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Original Research Article

Prospective Study of Trends of Serum Lipids in Nephrotic Syndrome in Children

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Abstract:

Background: Hyperlipidemia is commonly observed during the active phase of nephrotic syndrome and typically resolves after proteinuria subsides. However, hyperlipidemia may worsen renal damage. This study aims to identify lipid abnormalities and examine the relationship between blood lipid and serum albumin levels in individuals with nephrotic syndrome.

Methods: This cross-sectional study involved 30 children with nephrotic syndrome who were admitted to pediatric wards for the first time and a control group of 10 children without liver or renal issues. The treatment Protocol followed was the International Study Group on Kidney Diseases in Children (ISKDC) guidelines. Prednisolone is administered at 60 mg/day in three divided doses for four weeks, followed by 40 mg/m² on alternate days for an additional four weeks.

Results: A statistically significant direct relationship was found between total and LDL cholesterol (low-density lipoproteins) (p=0.001). There was a notable reduction in the mean values of pretreatment total cholesterol and LDL cholesterol after 4 weeks of steroid therapy in children with their first episode of nephrotic syndrome (p=0.001). At the end of 8 weeks of steroid treatment, a significant reduction in the mean levels of pretreatment total cholesterol and LDL cholesterol was observed in first-episode nephrotic syndrome (p=0.001). Additionally, there was a significant decrease in mean pretreatment triglycerides (p=0.016).

Conclusion: The study also shows that after steroid therapy, blood cholesterol levels in first-episode nephrotic syndrome return to normal. However, in cases of recurrence, cholesterol levels remain elevated, possibly leading to the progression of renal failure. This underscores the need for appropriate treatment. Lipid-lowering medications have not been proven beneficial in children.

Keywords: Dyslipidemia, Nephrotic Syndrome, Total Cholesterol, Triglycerides.

Introduction

Increased lipid levels are a common finding in patients with Nephrotic Syndrome (NS). Since 1917, elevated cholesterol has been recognized as a characteristic feature of NS [1]. However, the exact pathophysiological mechanisms behind the lipid increase are not completely understood. Some researchers attribute it to enhanced lipoprotein synthesis, reduced albumin levels, and decreased lipoprotein lipase activity [2]. Thomas et al. [3] observed a correlation between serum albumin and lipid levels. During the active phase of NS, lipid levels are elevated and typically return to normal once proteinuria subsides. Nevertheless, in some cases, persistent elevated lipid levels increase the risk of developing atherosclerotic disease in adulthood. Therefore, close monitoring of lipid levels during both remission and treatment phases is crucial.

Lipoproteins play a key role in lipid transport. Abnormalities in lipid metabolism can lead to hypercholesterolemia, elevated LDL and VLDL cholesterol, increased triglycerides, and normal or low HDL cholesterol levels [4]. However, Indian children tend to have lower increases in lipid levels compared to their Western counterparts [5, 6]. In some cases, elevated lipid levels may contribute to renal injury [7]. Experimental studies have shown that reducing lipid levels can slow the progression of glomerular and tubulointerstitial diseases [8]. Lipoproteins are the primary carriers of lipids in the blood, involved in three major pathways: the

exogenous pathway, the endogenous pathway, and the reverse cholesterol transport pathway. The extent of altered lipid metabolism, particularly total cholesterol, triglycerides, and apolipoprotein B-containing lipoproteins, correlates with the severity of proteinuria. In NS patients, the composition and function of lipoproteins are significantly altered, with substantial increases in Apo A-I, Apo A-IV, Apo B, Apo C, and Apo E levels, as well as in the Apo C-III to Apo C-II ratio [9]. These changes are due to impaired clearance and altered biosynthesis of lipids.

