

# A Study of Lipid Profile and CRP in Children with Nephrotic Syndrome.

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## ABSTRACT

**Background:** Nephrotic syndrome (NS) is more common in children in comparison of adults. Dyslipidaemia is considered one of the common findings of nephritic syndrome. C reactive protein (CRP) is considered one of the important marker of inflammation as well as cardiovascular disease. Therefore the present study was designed to assess if there is any relation between lipid profile, CRP and nephrotic syndrome. **Methods:** Present study included thirty (30) children suffering from nephritic syndrome and thirty (30) healthy children as from pediatric wards of Rajshree Medical Research Institute, Bareilly. All children with nephrotic syndrome and control group were between 2-12 years of age. Serum cholesterol level (normal range 150-200 mg/dl), triglycerides (normal range 60-165 mg/dl) and serum VLDL were measured by Enzymatic colorimetric method. Whereas HDL (normal range 30-70 mg/dl) was measured by Phosphotungstate method. Serum LDL was calculated by Friedewald's equation. Serum Albumin (normal range 3.5-5 gm/dl) was measured by Photometric method. CRP was estimated by enzyme linked immune assay (ELISA) method. **Results:** TC ( $p < 0.01$ ), LDL ( $p < 0.01$ ) and TG ( $p < 0.01$ ) were significantly high in patients of nephrotic syndrome in comparison of control children. Further, HDL ( $p < 0.01$ ) was significantly low in nephrotic syndrome group in comparison of control group. There was an insignificant difference in VLDL of both groups. There was an insignificant difference in CRP ( $p < 0.07$ ) of both groups. **Conclusion:** Findings of the current study suggest that dyslipidaemia is associated with nephritis syndrome. This high level of lipoprotein can lead to future cardiovascular diseases in nephrotic syndrome patients. Although, CRP level which is a strong predictor of CVD is normal; nevertheless, risk of future CVD cannot be ruled out. However, more studies on large populations are required to establish a relation between lipid profile, CRP and nephrotic syndrome.

**Keywords:** Nephrotic syndrome, dyslipidaemia, CRP, CVD.

## INTRODUCTION

Nephrotic syndrome (NS) is more common in children in comparison of adults.<sup>[1]</sup> Nephrotic syndrome is manifestation of glomerular disorder which is characterized by profuse proteinuria the, edema, hypoalbuminemia and hyperlipidaemia.<sup>[2,3]</sup> Dyslipidaemia is considered one of the common findings of nephritic syndrome.<sup>[4]</sup> An essential role has been played by lipoproteins in transport of plasma lipids.<sup>[5]</sup>

Various studies suggested low serum albumin level induces synthesis of lipoprotein and decreases brake down of lipids.<sup>[6,7]</sup> Although, hyperlipidaemia is more severe in children of western countries compare to Indian children.<sup>[8,9]</sup> Dyslipidaemia may leads to renal injury along with increased risk of

atherosclerosis and cardiovascular diseases. 10 C reactive protein (CRP) is considered one of the important marker of inflammation as well as cardiovascular disease (CVD).<sup>[11]</sup>

Therefore the present study was designed to assess if there is any relation between lipid profile, CRP and nephrotic syndrome.

## MATERIALS AND METHODS

The present study was a cross section type of study which was approved from the ethical committee of Rajshree Medical Research Institute, Bareilly. Present study included thirty (30) children suffering from nephritic syndrome and thirty healthy children as from pediatric wards of Rajshree Medical Research Institute, Bareilly. All children with nephrotic syndrome and control group were between 2-12 years of age. Children having any type of liver disorders, oedema due to Kwashiorkor or congestive cardiac failure were excluded from the study. The Nephrotic cases were selected according to criteria proposed by International study of kidney disease in children that is children with edema, proteinuria

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(more than 3 gm in 24 h/1.73m<sup>2</sup>), hypoproteinemia (serum albumin less than 2.5 gm/dl, hyperlipidemia.

