A CROSS-SECTIONAL STUDY TO ASSESS PROTEINURIA AND LIPOPROTEIN (A) LEVELS IN CHRONIC KIDNEY DISEASE

Subalakshmi Radhakrishnan Department of Biochemistry¹

Vijayapriya Indirajith Department of Biochemistry²

Periyandi Chandran Department of Biochemistry¹

Ganesan Subramanyam Professor, HeadDepartment of Biochemistry²

> Menaha Ramu Department of Biochemistry³

Kandhi Suganya

Department of Physiology Sri Ramachandra Institute of Higher Education & Research 1, Mount Poonamallee Rd, Sri Ramachandra Nagar, Ramachandra Nager, Chennai, Tamil Nadu, India, 600116

> Pothanur Mayavan Sasikala⊠ Department of Biochemistry³ sasisakthi@dr.com

¹Government Sivagangai Medical College & Hospital Mela-Vaniangudi, Manamadurai – Tanjavur Rd, Rajaduraisingham, Sivaganga, Tamil Nadu, India, 630561

> ²Dhanalakshmi Srinivasan Medical College & Hospital Siruvachur, Perambalur, India, 621113

³Government Medical College & Hospital Karur 6th Cross Rd, Gandhi Gramam, North Gandhi Gramam, Karur, Tamil Nadu, India, 639004

Corresponding author

Abstract

Chronic kidney disease (CKD) is a reduced glomerular filtration rate and/or increased urinary albumin excretion. The worldwide prevalence of chronic kidney disease (CKD) ranges from 8 to 16 %, and the prevalence of CKD is rising.

The aim: To study the association between CKD stages, proteinuria, and lipoprotein (a) levels among the study participants. Materials and methods: This study was an institution-based observational case-control study involving CKD patients as study group and healthy volunteers as control one. Blood samples were tested for urea, serum creatinine, uric acid levels, triglycerides, total cholesterol, HDL cholesterol, VLDL cholesterol and serum lipoprotein. Statistical analysis was done with SPSS version 20.0.

Result: In our study, the most common age group affected among cases was 41 to 50 years (5th decade), and there was a male preponderance in CKD. CKD patients had a higher mean protein creatinine ratio than controls, and this difference was statistically significant. In addition, CKD patients had significantly higher total cholesterol, triglyceride levels and lower HDL cholesterol levels than controls. Also, they had significantly elevated serum lipoprotein (a) levels than controls.

Conclusion: Based on our study findings, we can conclude that because of the potential role of lipoprotein (a) in the development of cardiovascular disease, it is imperative to include an estimation of lipoprotein (a) levels in all CKD patients, especially in later stages to give a targeted therapy for dyslipidemia among CKD patients.

Keywords: proteinuria, lipoprotein, chronic kidney disease, glomerular filtration rate.

DOI: 10.21303/2504-5679.2022.002567

1. Introduction

Chronic kidney disease (CKD) is defined as a reduced glomerular filtration rate $(GFR < 60 \text{ ml/min/}1.73 \text{ m}^2)$ and/or increased urinary excretion of albumin (albuminuria). It is characterized based on its cause, stage of diseases (defined by GFR) and the severity of albuminuria [1]. The worldwide prevalence of chronic kidney disease (CKD) ranges from 8 to 16 %, and the prevalence of CKD is on the rise [2]. The rise in the prevalence of CKD correlates with the incidence of its risk factors, such as diabetes mellitus and essential hypertension [3]. In addition, research has shown that the dominance of dyslipidemia among patients with CKD translates to an increased risk of cardiovascular disease (CVD) [4, 5]. Lipoprotein (a) also known as Lp(a) is synthesized by the liver and several research studies have revealed that elevated lipoprotein (a) levels have been correlated with reduced estimated GFR (eGFR) and this holds true even in the earliest and milder stages of renal impairment [6, 7].

Hence the aim of our study is to determine lipoprotein levels among CKD patients and to study the association between CKD staging with levels of proteinuria and lipoprotein levels.