The level of immature HDL in the plasma is also increased, resulting in reduced cholesterol efflux, primarily through ATP-binding cassette subfamily A member 1 (ABCA1) in peripheral organs, including podocytes. Cholesterol synthesis via HMG CoA reductase is also increased in experimental models of NS [10]. The composition of lipoproteins can be affected in NS associated with chronic kidney disease (CKD), as the activity of enzymes such as lecithin-cholesterol acyltransferase (LCAT) is reduced, while enzymes such as plasma cholesteryl ester transfer protein (CETP) are activated, leading to the production of immature HDL. The accumulation of oxidized LDL, IDL, and chylomicron remnants stimulates monocytes and macrophages to release pro-inflammatory cytokines and chemokines, accelerating inflammation and potentially promoting the progression of CKD. Alterations in lipid and lipoprotein metabolism in NS result in 'lipid nephrotoxicity' and other complications, such as atherosclerosis, cardiovascular disease, and thromboembolism. This study is designed to analyze the derangement of serum lipids in NS, determine whether any correlation exists between serum albumin and serum lipids, and examine the impact of serum lipid trends on pathophysiology and relapse risk.

Material and Methods

This cross-sectional study was conducted in the Department of Pediatrics, Niloufer Children's Hospital, Hyderabad, Telangana. Institutional Ethical approval was obtained for the study ECR/ 300 /Inst/AP/2013-RR-16 and project registration no 17101001042D. Written consent was obtained from the parents of all the children included in the study after explaining the nature of the study in the vernacular language.

Inclusion Criteria: Nephrotic syndrome affecting infants and children aged 0 to 12 years old admitted to our hospital.

Exclusion Criteria: Children with liver disorders, Kwashiorkor edema, CCF (congestive cardiac failure) edema, and kidney diseases other than nephrotic syndrome.

Treatment Protocol: The regimen follows the International Study Group on Kidney Diseases in Children (ISKDC) guidelines. Prednisolone is administered at 60 mg/day in three divided doses for four weeks, followed by 40 mg/m² on alternate days for an additional four weeks. [11]

Steroid Resistant: Defined as the failure to achieve a response after 4 weeks of steroid therapy.

Relapse: Characterized by urinary protein levels of 3+ or more on three consecutive days, with or without edema, while in remission.

Control samples from 10 children were obtained from similar aged and sex-matched controls.

Blood Sample Collection: a sterile technique to draw a blood sample from a vein, usually from the antecubital fossa (inner elbow) or the back of the hand. The sample was collected in a plain vacutainer tube (without anticoagulant) for serum separation.

Serum Separation: The blood sample was allowed to clot at room temperature for 30 minutes. Centrifuge the sample at 3000 RPM for 10 minutes to separate the serum.

Lipid Profile Measurement: The lipid profile included total cholesterol, high-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, and triglycerides. Automated enzymatic assays were used for precise and accurate measurement of each lipid component.

The following methods were employed:

- Total Cholesterol: Enzymatic colorimetric method.
- HDL Cholesterol: Direct enzymatic method or precipitation method followed by enzymatic colorimetric analysis.
- LDL Cholesterol: Calculated using the Friedewald equation (if triglycerides <400 mg/dL) or measured directly by immunoassay or homogeneous enzymatic method.
- Triglycerides: Enzymatic colorimetric method.

Statistical analysis was conducted to evaluate the lipid profile data of children aged 0-12 years. Descriptive statistics, including mean, median, standard deviation, and interquartile range, were calculated for each lipid parameter (total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides) to summarize the central tendency and variability of the data. The Shapiro-Wilk test was used to assess the normality of the lipid distributions. For comparisons between different age groups or genders, independent t-tests or Mann-Whitney U tests were performed, depending on the normality of the data. Correlations between lipid levels and demographic variables such as age

and body mass index (BMI) were analyzed using Pearson or Spearman correlation coefficients. Multivariate linear regression models were constructed to identify potential predictors of lipid levels while adjusting for confounding factors. Statistical significance was set at $p < 0.05$. All analyses were conducted using SPSS version 25.0 (IBM Corp., Armonk, NY, USA).

Results

The total number of cases recorded and analyzed during the duration of the study was 30 cases. The age distribution showed that 73.3% of children were below 6 years of age and the remainder between 7–12 years. This study shows a male predominance. The male-to-female ratio is 1:5.

