Thyroid Dysfunction in Patients with Nephrotic Syndrome

Merna Makram Asham Yassa*, Mostafa Abdullah Mohamed Haridi, Ghada Abd El-Rahman Mohamed, Nashwa M.Abd Elmonem Azoz
Department of Internal Medicine, Faculty of Medicine, Assiut University, Assiut, Egypt.
Corresponding Author: Merna Makram Asham Yassa
E-mail: *merna.makram622@yahoo.com*

Abstract:

Background: The relationship between thyroid dysfunction and nephrotic syndrome (NS) is complex, with limited data on the prevalence of various types of thyroid disorders and their association with the pathological characteristics of NS.

Aim of work: To evaluate the thyroid hormone profile in NS patients and identify clinical predictors of thyroid dysfunction in this population.

Methods: This cross-sectional study included 150 NS patients (50 with normal thyroid function and 100 with thyroid dysfunction) at the Nephrology Unit of Assiut University Hospital between 2020 and 2022. Participants underwent comprehensive clinical evaluation and laboratory tests, including thyroid hormones and thyroid peroxidase antibodies. Renal histopathological types were also recorded.

Results: Patients with thyroid dysfunction had significantly higher mean age, creatinine, cholesterol, and low-density lipoprotein levels, as well as lower high-density lipoprotein levels and glomerular filtration rates compared to those with normal thyroid function. Thyroid peroxidase antibody levels were significantly higher in the thyroid dysfunction group.

($P < 0.001$). No significant differences were found between the groups regarding renal biopsy findings. Glomerular filtration rate and cholesterol levels were the only predictors of thyroid dysfunction in NS patients.

Conclusion: Thyroid dysfunction is prevalent in NS patients, and interpreting the interactions between thyroid and renal function is challenging. Close collaboration between endocrinologists and nephrologists is essential for accurately interpreting thyroid and kidney function changes in this patient population.

Keywords: Nephrotic syndrome; Thyroid dysfunction; Thyroid peroxidase antibodies; Renal histopathology.

Introduction:

Nephrotic syndrome (NS) is a complex disorder characterized by substantial proteinuria, hypoalbuminemia, edema, and hyperlipidemia. Endocrine abnormalities, particularly thyroid dysfunction, are frequently observed in NS patients.

The intricate relationship between the thyroid gland and the kidneys is well-established, with the thyroid playing a crucial role in kidney growth and function, while the kidneys are essential for the metabolism and elimination of thyroid hormones. The prevalence of

hypothyroidism in NS is higher compared to the general population, with subclinical cases being more common than overt hypothyroidism [1].

The pathophysiology of thyroid dysfunction in NS can be attributed to the loss of thyroid hormones and thyroid-binding globulin in the urine due to proteinuria, leading to alterations in thyroid hormone levels and increased thyroid-stimulating hormone (TSH) secretion [2]. Additionally, autoimmunity may play a role in the association between NS and thyroid dysfunction, as it can affect both organs simultaneously [2].

Objectives:

This study aimed to evaluate the thyroid hormone profile in NS patients and identify clinical predictors of thyroid dysfunction to better understand the relationship between these two conditions and provide more targeted care for affected patients.

Patients and Methods:

Clinical trial number: NCT043233007

Study Design and Setting

This cross-sectional study was conducted at the Nephrology Unit of the Internal Medicine Department, Assiut University Hospital, between 2020 and 2022.

Study Population

150 NS patients were enrolled, including 50 with normal thyroid function and 100 with thyroid dysfunction.

Inclusion and Exclusion Criteria

Patients with NS, defined by generalized edema, urine protein excretion > 3.5 g/24 hours, urine protein to creatinine ratio > 3,500 mg/g, and serum albumin level < 30 g/L [3] were included in the study. Patients aged < 18 years, those with thyroid diseases or a history of thyroid hormone or antithyroid drug intake, diabetes mellitus, autoimmune diseases such as systemic lupus erythematosus, liver cirrhosis, or chronic liver disease were excluded.

Sample Size Calculation

Based on a population size of 150 patients attending the study site (both inpatients and outpatients) in one year, an expected frequency of thyroid dysfunction of 82.0% [4], and a confidence level of 95%, the minimal sample size was calculated to be **90** patients.

