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Original Research Article

STUDY OF CLINICAL AND BIOCHEMICAL PROFILE OF NEPHROTIC SYNDROME IN CHILDREN WITH SPECIAL REFERENCE TO LIPID PROFILE AND THYROID FUNCTION

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ABSTRACT

Background: Idiopathic nephrotic syndrome (NS) in children is characterized by massive proteinuria, hypoalbuminemia, edema and hyperlipidemia[1,2]. Most pediatric cases (≈90%) are steroid-sensitive (often minimal-change disease), with only 10–20% steroid-resistant. Loss of albumin and thyroxine-binding globulin (TBG) in NS can lead to lipid abnormalities and thyroid dysfunction[5,7]. However, data on lipid profiles and thyroid status in different NS types are limited. This study aimed to evaluate the clinical features, lipid parameters and thyroid function in 100 children with NS, and to correlate these findings with disease course. **Materials and Methods:** In this prospective observational study (August 2022–July 2023), 100 children (age 6 months–12 years) with confirmed NS admitted to KGH, Visakhapatnam, were enrolled. Demographic and clinical data were recorded. Fasting serum lipid profile (total cholesterol, LDL, HDL, triglycerides) and thyroid function tests (T3, T4, TSH) were measured during relapse and in remission. Patients were classified as first-episode NS, frequently relapsing NS (FRNS) or infrequently relapsing NS (IFRNS) by ISKDC criteria. Laboratory means were compared by ANOVA and t-tests; categorical data by Chi-square; correlations by Pearson's test. A p-value<0.05 was considered significant. **Results:** Out of 100 children (mean age 4.8±2.6 years, 60% boys), 35% were first-episode NS, 26% FRNS and 39% IFRNS. Children <5 years comprised 60%; mean age was lower in FRNS vs first-episode (4.2 vs 5.1 yrs). Edema (facial/pedal) was universal, and common symptoms included abdominal distension (43%) and oliguria (29%). Serum albumin was significantly lowest in FRNS (mean 2.0 g/dL) compared to first-episode NS (2.8 g/dL; p=0.006). Mean total cholesterol was elevated in all groups (overall mean 359 mg/dL). FRNS had the highest cholesterol (396 mg/dL) vs first-episode (353 mg/dL) and IFRNS (328 mg/dL) (p=0.039). Similarly, LDL and triglycerides were highest in FRNS, while HDL was low in all. Hypothyroidism was common: overall, 24% of children had subclinical and 4% overt hypothyroidism, mostly in relapsing cases. In the active NS phase, 72% were euthyroid, 24% subclinical hypo, 4% hypothyroid; almost all normalized in remission. Serum albumin correlated inversely with cholesterol (r≈–0.5) and directly with TSH (p<0.01). **Conclusion:** Nephrotic children exhibited pronounced dyslipidemia and frequent thyroid abnormalities, especially in relapsing NS. Low serum albumin was associated with higher cholesterol and TSH. These findings underscore the need for routine lipid and thyroid screening in pediatric NS. Lipid abnormalities contribute to cardiovascular risk[4], and thyroid dysfunction (loss of hormones in urine) occurs in over half of NS cases[5]. Early identification and management of hyperlipidemia and hypothyroidism may improve outcomes in NS patients.[3,4]

INTRODUCTION

Nephrotic syndrome (NS) in children is defined by heavy (>40 mg/m²/hr) proteinuria leading to hypoalbuminemia, edema and secondary hyperlipidemia.[1,2] In pediatric practice, idiopathic NS accounts for ~90% of cases, the majority being minimal-change disease. Incidence is low (≈2–7

cases per 100,000 children per year).[1] NS typically presents between ages 2–7, with facial puffiness, generalized edema and ascites.[1] The hallmark biochemical abnormality is massive proteinuria with serum albumin <2.5 g/dL, which drives hepatic lipoprotein overproduction and lipid abnormalities.[1,7] Total cholesterol and LDL-cholesterol are classically elevated in NS,[3] while HDL may be normal or low. Dyslipidemia in NS increases atherogenic risk and may exacerbate glomerular injury.[3]

In addition, urinary loss of thyroid-binding proteins (thyroxine binding globulin, transthyretin) and albumin can lead to thyroid hormone loss in NS.[1] Many studies report frequent subclinical and overt hypothyroidism in NS. For example, Jung et al. found 51.6% of nephrotic children had thyroid abnormalities (8 overt, 8 subclinical out of 31). Loss of free hormone and binding proteins can impair thyroid status until remission.[5] Abnormal thyroid profiles have been linked to the degree of proteinuria and low albumin.