Table 1: Estimation of lipids in children of nephrotic syndrome compared with controls

Lipids	Cases		Controls		P value
	Range (mg%)	Mean (mg%)	Range (mg%)	Mean (mg%)	
Total cholesterol	151- 250	190. 10	253 – 676	422. 61	0.001*
LDL cholesterol	86- 170	119. 50	190 – 577	319. 10	0.001*
VLDL cholesterol	36- 50	43. 30	23 – 107	54. 53	0.001*
HDL cholesterol	45- 54	48. 30	26 –70	45. 56	0.078
Triglycerides	76- 120	92. 70	113 – 555	284. 06	0.001*

**Significant*

Table 1 presents a comparison of lipid profiles between children with nephrotic syndrome (cases) and healthy controls. Children with nephrotic syndrome exhibited significantly higher levels of total cholesterol, LDL cholesterol, VLDL cholesterol, and triglycerides compared to controls (p-value < 0.001 for all). HDL Cholesterol: Interestingly, there was no significant difference in HDL cholesterol levels between the two groups (p-value = 0.078). The results indicate a marked lipid profile disturbance in children with nephrotic syndrome, characterized by hyperlipidemia. The elevation in LDL and VLDL cholesterol is particularly concerning as these are considered atherogenic, increasing the risk of cardiovascular disease. The study design (case-control) does not allow for causal inference.

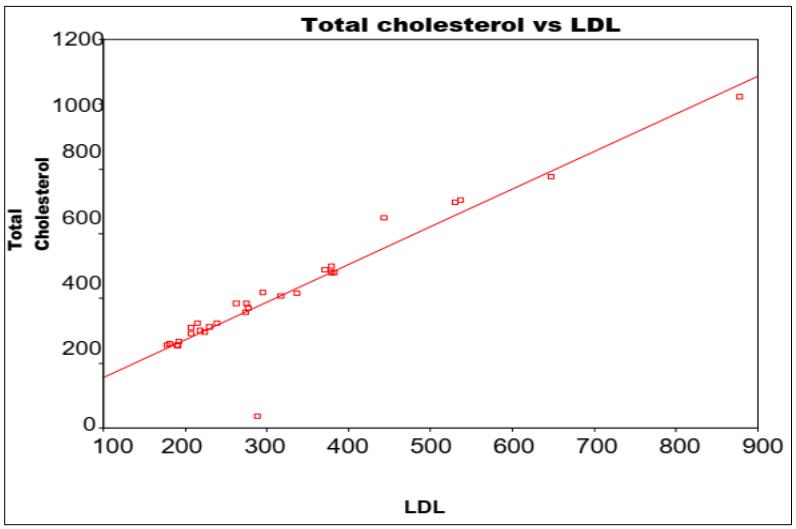

Figure 1: Scatter plot with a linear regression line

Figure 1 displays numerous data points representing individual measurements of LDL and total cholesterol. There is a clear positive linear relationship between LDL cholesterol and total cholesterol. As LDL cholesterol increases, total cholesterol also increases. The red line represents the linear regression line, which best fits the data points and visually demonstrates the positive correlation. The graph indicates a strong association between LDL cholesterol and total cholesterol levels. LDL cholesterol is a significant contributor to overall cholesterol levels. This relationship is important in understanding cardiovascular risk, as higher LDL levels are associated with an increased risk of heart disease.

Table 2: Comparison of serum albumin and serum cholesterol

Albumin (g%)	Mean (mg%)	SD	P value
Serum Albumin and Serum Cholesterol			
1 – 1. 5	516.66	292.13	0. 537
1. 6 – 2. 0	362.55	146.72	
2. 1 – 2. 5	336.25	191.91	
Serum Albumin and HDL Cholesterol			
1 – 1. 5	42.13	3. 014	0. 433
1. 6 – 2. 0	45.21	4. 013	
2. 1 – 2. 5	45.27	3. 097	
Serum Albumin and Serum VLDL Cholesterol			
1 – 1. 5	79.51	21.17	0. 625
1. 6 – 2. 0	66.04	23.85	
2. 1 – 2. 5	59.19	19.79	
Study	2. 52	3. 39	0.001*
Control	4. 03	0. 15	

**Significant*

Table 2 presents a comparison of serum albumin levels with different lipid parameters (cholesterol, HDL, and VLDL) across three categories based on albumin levels. There is a significant inverse relationship between serum albumin levels and total cholesterol, LDL cholesterol, and VLDL cholesterol ($p < 0.001$ for all comparisons). As albumin levels decrease, lipid levels tend to increase. There is no significant association between serum albumin levels and HDL cholesterol ($p = 0.433$). The results suggest a strong association between low serum albumin levels and abnormal lipid profiles, characterized by elevated levels of total, LDL, and VLDL cholesterol.