**Methodology**

Serum cholesterol level (normal range 150-200 mg/dl), triglycerides (normal range 60-165 mg/dl) and serum VLDL were measured by Enzymatic colorimetric method. Whereas HDL (normal range 30-70 mg/dl) was measured by Phosphotungstate method. Serum LDL was calculated by Friedewald's equation. Serum Albumin (normal range 3.5-5 gm/dl) was measured by Photometric method. CRP was estimated by enzyme linked immune assay (ELISA) method.

**Statistical analysis**

Results of the present were analyzed by using spss v21 manufactured by USA. Paired students t test was used to assess if there is any significant difference between lipoproteins and CRP of nephrotic syndrome group and control group. A p value <0.05 was considered as statistically significant.

**RESULTS**

**Table 1: Comparison of lipid profile in both groups.**

	Group I (NS Patients)	Group II (Control)	p value
Total Cholesterol (mg/dl)	328±35.6	189.6±28.6	<0.01*
High Density Lipids (mg/dl)	25.6±8	46.4±6.4	<0.01*
Low Density Lipids (mg/dl)	266.8±31.4	125.6±18.5	<0.01*
Very Low Density Lipid (mg/dl)	42.4±7.6	41±6	<0.06NS
Triglycerides (mg/dl)	178±20	87.8±13	<0.01*

\*= significant, NS= non significant.

**Table 2: Comparison of different types of protein in both groups.**

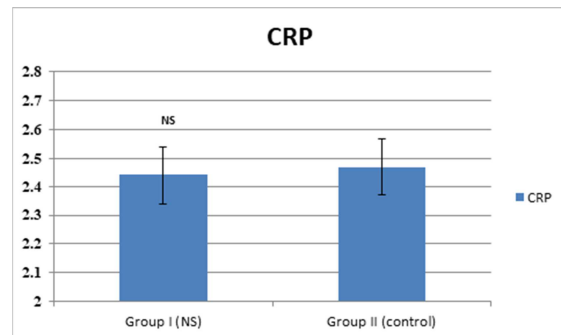
	Group I Patients	Group II Normal	p value
Serum Total Protein(g/dl)	3.65±0.48	7.2±0.4	<0.01*
Serum Albumin(g/dl)	1.7±0.26	4.10±0.3	<0.01*
Serum Globulin(g/dl)	2.29±0.24	3.30±0.25	<0.01*

\*= significant

All the results of the present study have been expressed as mean±SD. [Table 1] shows that TC (p<0.01), LDL (p<0.01) and TG (p<0.01) were significantly high in patients of nephrotic syndrome in comparison of control children. Further, HDL (p<0.01) was significantly low in nephrotic

syndrome group in comparison of control group. However, there was an insignificant difference in VLDL of both groups.

[Table 2] shows that serum total protein (3.65±0.48 g/dl vs 7.2±0.4 g/dl, p<0.01), serum albumin (1.7±0.26 g/dl vs 4.10±0.3 g/dl, p<0.01) and serum globulin (2.29±0.24 g/dl vs 3.30±0.25 g/dl, p<0.01) were significantly low in nephrotic syndrome patients compare to control group. It is evident from figure 1 that there was an insignificant difference in CRP (p<0.07) of both groups.



**Figure 1: Comparison of CRP in both groups.**

NS = non significant.

