VOLUME - 11, ISSUE - 12, DECEMBER - 2022 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

General Medicine

STUDY OF ATHEROGENIC INDEX OF PLASMA IN NON - DIABETIC CKD PATIENTS

Dr. S. Thenarvi	Post Graduate, Department of General Medicine, Rajah Muthiah Medical College and Hospital, Annamalai University
Dr. Saritha. K. Narayanan	M.D., Associate Professor, Department of General Medicine, Rajah Muthiah Medical College and Hospital, Annamalai University
Prof. Dr. M. Ramakrishna Rao	M.D., Professor, Department of General Medicine, Rajah Muthiah Medical College and Hospital, Annamalai University

ABSTRACT **Objective:** To calculate the atherogenic index of plasma in non- diabetic chronic kidney disease patients and correlate it with the various stages of chronic kidney disease. **Methods:** We conducted a hospital based observational study of 60 non diabetic CKD patients. Fasting lipid profile was estimated in those patients and atherogenic index of plasma (AIP) was calculated. AIP was correlated with various stages of chronic kidney disease. **Conclusion:** With decreasing GFR values, there is an increase in the AIP values.

KEYWORDS : CKD, Atherogenic index of plasma, fasting lipid profile

INTRODUCTION

In the current scenario, with increasing life expectancy and increased incidence of non-communicable diseases, the incidence and prevalence of chronic kidney disease is increasing. CKD is defined as abnormalities of kidney structure or function, present for >3 months, with implications for health and CKD is classified based on cause, GFR category, and albuminuria category.[1]

Patients with chronic kidney disease are at a heightened risk of developing cardiovascular disease, because of the many known risk factors associated with CKD. These risk factors include age, male sex, smoking, dyslipidemia, obesity, hypertension, diabetes and non-traditional risk factors related to uraemia such as hyper-homocysteinemia, anaemia, mineral bone disease – CKD with hyper-parathyroidism, hypo-albuminemia, oxidative stress and chronic inflammation. There is a higher likelihood for a CKD patient to die because of cardiovascular disease before progressing to end stage renal disease and thus requiring renal replacement therapy.[2,3]

Dyslipidemia is one of the serious complications of chronic kidney disease. Alterations in the metabolism of lipoproteins could be seen at the very early stages of chronic kidney disease and it usually follows a worsening course that is similar to the deterioration in the renal function.[4] The disparity that exists between the production of the lipoproteins and the degradation of the lipoproteins accounts for the profound dyslipidemia that is observed in the chronic kidney disease patients.

The various studies which were published recently indicate that dyslipidemia proves to be a major culprit in the pathogenesis of cardiovascular disease and also in the worsening of renal function. However there appears to be some major differences in the pattern of the dyslipidemia that was observed by the various researchers.[5]

This study was conducted to calculate the atherogenic index of plasma (AIP) in non- diabetic CKD patients and to correlate AIP with various stages of chronic kidney disease.

Inclusion Criteria

1. Patients between age group of 20 to 80 years.
2. All patients who have given written consent for this study.
3. Patients with H/O CKD who are on conservative management and/or hemodialysis.

Exclusion Criteria

1. Patients with already diagnosed dyslipidemia on treatment.
2. Patients who refused to give consent for the study.
3. Patients in ICU admission.
4. Other conditions causing dyslipidemia patients with hypothyroidism as per TSH and T4 levels pregnancy.
5. Patients with blood sugar levels above the normal range (FBS ≥126mg/dl or PPBS ≥200mg/dl or HbA1C ≥6.5)

RESULTS

Table 1: Distribution according to severity of renal disease

Variable		Frequency (n=60)	Percentage (%)
Severity of renal disease	Stage 5	28	46.7
	Stage 4	15	25
	Stage 3	17	28.3

Table 2: Atherogenic Index of Plasma (AIP)

CKD STAGE	N	MEAN	STANDARD DEVIATION
3	17	0.607	0.14
4	15	0.692	0.128
5	28	0.814	0.138

From the above table, it is evident that the mean AIP increased with later stages of CKD.

EGFR VS AIP

CKD STAGE	N	MEAN	STANDARD DEVIATION
3	17	0.607	0.14
4	15	0.692	0.128
5	28	0.814	0.138

The Pearson Correlation test was done to analyse the relationship between EGFR and AIP, which showed a significant negative correlation [with decreasing GFR values, there is an increase in the AIP values].