Despite these known associations, few large pediatric series have simultaneously assessed lipid and thyroid changes across NS subtypes. This study therefore examined the clinical spectrum, lipid parameters and thyroid function in children with first-episode vs relapsing NS. Our objectives were to characterize dyslipidemia and hypothyroidism in NS, and to identify correlations with albumin and disease course. We hypothesized that relapsing NS (FRNS) would have more severe hyperlipidemia and thyroid dysfunction than first-episode cases, due to sustained protein loss.

MATERIALS AND METHODS

Study design and patients: A prospective observational study was conducted in the Pediatric ICU and Nephrology Unit of King George Hospital, Visakhapatnam, from August 2022 to July 2023. Consecutive children (6 months–12 years) with idiopathic NS were enrolled after parental consent. NS was diagnosed by standard criteria (proteinuria ≥3+ on dipstick or ≥50 mg/kg/day). Exclusion criteria included secondary NS (e.g. lupus, infection, diabetes), steroid-resistant NS, chronic kidney disease, or other causes of edema. The study was approved by the institutional review board.

Clinical classification: Patients were categorized by disease course: first-episode NS, frequently relapsing NS (FRNS: ≥2 relapses in 6 months or ≥4 in 12 months), and infrequently relapsing NS (IFRNS). Baseline data included age, sex, and clinical features (edema, oliguria, ascites, etc.). Height, weight and blood pressure were recorded.

Laboratory measurements: At admission (active disease), fasting blood samples were collected for serum creatinine, albumin, total protein, total cholesterol, LDL-C, HDL-C, triglycerides, and thyroid hormones (total T3, total T4, free T4, TSH). Serum albumin was measured by bromocresol green method. Lipid profile was assayed enzymatically. Thyroid function tests were by chemiluminescent immunoassays. In cases achieving remission during the study period (n≈80%), repeat thyroid tests were obtained 4–6 weeks after remission (defined as normalization of proteinuria). All assays used hospital laboratory reference ranges.

Statistical Analysis: Data were analyzed using SPSS v25. Continuous variables were tested for normality. Means (±SD) were compared by Student's t-test or ANOVA; categorical variables by Chi-square or Fisher's exact test. Correlation between albumin and lipid/TSH was assessed by Pearson's or Spearman's correlation as appropriate. A p-value <0.05 was considered statistically significant. Tables and figures were prepared to summarize demographic, clinical, lipid, and thyroid data.

RESULTS

Patient characteristics: One hundred children with NS were studied. The mean age was 4.8±2.6 years (range 0.6–12), and 60 (60%) were boys (M:F=1.5:1). Age distribution showed 2% <1 yr, 58% between 1–5 yrs, 34% between 5–10 yrs, and 6% >10 yrs; mean age did not differ significantly by NS type (p>0.1). Thirty-five children (35%) were first-episode NS, 26 (26%) were FRNS, and 39 (39%) IFRNS. (Note: in relapsed patients, FRNS accounted for 40% of relapses and IFRNS 60%, by definition.)

Clinical profile: All children had edema (100% facial/pedal edema). The most common additional symptom was abdominal swelling (ascites) in 43%, followed by oliguria (29%), dysuria (27%) and abdominal pain or cough/fever (each 21%). Hypertension was relatively uncommon (16%) and anemia (pallor) in 42%. Clinical signs included ascites (43%), pallor (42%), pleural effusion (30%), generalized anasarca (21%) and hypertension (16%). These features align with prior reports that edema and effusions are typical in pediatric NS.