Data Collection and Measurements

Eligible patients were subjected to the following preliminary evaluation

1. **Full history taking and clinical evaluation,** including measurement of BMI [weight divided by squared height (kg/m^2)].
2. **Vital sign examination:** including blood pressure (mmHg) measured twice in the upper arm after a 10-minute rest period and an average.
3. **Laboratory tests:** 3 venous blood samples were collected from all studied participants to evaluate:
   - Random blood glucose (by fully automated chemistry analyzer, Advia 1800_2).
   - Complete urine analysis for detection of red blood corpuscles and/or pus, volume (ml), gravity, and for 24 hours urinary proteins (by (fully automated chemistry analyzer, Advia 1800_2)
   - Complete blood count (CBC): The CELL-DYN Ruby analyzer was used for analysis.
   - Serum Lipogram (by fully automated chemistry analyzer, Advia 1800_2).
   - Kidney function tests [urea, creatinine, glomerular filtration rate (GFR)]: Advia 1800™, based on photometry, was used for analysis.
   - Liver function tests (total protein, albumin, total bilirubin, direct bilirubin, alkaline phosphatase, alanine transaminase, and aspartate transaminase): Advia 1800™ based on photometry was used for analysis.

- Thyroid function tests [triiodothyronine (fT3) and tetraiodothyronine (fT4), and serum concentration of thyroid-stimulating hormone (TSH)].
- Immunological markers (ANA and Anti-dsDNA) for suspected cases.
- Other investigations included antithyroperoxidase antibody (IU/mL) and renal biopsy.

Statistical Analysis

Data were analyzed using appropriate statistical methods to compare the characteristics of patients with normal thyroid function and those with thyroid dysfunction. Logistic regression analysis was performed to identify clinical predictors of thyroid dysfunction in NS patients.

Ethical Considerations:

- Approval from the Ethics of Scientific Research Committee, Faculty of Medicine, Assiut University, was obtained.
- Verbal and written consents were obtained from all the patient's caregivers.
- The privacy and confidentiality of all information obtained were observed without intervention in the prescribed treatment.

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Results

Baseline data of the studied groups (Table 1):

It was found that patients with thyroid dysfunction had a significantly higher mean age (33.13 ± 8.41 vs. 29.82 ± 7.69 (years); *p*= 0.021) in comparison to those with normal thyroid function, with no significant difference between both groups as regards sex (p= 0.356).

Baseline data among the studied groups (Table 2):

Both groups of patients with thyroid dysfunction and those with normal thyroid function had insignificant differences regarding baseline data, except that patients with thyroid dysfunction had significantly higher and lower GFR values.

Liver function tests and lipid profile among the studied groups (Table 3):

Patients with thyroid dysfunction had significantly higher cholesterol (139.76 ± 17.51 vs. 115.88 ± 33.17 (mg/dl); $p = 0.000$) and low-density lipoproteins (108.22 ± 11.12 vs. 97.02 ± 18.87 (mg/dl); $p= 0.000$) and significantly lower high-density lipoproteins (62.64 ± 13.30 vs. 79.52 ± 11.04 (mg/dl); $p = 0.000$).

Thyroid function tests among the studied groups (Table 4, Figure 1):

Patients with thyroid dysfunction had significantly higher TSH (15.61 ± 12.85 vs. 2.81 ± 1.16 (mIU/ml); $P = 0.000$) with significantly lower free T3 (2.70 ± 3.41 vs. 3.42 ± 0.65 (pg/ml); $p= 0.000$) and free T4 (0.86 ± 1.35 vs. 1.23 ± 0.30 (ng/dl); $p = 0.000$) in comparison to those with normal thyroid function.

Also, patients with thyroid dysfunction had significantly higher thyroid peroxidase antibodies (81.49 ± 26.95 vs. 32.24 ± 5.13 (IU/ml); $P= 0.000$) than those with normal thyroid function.

Ultrasound findings and renal biopsy among the studied groups (Table 5, Figure 2):

Most patients in both groups had echogenicity grade-I, and based on renal biopsy, the most frequent histopathological types in both groups were FSGN and MPGN. There were insignificant differences between the groups regarding ultrasound ($p = 0.813$) and renal biopsy ($p = 0.354$).

Predictors for thyroid dysfunction in patients with nephrotic syndrome (Table 6):

Based on the current study, the only predictors for thyroid dysfunction in patients with nephrotic syndrome were glomerular

filtration rate (odd's ratio = 2.22) and cholesterol (odd's ratio = 3.01).