2. Materials and methods

The study was conducted in the Department of Biochemistry and Department of Nephrology, Dhanalakshmi Srinivasan Medical College and Hospital in Perambalur, Tamilnadu. This study was an institution-based observational case-control study involving CKD patients as study group and healthy volunteers as control. The study timeline is from June 2018 to July 2019. The study sample included 40 known cases of chronic kidney disease (CKD) attending the Department of Nephrology, Dhanalakshmi Srinivasan Medical College and Hospital in Perambalur for treatment and 40 healthy volunteers with no history of any renal or hepatic disorders and other co-morbidities visiting the hospital as patient attendees or for routine health screening/check-up. The study was conducted after obtaining proper institutional ethics clearance (IECHS/DSMCH/077) and consent forms from the study participants.

Inclusion criteria:

- Patients with a history and physical findings of CKD for more than 6 months, irrespective of the stage of the disease.

- Biochemical analysis suggesting a diagnosis of CKD.

- Sonological findings (radiological opinion) suggestive of CKD.

- Healthy volunteers with no history of kidney disease and without any systemic medical illness. Exclusion criteria:

- CKD patients with diabetes mellitus.
- CKD patients with any other hepatic disease.
- CKD patients with a history of CVD or any CVS complication.
- CKD patients with less than 6 months duration.
- Unwilling patients.

After history and physical examination, blood and urine samples were collected from all the 80 study participants. For all study participants, blood samples were tested for Urea (Urease method), Serum creatinine (Jaffe's method), Serum uric acid levels (Uricase method), Serum triglycerides (Glycerol-3-phosphate oxidase method), Total cholesterol (CHOD-PAP method), HDL cholesterol (Precipitation and CHOD-PAP method), VLDL cholesterol (Friedwald's formula) and Serum lipoprotein (a) levels (Immunoturbidimetric Method) [8]. Also, Urinary protein excretion (mg/dl) (Sulfosalicylic method), Urine creatinine (Jaffe's method) was estimated, and the urine protein-creatinine ratio was calculated for all study participants.

Statistical analysis

Statistical analysis was done using SPSS version 20.0. Student 't' test was used to compare the means of continuous variables after ensuring that the variable followed normal distribution using the Kolmogorov Smirnov Test. Since the test was statistically insignificant (p = 0.124), the data was assumed to follow a normal distribution. χ^2 test was used to compare the various categorical variables. Oneway ANOVA was employed to test for differences in mean lipoprotein (a) levels (Within subjects factor) between severity groups of CKD based on eGFR staging (Between subjects factor). Bonferroni Post-Hoc test was used to test the difference in mean lipoprotein (a) levels between individual group-wise comparisons. Pearson's correlation was used to study the linear relationship between continuous variables such as age, eGFR, Blood urea, Serum Creatinine, protein-creatinine ratio and serum Lipoprotein (a) levels. The p-value of < 0.05 was used to reject the null hypothesis in all the statistical tests of significance.

3. Result

.....

Table 1 shows the comparison of mean age between cases and controls (n = 80), with the mean age of cases being 55.2 ± 11 years while that of controls is 52.9 ± 11.3 years which shows no statistically significant difference in mean age between the cases and controls as p > 0.05 and hence they are comparable.

A comparison of mean weight between cases and controls (n = 80) was shown in **Table 2** where the mean weight in cases was 60.45 ± 8.74 and among controls was 61.89 ± 8.5 . Since no statistically significant difference was seen in mean weight between the cases and controls as p > 0.05, the variable was highly comparable.

| Group | Ν | Mean age (years)±S.D | Mean difference | 't-test p-value |
|----------|----|----------------------|-----------------|--------------------|
| Cases | 40 | 55.28 ± 11.07 | 2 225 | 0.250 |
| Controls | 40 | 52.9±11.3 | 2.325 | 0.350 |

| Comparison | of mean | weight |
|------------|---------|--------|

| Group | Ν | Mean weight (Kg)±S.D | Mean difference | 't-test p-value |
|----------|----|----------------------|-----------------|--------------------|
| Cases | 40 | 60.45 ± 8.74 | 1 442 | 0.458 |
| Controls | 40 | 61.89 ± 8.53 | 1.442 | 0.438 |

