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Review

Dyslipidemia Associated With Chronic Kidney Disease – An Overview

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Abstract

Chronic kidney disease (CKD) is a common disorder that results in severe morbidity and mortality. Dyslipidemia is a common CKD consequence that causes changes in lipid metabolism and increases the risk of cardiovascular disease (CVD). The efficacy and safety of CKD patients are still being debated because to concerns about potential side effects and changed pharmacokinetics in this population. Current CKD management may have an impact on lipid levels as well. This describes the modifications made to both hemodialysis and peritoneal dialysis in CKD patients. Statin medication is an important consideration in the treatment of dyslipidemia in CKD patients. Niacin and fibrates are effective lipid-lowering agents in CKD and appear to have some cardiovascular advantages. Nephrotic syndrome causes hyperlipidemia and significant changes in lipid and lipoprotein metabolism. The review summarises current information on the roles of lipids in CKD patients, providing an overview of lipoprotein metabolism, emphasising CKD-induced changes and the safety impact of Statin medication on CKD patients.

Keywords: Dyslipidemia, Pathogenesis of CKD, Lipid management, Hemodialysis and peritoneal dialysis, Nephrotic syndrome, First line and Second line drug.

INTRODUCTION

Cardiovascular disease is the main cause of death and morbidity in persons with renal failure. Many CKD patients succumbed from cardiovascular illness before they require dialysis, and some acquire End Stage Renal illness (ESRD).Microalbuminuria in the absence of evident decline in renal function or diabetes predicts increased cardiovascular disease and death [1]. One to four out of every ten people are experiencing chronic kidney disease. Dyslipidemia provides a significant risk and is a highly predictive risk factor for cerebral vascular disorders, atherosclerosis, and coronary artery disease. Hypertriglyceridemia, hypercholesterolemia, lower high-thickness lipoproteinemia, and mixed hyperlipidemia are all combined to form dyslipidemia [2]In stages 3-5 of CKD, dyslipidemia is a risk factor for poor renal outcomes.

One symptomatic issue for the first set of patients is nephrotic syndrome.[3]. Because of the liver's non-

specific increase in protein secretion to make up for the enormous loss of serum proteins into urine that causes hypoalbuminemia, the production of Very Low Density Lipoprotein (VLDL) in the liver is enhanced. Patients in the second category suffer from chronic renal failure. Renal insufficiency does not cause an increase in hepatic VLDL synthesis, but it does cause problems with TG-rich lipoprotein breakdown [4]. While endogenous TGs are carried from the liver to the circulation by VLDL, dietary TGs and cholesterol are delivered by chylomicrons. There is no rise in low-density lipoprotein cholesterol, and there is no significant correlation between LDL levels and results. High-density lipoprotein cholesterol is inversely correlated with cardiovascular risk and decreases in variation. The most prevalent anomaly of plasma lipids in individuals with renal failure is hypertriglyceridemia, which occurs in conjunction with normal cholesterol levels. In a significant portion of cases with nephrotic range proteinuria, hypertiglyceridemia—this results from the buildup of VLDL and residual lipoproteins such intermediate-density lipoprotein (IDL)—also represents the primary lipoprotein abnormality. [5]. This dyslipidemia is caused by both elevated VLDL synthesis and decreased VLDL clearance.

Etiology of dyslipidemia in ckd

A persistent increase in insulin levels may result in dyslipidemia. Dyslipidemia has been linked to elevated O-GlcNactransferase (OGT) levels. A persistent increase in insulin levels may result in dyslipidemia [6]. The degree of proteinuria and renal function both affect the lipid profile. Idiopathic hyperlipidemia refers to hyperlipidemia with no known cause. Dyslipidemia can result from TG and LDL cholesterol overproduction, faulty clearance, or increased clearance of HDL due to one or more mutations. Sedentary lifestyles with high dietary intakes of saturated fat, cholesterol, and Trans fats (polyunsaturated and monounsaturated fatty acids) are the most significant secondary causes in certain industrialised countries. The degree of proteinuria and renal function both affect the lipid profile. Idiopathic hyperlipidemia refers to hyperlipidemia with no known cause. Dyslipidemia can result from TG and LDL cholesterol overproduction, faulty clearance, or increased clearance of HDL due to one or more mutations. Sedentary lifestyles are the most significant secondary cause in certain wealthy countries [7]. This dyslipidemia may be further impacted by hyperparathyroidism, which is common in patients with chronic kidney disease (CKD). This condition increases the buildup of calcium in the liver and adipose tissue cells.