Discussion

The complex interplay between thyroid hormones and renal physiology is well-documented, underscoring the critical role these hormones play not only in the developmental stages of renal architecture but also in the regulation of renal functions such as filtration, tubular reabsorption, and the maintenance of electrolyte equilibrium [5]. Conversely, renal processes are vital for the metabolic breakdown and excretion of thyroid hormones.

The link between diminished thyroid hormone levels and the manifestation of nephrotic syndrome is more pronounced than the connection to autoimmune thyroid conditions [6]. Despite the prevalence of anecdotal evidence linking hypothyroidism with nephrotic syndrome, there remains a significant gap in comprehensive, systematic research in this area. This study endeavors to bridge this gap by examining thyroid dysfunction within a cohort of nephrotic syndrome patients.

Our analysis revealed a notable difference in the average age between patients exhibiting thyroid dysfunction (33.13 ± 8.41 years) and those with normal thyroid function (29.82 ± 7.69 years; $p = 0.021$), suggesting an age-related predisposition to thyroid dysfunction in this patient population. Gender distribution showed no significant variance, with a slight male predominance (52%) in the thyroid dysfunction group and a female majority (56%) in the euthyroid group. These observations align with the findings of Keunmoe et al. [7], who analyzed a similar patient cohort without finding significant differences in gender distribution. However, our study presents a contrasting view of age-related differences. Further, our study highlighted that patients with thyroid dysfunction presented with elevated creatinine levels (94.56 ± 10.82 vs. 82.09 ± 19.14 umol/l; $p < 0.001$) and reduced GFR (95.90 ± 17.92 vs. 107.70 ± 19.91 ml/min/1.73m2; $p = 0.001$), corroborating the findings of Keunmoe et al. [7] and Li et al. [4]. However, Singh et al. [8] reported no significant differences in serum creatinine levels, possibly due to their smaller sample size.

The observed increase in creatinine among patients with thyroid dysfunction is likely attributable to elevated TSH levels, which inversely correlate with GFR. This significant relationship may involve decreased β-adrenergic responsiveness, reduced renin secretion, and a compromised RAAS [6]. Moreover, our findings indicate that patients with thyroid dysfunction also had significantly higher cholesterol (139.76 ± 17.51 vs. 115.88 ± 33.17 mg/dl; $p < 0.001$) and LDL (108.22 ± 11.12 vs. 97.02 ± 18.87 mg/dl; $p < 0.001$) levels, alongside lower HDL (62.64 ± 13.30 vs. 79.52 ± 11.04 mg/dl; $p < 0.001$) levels compared to their euthyroid counterparts, aligning with Li et al.'s research [4].

Patients with thyroid dysfunction typically exhibit elevated TC, TG, and LDL-C levels alongside reduced HDL-C levels. The influence of T3 on lipid metabolism, including its effect on gene expression for key enzymes, may explain these lipid profile changes. Additionally, this study noted significantly higher TSH levels (15.61 ± 12.85 vs. 2.81 ± 1.16 mIU/ml; $p < 0.001$) and lower free T3 (2.70 ± 3.41 vs. 3.42 ± 0.65 pg/ml; $p < 0.001$) and free T4 levels (0.86 ± 1.35 vs. 1.23 ± 0.30 ng/dl; $p < 0.001$) in the thyroid dysfunction group, consistent with prior studies [4].

Furthermore, significantly higher levels of thyroid peroxidase antibodies (81.49 ± 26.95 vs. 32.24 ± 5.13 IU/ml; $p < 0.001$) were observed in the thyroid dysfunction group, suggesting an autoimmune component, as supported by Jain et al. [6].

In the current study, most patients in both groups had grade I echogenicity on

ultrasound, and the most frequent histopathological types based on renal biopsy were focal segmental glomerulosclerosis (FSGN) and membranoproliferative glomerulonephritis (MPGN). There were no significant differences between the groups regarding ultrasound findings ($p = 0.813$) or renal biopsy results ($p = 0.354$), indicating that the type of nephrotic syndrome may not directly influence the occurrence of thyroid dysfunction. Few studies have investigated the association between thyroid dysfunction and various types of nephrotic syndrome. A retrospective study on 28 patients with Hashimoto's thyroiditis revealed that the most associated kidney disease was membranous glomerulonephritis. Hashimoto's thyroiditis has also been found in patients with lupus nephritis, minimal change disease, IgA nephropathy, MPGN, and FSGN [14]. Li et al. [4] also observed that membranous nephropathy and minimal change disease were prevalent in both thyroid function groups, with no significant differences in renal pathology.