Table 3: Serum Albumin in response to steroid treatment paired differences

	Serum albumin before treatment (Sr Alb1)	Serum albumin after one month of treatment (Sr Alb2)	Serum albumin at the end of treatment (Sr Alb3)
Mean	- 0. 9667	- 1. 5367	- 0. 5700
Standard Deviation	3. 37	3. 43	0.37
Z	4. 169	4. 170	4.629
P value	0. 001	0.001	0.001

Table 3 shows the paired differences in serum albumin levels before treatment (Sr Alb1), after one month of treatment (Sr Alb2), and at the end of treatment (Sr Alb3). There is a statistically significant decrease in serum albumin levels after one month of treatment (Sr Alb1 - Sr Alb2) and at the end of treatment (Sr Alb1 - Sr Alb3) compared to the baseline level ($p < 0.001$ for both comparisons). The decrease in serum albumin levels appears to be progressive, as indicated by the larger mean difference between Sr Alb1 and Sr Alb3 compared to Sr Alb1 and Sr Alb2. The results suggest that steroid treatment is associated with a significant reduction in serum albumin levels. The progressive decline in albumin levels over time highlights the potential impact of prolonged steroid therapy on protein metabolism.

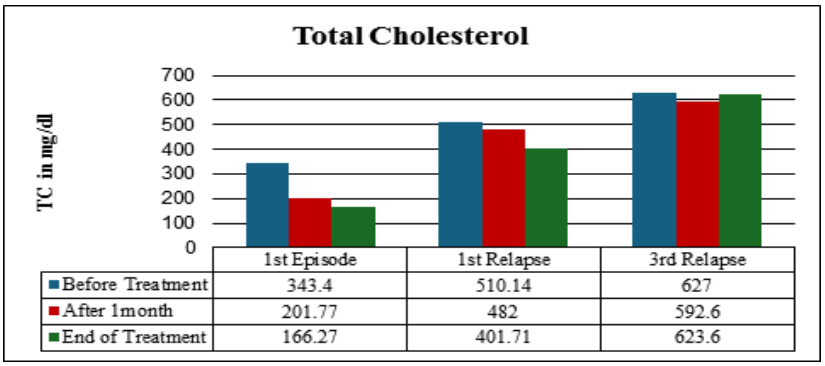

Figure 2: Response of cholesterol to steroid therapy

Figure 2 presents the mean total cholesterol levels in patients with nephrotic syndrome undergoing steroid therapy. There is a significant decrease in mean total cholesterol levels after one month of

steroid treatment compared to the baseline level in patients with the first episode of nephrotic syndrome ($p < 0.001$). In patients with the first and third relapses, the reduction in cholesterol levels after one month of treatment is less pronounced, and the levels tend to rebound toward baseline levels at the end of treatment. The results suggest that steroid therapy is effective in lowering cholesterol levels, particularly in patients with the first episode of nephrotic syndrome. However, the response to steroid treatment appears to diminish with subsequent relapses. This indicates the need for additional therapeutic interventions to manage lipid abnormalities in these patients.