Anti-psychotic medications such as risperidone, olanzapine, quetiapine, and zotepine have been implicated with raising the risk of developing chronic kidney disease, particularly when coexisting with other conditions like dyslipidemia, obesity, and diabetes mellitus [8]. Chronic renal disease has been linked to long-term SGAP use. The psychiatric medication lithium impairs the kidney's capacity to concentrate urine, which increases the risk of dehydration and acute kidney injury (AKI). Lithium is frequently used to treat bipolar affective disorder (BAD) and, to a lesser extent, unipolar depression [9]. Patients with chronic kidney disease (CKD) experience complicated qualitative and quantitative abnormalities in lipid and lipoprotein metabolism, regardless of the underlying cause of their condition.

Pathogenesis of ckd in dyslipidemia

Dyslipidemia clarifies the function of a range of related metabolic and cardiovascular disorders. TGs, phospholipids, cholesterol esters, cholesterol, and apolipoproteins make up lipoproteins. The latter give lipoproteins their functional specialisation and are crucial in activating lipolytic enzymes and cell surface receptor recognition sites [10]. In addition to increasing the degree of lipoprotein atherosclerosis in glomerular structures, dyslipidemia and chronic kidney disease (CKD) also trigger growth factors and cytokines that cause inflammation and fibro genesis [11,12]. Changes in reverse transport, metabolism, and cholesterol structure are caused by renal failure. In CKD, oxidative stress is elevated and impacts lipid homeostasis [13]. Atherosclerosis can develop in the absence of hypercholesterolemia, as many individuals with non-nephrotic CKD or type 2 diabetes mellitus attest to.[14]

Lipoprotein with progression of ckd

The cumulative kidney end point (unadjusted and adjusted) and lipoproteins are found to be associated. The renal end goal was not independently predicted by total cholesterol, LDL-C, HDL-C, triglycerides, apoA -I, apoB, and LP (a) levels, despite a correlation in unadjusted models following covariate correction [15]. Increased blood triglyceride (TG) levels, decreased high-density lipoprotein concentration and functionality, and elevated levels of atherogenic small, dense, low-density lipoproteins are all signs of altered lipid metabolism in patients with chronic kidney disease (CKD) [16]. The liver and stomach create Apo AI, phospholipids, and cholesterol, which are combined to form HDL. Lecithin-Cholesterol-Acyltransferase Enzyme (LCAT) deficiency, higher concentrations of Apo-B, decreased plasma levels of Apo-I and Apo-II, primary proteins producing HDL, and increased serum concentrations of triglycerides (TG) due to defective clearance are the causes of these conditions [17]. Lower levels of apolipoprotein A-I, the main cofactor for the LCAT, are associated with chronic kidney disease. Progressive renal disease is the outcome of the correlation between a hereditary LCAT deficit and a significant drop in HDL cholesterol as well as decreased HDL-mediated reverse cholesterol transfer. The variables that lead to atherogenic diathesis and the increased risk of cardiovascular disease in people with chronic kidney

disease (CKD) include hypertension, oxidative stress, inflammation, and altered metabolism of lipids, calcium, and phosphate [18]. Patients suffering from End Stage Renal Disease (ESRD) have significantly lower serum levels of LCAT activity. Many bioactive lipids, micro RNAs, other short RNAs, proteins, hormones, etc. are transported by lipoprotein particles. Since lipid species have been shown to possess either pro- or anti-atherogenic qualities, they may have a direct impact on atherogenesis [19] .