In **Table 3**, cases had a higher systolic blood pressure where the mean SBP was 139.7 ± 15.6 when compared to controls 115.2 ± 11.2 (Mean difference: 24.5 mmHg) and the mean diastolic blood pressure of the cases was 139.7 ± 15.6 than controls 76.45 ± 7.3 (Mean difference: 8.5 mmHg). The difference in cases' and controls' systolic and diastolic blood pressures was statistically significant as p < 0.05.

Table 3

| Comparison | of blood | pressure |
|------------|----------|----------|
| Comparison | 01 01000 | pressure |

| BP | Group | Ν | Mean (mm Hg) ±SD | Mean difference | ʻt-test p-value |
|-----------|----------|----|------------------|-----------------|--------------------|
| Systolic | Cases | 40 | 139.7 ± 15.6 | 24.55 | < 0.001 |
| | Controls | 40 | 115.2 ± 11.2 | 24.55 | < 0.001 |
| Diastolic | Cases | 40 | 84.95 ± 10.5 | 0.5 | < 0.001 |
| | Controls | 40 | 76.45 ± 7.3 | 8.3 | < 0.001 |

Table 4 compares renal parameters between cases and controls (n = 80). Cases had higher blood urea and serum creatinine levels than controls, as expected, and this difference was statistically significant as p < 0.05. Also, there were statistically significant higher mean serum uric acid levels than controls (p < 0.05). In addition, the mean protein creatinine ratio was significantly higher than controls (p < 0.05).

| Parameter | Group | Ν | Mean(mg %)±S.D | Mean difference | 't'test p-value | |
|--------------------------|----------|----|-------------------|-----------------|--------------------|--|
| Duraa | Cases | 40 | 65.15 ± 30.8 | 17 62 | < 0.001 | |
| B.urea | Controls | 40 | 17.51 ± 2.57 | 47.05 | | |
| | Cases | 40 | 4.540 ± 4.06 | 2 905 | < 0.001 | |
| S.creatinine | Controls | 40 | 0.735 ± 0.115 | 3.805 | | |
| Mean uric acid | Cases | 40 | 6.488 ± 1.688 | 1 244 | < 0.001 | |
| | Controls | 40 | 5.243 ± 0.715 | 1.244 | | |
| D | Cases | 40 | 2.760 ± 1.676 | 2 595 | < 0.001 | |
| Protein-creatinine ratio | Controls | 40 | $0.175 \pm .043$ | 2.385 | < 0.001 | |

Table 4

Comparison of renal parameters

In terms of comparing lipid parameters (**Table 5**) between cases and controls (n = 80), the serum total cholesterol levels of cases were $167.2\pm40 \text{ mgs }\%$. In comparison, controls were $163.6\pm24 \text{ mgs }\%$ which was not statistically significant. The serum triglyceride levels of cases were high ($176\pm43 \text{ mgs }\%$) when compared to the controls ($139.8\pm12 \text{ mgs }\%$), which elevated the VLDL levels too. The serum LDL cholesterol levels of cases were $94\pm35 \text{ mgs }\%$ while that of controls was $83.1\pm24 \text{ mgs }\%$. Whereas the serum HDL cholesterol levels of cases were reduced ($37.5\pm6.8 \text{ mgs }\%$) while controls ($52.4\pm7.08 \text{ mgs }\%$) and shown to be statistically significant. The mean serum lipoprotein (a) levels of cases were $39.7\pm19.6 \text{ mgs }\%$ while controls were $23.4\pm6.4 \text{ mgs }\%$. Cases had higher mean serum lipoprotein (a) levels than controls, which was statistically significant.