Our study identifies GFR and cholesterol levels as significant predictors of thyroid dysfunction in nephrotic syndrome patients, echoing Li et al.'s findings [4].

Despite limitations such as a small sample size and the single-center nature, this research contributes valuable insights into the complex relationship between thyroid dysfunction and nephrotic syndrome, underscoring the need for comprehensive thyroid function monitoring in this patient population. Further research is warranted to elucidate the underlying mechanisms and explore potential therapeutic interventions.

Conclusion

Thyroid dysfunction is common in patients with nephrotic syndrome, and the type of renal pathology differs among thyroid dysfunction subgroups. Interpreting the interactions between thyroid and renal function is challenging for clinicians treating patients with nephrotic syndrome. Long-term follow-up of patients with thyroid dysfunction after management is essential to evaluate the effect of medical therapy on these patients.

List of Tables and Figures:

Table (1): Baseline data of the studied groups

	Thyroid dysfunction (n= 100)	**Normal thyroid function (n= 50)**	**P-value**
Age: (years) Mean ± SD Range	 33.13 ± 8.41 19.0-54.0	 29.82 ± 7.69 19.0-47.0	**0.021***
Gender: No. (%) Male Female	 52 (52.0%) 48 (48.0%)	 22 (44.0%) 28 (56.0%)	0.356

Data is expressed as frequency (percentage) and mean (SD). *P*-value was significant if < 0.05

Table (2): Baseline data among the studied groups

	Thyroid dysfunction (n= 100)	**Normal thyroid function (n=50)**	**P-value**
Random blood sugar (mg/dl)			
Mean ± SD	99.07 ± 12.21	96.60 ± 10.64	0.225
Range	76.0-129.0	76.0-117.0	
Complete blood picture			
Haemoglobin (g/dl)			

Mean ± SD	9.73 ± 1.44	9.92 ± 1.13	0.406
Range	7.5-14.0	8.0-12.0	
	Thyroid dysfunction (n= 100)	**Normal thyroid function (n=50)**	**P-value**
MCV: (fl)			
Mean ± SD	81.53 ± 9.34	82.20 ± 6.66	0.651
Range	62.0-99.0	70.0-96.0	
MCH: (g/dl)			
Mean ± SD	28.73 ± 4.16	28.00 ± 4.18	0.313
Range	18.0-39.0	20.0-35.0	
Platelets (10^3\ul)			
Mean ± SD	302.86 ± 100.92	318.70 ± 78.72	0.333
Range	150.0-546.0	174.0-440.0	
Leucocytes (10^3\ul)			
Mean ± SD	7.15 ± 1.93	7.26 ± 1.93	0.743
Range	3.2-11.7	4.0-10.5	
Kidney function test			
Urea (mmol\L)			
Mean ± SD	5.45 ± 2.33	4.95 ± 1.32	0.167
Range	1.0-13.0	2.5-7.2	
	Thyroid dysfunction (n= 100)	**Normal thyroid function (n=50)**	**P-value**
Creatinine (umol\l)			
Mean ± SD	94.56 ± 10.82	82.09 ± 19.14	**0.000***
Range	75.0-115.0	40.0-114.0	
GFR (ml/min/1.73m)			
Mean ± SD	95.90 ± 17.92	107.70 ± 19.91	**0.001***
Range	65.0-134.0	64.0-147.0	
Urine analysis			
Red blood corpuscles			
Mean ± SD	7.61 ± 3.56	7.82 ± 3.85	0.458
Median (Range)	4.0 (0.0-17.0)	8.0 (0.0-17.0)	
Pus cells:			
Mean ± SD	8.85 ± 3.97	8.82 ± 4.45	0.841
Median (Range)	8.5 (2.0-20.0)	8.0 (3.0-20.0)	
Specific gravity			
Mean ± SD	1018.16 ± 3.87	1018.60 ± 3.10	0.486
Range	1003.0-1025.0	1015.0-1025.0	
Volume: (CC)			
Mean ± SD	1420.20 ± 426.85	1338.40 ± 361.52	0.247
Range	750.0-2500.0	750.0-2500.0	
Protein in 24 hrs: (gm)			
Mean ± SD	5.77 ± 1.71	6.10 ± 1.96	0.297
Range	3.2-10.0	3.2-10.0	