**DISCUSSION**

Dyslipidaemia is one of the foremost causes of CVD.<sup>[5]</sup> Hyperlipidaemia has been found associated with hypertension as well as CVD.<sup>[12]</sup> Findings of the present study have shown that TC, TG and LDL were significantly high in NS children compare to control group. These findings are consistent with the findings of previous studies of Krishnaswany D et al,<sup>[13]</sup> Appel GB et al and Alexander JH et al as they recorded significantly high level of these lipoproteins in their studies. This high level of TC,<sup>[14,15]</sup> TG and LDL in NS children may be due to decrease of protein in body during nephrotic syndrome as hypoproteinemia induces protein synthesis in liver which may leads to hyperlipidaemia.<sup>[16]</sup> NS causes increase of LDL due to higher protein receptors and increased mRNA receptors along with enhanced gene translation rate.<sup>[17]</sup> This increased level of LDL may leads to impairment of endothelium which may result in loss of dilating ability of arteries. This derangement of endothelium function causes hypertension.<sup>[12]</sup> Apart from this findings of the current studies have shown that HDL was significantly low in NS group in comparison of control group which is very similar to the previous study of Adekoya A.O et al,<sup>[18]</sup> This low level of HDL in NS group may be due to high level of LDL leads decrease of HDL due to lipid peroxydation.<sup>[17]</sup> Further, it has been suggested that dyslipidaemia and proteinuria are related with NS as oncotic pressure is decreased due to hypoalbuminemia results in increases synthesis of protein from the liver.<sup>[19]</sup> Furthermore, lippoprotein catabolism can be affected by NS as this increased

level of lipids may increase the catabolism of lipoprotein in kidney.<sup>[20]</sup>

Higher level of lipoprotein induces various inflammatory processes along with arterial inflammation.<sup>[21]</sup> In addition, higher lipoprotein besides various inflammatory pathways may lead to atherosclerosis.<sup>[22]</sup>

CRP is abundantly produced by hepatocytes in acute response, on stimulation various inflammatory markers like IL6, tumor necrosis factor etc.<sup>[23]</sup> CRP level may be analogous to lipids level in predicting the short term and long term cardiovascular outcomes. Moreover CRP is considered as strong predictor of CVD.<sup>[24]</sup> However, there was an insignificant difference in CRP level in both groups. Which is similar to the previous study of Shostak E et al,<sup>[25]</sup> as they did not observed any significant difference in CRP of NS group and control group. This normal level of CRP in NS group may be due to an insignificant loss of CRP in urine.<sup>[25,26]</sup>

## CONCLUSION

Findings of the current study suggest that dyslipidaemia is associated with nephritis syndrome. This high level of lipoprotein can lead to future cardiovascular disease in nephrotic syndrome patients. Although, CRP level which is a strong predictor of CVD has been found in normal limits in the present study; nevertheless, risk of future CVD cannot be ruled out. However, more studies on large populations are required to establish a relation between lipid profile, CRP and nephrotic syndrome.