Laboratory findings – Albumin: The overall mean serum albumin was 2.4±0.9 g/dL (normal 3.5–5.0). Mean albumin differed by NS type: FRNS patients had the lowest mean (2.0±0.90), vs first-episode NS (2.8±1.02) and IFRNS (2.4±0.90) (p=0.006 by ANOVA). Thus, relapsing NS was associated with more severe hypoalbuminemia. We also examined complications: children with albumin <1.5 g/dL were significantly more likely to have ascites (p=0.006) and anasarca (p=0.006) than those with higher albumin.

Lipid profile: Hyperlipidemia was universal. Overall, mean total cholesterol was 359±110 mg/dL. By NS subtype, FRNS patients had significantly higher cholesterol (mean 396±128) than first-episode NS (353±103) or IFRNS (328±87) (ANOVA p=0.039). Similarly, mean LDL-C and triglycerides were highest in FRNS (LDL ~260 mg/dL, TG ~180

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mg/dL) and lowest in IFRNS, while HDL-C was reduced in all groups. The total cholesterol level correlated inversely with serum albumin (r≈–0.50, p<0.01). In fact, children with albumin <1.5 g/dL had mean cholesterol ≈510 mg/dL, versus ~370 mg/dL if albumin 1.6–2.0 and 326 mg/dL if 2.1–2.5 (p=0.003). This inverse albumin–cholesterol relationship, noted over 40 years ago, was confirmed: lower albumin predicted higher lipids. Dyslipidemia was also more severe in relapsing NS. These findings agree with literature that NS causes marked elevation of total and LDL cholesterol, especially in relapse, and that lipid levels only normalize when proteinuria remits.

Thyroid function: Thyroid abnormalities were common during the nephrotic phase. At presentation, mean serum free T4 was 1.0±0.3 ng/dL (normal 0.8–1.8) and TSH was 6.1±3.2 µIU/mL (normal 0.5–5.0). Overall, 28 children (28%) had subclinical hypothyroidism (elevated TSH with normal T4) and 4 (4%) had overt hypothyroidism (TSH elevated, T4 low). The distribution by NS type showed FRNS had the majority of cases: of the 24 subclinical cases, 15 were FRNS, 6 first-episode, 3 IFRNS; of the 4 overt cases, 2 were FRNS, 1 first-episode, 1 IFRNS. In the nephrotic (active) phase, 72% of patients were euthyroid, 24% subclinical and 4% overt. By contrast, after remission (in ~80 patients), 96% were euthyroid and only 4% remained hypothyroid (overt)—none had subclinical disease. Thus, most thyroid disturbances resolved with remission of NS. Serum albumin again showed correlation: lower albumin was associated with higher TSH (albumin <1.5 g/dL mean TSH 7.52 µIU/mL vs albumin 2.1–2.5 g/dL mean TSH 4.94 µIU/mL; p=0.003) (Table 11). These results confirm that proteinuria in NS can cause urinary loss of thyroid hormones and binding proteins[5], leading to hypothyroidism that often normalizes after remission.

Figures and Tables: The main results are summarized in Tables 1–3 (see below) and Figures 1–3. Table 1 shows demographics and albumin by NS type. Table 2 shows lipid parameters (cholesterol, LDL, HDL, triglycerides) by group. Table 3 shows thyroid status by NS type. Figure 1 (bar chart) illustrates the frequency of first-episode, FRNS and IFRNS. Figure 2 plots mean serum cholesterol in each NS group. Figure 3 compares thyroid status (euthyroid, subclinical hypo, overt hypo) during active disease vs remission.

