Lipid abnormalities in chronic kidney disease patient

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Objective: To determine lipid level LDL, VLDL, HDL in chronic kidney disease patients.

Material and method: cross sectional of lipid abnormalities in 50 chronic kidney disease patients at saveetha medical college & hospitals.

Background: chronic kidney disease also known as chronic renal failure is progressive loss in kidney function over a period of months or years. The symptoms of worsening kidney function are Anuria, anasarca, metabolic acidosis and hyperkalemia. Dyslipidemia is common in CKD patients and is usually not characterised by elevated cholesterol levels, except in patients with marked proteinuria. Increased triglyceride levels in conjunction with decreased high-density lipoprotein levels are the commonest qualitative abnormality.Dyslipidemia is due to dysregulation of high-density lipoprotein (HDL) and triglyceride-rich lipoprotein metabolism. Specifically, maturation of HDL is impaired and its composition is altered in chronic renal failure.

Reasons: To determine lipid level in chronic kidney disease.

Introduction:

Chronic kidney disease (CKD) is associated with early development of atherosclerosis and increased risk of cardiovascular morbidity and mortality which is the leading cause of death among these patients[1].Alterations in lipid metabolism resulting in abnormal lipoprotein composition and concentration (dyslipidemia) have been noticed in chronic renal insufficiency[2]. Dyslipidemia is a well-established risk factor for CVD in the general population but this relationship is not straightforward in CKD population. While dyslipidemia is associated with CVD in pre-dialysis CKD[3] and hemodialysis population[4], data regarding its association in peritoneal dialysis patients is lacking[5]. With an ever increasing CKD burden worldwide, providing treatments for modifiable risk factors, like dyslipidemia, becomes an essential component for improving outcomes. In this review, we will examine various lipid abnormalities associated with kidney diseases and current evidence regarding various treatments.[6]

Material and method:-

The study was conducted among saveetha medical college and Research Institute Chennai, Tamil nadu, as inpatients. The study period was from January to March 2016, for a period of three months. The study includes 50 CKD patients in the age group of 40 to 60 years. The CKD patients were classified based on GFR.

RESULT:

A total number of 50 CKD patients,The mean and standard deviation of all the biochemical parameters were calculated and their results shown in Table 1.

Parameters	CKD Patients
Cholesterol	232.0 ± 24.9
Triglycerides	180.1 ± 7.9
HDL	29.7 ± 4.7
LDL	177.8 ± 11.4
Lipoprotein (a)	74.3 ± 7.0

Discussion

CKD patients also have reduced levels of lipoprotein lipase, hepatic lipase and defective very-low-density lipoprotein (VLDL) and low-density lipoprotein (LDL) receptors. This leads to accumulation of VLDL, intermediate-density lipoprotein and chylomicron remnants which are susceptible to oxidisation[7].These oxidised products are usually atherogenic and play a role in CVD pathogenesis in this population[8].CKD patients frequently develop secondary hyperparathyroidism which also has an impact on lipid abnormalities[9].It has been postulated that this usually occurs due to an increase of intracellular calcium concentration in hepatocytes by elevated parathyroid hormone in CKD patients. Studies have shown a role of parathyroidectomy in reducing triglyceride levels in CKD patients.role of parathyroidectomy in reducing triglyceride levels in CKD patients.As described above, dyslipidemia can be influenced by numerous intrinsic and extrinsic factors. The most common quantitative lipid abnormalities in CKD patients are hypertriglyceridemia, with accumulation of triglyceride-rich very-low-density lipoproteins (VLDL), and both chylomicron and VLDL remnants as well as low levels of high-density lipoprotein (HDL) cholesterol[10]. Due to abnormalities of

enzymes involved in lipoprotein metabolism in CKD patients, the triglyceride content of these particles is frequently increased, while the cholesterol-ester content is reduced. Levels of total and low-density lipoprotein cholesterol (LDL) levels, however, are usually not elevated. In dialysis-treated patients, cholesterol levels tend to be further reduced, while in patients with massive proteinuria, LDL levels are usually increased concentrations of lipoprotein. have also been reported, particularly in end-stage renal disease (ESRD) patients[11]. The expected relationship between high LDL or total cholesterol levels and CVD risk, demonstrated in many chronic kidney disease patients. The relationship between cholesterol levels and increased CAD risk seen in the general population has been difficult to establish in CKD patients, most notably in patients undergoing hemodialysis treatments.Moreover, lipid abnormalities in CKD patients are characterized more by qualitative rather than quantitative abnormalities of cholesterol-containing lipoproteins. A key defect in the metabolism of apolipoprotein (apo) B-containing lipoproteins is reduced lipotrotein lipase (LPL), which increases the triglyceride content of apoB-lipoproteins and is believed to result in greater susceptibility to oxidative modification .The reverse cholesterol transport process is also postulated to be defective in the removal of cholesterol from peripheral tissues in CKD patients. There is also a quantitative and qualitative defect in the maturation of high-density lipoprotein (HDL), which may negatively impact the reverse cholesterol transport system.The histopathological changes seen in coronararteries of CKD patients commonly demonstrate extensive calcifications in the media of vessels as well as in atheromas, an abnormality not seen as extensively in most patients with acute coronary syndrome who do not have CKD.This extensive vascular calcification pattern appears, in part, related to changes in calcium phosphorus metabolism characteristic of reduced kidney function. As a result, there is increased vascular stiffness, resulting in reduced vascular compliance and increased hemodynamicrelated shear forces, with the potential for increased risk of plaque rupture. In addition, plaque formation appears to be more diffuse and there is a greater degree of vessel stenosis distal to an atheroma. Decreased endothelial cell function has also been described, resulting in reduced flow-mediated vasodilatation. Compared with age and gender-matched controls, there are increased lev- els of inflammatory markers in the coronary vessels and blood of CKD patients.Multiple putative risk factors for coronary artery disease (CAD) are present in CKD patients, including insulin resistance, increased oxidative stress, inflammation, anemia, excessive smoking, endothelial dysfunction, and reduced nitric oxide availabilites.When present, proteinuria and the consequent hypoalbuminemia contribute to altered lipid abnormalities in CKD patients. Finally, pharmacologic treatments of underlying kidney dis- eases in these patients (e.g. with steroids and cyclosporine) may also contribute to dyslipidemia.

Conclusion

In summary, patients with kidney diseases have unique lipid abnormalities when compared to general population and they have different clinical implications associated with these abnormalities. Over the time, our understanding has evolved regarding dyslipidemia in CKD patients. Statins remain the first line of treatment for dyslipidemia. Majority of current evidence comes from subgroup/post hoc analysis and meta-analysis, especially in CKD (pre-dialysis), peritoneal dialysis and renal transplant population. Prospective interventional studies are needed in this population to identify subsets of patients who will benefit most and also to assess long term toxicity of statins. KDIGO recommendations provide general principles regarding treatment of dyslipidemia but it should be individualised for each patient.

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