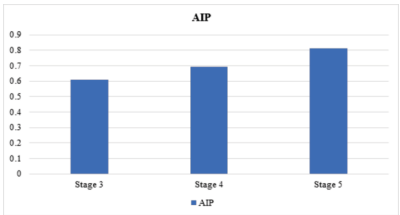

Figure 1: Bar chart showing mean AIP for different categories of chronic kidney disease

DISCUSSION

The leading etiology of hospitalization and mortality in chronic kidney disease patients is cardiovascular disease[6].

The spectrum of cardiovascular disease likely begins in the early stages of chronic kidney disease to reach critical level at the commencement of renal replacement therapy[7,8] Dyslipidemia, one of the traditional risk factors for cardiovascular disease occurs frequently in chronic kidney disease[9,10].

However, the pattern of dyslipidemia in chronic kidney disease patients differs from the general population. Several studies have been conducted which analyses the pattern of dyslipidemia in pre –dialysis, dialysis and kidney transplanted patients. These type of studies will help in the better understanding of the lipid status in this high risk group and the subsequent cardiovascular risk.

In this study, the atherogenic index of the plasma [a value based on the triglyceride and HDL-C levels] is calculated and correlated with various stages of the kidney disease.

Atherogenic Index of Plasma (AIP) is a strong predictor of the risk of atherosclerosis and coronary heart disease. AIP demonstrates the true relationship between the protective and the atherogenic lipoprotein and is related to the size of pre- and the anti- atherogenic lipoprotein particle. AIP is calculated as logarithmic ratio of triglycerides to high density lipoprotein cholesterol log (TG/HDL-C)

Low risk of CVD → AIP < 0.11
Intermediate risk → AIP 0.11 – 0.21
Increased risk → AIP > 0.21(11)

In our study, among the sixty patients studied, forty-seven patients are male and thirteen patients are female. The mean age of the study population is 48.8. Among the study population, all the sixty patients are hypertensives. Twenty-eight patients are treated with hemodialysis and the remaining thirty-two patients are conservatively managed. Regarding the stages of CKD, seventeen patients belonged to stage 3 with eGFR between 30 and 59ml/min/1.73m^2. Fifteen patients belonged to stage 4 with eGFR between 15 and 29ml/min/1.73m^2. Twenty-eight patients belonged to stage 5 with eGFR less than 15ml/min/1.73m^2.

In our study population, the mean value of total cholesterol is 190.40 mg/dl. The mean value of triglycerides is 221.92mg/dl. The mean value of LDL-C is 105.38mg/dl. The mean value of HDL-C is 40.65mg/dl. The mean value of VLDL is 44.38mg/dl. Regarding the prevalence of dyslipidemia, 88.3% participants were having dyslipidemia. 49.1% of stage 5 CKD patients were having dyslipidemia. 28.3% of stage 4 CKD patients were having dyslipidemia. 22.6% of stage 3 CKD patients were having dyslipidemia. The mean value of AIP in stage 3,4,5 kidney disease are 0.607, 0.692 and 0.814 respectively. The mean value of the various lipids and AIP is higher in the group on hemodialysis, whereas mean HDL –C is lower in the group on hemodialysis. This difference is statistically significant with p value less than 0.05.

High triglyceride levels in CKD can be explained by significant increases in the levels of apolipoprotein C-III, which is a potent inhibitor of Lipoprotein lipase[12,13]. LPL, located in the capillary endothelium is an enzyme that degrades circulating triglycerides in the bloodstream. These triglycerides are embedded in VLDL and in chylomicrons that travel through bloodstream[14,15].

High levels of lipoprotein Lp(a) leads to atherosclerosis and cardiovascular disease in chronic kidney disease patients. Lp(a) is an LDL-like particle in which the specific apolipoprotein(a) is linked to apoB-100 by a single disulfide bond[16]. In CKD, plasma Lp(a) levels are greatly influenced by the glomerular filtration rate (GFR), and are elevated in the earlier stages of renal impairment[17].
The presence of up-regulated HMG-CoA reductase and elevated acetyl-coenzyme A acetyl-transferase (ACAT)-2 increase the accumulation of esterified cholesterol and hence the production of lipoproteins containing apo-B, such as LDL and VLDL[18].

CKD is associated with a decreased activity of lecithin cholesterol acyl-transferase (LCAT), which is linked to HDL and it is responsible for converting cholesterol into esterified forms of cholesterol. This cholesterol esterification enhances the hepatic removal of cholesterol. LCAT dysfunction will hamper the cholesterol metabolism[19,20].

The down-regulation of LP and LPL can be induced by a poorly understood secondary hyperparathyroidism, a commonly observed finding in CKD which can also cause worsening of dyslipidemia[21].

