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Evaluation of Hyperlipidemia in Nephrotic Syndrome Patients Especially the High-Density Lipoprotein

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ABSTRACT

Background: Lipid metabolism impairment leading to dyslipidemia is almost always present in nephrotic syndrome patients. The elevation of serum cholesterol, LDL, and triglyceride have been recognized and studied. However, the association between different nephrotic syndrome histological types and dyslipidemia is not well studied, in addition to an unclear function of HDL in nephrotic syndrome patients.

Aim: Highlight the lipid abnormality of primary nephrotic syndrome especially HDL level in our community.

Methods: 40 participants included in an observational cross-sectional study in the Nephrology Department of the Ibn-Sina Teaching Hospital in Mosul city. The study conducted from October 2024 to October 2025. Inclusion criteria involved 18 years or older with a confirmed diagnosis of primary nephrotic syndrome. The patients were assessed by clinical, biochemical, lipid profile, and renal biopsy.

Results: The number of patients who had primary focal segmental glomerulosclerosis (FSGS) was 17(42.5%), and 14(35%) had primary membranous nephropathy (MN), and 9(22.5%) had other histological types. The number of patients who had normal or low HDL levels were 29, while the number of patients who had high HDL levels was 11. The high HDL levels group has 5 patients with FSGS, 3 patients with MN, and 3 patients with other histological subtypes. Both the high HDL and the low or normal HDL groups share the same characteristics apart from the difference with regards to B. urea, UPCR and of course HDL levels. The difference between the high HDL and the low or normal HDL groups with regards to the values of UPCR was significant with P-value of 0.01. The low or normal HDL group showed higher UPCR (3902.95± 2511.29mg/dl) compared with the high HDL group (1761.71± 759.37mg/dl).

Conclusion: Dyslipidemia in nephrotic syndrome (NS) is a very common feature, marked by high cholesterol, triglycerides, LDL, VLDL, and lipoprotein, driven by the liver overproducing lipids due to low albumin and impaired clearance. The most common histologic subtype of primary nephrotic syndrome is FSGS. HDL levels are elevated in minority of patients with nephrotic syndrome who probably develop less severe form of nephrotic syndrome.

INTRODUCTION

Background

Nephrotic syndrome is a common disease in both the pediatric and adult populations with consistent features which include oedema, proteinuria, and hypo-albuminaemia. (1)

The reported incidence of nephrotic syndrome is 2 to 7 new cases and prevalence of 16 cases per 100,000 children, and the annual incidence in adults is about 3 new cases per 100,000. The prevalence of nephrotic syndrome in adults remains a challenge to accurately estimate due to the fact there is usually an underlying cause and overlapping features. (2-5)

The response to initial management by steroid is good with the majority of children and adults will have clinical remission, however, a significant proportion (20% of children and 50% of adults) of the patients with nephrotic syndrome will have steroid resistance requiring a second line of management and perhaps prolonged treatment. The persistence of nephrotic syndrome subjects the patients to the long-term complications of nephrotic syndrome which

may arise from the prolonged course of nephrotic syndrome or as results of toxicity of drugs used to control nephrotic syndrome. [6,7]

Lipid metabolism impairment leading to dyslipidemia is almost always present in nephrotic syndrome patients. The elevation of serum cholesterol and triglyceride have been recognized and studied. However, the association between different nephrotic syndrome histological types and dyslipidemia is not well studied, in addition to the unclear function of HDL in nephrotic syndrome patients. [8]

Histologic Patterns and Features of Primary Nephrotic Syndrome

The histological pattern of nephrotic syndrome differs according to age, sex, race and geographic location. There has been reported difference in the trending pattern recently. [9]

Focal segmental glomerulosclerosis (FSGS):

The key histological feature is sclerosis and hyalinosis of segments in less than 50% of all glomeruli on electron microscopy. The patient may have hypertension, hematuria, elevated renal indices, and renal insufficiency. [9-15]

Membranous nephropathy (MN):

The key histological feature is thickening of the glomerular basement membrane on electron microscopy; immunoglobulin G and C3. The patients are usually at the third to fifth decade, microscopic hematuria, underlying systemic disease present in 25%. [9-15]

Minimal change disease (MCD):