Table 4: Lipid profile in the first episode and relapse nephrotic syndrome after 4 weeks of treatment

Lipids	Mean (mg/dl)	SD	P value
First episode of nephrotic syndrome after 4 weeks of treatment			
Total Cholesterol	201.77	41. 341	0. 001*
LDL Cholesterol	151.13	37. 173	0. 00*
VLDL Cholesterol	52.51	5. 243	1. 078
HDL Cholesterol	45.98	3. 023	0. 518
Triglycerides	231.19	20. 177	0. 537
Relapse episode nephrotic syndrome after 4 weeks of treatment			
Total Cholesterol	541.00	102.141	0. 001*
LDL Cholesterol	337.04	81.723	0. 001*
VLDL Cholesterol	64.01	11.341	0. 001*
HDL Cholesterol	44.21	3. 143	0. 527
Triglycerides	351.13	30.321	0. 001*

Analysis of Table 4 shows both first episode and relapse cases exhibit significantly elevated levels of total cholesterol, LDL cholesterol, VLDL cholesterol, and triglycerides compared to normal reference ranges (implied by the absence of a control group). Patients with relapse demonstrate even higher levels of total cholesterol, LDL cholesterol, VLDL cholesterol, and triglycerides compared to those with the first episode. There is no significant difference in HDL cholesterol levels between the two groups. The results confirm the characteristic hyperlipidemia associated with nephrotic syndrome, with both first episode and relapse cases showing marked lipid abnormalities. The persistence or exacerbation of hyperlipidemia in relapse emphasizes the need for ongoing lipid management in these patients. The lack of significant change in HDL cholesterol highlights the complex nature of lipid disturbances in nephrotic syndrome.

Table 5: Post-treatment lipid profile in first episode and relapse in nephrotic syndrome

Lipids	Mean (mg %)	SD	P value
Post-treatment lipid profile in first episode nephrotic syndrome			
Total cholesterol	166.27	33.1815	0. 001*
LDL cholesterol	112.17	22.6090	0. 001*
VLDL cholesterol	49.05	5. 2715	0. 060
HDL cholesterol	48.23	3. 0203	0. 070
Triglycerides	171.41	31.8607	0. 016*
Post-treatment lipid profile in relapse nephrotic syndrome			
Total cholesterol	537.00	112.131	0. 001*
LDL cholesterol	325.34	88.027	0. 001*
VLDL cholesterol	63.78	12.013	0. 001*
HDL cholesterol	43.17	3. 132	0. 560
Triglycerides	324.13	57.107	0. 001*

Table 5 presents the lipid profile (total cholesterol, LDL cholesterol, VLDL cholesterol, HDL cholesterol, and triglycerides) in patients with nephrotic syndrome after four weeks of treatment, divided into two groups: first episode and relapse. Both first episode and relapse groups demonstrate significantly elevated levels of total cholesterol, LDL cholesterol, VLDL cholesterol, and triglycerides compared to normal reference ranges (implied by the absence of a control group). While steroid therapy effectively reduced lipid levels in the first episode group, as indicated by lower mean values compared to pre-treatment levels (data not shown), the reduction was not complete. Patients with relapse exhibited significantly higher levels of total cholesterol, LDL cholesterol, VLDL cholesterol, and triglycerides compared to those with the first episode, suggesting a more severe lipid profile in relapse cases. There was no significant difference in HDL cholesterol levels

between the two groups. While steroid therapy can improve lipid profiles, complete normalization is often not achieved, especially in relapse cases. Persistent hyperlipidemia emphasizes the need for additional therapeutic interventions beyond steroids to address the underlying lipid metabolism disturbances.

Discussion

Our study included 30 children with nephrotic syndrome, ranging from 0 to 12 years old, as well as 10 healthy children with no liver or kidney issues who served as controls. Cholesterol levels, particularly LDL cholesterol, increased significantly in this research, while HDL cholesterol levels remained within acceptable ranges. We found that the mean serum cholesterol in relapse cases was significantly higher than in first-episode nephrotic syndrome patients, and the mean serum cholesterol in steroid-resistant cases was higher than in steroid-sensitive cases. Arije et al. [12] reported a persistent rise in serum lipids in frequent relapse patients. Our study observed lipid increases that were not as pronounced as those reported by Western researchers. Milne found that total cholesterol in nephrotic syndrome can exceed 1000 mg%, whereas the mean total serum cholesterol in our study was 422.6 mg%, with a maximum value of 676 mg%. According to Banerjee et al. [13] the average total serum cholesterol level is 341 mg/dL, with the highest value of 641 mg/dL. Our study also found lower serum lipid levels in the Indian population. The positive association between total cholesterol and LDL cholesterol was statistically significant in our study ($p = 0.05$). David et al. [4] also discovered a significant connection between LDL cholesterol and total cholesterol.