Data expressed as a mean (SD) range. *P*-value was significant if < 0.05. **MCV**: mean corpuscular volume; **MCH**: mean corpuscular haemoglobin; **GFR**: glomerular filtration rate

Table (3): Liver function tests and lipid profile among the studied groups

	Thyroid dysfunction (n= 100)	Normal thyroid function (n=50)	P-value
Plasma lipid levels			
Triglyceride: (mg\dl)			
Mean ± SD	98.52 ± 22.96	98.28 ± 13.27	0.946
Range	52.0-167.0	75.0-123.0	
Cholesterol: (mg/dl)			
Mean ± SD	139.76 ± 17.51	115.88 ± 33.17	**0.000***
Range	87.0-168.0	67.0-195.0	
High-density lipoproteins (mg\dl)			
Mean ± SD	62.64 ± 13.30	79.52 ± 11.04	**0.000***
Range	40.0-89.0	60.0-98.0	
Low-density lipoproteins (mg/dl)			
Mean ± SD	108.22 ± 11.12	97.02 ± 18.87	**0.000***
Range	85.0-129.0	45.0-139.0	
Liver function tests			
Total protein: (g/l)			
Mean ± SD	71.58 ± 6.55	70.42 ± 5.96	0.294
Range	59.0-86.0	59.0-83.0	
Albumin: (g/l)			
Mean ± SD	23.54 ± 4.84	23.80 ± 4.95	0.759
Range	15.0-34.0	15.0-34.0	
Total bilirubin: (umol\l)			
Mean ± SD	13.92 ± 5.79	13.04 ± 5.44	0.372
Range	3.0-25.8	3.0-21.0	
Direct bilirubin: (umol\l)			
Mean ± SD	2.10 ± 0.89	2.16 ± 0.87	0.705
Range	0.2-3.8	0.2-3.5	
Aspartate transaminase (U/L)			
Mean ± SD	20.80 ± 8.71	18.91 ± 8.55	0.211
Range	3.7-36.0)	3.7-34.0	
Alanine transaminase (U/L)			
Mean ± SD	28.75 ± 10.36	30.52 ± 10.20	0.323
Range	7.0-46.0	7.0-46.0	
Alkaline phosphatase: (U/L)			
Mean ± SD	77.04 ± 18.81	78.96 ± 19.71	0.563
Range	45.0-114.0	50.0-114.0	

Data expressed as a mean (SD) range. *P*-value was significant if < 0.05.

Table (4): Thyroid function tests among the studied groups

	Thyroid dysfunction (n= 100)	Normal thyroid function (n=50)	P-value
Thyroid function tests			
TSH: (mlU\ml)			
Mean ± SD	15.61 ± 12.85	2.81 ± 1.16	**0.000***
Median (Range)	14.3 (0.0-60.0)	2.9 (0.6-4.5)	
Free T3: (pg\ml)			
Mean ± SD	2.70 ± 3.41	3.42 ± 0.65	**0.000***
Median (Range)	1.4 (0.0-12.7)	3.5 (2.3-4.4)	
Free T4: (ng\dl)			
Mean ± SD	0.86 ± 1.35	1.23 ± 0.30	**0.000***
Median (Range)	0.2 (0.0-4.6)	1.2 (0.8-1.9)	
Thyroid Peroxidase Antibodies (IU/mL)			
Mean ± SD	81.49 ± 26.95	32.24 ± 5.13	**0.000***
Range	21.0-120.0	23.0-40.0	

Data is expressed as mean (SD) and range. *P*-value was significant if < 0.05

Table (5): Abdominal ultrasound and renal biopsy among the studied groups

	Thyroid dysfunction (n= 100)		Normal thyroid function (n=50)		P-value
	No.	%	No.	%	
Abdominal ultrasound (Echogenicity)					
Grade I	62	62.0	30	60.0	0.813
Grade II	38	38.0	20	40.0	
Renal biopsy					
FSGN	36	36.0	16	32.0	0.354
FSGS	15	15.0	6	12.0	
MPGN	33	33.0	18	36.0	
Mesangio Proliferative GN	8	8.0	5	10.0	
Minimal change disease	8	8.0	5	10.0	

Data expressed as frequency (percentage). *P*-value was significant if < 0.05. **FSGN**: focal segmental glomerulonephritis; **FSGS**: focal segmental glomerulosclerosis; **MPGN**: memeberano-proliferative glomerulonephritis; **GN**: glomerulonephritis