The key histological feature is normal appearing glomeruli in microscopy, effacement of foot processes on electron microscopy. The course of the disease is usually benign and usually following upper respiratory tract infection. [9-15]

Pathophysiology of dyslipidemia in nephrotic syndrome

The exact pathophysiology remains uncertain, however, the proposed cause of dyslipidemia in nephrotic syndrome patients might be explained by the increased production and reduction in the clearance of lipoproteins as indicated by the elevated levels of total serum cholesterol and low-density lipoprotein (LDL) cholesterol as the consistent finding in dyslipidemia in nephrotic syndrome patients. Triglyceride levels that are raised, increased concentrations of apolipoprotein B, apolipoprotein C, and apolipoprotein E, and decreased levels of apolipoprotein A-I and apolipoprotein A-II are all possible characteristics of hyperlipoproteinemia. There are a number of mechanisms that are somewhat unknown that are associated with proteinuria, hypoalbuminemia, and probably an enhanced availability of mevalonate as a substrate for cholesterol synthesis. These mechanisms are responsible for the increased lipoprotein synthesis. Decreased activity of lipolytic enzymes and reduced clearance of cholesterol- and triglyceride-rich lipoproteins of lower densities, as well as an altered composition of high-density lipoprotein (HDL) may be the result of urinary loss of high-density lipoprotein (HDL) components and other lipo-regulatory variables. One possible explanation for the related variation in lipoprotein profiles among individuals diagnosed with nephrotic syndrome is that these two metabolic disorders are characterized by a degree of variability. Fig.(1) [16]

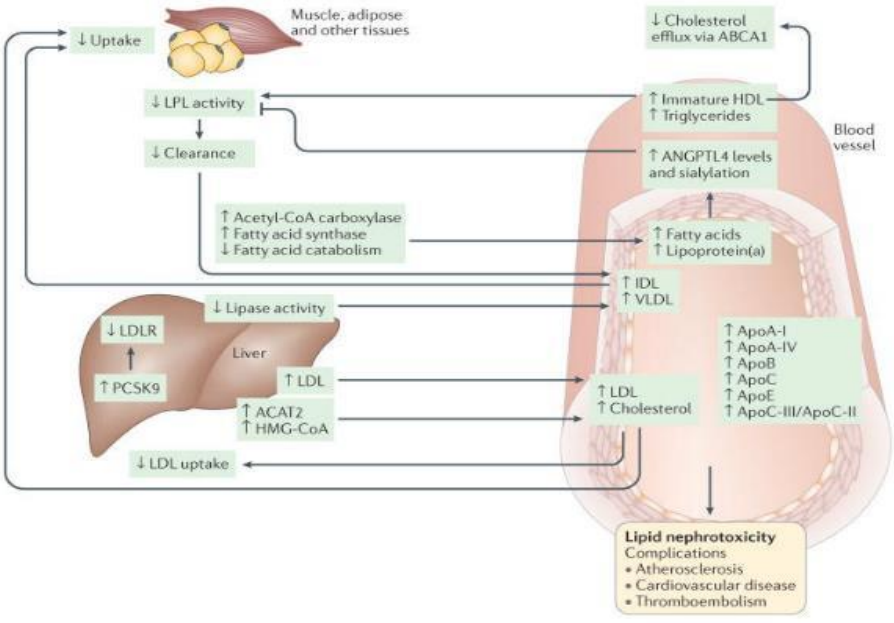

Fig 1. Pathophysiology of dyslipidemia in nephrotic syndrome. [16]

The role of LDL and HDL

Both the heightened production and reduced breakdown of LDL contribute to the elevated levels of LDL and cholesterol seen in individuals with nephrotic syndrome. Additionally, an increased expression of proprotein convertase subtilisin/Kexin type 9 (PCSK9) leads to greater degradation of the LDL receptor, resulting in diminished LDL uptake by the liver. [17] Genetic deletion of podocytes in mice, as well as exposure to nephrotoxic serum, induces hypercholesterolemia and raises PCSK9 levels. [18] Notably, specific deletion of PCSK9 in hepatocytes protects mice from hyperlipidemia triggered by nephrotoxic serum. [19] Moreover, patients experiencing active nephrotic syndrome exhibit higher plasma concentrations of both cholesterol and PCSK9, which have been observed to normalize during disease remission. [20]