Table 1: Course of nephrotic syndrome in relation to serum albumin

Type of nephrotic syndrome	Mean serum albumin (g/dL)	SD	p value
First episode	2.8	1.021	0.006
FRNS	2.0	0.9024	
IFRNS	2.4	0.9006	
Total	**2.4**	—	—

FRNS: Frequently relapsing nephrotic syndrome
IFRNS: Infrequently relapsing nephrotic syndrome

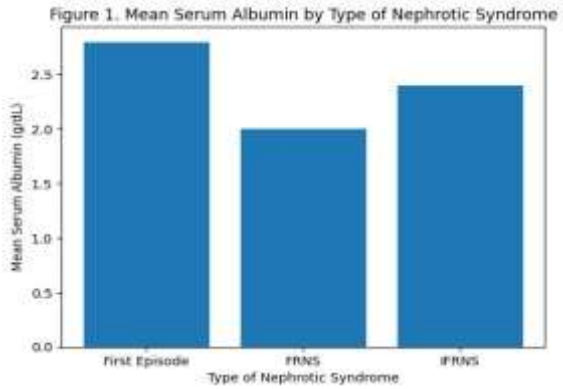
Table 2: Course of nephrotic syndrome in relation to serum cholesterol

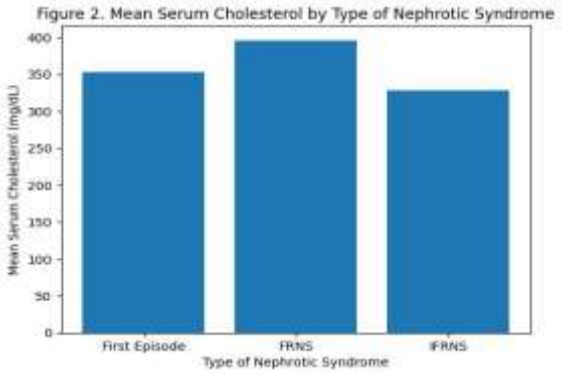
Type of nephrotic syndrome	Mean serum cholesterol (mg/dL)	SD	p value
First episode	353	102.5607	0.0399
FRNS	396	127.6928	
IFRNS	328	86.9831	
Total	**359**	—	—

FRNS: Frequently relapsing nephrotic syndrome
IFRNS: Infrequently relapsing nephrotic syndrome

Table 3: Thyroid function according to disease phase among nephrotic children

Thyroid status	Active phase n (%)	Remission phase n (%)
Euthyroidism	72 (72%)	96 (96%)
Subclinical hypothyroidism	24 (24%)	0 (0%)
Hypothyroidism	4 (4%)	4 (4%)





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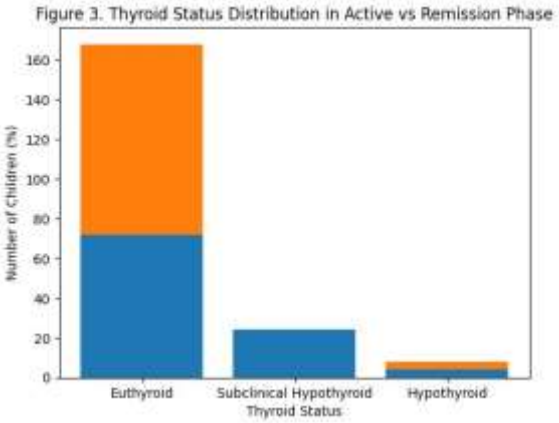

DISCUSSION

This study confirms that nephrotic syndrome in children is associated with significant dyslipidemia and thyroid abnormalities, especially in relapsing cases. The classic finding of hypercholesterolemia and hypoalbuminemia in NS,[1,2] was replicated: 100% of our patients had hyperlipidemia, and mean serum albumin was very low (2.4 g/dL). We observed that FRNS children (multiple relapses) had more severe hypoalbuminemia (mean 2.0 g/dL) and correspondingly higher lipids than first-episode cases. The inverse albumin–cholesterol relationship (Pearson $r \approx -0.5$, $p<0.01$) is well documented,[7] and reflects the liver's compensatory overproduction of lipoproteins in hypoalbuminemia. Our FRNS patients had the highest lipids (total cholesterol ~396 mg/dL) similar to previous reports of relapsing NS. Notably, dyslipidemia remained marked until remission, consistent with other series. HDL cholesterol was low in most patients, removing a cardioprotective factor. These patterns are of clinical importance, as prolonged hyperlipidemia in childhood NS is a risk factor for premature atherosclerosis.[3,4]