Table (6): Predictors for thyroid dysfunction in patients with nephrotic syndrome

	Odd's ratio	95% confidence interval	P value
Age	1.23	0.90-3.98	0.5
GFR	2.22	1.11-4.89	**0.01**
Cholesterol	3.01	2.90-7.54	< **0.001**
HDL	1.09	0.79-2.19	0.47
LDL	0.87	0.22-1.198	0.19

GFR: glomerular filtration rate; **HDL**: high-density lipoproteins; **LDL:** low-density lipoproteins

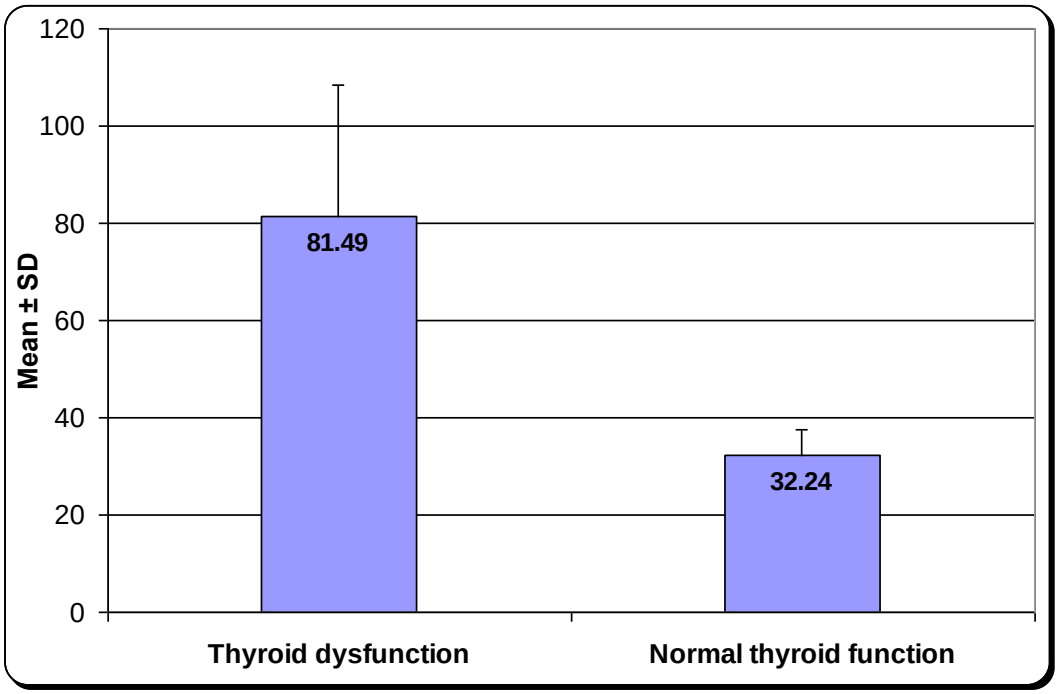

Figure 1: Thyroid peroxidase antibodies among the studied groups

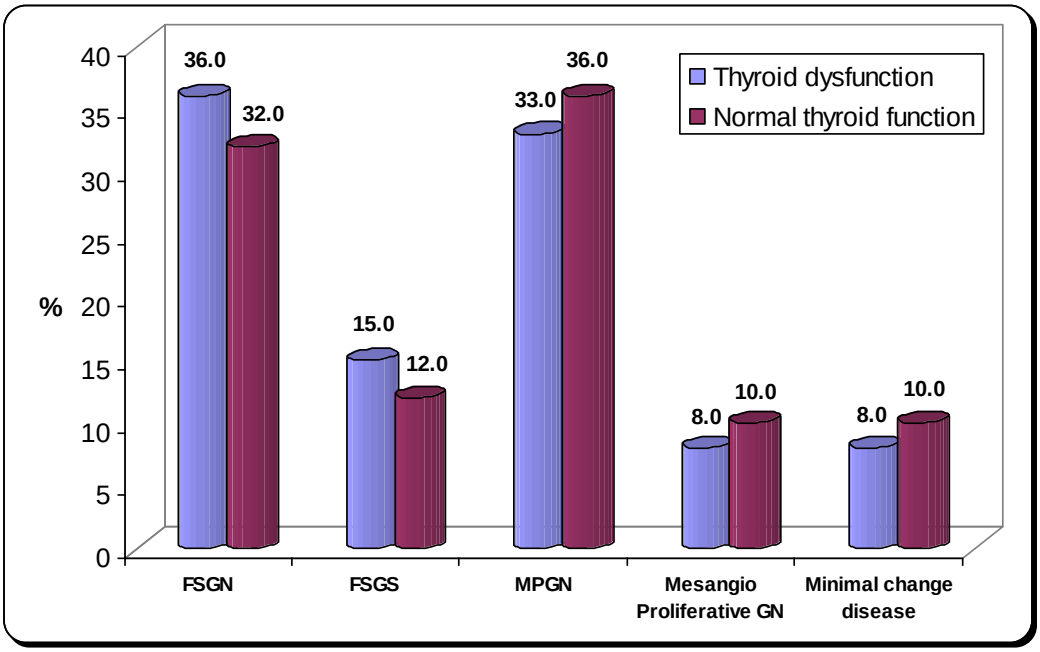

Figure 2: Renal biopsy among the studied groups

References:

1. Rapa A. Subclinical hypothyroidism in children and adolescents: a wide range of clinical, biochemical, and genetic factors involved. *J Clin Endocrinol Metab*. 2009;94(7):2414-2420. doi:10.1210/jc.. 2008-2414.
2. Shin DH, Lee MJ, Kim SJ, Oh HJ, Kim HR, Han SH, et al. Preservation of renal function by thyroid hormone replacement therapy in chronic kidney disease patients with subclinical hypothyroidism. *J Clin Endocrinol*

Metab. 2012;97(8):2732-2740. doi:10.1210/jc.2012-1663.
3. Hull RP, Goldsmith DJA. Nephrotic syndrome in adults. *BMJ*. 2008;336(7654):1185-1189. doi:10.1136/bmj.39568.662384.80.
4. Li L-Z, Wang Y, Chen J, Zhang Y, Zhang Y, Liu Y, et al. The relationship between thyroid dysfunction and nephrotic syndrome: a clinicopathological study. *Sci Rep*. 2019;9(1):6421. doi:10.1038/s41598-019-42902-7.
5. Hussein SZ, Ali SJ. Serum thyroid hormones and cortisol in patients with chronic renal failure. *Med J Tikrit Univ*. 2019;25(1):1-6.