Hypercholesterolemia and elevated LDL levels are associated with the increased expression and activity of liver acetyl-CoA acetyltransferase 2 (ACAT2), leading to greater cholesterol esterification and a decrease in intracellular free cholesterol levels. [21] Additionally, studies indicate that cholesterol synthesis via 3-hydroxy-3-methylglutaryl-CoA (HMG-CoA) reductase is heightened in experimental models of nephrotic syndrome. [22] Furthermore, there is evidence suggesting that lipoprotein(a) [23] may enhance LDL oxidation in nephrotic syndrome, with its levels being notably elevated in this condition. [24]

Patients diagnosed with nephrotic syndrome may exhibit varying levels of HDL cholesterol, though it is common for the ratio of HDL cholesterol to be lower. [25] HDL serves as the primary lipoprotein responsible for facilitating the efflux of cholesterol from peripheral tissues and transporting it to hepatocytes, where it is transformed into bile acids. The main function of HDL is to encourage cholesterol efflux, mainly through the action of membrane-associated transporters such as ATP-binding cassette subfamily A member 1 (ABCA1) and ATP-binding cassette subfamily G member 1 (ABCG1). [26] Beyond its role in reverse cholesterol transport, HDL possesses antioxidant functions, anti-inflammatory effects, and helps prevent endothelial dysfunction by interacting with scavenger receptor B1 to stimulate endothelial nitric oxide synthase. [27-29]

The levels of high-density lipoprotein (HDL) can be low, normal or high with a significant change in the composition of the HDL molecule. Specifically, there is a reduction in the cardioprotective HDL2 fraction and an increase in the cholesterol-poor HDL3 particles. This disruption in the maturation process of HDL from lipid-poor to cholesterol-rich forms, combined with compromised function of lecithin-cholesterol acyltransferase (LCAT) and altered activity of cholesteryl ester transfer protein (CETP), elevates the risk of cardiovascular disease, even when HDL levels remain normal or diminished. [30]

Clinical consequences of dyslipidemia

The clinical presentations of dyslipidemia in nephrotic syndrome are varied in general and may differ from patient to patient. The major impact of dyslipidemia in nephrotic syndrome is the acceleration of atherosclerosis, which may lead to various clinical consequences including myocardial infarction and stroke. Additionally, there has been established that dyslipidemia has a causative role in the elevated risk of thrombosis in nephrotic syndrome. Therefore, dyslipidemia is considered as one of the major risk factors for the development of atherothrombotic disorders. Hyperactive platelets is usually accompanied atherosclerosis which may lead to the elevated risk of thrombosis. [31] Furthermore, the products of LDL oxidation enhance platelet activation and thrombus formation. [32] Dyslipidemia in the context of nephrotic syndrome is notably linked to a heightened risk of nephrotoxicity, which may present as advancing kidney disease. This progression could arise from glomerulosclerosis, resulting from damage to podocytes and/or the proliferation of mesangial cells, along with injury to proximal tubular cells. [33]

Aim of the study:

Highlight the lipid abnormality of primary nephrotic syndrome especially HDL level in our community.

PATIENTS AND METHODS

Study design & setting

- Cross-sectional study
- Observational study
- Nephrology Department of the Ibn-Sina Teaching Hospital in Mosul city.
- The study conducted from October 2024 to October 2025.
- Number of participants: 40

Official approvals & statistical analysis

The approval was obtained from the Scientific Council of Medicine of the Arab Board of Health Specializations.
Informed consent was obtained from the participants with a clear explanation of the study.

The descriptive statistical analysis (mean, standard deviation, and others) was used to describe the data obtained from the participants including social, demographic, biochemical, lipid profile, and histopathological results.

Both independent t-test and chi-square test were used to assess the statistical significance between both groups; all analysis was carried out using SPSS version 31.0. The P-value was considered to be significant if <0.05.