Pearson's correlation showed the linear relationship between continuous variables such as age, eGFR, blood urea, serum creatinine, protein-creatinine ratio and serum lipoprotein (a) levels. All parameters were significantly correlated except the age factor (**Table 6**).

| Parameter | Group | Ν | Mean (mg %) | S.D | Mean difference | ʻt-test p value |
|--------------------------|----------|----|-------------|--------|-----------------|--------------------|
| Maan total abalactoral | Cases | 40 | 167.28 | 40.56 | 3.651 | 0.626 |
| Weall total cholesterol | Controls | 40 | 163.63 | 24.16 | | |
| Maan aanun tuialaaanidaa | Cases | 40 | 176.65 | 43.66 | 26 775 | 0.007 |
| Mean serum triglycerides | Controls | 40 | 139.88 | 12.34 | 36.775 | |
| | Cases | 40 | 94.38 | 35.23 | 11.196 | 0.101 |
| Mean LDL cholesterol | Controls | 40 | 83.18 | 24.03 | | |
| Mean serum HDL level | Cases | 40 | 37.58 | 6.883 | 14.90 | < 0.001 |
| | Controls | 40 | 52.48 | 7.089 | | |
| | Cases | 40 | 35.33 | 16.73 | 7.355 | 0.009 |
| Mean VLDL cholesterol | Controls | 40 | 27.97 | 2.468 | | |
| | Cases | 40 | 39.69 | 19.609 | 16.050 | < 0.001 |
| MeanLp(a) (mgs %) | Controls | 40 | 23.417 | 6.402 | 16.279 | |

Table 5

Comparison of lipid parameters

Table 6

Pearson's correlation between continuous variables

| Lipoprotein (a) level versus | Pearson Correlation | p-value |
|------------------------------------|---------------------|---------|
| Age in years | 0.172 | 0.127 |
| eGFR (mL/min/1.73 m ²) | -0.555 | < 0.001 |
| Blood urea | 0.507 | < 0.001 |
| Serum creatinine | 0.360 | 0.001 |
| Protein creatinine ratio | 0.336 | 0.002 |

4. Discussion

This institution-based observational case-control study involving CKD patients as cases and healthy volunteers as controls was done to determine and compare the levels of lipoprotein (a) among CKD patients and healthy controls in addition to studying any association between proteinuria and LP(a) levels among CKD patients.

The demographic profile of our study showed that the most common age group affected among cases was 41 to 50 years followed by 51 to 60 years **Table 1**. There was no statistically significant difference in age groups between the cases and controls, and hence they are comparable. The study findings can be compared with a mean age of 58.2 years, as reported by Rahman et al. [9] and 48.99 ± 16.74 years, as reported by Choudhary et al. [10]. Also, according to the, 23.47 % of cases were in the fifth decade.

There was a male preponderance among CKD cases, with male to female ratio being 6.5:3.5 and similarly matched controls were taken with a ratio of 3:1. There was no statistically significant difference in the distribution of gender between the cases and controls, and hence they are comparable. The study findings corroborate with Choudhary et al. [10], in which the male-to-female ratio was 1.21:1.

The finding of blood pressure in our study **Table 3** is on the expected lines, as hypertension is our population's most common risk factor for CKD. However, there was no statistically significant difference in mean weight between the cases and controls as p > 0.05.

Cases had higher blood urea (47.6 mgs %) and serum creatinine (3.8 mgs %) levels than controls. This difference was statistically significant as p < 0.05 **Table 4**. In addition, cases had higher mean serum uric acid levels (1.24 mgs %) than controls, which was statistically significant as p < 0.05. Finally, cases had a higher mean protein creatinine ratio (2.76 vs 0.175) than controls, which was statistically significant as p < 0.05 as shown in **Table 4**.

Cases had higher mean serum total cholesterol levels than controls. However, this difference was not statistically significant as p > 0.05 as shown in **Table 5**. Cases had higher mean serum triglyceride levels than controls, and this difference was statistically significant. Furthermore, cases had higher mean serum VLDL cholesterol levels than controls, as expected from the serum triglyceride levels, and this difference was statistically significant as shown in **Table 5**.