6. Jain D, Aggarwal HK, Jain P, Yadav RK, Garg S. Evaluation of thyroid dysfunction in patients with nephrotic syndrome. *Med Pharm Rep*. 2019;92(2):139-143. doi:10.15386/mpr-1203.
7. Keunmoe P, Njamen TN, Dehayem MY, Mbanya JC. The spectrum of thyroid function abnormalities and associated biochemical factors in patients with chronic kidney disease in Cameroon. *Am J Biomed Sci Res*. 2020;8(5):387-396. doi:10.34297/AJBSR.2020.08.001291.
8. Singh S. Thyroid function in patients with idiopathic nephrotic syndrome. *Int Urol Nephrol*. 2021;53(1):1-6. doi:10.1007/s11255-020-02618-4.
9. Haghi AR, Solhjoo M, Tavakoli MH. Correlation between subclinical hypothyroidism and dyslipidemia. *Iran J Pathol*. 2017;12(2):106-111. doi:10.30699/ijp.2017.24480.
10. Unal E, Akın A, Yıldırım R, Demir V, Yıldız İ, Haspolat YK. Association of subclinical hypothyroidism with dyslipidemia and increased carotid intima-media thickness in children. *J Clin Res Pediatr Endocrinol*. 2017;9(2):144-149. doi:10.4274/jcrpe.3837.
11. Azhar H. The effects of dyslipidemia in subclinical hypothyroidism. *Cureus*. 2019;11(11):e6247 . doi:10.7759/cureus.6247.
12. Dey A, Kanneganti V, Das D. A study of the cardiac risk factors emerging out of subclinical hypothyroidism. *J Family Med Prim Care*. 2019;8(7):2439-2444. doi:10.4103/jfmpc.jfmpc_331_19.
13. Kalra S. Thyroid dysfunction and dysmetabolic syndrome: the need for enhanced thyrovigilance strategies. *Int J Endocrinol*. 2021;2021:9641846. doi:10.1155/2021/9641846.
14. Santoro D, Vita G, Santoro D, Vita G. Autoimmune thyroiditis and glomerulopathies. *Front Endocrinol (Lausanne)*. 2017;8:265518. doi:10.3389/fendo.2017.00265.