CONCLUSION

Lipid metabolism is significantly altered in most patients with renal failure. Our results indicate that chronic kidney disease patients show significant abnormalities of lipid metabolism such as hypertriglyceridemia, hypercholesterolemia and low HDL cholesterol. Atherogenic Index of Plasma (AIP) can be used as a strong predictor of the risk of atherosclerosis and coronary heart disease in chronic kidney disease patients. In this study, with decreasing GFR values, there is an increase in the AIP values. As a first step of controlling dyslipidemia, bodyweight control, dietary regulation and regular exercise should be applied. Supplementing the diet with polyunsaturated fatty acids from fish oil to reduce triglycerides may be helpful. Statins can be used with careful monitoring.

REFERENCES

1. KDIGO 2012. Clinical Practice Guidelines for the evaluation and management of chronic kidney disease. Volume – 3.Issue – 1. Jan 2013.
2. Ivana Mikolasevic et al. Dyslipidemia in patients with chronic kidney disease: etiology and management. International Journal of Nephrology and Renovascular Disease. Feb 2017. 35-45.
3. Parmar JA et al,. Dyslipidemia and Chronic Kidney Disease. ISRJ. 2014; 3:396 – 397
4. Magar et al. A Study of lipid profile in chronic renal failure patients undergoing hemodialysis. IOSR – JDMS. Volume – 15. Issue 6. June 2016. PP 01 -03.
5. Balode et al. Serum lipid profile in chronic kidney disease patients on hemodialysis. Indian Journal of Applied Research. Volume :3. Issue : 8. August 2013.(20 – 22).
6. Rayner HC, Pisoni RL, Bommer J, Canaud B, Hecking E, Locatelli F, et al. Mortality and Hospitalization in haemodialysis in five European countries. Results from dialysis outcome and practice patterns study (DOPPS) Nephrol Dial Transplant. 2004;19:108–120.
7. Kasper et al. Harrison s Principles of Internal Medicine. 20th edition.
8. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, et al. INTERHEART Study Investigators, author. Effect of potentially modifiable risk factors associated with MI in 52 countries (The INTERHEART Study): case-control study. Lancet. 2004;364(9438):937–952.
9. Locatelli F, Pietro Pozzoni, Tentori F, Vecchio LD. Epidemiology of cardiovascular risk in patients with chronic kidney disease. Nephrol Dial Transplant. 2003;18(7):2–9.
10. Akpan EE, Ekrikpo UE, Effa EE, Udo AA, Kadiri S. Assessment of dyslipidemia in pre-dialysis patients in south-west Nigeria. Niger Med J. 2014; 55(3):214–219.
11. Shabnam Niroumand et al. Atherogenic Index of Plasma (AIP): A marker of cardiovascular Disease. Medical journal of Islamic Republic of Iran. July2015.
12. Visconti et al. Lipid disorders in patients with renal failure: role in cardiovascular events and progression of chronic kidney disease. Journal of clinical and translational endocrinology. Vol 6. 2016. 8 –14.
13. V. Pandya, Rao A., K. Chaudhary Lipid abnormalities in kidney disease and management strategies World J Nephrol, 4 (2015), pp. 83-91.
14. J.R. Mead, S.A. Irvine, D.P. Ramji Lipoprotein lipase: structure, function, regulation, and role in disease. J Mol Med, 80 (2002), pp. 753-769.
15. B.G. Nordestgaard, A. Tybjaerg-Hansen IDL, VLDL, chylomicrons and atherosclerosis. Eur J Epidemiol, 8 (1992), pp. 92-98.
16. K.M. Kostner, G.M. Kostner Lipoprotein(a): still an enigma? Curr Opin Lipidol, 13 (2002), pp. 391-396.
17. H.J. Milionis, M.S. Elisaf, A. Tselepis, E. Bairaktari , S.A. Karabina, K.C. SiamopoulosApolipoprotein(a) phenotypes and lipoprotein(a) concentrations in patients with renal failure. Am J Kidney Dis, 33 (1999), pp. 1100-1106.

18. L.L. Rudel, G.S. Shelness. Cholesterol esters and atherosclerosis-a game of ACAT and mouse. Nat Med, 6 (2000), pp. 1313-1314.
19. N.D. Vaziri, Liang K., J.S. Parks. Down-regulation of hepatic lecithin: cholesterol acyl transferase gene expression in chronic renal failure. Kidney Int, 59 (2001), pp. 2192-2196.
20. A. Jonas Lecithin cholesterol acyl transferase, Biochim Biophys Acta, 1529 (2000), pp. 245-256.
21. Liang K., F. Oveisi, N.D. Vaziri Role of secondary hyperparathyroidism in the genesis of hypertriglyceridemia and VLDL receptor deficiency in chronic renal failure. Kidney Int, 53 (1998), pp. 626- 630.