Lipids in hemodialysis and peritoneal dialysis

For the treatment of uremic symptoms and some aspects of uremic toxicity, dialysis is quite helpful. During long-term hemodialysis, the lipid and apolipoprotein features associated with predialytic renal failure largely do not change. The effects of hemodialysis and peritoneal dialysis on lipid profiles vary [20].Hemodialysis highlights the peroxidation of proteins and lipids. Serum total cholesterol levels in HD patients are usually normal or low, and protein oxidation is aggregated during peritoneal dialysis. Both dialysis treatments change the activity of antioxidant enzymes [21] . Patients on hemodialysis who do not have hyperlipidemia have higher levels of VLDL and Apo C-III cholesterol and lower levels of HDL cholesterol [22,23] . An increase in Apo C-III has led to the occurrence of lipoproteins carrying Apo-B [22] . Apo C-III is distributed similarly in lipoprotein fractions as it was in patients prior to dialysis [24,25] Hemodialysis patients' HDL cholesterol was lower than that of the non-uremic controls[26,27]. Because plasma triglycerides and HDL cholesterol have an inverse relationship, hypertriglyceridemia plays a significant role in the explanation of lower HDL cholesterol levels in hemodialysis patients. Different levels of various lipoproteins were mirrored in the triglyceride and total cholesterol levels between the hemodialysis and control groups [28,29].

Patients receiving peritoneal dialysis frequently have higher levels of lipoprotein (a), apoB, triglycerides, total cholesterol, and low-density lipoprotein (LDL) cholesterol. Apolipoprotein A1 and high-density lipoprotein (HDL) levels are typically low. Patients on hemodialysis have lower levels of LDL cholesterol and apoB protein than do those on PD.PD patients also have increased levels of intermediate-density lipoprotein [30]. It is yet unknown why PD causes an excess of LDL particles to be produced.

A possible cause of the aberrant lipid profile is hypoalbuminemia brought on by peritoneal protein losses. Due to the glucose, carbohydrate, or amino acid loading from PD solutions, which affects LDL cholesterol measurement, PD patients practically never fast. Patients receiving hemodialysis and the general population both had comparatively greater atherogenic lipid profiles as compared to those receiving peritoneal dialysis [31].

Dialysis causes increased morbidity and mortality along with the progressive return of uremic symptoms over an extended period of time. Patients experience the effects when their remaining renal function decreases, a condition that is sometimes referred to as "the end of the honeymoon period."[32]. Patients on peritoneal dialysis may be compared to those with nephrotic syndrome. Given their greater atherogenic lipid profile, Statin medication would have been beneficial for PD patients [30]. Compared to HD patients, PD patients have much greater levels of apo-B-containing lipoproteins that are rich in triglycerides and cholesterol [33]. Numerous aspects of PD treatment that could have an immediate impact on lipoprotein metabolism [34].

Treatment stratergy

First line drug

The mainstay of managing dyslipidemia, statins have been extensively utilised to lower CVD in the general population. For type IIa, type IIb, and secondary hyperlipoproteinemia (a condition in which cholesterol levels are elevated above total cholesterol), statins are the first-line treatment. Effects of statin therapy (pravastatin, simvastatin, and atrovastatin) on peritoneal dialysis patients' C-reactive protein (CRP) levels [35]. Statins work by competitively inhibiting this enzyme, which lowers the liver's production of cholesterol. The most effective LDL-lowering medications are statins. Additionally, drugs raise HDL somewhat and decrease TG, IDL, and VLDL. These medications do not, however, impact lipoprotein. Moreover, statins have anti-inflammatory, anti-proliferative, and antioxidant qualities. The clinical advantages of statin or Statin/ezetimibe medication are unknown in dialysis patients with a towering probability of cardiovascular disease, according to KDIGO guidelines. For the majority of adult dialysis-dependent CKD patients, this treatment was not advised. Additionally, these guidelines recommend Statin therapy for adult recipients of kidney transplants [36].

