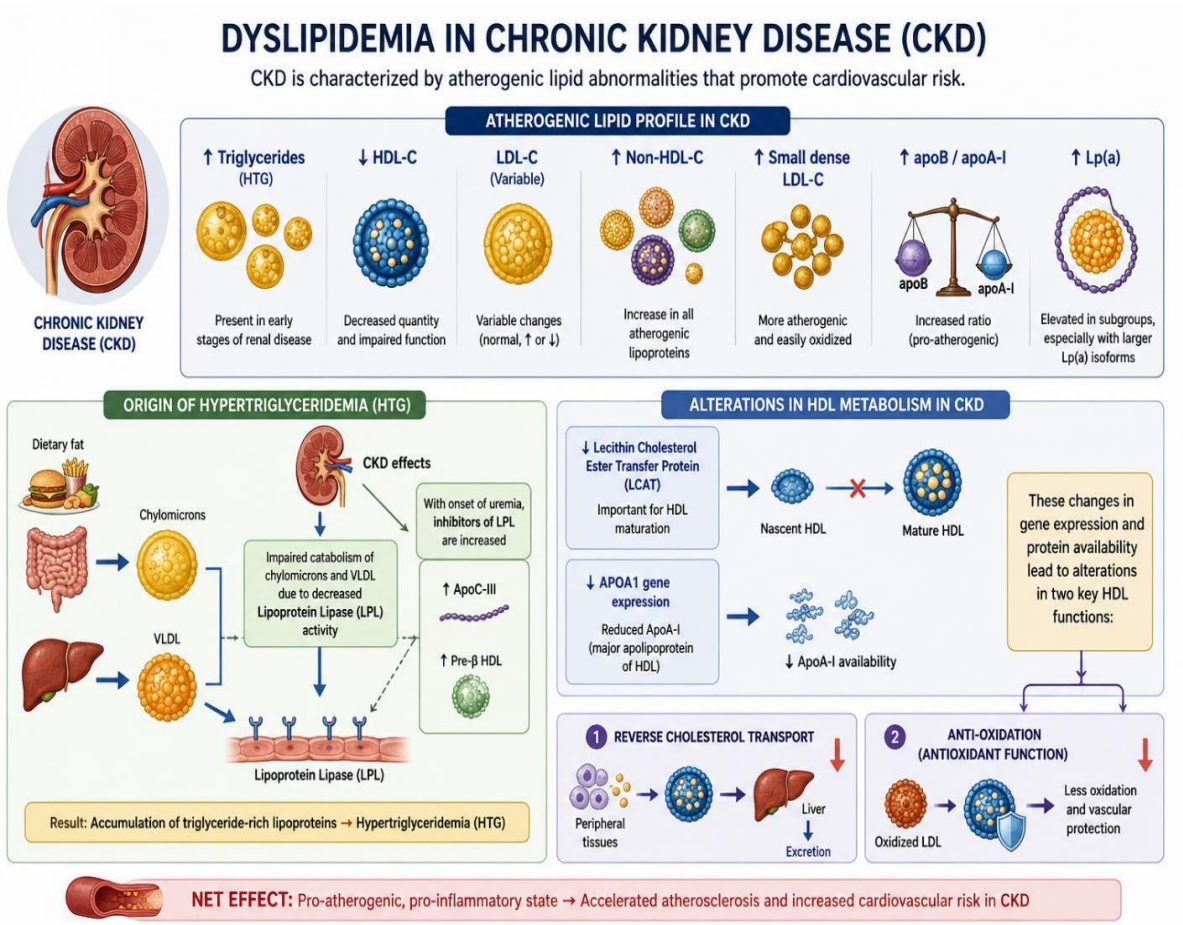

Figure 1: Pathogenesis of dyslipidaemia in CKD.

CKD'S IMPACT ON LIPOPROTEIN COMPOSITION

Beyond merely assessing lipid levels, new research suggests that lipoprotein particle size and composition are changed in CKD, with CKD participants having more tiny dense LDL and fewer bigger LDL particles than controls. Compared to bigger LDL particles, small dense LDL is believed to be more atherogenic. According to a new theory, the lipoprotein particle "cargo" can influence the onset and course of atherosclerosis in addition to cholesterol levels or lipoprotein size. Numerous bioactive lipids, proteins, hormones, microRNAs, and other small RNAs are transported by lipoprotein particles. For example, a recent study compared LDL particle composition between subjects with stage 4/5 CKD and non-CKD controls, and found similar total lipid and cholesterol content, but altered content of various lipid subclasses, for example, decreased phosphatidylcholines, sulfatides, and ceramides and increased N-acyltaurines. Many of these lipid species are known to have either pro- or anti-atherogenic properties and thus could directly affect atherogenesis.[6]

TRIGLYCERIDE-RICH LIPOPROTEIN METABOLISM CHANGES IN CKD PATIENTS

One of the most prevalent quantitative lipid abnormalities in CKD patients is hypertriglyceridemia. In the early stages of chronic kidney disease (CKD), the quantities of triglyceride-rich lipoproteins—very-low-density lipoprotein (VLDL), chylomicrons, and their remnants—begin to rise. Numerous investigations have demonstrated that even when serum creatinine levels are within acceptable ranges, patients with compromised renal function have higher triglyceride concentrations. Additionally, after a high-fat meal, people with CKD typically experience abnormal increases in serum triglyceride levels (postprandial hypertriglyceridemia). Delayed catabolism is the main mechanism causing elevated triglyceride-rich lipoprotein concentrations in predialysis patients. Because the enzyme gene is downregulated and lipase inhibitors are present, the decreased catabolic rate is probably caused by decreased lipoprotein lipase activity.[7]