Thyroid dysfunction in NS is increasingly recognized. Proteinuria can lead to urinary loss of T4 and TBG, causing "nephrotic hypothyroidism".[5] We found 28% of patients with subclinical and 4% with overt hypothyroidism during relapse, a rate comparable to other studies (50–70% with any abnormality.[5] Importantly, the vast majority normalized after proteinuria resolved (remission), as reported by Jung et al.[5] This implies that many thyroid abnormalities in NS are transient consequences of protein loss, rather than intrinsic thyroid disease. We also found a strong albumin–TSH correlation (higher TSH with lower albumin), reflecting that worse proteinuria yielded more hormone loss. From a management perspective, these data suggest screening thyroid levels in relapsing NS, and treating with levothyroxine if persistent hypothyroidism is documented.[5]

Our findings align with existing literature: Pediatric NS is characteristically idiopathic (mostly minimal-change) and steroid-responsive. In general, ~80–90% of children achieve remission with corticosteroids. Our cohort's distribution (35% first-episode, 65% relapsing [26% frequent, 39% infrequent]) reflects a tertiary-care referral bias toward complicated cases. The Male:Female ratio of 1.5:1 and peak age of 2–7 years are consistent with known epidemiology.[1,6]

Limitations: Being a single-center study of 100 patients, generalizability is limited. We did not include steroid-resistant cases or compare treatment outcomes. We also lacked a control group of healthy children or NS patients in long-term remission. Future studies should include larger multicenter cohorts and longer follow-up to assess cardiovascular endpoints.

CONCLUSION

In summary, children with idiopathic nephrotic syndrome exhibit marked dyslipidemia and frequent thyroid hormone abnormalities during active disease, especially in relapsing cases. Low serum albumin correlates with higher cholesterol and TSH levels. Given these findings, routine monitoring of lipid profiles and thyroid function is recommended in pediatric NS. Early management of hyperlipidemia (e.g. dietary measures, statins) and hypothyroidism may reduce long-term morbidity. Further research should address whether correcting these metabolic derangements improves outcomes in childhood NS.

REFERENCES

1. National Institute of Diabetes and Digestive and Kidney Diseases. (n.d.). Nephrotic syndrome in children. U.S. Department of Health and Human Services. https://www.niddk.nih.gov/health-information/kidney-disease/children/nephrotic-syndrome-children. (Accessed 2026)
2. Tapia C, Bashir K. Nephrotic Syndrome. [Updated 2023 May 29]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK470444/
3. Hassan Thabet, M.A.E., Salcedo, J.R. & Chan, J.C.M. Hyperlipidemia in childhood nephrotic syndrome. Pediatr Nephrol 7, 559–566 (1993). https://doi.org/10.1007/BF00852550.
4. Dowerah P, Gogoi A, Shira CD, Sarkar B, Mazumdar S. A study of dyslipidemia and its clinical implications in childhood nephrotic syndrome. Cureus 2023;15(10):e47434. doi:10.7759/cureus.47434.
5. Sun Hee Jung, Jeong Eun Lee, Woo Yeong Chung. (2019). Changes in the thyroid hormone profiles in children with nephrotic syndrome. Korean Journal of Pediatrics 62(3):85-89. https://doi.org/10.3345/kjp.2018.06891
6. Agrwal, Shipra & Mantan, Mukta & Dabas, Aashima & Batra, VineetaV. (2022). Childhood Steroid-resistant Nephrotic Syndrome: Long-term Outcomes from a Tertiary Care Center. Indian Journal of Nephrology. 32. 10.4103/ijn.ijn_258_21.
7. Edgard E. Delvin, Aicha Merouani, Emile Levy. (2003). Dyslipidemia in pediatric nephrotic syndrome: causes revisited. Clinical Biochemistry. 36(2). 95-101. https://doi.org/10.1016/S0009-9120(02)00433-2.

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