## REFERENCES

- Priya Pias, Ellis D Avner (2015) Nephrotic Syndrome. Nelson Textbook of Pediatrics. (20th edn), Philadelphia, WB Saunders, USA, pp. 2521-2523.
- Abraham. M. Rudolph – Textbook of pediatrics 21st edition
- Nelson Text book of pediatrics – 20th edition
- A.A Epstein, The nature and treatment of Nephrosis, JAMA 69, 1917, 444-47.
- B. Bhandari, S.L.Mandowara, Lipoprotein profile in nephrotic syndrome, Indian pediatrics, 17, 1980, 416-19.
- E.M.Thomas, A.H Rosenblum, H.B Lander, R Fisher, Relationship between blood lipid and blood protein levels in nephrotic syndrome, Amer J Dis. Child, 81, 1951, 207.
- J.P Peters, E.B Man, The inter relationship of Serum lipids in patients with diseases of kidneys, J Clin Invest, 22, 1943,721.
- D.G Benakappa, A Subba Rao, N.S.C Sastry, Low density lipoprotein levels in children with nephrotic syndrome, Indian pediatrics, 13 (4), 1976, 287-89.
- J.F Moorhead, M.K Chan, A.M Nahas, Z Varghese, Lipid nephrotoxicity in chronic progressive glomerular and tubulo interstitial disease, Lancet , 2, 1982, 1309-11
- Galvano F, Malaguarnera M, Vacante M, Motta M, Russo C, Malaguarnera G, et al. (2010): The physiopathology of lipoprotein (a). Front Biosci (Schol Ed). 2:866-75.
- Jabs WJ, Logering BA, Gerke P, Kreft B, Wolber EM, Klinger MH, Fricke L, Steinhoff J (2003) The kidney as a second site of human C-reactive protein formation in vivo. Eur J Immunol 33:152–161.
- Sarvottam K, Yadav RK. Obesity-related inflammation & cardiovascular disease: Efficacy of a yoga-based lifestyle intervention. Indian J Med Res. 2014; 139: 822-834.
- Krishnaswamy D , Indumati V , Satihkumar D ,Vijay V, Maharudra S , Amareshwara M and Rajeshwari V. Serum proteins, initial and follow-up lipid profile in children with nephrotic syndrome. IJABPT 2011 ;2:59-63.
- Appel G.B, Blum C.B, Chein S, Kunis C.L and Appel A.S. The hyperlipidaemia of the nephrotic syndrome: relation to plasma albumin concentration, oncotic pressure, and viscosity. N Engl J Med 1985;312:1544-1548.
- Alexander J.H, Schapel G.J and Edwards K.D. Increased incidence of coronary heart disease associated with combined elevation of serum triglycerides and cholesterol concentrations in the nephrotic syndrome in man. Med J Aust 1974; 2:119-122.
- Conde GJ, Sánchez MJ, Macías GJ. Fisiopatología glomerular”. Patología General. Semiología Clínica y Fisiopatología. McGraw - Hill Interamericana. 1995. ISBN 8448600932.
- Warwick G.L, Packard C.J, Demant T, Beedford D.C, Bunton J.M and Shepherd J. Metabolism of apolipoprotein B-containing lipoproteins in subjects with nephrotic range proteinuria. Kidney Int 1991;40:129-138.
- Adekoya A.O, Adekoya B.J, Desalu O.O and Aderibigbe A . Pattern of lipid profile in adult nephrotic syndrome patients in Nigeria. Int J Bio Med Res 2011;2:954-960.
- Crew RJ, Radhakrishnan J, Appel G. (2004): Complications of the nephrotic syndrome and their treatment. Clin Nephrol. 62(4):245-59.
- Doucet C, Mooser V, Gonbert S, Raymond F, Chapman J, Jacobs C, et al. (2000): Lipoprotein(a) in the nephrotic syndrome: molecular analysis of lipoprotein(a) and apolipoprotein(a) fragments in plasma and urine. J Am Soc Nephrol. 11(3):507-13.
- Alexander RW. Oxidative stress and the mediation of arterial inflammatory response: A new prospective. Hypertension. 1996; 25:155-161.
- Walter MF, Jacob RF, Jeffers B, Ghadanfar MM, Preston GM, Buch J, Mason PR; Serum levels of thiobarbituric acid reactive substances predict cardiovascular events in patients with stable coronary artery disease A longitudinal analysis of the prevent study. J Am Coll Cardiol. 2004; 44(10):1996-2002.
- Vermeire S, Van Assche G, Rutgeerts P The role of C-reactive protein as an inflammatory marker in gastrointestinal diseases. Nat Clin Pract Gastroenterol Hepatol. 2005 Dec;2(12):580-6.
- de Ferranti S, Rifai N. C-reactive protein and cardiovascular disease: a review of risk prediction and interventions. Clin Chim Acta. 2002 Mar;317(1-2):1-15.
- Shostak E, Krause L , Dagan A, Ben Dor A, Keidar M, Davidovits M. Is serum CRP level a reliable inflammatory marker in pediatric nephrotic syndrome? Pediatr Nephrol. 2016 Aug;31(8):1287-93.
- Asuthi KD, Muryawan MH, Mellyana O. Correlation between lipid profile and C-reactive protein in children with nephrotic syndrome. Paediatrica Indonesiana.2015 Vol 55 (1).

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