Inclusion criteria
Eighteen years or older with a confirmed diagnosis of primary nephrotic syndrome

Exclusion criteria
1. Less than 18 years old
2. CKD
3. Nephrotic syndrome secondary to other causes

Assessment
The assessment of the patients included the demographic and social data which included:
1. Age
2. Sex
3. BMI
4. Smoking status

The disease specific data and past medical history were obtained and included:
1. Onset
2. Duration
3. Predisposing factors
4. Comorbidities

A detailed physical examination was conducted to assess the following:
1. Vital signs including blood pressure, respiratory rate, heart rate, and temperature
2. General appearance including pallor, jaundice, and nutrition status
3. Edema assessment which was graded from 0 to 4+
4. Abdominal examination to document distension, ascites, or flank fullness
5. Cardiovascular examination to document any signs of pleural effusion or fluid overload
6. Any signs of steroid related physical findings

The patients were assessed by a standard laboratory analysis which included the following:
1. Renal function test (B. urea and S. creatinine) to calculate the estimated glomerular filtration rate (eGFR) using the CKD-EPI equation.
2. Urine protein to creatinine ratio (UPCR) to quantify albuminuria in a spot urine sample
3. Serum albumin to determine hypalbuminemia
4. Other laboratory tests as indicated such as S. electrolytes or blood glucose
5. Lipid profile including total cholesterol, HDL, LDL , VLDL, and triglycerides

The diagnosis of the histological subtype was confirmed by histopathological examination of the tissue obtained during renal biopsy. The histopathological examination was performed in the same lab by the same renal histopathologist for all the patients. The histopathologist used light microscopy, immunofluorescence, and electron microscopy. The quality and adequacy (sufficient glomeruli, preservation of the architecture of the tissue) of the renal biopsy was recorded.

Reference ranges and units used
B. urea: 15–40 mg/dL
S. creatinine: 0.7–1.3 mg/dL for adult men and 0.5–1.1 mg/dL for adult women
Serum albumin: 35 to 50 g/L

UPCR:
- Normal (Normoalbuminuria): < 150 mg/g
- Moderate (Microalbuminuria): 150–499 mg/g
- High (Macroalbuminuria/Proteinuria): ≥500 mg/g

Lipid profile
Total cholesterol:
- < 200 mg/dL: Desirable/Normal
- 200–239 mg/dL: Borderline High
- ≥ 240 mg/dL: High

LDL

- < 100 mg/dL: Optimal
- 100–129 mg/dL: Near Optimal/Normal

HDL

- ≥ 60 mg/dL: Best (Protective)
- < 40 mg/dL (Men) / < 50 mg/dL (Women): Low/Increased Risk

VLDL

- Normal/Healthy: Below 30 mg/dL (2-30 mg/dL).
- High/Increased Risk: 30 mg/dL or above.

Triglycerides

- Normal — Less than 150 mg/dL
- Borderline high — 150 to 199 mg/dL
- High — 200 to 499 mg/dL
- Very high — 500 mg/dL or above

RESULTS

The study included 40 patients who were presented with primary nephrotic syndrome confirmed by clinical, laboratory, and renal biopsy. showen in table 1.

Table 1. Study group demographic, clinical, and biochemical data

Index	Number
Patients	40
Age	35.89 ± 16.57 years
Sex	
Male	26(65%)
Female	14(35%)
Hypertension	5(12.5%)
Serum Albumin	27± 8.63 g/L
UPCR	3367.64 ± 2394.53 mg/g
B. Urea	49.28 ± 22.04 mg/dL
S. Creatinine	1.26 ± 0.78 mg/dL
Total Cholesterol	301.64± 117.45 mg/dL
Triglyceride	275.39± 152.28 mg/dL
HDL	52.32± 14.10 mg/dL
LDL	170.5± 96.47 mg/dL
VLDL	54.10± 31.26 mg/dL

The number of patients who had primary focal segmental glomerulosclerosis (FSGS) was 17(42.5%), and 14(35%) had primary membranous nephropathy (MN), and 9(22.5%) patients had other histological types. The type of nephrotic syndrome according to the renal biopsy performed to all the participants is shown in table 2.

Table 2. The type of nephrotic syndrome based on renal biopsy and demographic characteristics.

Characteristic	**FSGS**	**MN**	**Other**
Number (%)	17(42.5%)	14(35%)	9(22.5%)
Age(mean)	34.92± 15.68	41.33±17.02	33.5± 16.85
Sex			
Male	12	8	6
Female	5	6	3

Several blood, urine tests, and lipid profile were performed to see any correlation with specific subtypes of primary nephrotic syndrome as shown in table 3 and 4.