Cases had higher mean serum LDL cholesterol levels than controls, but this difference was not statistically significant. Conversely, cases had lower mean serum HDL cholesterol levels than controls, and this difference was statistically significant. Reduced HDL levels (males < 40 mgs %, females < 50 mgs %) were present in 75 % of cases and 15 % among controls, and this difference was statistically significant as shown in **Table 5**.

Hill et al. in 2016 [11] stated that the patients in the undialysed group (50 %) and in the dialyzed group (35 %) had elevated serum triglycerides which are similar to the current study findings. The prevalence of individual dyslipidemias was high total cholesterol in 50.44 %, high triglycerides in 67 %, high LDL-Cholesterol in 42 %, high VLDL-Cholesterol in 67 % and low HDL-Cholesterol in 73.9 % [10]. Moreover, the TC, TG, LDL-C and VLDL-C were in increasing trend with the progression of CKD stages (3–5) and increased in Subgroup II (ESRD) as compared to Subgroup I (Non-ESRD), the increase being significant in the case of TG and VLDL-C. HDL-C value was in decreasing trend with the progression of CKD stages.

Yun et al. [12] did not find any lipid abnormality in chronic renal failure patients either on regular hemodialysis or being managed conservatively. Rahman et al also reported that none of the lipoprotein or lipid measures was related to composite endpoint or rate of change in GFR. However, a similar study also observed an increase in the values of TC, TGL and VLDL-C while a decrease in HDL-C values with the progression of CKD stages [13]. Aharwar et al. [14] observed the TG, LDL-C, and VLDL-C to progressively increase with successive CKD stages while HDL-C value to decrease progressively with successive CKD stages. Garg et al. 2015 [15] showed a statistically significant rise in the values of TG, TC, VLDL-C and TC/HDL-C with the progression of the stages of CKD.

Elevated levels of lipoprotein (a) were present in 75 % of cases and 10 % among controls, and this difference was statistically significant as p < 0.05 Table 5.

Correlation between Lipoprotein (a) levels and other factors was studied using Pearson Correlation. It was observed that there was a statistically significant negative correlation between Lipoprotein (a) levels and eGFR, indicating that a decline in eGFR is associated with increased levels of Lp(a). In addition, there was a statistically significant positive correlation between lipoprotein (a) levels and renal function parameters such as blood urea, serum creatinine levels and Protein creatinine ratio. However, there was no statistically significant correlation between Lipoprotein (a) levels and age **Table 6**.

The study findings can be compared with that of Sudha Rani J et al. [16], in which the mean serum lipoprotein (a) levels of cases were 73.6 ± 3.8 mgs % (reference range: 62 to 82) while that of controls (reference range: 15 to 26) was 21.2 ± 3.3 mgs % and also the difference was statistically significant. In addition, according to Sharma H et al. [17], the mean serum lipoprotein (a) levels of CKD cases who were on hemodialysis was higher than that of controls. However, this difference was found to be not statistically significant. On the other hand, fewer studies [18–20] observed higher mean serum lipoprotein (a) levels among CKD cases than that of controls, and this difference was found to be statistically significant. Moreover, the 4 weeks of hemodialysis led to a significant fall in lipoprotein (a) levels by 24 % of CKD patients [21, 22].

Study Limitations

- Smoking, alcoholism and other confounding variables may alter the lipid pattern in the study group.

- Effect of hemodialysis or other forms of renal replacement therapy was not studied because of feasibility.

- CKD patients with pre-existing diabetes and cardiovascular diseases were excluded from the study and hence this may underestimate the dyslipidemia and levels of lipoprotein (a).

- Further studies with more sample size is advocated for potential effect of CKD progression on and effect of lipoprotein (a) on progression of CKD.

Prospects for further research. Further studies with more sample sizes are advocated for the potential effect of CKD progression on and the effect of lipoprotein (a) on the progression of CKD.