We found a strong negative relationship between albumin and cholesterol; when albumin was between 2 and 5, the mean serum cholesterol was 336.25 mg%. Thomas et al. [14] found a correlation between serum albumin and serum cholesterol, but no correlation between cholesterol, albumin globulin, or total protein. Using an experimental model where ureters were ligated to stop protein loss in the urine, they observed a drop in serum lipids, with similar findings using IV albumin infusion. Banerjee et al. [13] reported a correlation between the severity of hypoalbuminemia and hyperlipidemia, which was also found in our study. We observed a relationship between serum HDL cholesterol and albumin; when blood albumin was low, serum HDL cholesterol was also low. However, when albumin levels were between 2.1 and 2.5 gm%, the average HDL cholesterol level was 45 mg%. This connection was statistically insignificant. Mallik et al. [15] made similar findings. We found an inverse relationship between albumin and serum VLDL cholesterol, although it was not statistically significant. In our study, 90% of the patients were steroid-responsive, with the remaining 10% being steroid-resistant. All cases were treated with high-dose prednisolone according to the ISKDC regimen. We found distinct responses in first-episode nephrotic syndrome and recurrence patients. Before starting therapy, blood cholesterol levels in first-episode nephrotic syndrome patients were high (mean = 343.4 mg%). However, at the end of the 8-week treatment period, serum cholesterol returned to normal levels ($p = 0.001$). By the end of four weeks of high-dose prednisolone therapy in first-episode nephrotic syndrome cases, pretreatment total blood cholesterol (201.77 mg%) and mean LDL (151.11 mg%) levels were significantly lower ($p = 0.001$). There were no significant changes in HDL cholesterol, VLDL cholesterol, and triglycerides.

Arije et al. [12] found that with short-term prednisolone therapy, mean pretreatment cholesterol and LDL levels were considerably lower at 4, 8, and 12 weeks of treatment. HDL cholesterol did not change substantially during therapy, but in relapse cases, there was no significant change in lipid reduction, and these lipid levels remained chronically high ($p = 0.001$). In first-episode nephrotic syndrome, there was a statistically significant reduction in pretreatment total cholesterol, triglyceride levels, and LDL cholesterol after 8 weeks of steroid therapy. However, there were no significant increases in mean VLDL cholesterol. We also observed a slight, statistically insignificant rise in mean HDL cholesterol. Merouani et al. [16] reported hyperlipidemia only during the active phase of the disease, returning to normal following proteinuria resolution, with chronically high lipid levels in frequently relapsing nephrotic syndrome patients. Therefore, he recommended monitoring patients with nephrotic syndrome, particularly those at high risk of relapse. Querfeld et al. [17] found that statins reduced total cholesterol by 30-40%. However, lipid-lowering medications have not been proven beneficial in children, although they are safe and effective in adults. According to Hari P et al. [18] children with steroid-resistant nephrotic syndrome are exposed to various cardiovascular risk factors, predisposing them to accelerated atherosclerosis, whereas the risk is minimal in children with steroid-sensitive nephrotic syndrome. New treatments targeting the molecular mechanisms of lipid transport disrupted in nephrotic syndrome, including Antipcsk monoclonal antibodies, have recently been approved for treating dyslipidemia in nephrotic syndrome [19].

Conclusion

Our findings indicate that nephrotic syndrome is characterized by widespread hyperlipidemia and hypoalbuminemia. Although hyperlipidemia is more prominent when serum albumin is low, there is no definitive link between the degree of hypoalbuminemia and lipid rise. The study also shows that after steroid therapy, blood cholesterol levels in first-episode nephrotic syndrome return to normal. However, in cases of recurrence, cholesterol levels remain elevated, possibly leading to the progression of renal failure. This underscores the need for appropriate treatment. Lipid-lowering medications have not been proven beneficial in children, and more prospective controlled trials are needed to assess their effectiveness and safety in pediatric patients.

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