Table 3. The type of nephrotic syndrome based on renal biopsy and biochemical results

Characteristic	FSGS (n=17)	MN(n= 14)	Other (n= 9)	P-value
Blood urea	51.57±22.58	63.83±20.13	34.37±10.65	0.005
Serum creatinine	1.44± 0.98	1.43±0.37	0.81±0.23	0.07
Serum Albumin	27.64± 8.15	26.66± 10.73	26.12±7.54	0.90
UPCR	3630.85± 2703.46	3384.64 ± 1716.22	2894.25 ± 2619.71	0.75

Table 4. Correlations between baseline lipid levels and primary nephrotic syndrome type

Lipid profile	FSGS (n=17)	MN(n= 14)	Other (n= 9)	P-value
Cholesterol	316.5±122.47	251.83±113.56	313±99.92	0.26
LDL	178.64± 116.96	133.32± 51.17	184.12±91.77	0.31
VLDL	52.92± 28.81	68.16± 49.52	45.62± 18.98	0.30
HDL	52.5± 8.91	44.66± 18.72	57.75 ± 17.71	0.12
Triglyceride	270.28 ±133.04	349.67±218.55	228.62±89.44	0.19

The number of patients who had normal or low HDL levels were 29, while the number of patients who had high HDL levels was 11. The high HDL levels group has 5 patients with FSGS, 3 patients with MN, and 3 patients with other histological subtypes.

Both the high HDL and the normal or normal HDL group share the same characteristics apart from the difference with regards to B. urea, UPCR and of course HDL levels.

The difference between the high HDL and the normal or low HDL group with regards to the values of blood urea was significant with P-value of 0.03. The normal or low HDL group showed high blood urea levels (53.19± 23.21 mg/dl) compared with the high HDL group (37.57± 11.96 mg/dl).

The difference between the high HDL and the normal or low HDL group with regards to the values of UPCR was significant with P-value of 0.01. The normal or low HDL group showed higher UPCR (3902.95± 2511.29 mg/dl) compared with the high HDL group (1761.71± 759.37 mg/dl).showen in table 5.

Table 5. Comparison between the high HDL level group and the low or normal HDL level group with regards to age, sex, S. Creatinine, UPCR, cholesterol, LDL, VLDL, and triglyceride

Index	High HDL	Normal or Low HDL	P-value
Age	35.57 ± 7.34	35.32±17.39	0. 48
Sex			
Male	7	19	0.79
Female	4	10	
B. Urea	37.57± 11.96	53.19± 23.21	0.041
S. Creatinine	0.89± 0.23	1.38± 0.85	0.07
S. Albumin	29.71± 7.70	26.09± 8.74	0.17
UPCR	1761.71± 759.37	3902.95± 2511.29	0.01
Cholesterol	307±121.95	299.61±73.56	0.44
HDL	69.57±6.82	46.57±10.83	<0.001
LDL	152.28±54.19	176.57±106.21	0.58
VLDL	54±21.81	54.14±33.82	0.49
Triglyceride	253±90.57	282.84 ±167.22	0.29

DISCUSSION

Dyslipidemia in nephrotic syndrome is a common finding with variable and significant implications that may increase the risk of morbidity and mortality in patients with nephrotic syndrome. The less studied aspect of dyslipidemia in nephrotic syndrome is the level and composition of HDL in patients with nephrotic syndrome. [16,30]

In this study, the number of patients who had primary focal segmental glomerulosclerosis (FSGS) was 17(42.5%), and 14(35%) had primary membranous nephropathy (MN), and 9(22.5%) patients had other histological types. The predominance of FSGS is the emerging most common histopathological subtype in different studies. The other finding in this study is the higher percentage of male patients which is consistent with other epidemiological studies. [35-38]