5. Conclusion

In our study, the most common age group affected among cases was 41 to 50 years (5th decade), and there was a male preponderance in CKD. CKD patients had a higher mean protein creatinine ratio than controls, and this difference was statistically significant. CKD patients had significantly higher total cholesterol, triglyceride levels and lower HDL cholesterol levels than controls. Also, they had significantly elevated serum lipoprotein (a) levels than controls. Based on the above findings, we can conclude that because of the potential role of lipoprotein (a) in the development of cardiovascular disease, it is imperative to include an estimation of lipoprotein (a) levels in all CKD patients, especially in later stages to give a targeted therapy for dyslipidemia among.

Conflict of interest

The authors declare that there is no conflict of interest in relation to this paper, as well as the published research results, including the financial aspects of conducting the research, obtaining and using its results, as well as any non-financial personal relationships.

Financing

The study was performed with no additional financial support.

References

- KDIGO CKD Work Group. 2013. KDIGO 2012 clinical practice guideline for the evaluation and management of chronic kidney disease. Kidney International Supplements, 3, 1–150.
- [2] Jha, V., Garcia-Garcia, G., Iseki, K., Li, Z., Naicker, S., Plattner, B. et. al. (2013). Chronic kidney disease: global dimension and perspectives. The Lancet, 382 (9888), 260–272. doi: https://doi.org/10.1016/s0140-6736(13)60687-x
- [3] Hopewell, J. C., Haynes, R., Baigent, C. (2018). The role of lipoprotein (a) in chronic kidney disease. Journal of Lipid Research, 59 (4), 577–585. doi: https://doi.org/10.1194/jlr.r083626