The benefits of fluvostatin therapy on cardiovascular health and lipid reduction seen in the ALERT research are similar to those of Statin therapy on a broader population basis [37,38,39]. The two main side effects of these medications are hepatotoxicity and myopathy. If fibrates or niacin is also provided with these, the likelihood of myopathy increases. Renal shutdown may ensue after rhabdomyolysis in cases of myopathy. Taking statins when pregnant is not advised. Zetimibe is a medication for type IIa, type IIb, and hyperlipoproteinemia that can be taken either by itself or in combination with statins. Fluvastatin is regarded as the most potent antioxidant among all the statins. Pravastatin has the lowest potential for medication interactions because it is metabolised by sulfation, a non-microsomal process. In pre-end stage CKD and after transplant, robust clinical trials show that statins are safe and effective in decreasing cholesterol and preventing CVD events.

Second line drug

Fibrates Acid Derivatives

The preferred medication for hypertriglyceridemia (type III and IV) is a fibrate; in type IIb, it can be combined with other medications (fenofibrate, since it has the strongest effect on lowering LDL cholesterol).Since fibrates are metabolised by the kidney, they are often not recommended in patients with CKD. Clofibrate, ciprofibrate, bezafibrate, fenofibrate, and gemfibrozil are among the compounds. Because they are fleeting and moderate, most people can tolerate fibrates. The negative consequences of fibrates include elevated plasma levels of creatinine and homocysteine. Patients with reduced kidney function have higher blood levels of benzofibrate, clofibrate, and fenofibrate than controls with normal kidney function. Fenofibrates mainly lower total cholesterol (TG), which is found in VLDL, and raise HDL alongside the exception of bezafibrate, using them alongside statins increases the risk of myopathy.

Nicotinic Acid

Cheap medication niacin (vitamin B3) raises HDL cholesterol while lowering VLDL triglycerides and LDL cholesterol. It works by preventing adipose tissue lipolysis. Additionally, niacin can cause hepatotoxicity, which is seen by a decrease in HDL and LDL cholesterol.

Dyslipidemia in nephrotic syndrome

The term "dyslipidemia" describes abnormal blood lipid (triglyceride and cholesterol) levels. The diagnosis of nephrotic syndrome depends on the lipid profile, which is markedly atherogenic with elevated TC and LDL. Hypercholesterolemia will develop in people with CKD stages 1-4 who have nephrotic syndrome as a result of increased LDL production and impaired catabolism [40]. Patients with nephrotic syndrome have normal to low HDL cholesterol levels [41,42]. The main causes of impairment are elevated cholesterol ester transfer protein activity [43][44] acquired defects in LCAT activity, and downregulation of the scavenger receptor B-1 (hepatic HDL receptor), which has been seen in nephrotic syndrome patients.[45]. Nephrotic syndrome can be either temporary or chronic. Treatments for nephrotic syndrome dyslipidemia include lipid-lowering medications and those that particularly target the renal illness, such as glucocorticoids and renin-angiotensin system antagonists, which can help reduce proteinuria. Dysregulated lipid metabolism and dyslipidemia are associated with high risk of atherosclerosis and thrombosis, among other consequences of nephrotic syndrome. Regardless of their Apo (a) are forms, CKD patients with nephrotic range proteinuria unquestionably having higher LP (a) levels [46] . In patients with nephrotic syndrome, dyslipidemia may play a role in the advancement of renal illness as well as the higher risk of cardiovascular death in these individuals.
One of the primary signs and symptoms of nephrotic syndrome is hyperlipidemia [47]. For the treatment of dyslipidemia in nephrotic syndrome patients, statins are effective and safe.

CONCLUSION

A significant number of CKD patients have dyslipidemia. Research is beginning to show that statins can slow the progression of chronic kidney disease (CKD).Two distinct dialysis modalities have been evaluated and assessed. Patients with chronic renal illness are more likely to have peritoneal dialysis than hemodialysis.The higher than normal levels of apoprotein B, lipids, and LDL cholesterol in the serum of individuals with simple nephrotic syndrome. NS includes multiple secondary alterations in coagulation and lipids throughout the body, as well as severe proteinuria, or loss of protein in the urine. Statin usage is beneficial in lowering CVD events and is safe and effective in minimising CKD. This summary highlights the significance of incorporating lipid management into CKD treatment plans by demonstrating how early identification and proactive management of dyslipidemia can significantly improve outcomes for patients.

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