The finding of dyslipidemia in patients with nephrotic syndrome can by the result of hypoalbuminemia, which leads to the increased synthesis of hepatic lipoproteins and increased both cholesterol and triglycerides as a compensatory mechanism. Another theory involves the reduction of the clearance of lipoprotein as a result of the impairment of activity of the lipoprotein lipase enzyme or the dysregulation of the LDL receptor in the liver. The levels of HDL in patients with nephrotic syndrome is usually low or normal, and the HDL to cholesterol ratio is decreased with impairment of the maturation of cholesterol ester-rich HDL. However, a significant number of patients with nephrotic syndrome have high levels of HDL that can be explained by the result of the low albumin level that is required for free cholesterol transfer from the peripheral tissue to HDL. [16,30]

Dyslipidemia affects the cardiovascular system, and it is a well recognizable etiology in the development of cardiovascular disease. Therefore, understanding the clinical and biochemical presentations of dyslipidemia is critical in the management of nephrotic syndrome patients. [41]

The key aspect of dyslipidemia in nephrotic syndrome is dysfunctional HDL. The level of HDL is variable in nephrotic syndrome and even if the level of HDL is high, it does not exert its protective properties properly. [16,30]

In this study, individuals with high HDL levels differed from those with normal or low HDL in several key metabolic and renal parameters. Notably, the high-HDL group had significantly lower B. urea and a markedly lower UPCR, suggesting better renal handling or hydration status in this subset. Additionally, the serum creatinine in the high-HDL group is lower than the normal or low HDL group. These differences may be explained by the increased severity of nephrotic syndrome in the normal or low HDL group when compared with the high-HDL group.

Alqudsi et al. compared lipid profiles in 125 nephrotic-syndrome patients (FSGS, MCD, MN) and found that at baseline — after adjusting for serum albumin and proteinuria — total cholesterol and triglycerides were similar across groups, but LDL was higher in FSGS than MN ($p = 0.04$) and HDL was markedly higher in MCD than in FSGS or MN (both $p \leq 0.001$). After 12 months of follow-up, however, lipid values became comparable across all three groups regardless of statin use, suggesting that the baseline subtype-related differences resolve over time, likely as the underlying nephrotic state improves rather than as a specific response to statin therapy. [42]

The significant difference in BUN implies some divergence in kidney function or urea handling among disease groups. Since creatinine did not differ, the BUN difference might reflect factors such as hydration status, catabolism, or differential protein intake, rather than absolute renal filtration capacity. Clinically, this warrants a look at volume status, dietary protein, and concomitant therapies across groups. [43]

Higher HDL is often part of a broader favorable metabolic profile and has anti-inflammatory and endothelial-protective properties, which may translate to improved renal handling or hydration status. Conversely, elevated urea in the normal/low HDL group might indicate reduced renal clearance, higher protein catabolism, or confounding factors such as dehydration, differential hydration status, or diuretic use. [44]

The findings of this study raise the question of the role of the elevated HDL in the protection of the renal function or if the elevation is associated with less severe form of nephrotic syndrome.

LIMITATIONS OF THE STUDY

Although this study is unique in exploring the dyslipidemia in nephrotic syndrome specifically HDL role, it has several limitations that may affect the results of the study. The greater limitation of this study is the short duration and small number of participants that could impair the true estimation of the long-term complications in patients with dyslipidemia that usually require long time to develop. The HDL different subtypes testing was not included in this study. The confounding factors that may change the lipid profile and can affect the lipid profile in patients with nephrotic syndrome such as diet, drugs, alcohol, and exercise. The significant difference of B. urea may reflect a change in the protein intake and metabolism, and hydration status of the patients.

Conclusion

Dyslipidemia in nephrotic syndrome (NS) is a very common feature, marked by high cholesterol, triglycerides, LDL, VLDL, and lipoprotein, driven by the liver overproducing lipids due to low albumin and impaired clearance. The most common histologic subtype of primary nephrotic syndrome is FSGS. HDL levels are elevated in minority of patients with nephrotic syndrome who probably develop less severe form of nephrotic syndrome.

Recommendations

1. Larger and longer cohort studies to observe the long-term impact of the different HDL levels and the protective role of HDL in nephrotic syndrome
2. Evaluation of the different HDL subtypes in nephrotic syndrome and the correlation with the protective function against atherosclerosis
3. The consideration of other confounding factors in the development of atherosclerosis such smoking, obesity, and age.
4. The inclusion of different treatment options of nephrotic syndrome and its association with the development of dyslipidemia and HDL levels

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