- [4] Viney, N. J., van Capelleveen, J. C., Geary, R. S., Xia, S., Tami, J. A., Yu, R. Z. et. al. (2016). Antisense oligonucleotides targeting apolipoprotein (a) in people with raised lipoprotein (a): two randomised, double-blind, placebo-controlled, dose-ranging trials. The Lancet, 388 (10057), 2239–2253. doi: https://doi.org/10.1016/s0140-6736(16)31009-1
- [5] Tsimikas, S., Viney, N. J., Hughes, S. G., Singleton, W., Graham, M. J., Baker, B. F. et. al. (2015). Antisense therapy targeting apolipoprotein (a): a randomised, double-blind, placebo-controlled phase 1 study. The Lancet, 386 (10002), 1472–1483. doi: https://doi.org/10.1016/s0140-6736(15)61252-1
- [6] Kronenberg, F., Utermann, G. (2012). Lipoprotein (a): resurrected by genetics. Journal of Internal Medicine, 273 (1), 6–30. doi: https://doi.org/10.1111/j.1365-2796.2012.02592.x
- [7] Nordestgaard, B. G., Langsted, A. (2016). Lipoprotein (a) as a cause of cardiovascular disease: insights from epidemiology, genetics, and biology. Journal of Lipid Research, 57 (11), 1953–1975. doi: https://doi.org/10.1194/jlr.r071233
- [8] Thanassoulis, G. (2016). Lipoprotein (a) in calcific aortic valve disease: from genomics to novel drug target for aortic stenosis. Journal of Lipid Research, 57 (6), 917–924. doi: https://doi.org/10.1194/jlr.r051870
- [9] Rahman, M., Yang, W., Akkina, S., Alper, A., Anderson, A. H., Appel, L. J. et. al. (2014). Relation of Serum Lipids and Lipoproteins with Progression of CKD: The CRIC Study. Clinical Journal of the American Society of Nephrology, 9 (7), 1190–1198. doi: https://doi.org/10.2215/cjn.09320913
- [10] Choudhary, Dr. N. (2019). A study of lipid profile in chronic kidney disease in pre- dialysis patients. International Journal of Medical Research and Review, 7 (3), 150–156. doi: https://doi.org/10.17511/ijmrr.2019.i03.01
- [11] Hill, N. R., Fatoba, S. T., Oke, J. L., Hirst, J. A., O'Callaghan, C. A., Lasserson, D. S., Hobbs, F. D. R. (2016). Global Prevalence of Chronic Kidney Disease – A Systematic Review and Meta-Analysis. PLOS ONE, 11 (7), e0158765. doi: https:// doi.org/10.1371/journal.pone.0158765
- [12] Yun, J.-S., Ahn, Y.-B., Song, K.-H., Yoo, K.-D., Park, Y.-M., Kim, H.-W., Ko, S.-H. (2015). Lipoprotein (a) predicts a new onset of chronic kidney disease in people with Type 2 diabetes mellitus. Diabetic Medicine, 33 (5), 639–643. doi: https://doi.org/10.1111/dme.12862
- [13] Noor, S., Zuberi, N. A., Fatima, F., Iqbal, T. (2014). Status of Lipid Profile in Different Stages of Chronic Kidney Disease. Annals of AbbasiShaheed Hospital & Karachi Medical & Dental Collegem, 19 (2). 73.
- [14] Aharwar, S., Lahariya, D. (2015). A study of lipid profile in chronic kidney disease in non-diabetic patients. Hemoglobin, 8 (2.34), 11–51.
- [15] Garg, G., Chawla, S. M., Kaur, S. (2015). A Clinical Study Of Dyslipidemia In Patients Of Chronic Kidney Disease. International Journal of Bioassays, 4 (3), 3732–7. 75.
- [16] Rani, D. J., Raju, D. S. (2016). A Study of Lipid Profile in Chronic Renal Disease with Special Reference to LP (a). Scholars Academic Journal of Biosciences, 4 (10A), 832–839.
- [17] Sharma, H., J. Shah, T., H. Gorasia, J., P. Baria, D. (2012). Lipid profile and lipoprotein (a) in chronic renal failure patients with and without hemodialysis. International Journal of Medicine and Public Health, 2 (4), 28–31. doi: https://doi.org/10.5530/ijmedph.2.4.6
- [18] Xuan, L., Wang, T., Dai, H., Wang, B., Xiang, J., Wang, S. et. al. (2020). Serum lipoprotein (a) associates with a higher risk of reduced renal function: a prospective investigation. Journal of Lipid Research, 61 (10), 1320–1327. doi: https://doi.org/10.1194/jlr.ra120000771
- [19] Hsu, R. K., Powe, N. R. (2017). Recent trends in the prevalence of chronic kidney disease. Current Opinion in Nephrology and Hypertension, 26 (3), 187–196. doi: https://doi.org/10.1097/mnh.00000000000015
- [20] Tada, H., Yamagami, K., Nishikawa, T., Yoshida, T., Teramoto, R., Sakata, K. et. al. (2020). Lipoprotein (a) and the Risk of Chronic Kidney Disease in Hospitalized Japanese Patients. Internal Medicine, 59 (14), 1705–1710. doi: https://doi.org/10.2169/ internalmedicine.4503-20
- [21] Gulayin, P. E., Lozada, A., Schreier, L., Gutierrez, L., López, G., Poggio, R. et. al. (2022). Elevated Lipoprotein (a) prevalence and association with family history of premature cardiovascular disease in general population with moderate cardiovascular risk and increased LDL cholesterol. IJC Heart & Vasculature, 42, 101100. doi: https://doi.org/10.1016/j.ijcha.2022.101100
- [22] Moriarty, P. M., Gray, J. V., Gorby, L. K. (2019). Lipoprotein apheresis for lipoprotein (a) and cardiovascular disease. Journal of Clinical Lipidology, 13 (6), 894–900. doi: https://doi.org/10.1016/j.jacl.2019.09.010

Received date 26.05.2022 Accepted date 12.07.2022 Published date 31.07.2022 © The Author(s) 2022 This is an open access article under the Creative Commons CC BY license

How to cite: Radhakrishnan, S., Indirajith, V., Chandran, P., Subramanyam, G., Ramu, M., Suganya, K., Sasikala, P. M. (2022). A cross-sectional study to assess proteinuria and lipoprotein (a) levels in chronic kidney disease. EUREKA: Health Sciences, 4, 32–38. doi: http://doi.org/10.21303/2504-5679.2022.002567