ALTERATION IN HIGH DENSITY LIPOPROTEIN IN CKD

The proinflammatory milieu, elevated oxidative stress, and uremic toxins are some of the variables that alter the makeup of the HDL particle in CKD. These elements cause a significant remodelling of HDL particles, changing the composition of HDL's proteome and lipidome and causing posttranslational changes to the protein cargo of HDL. Moreover, the functional alterations of HDL are significantly influenced by the build-up of uremic toxins, such as symmetrical dimethylarginine, in progressive CKD.[8]

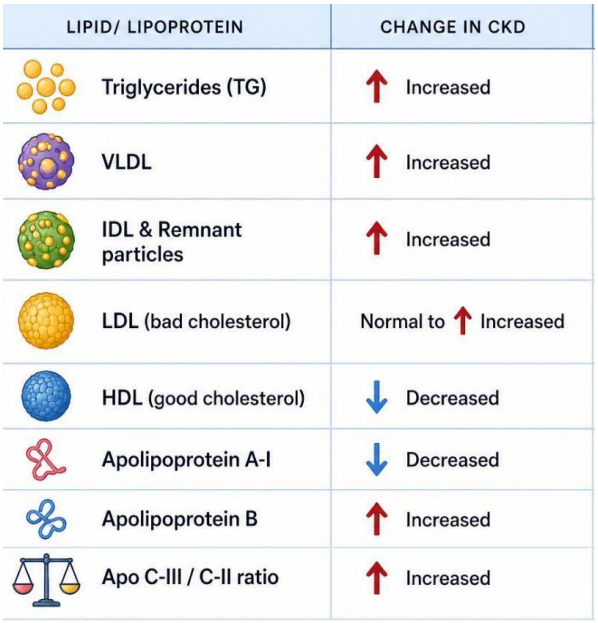

Figure 2: Lipid profile in CKD.

BENEFITS OF LIPID LOWERING IN CKD PATIENTS AND RISK ASSOCIATED WITH POOR CHOLESTEROL PROFILE

Oxidative stress and inflammation are linked to an increase in cardiovascular morbidity and mortality in patients with chronic kidney disease. While the latter is connected to the generation of reactive oxygen species (ROS), the former state activates transcription factors that result in the release of proinflammatory cytokines and the activation of macrophages. Higher levels of uremic toxins are thought to be linked to a state of elevated oxidative stress in CKD patients. Additionally, it has been demonstrated that CKD patients have lower levels of antioxidant enzymes, such as glutathione peroxidase, catalase, and superoxide dismutase.[9]

ASSOCIATION BETWEEN CKD AND CARDIOVASCULAR DISEASES

Hypertriglyceridemia, low HDL cholesterol, variable (usually normal) LDL cholesterol levels, and high lipoprotein (a) [Lp(a)] are the hallmarks of dyslipidaemia in CKD patients. In CKD, the metabolism of LDL (atherogenic small dense LDL particles), intermediate-density lipoprotein (IDL), and triglyceride-rich LDL (VLDL) is changed.

Additionally, this disease impairs reverse cholesterol transfer, which is mediated by HDL cholesterol. Patients with chronic kidney disease (CKD) have altered HDL and LDL, which increases their atherogenic propensity.[10]

MANAGEMENT STRATEGIES

Statins: While statins have been shown to reduce CVD events and mortality in the general population, their effects in the CKD population have been inconsistent.

Simvastatin with ezetimibe did not significantly affect CV mortality or all-cause mortality, although it did considerably reduce severe atherosclerotic events.

Fibrates: Fibrates primarily lower triglyceride levels and raise HDL cholesterol. Fibrates can raise HDL cholesterol by 10% and lower triglyceride levels by 18%–45%.[11]

DISCUSSION

Several lipid species, including cholesterol, triglycerides, fatty acids, and phospholipids, are dysregulated in tubular, endothelial, and podocyte cells and contribute to the advancement of chronic kidney disease.[12] One important modifiable risk factor for CKD is dyslipidaemia, which increases the risk of cardiovascular disease and accelerates the illness's course. Statins are still the mainstay of treatment for early-to-moderate CKD, but as the disease progresses, their effectiveness declines. The development of new treatment options and a better comprehension of the pathophysiology of dyslipidaemia may lead to better results.[13]

CONCLUSION

The occurrence of these lipoprotein changes in a biological context characterised by increased inflammation and oxidative stress, and the rising risk of non-atherosclerotic cardiovascular mortality with declining kidney function, has substantial therapeutic implications.[14] The effects of lipid accumulation in the kidneys are presented, along with possible molecular mechanisms underlying the link between renal lipid accumulation and CKD, providing theoretical bases for lowering circulating lipid levels in patients with CKD. It is generally accepted that excessive lipid accumulation in the renal parenchyma is relevant to CKD development and can exacerbate damage at the tubular and glomerular levels. Such evidence indicates that a lipid-lowering pharmacological approach, combined with substantial lifestyle changes, should be considered part of CKD therapy.[15]

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