ORIGINAL RESEARCH

Clinical profile and treatment response in patients with nephrotic syndrome

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ABSTRACT

Background: Nephrotic syndrome is characterized by substantial proteinuria causing hypoalbuminemia, hyperlipidemia, edema, and diverse complications. This study aimed to explore the clinical profile of nephrotic syndrome and to assess the clinical response to the given treatment. **Methods:**This prospective hospital-based study was conducted at a tertiary care hospital between December 2014 and 2016. Patients diagnosed with proteinuria, hypoalbuminemia, edema and hyperlipidemia were included in this study. **Results:**A total of 100 patients, with a mean age of 33.6 years were included. Among these, 88 patients had primary, while12 patients had secondary glomerular disease. Focal segmental glomerulosclerosis(FSGS)was the most common (n=24) primary glomerular lesion. All patients with IgA nephropathy were belonged to the age group of<40 years. Edema was observed in all patients with nephritic syndrome. Microscopic hematuria was present in all patients with diffuse proliferative glomerulonephritis(DPGN). About 86 patients were followed up for > 6 month.Steroid was given in all patients with minimal change disease (MCD), FSGS, membranoproliferative glomerulonephritis (MPGN), immunoglobulin M (IgM) nephropathy, C1q nephropathy and DPGN.Patients with MCD had higher complete remission rate(70.6%). About 50% patients with IgM nephropathy had relapse. Among 86 patients, 7 patients affected with infection. **Conclusion:**FSGS stands out as the most predominant glomerular lesion among adult with nephrotic syndrome. Patients with MCD typically respond well to treatment, whereas those with DPGN often face a poor prognosis.

Keywords: Adult nephrotic syndrome, glomerular disease, focal segmental glomerulosclerosis, clinical outcome

INTRODUCTION

Nephrotic syndrome, while uncommon, holds significant importance within the spectrum of kidney diseases.[1]It constitutes a clinical condition characterized by substantial proteinuria responsible for hypoalbuminemia, with resulting hyperlipidemia, edema, and various complications. [2]Globally, nephrotic syndrome stands as a substantial contributor to end-stage renal diseases among adult patients.[3]Idiopathic cases comprise approximately 80% to 90% of nephrotic syndrome diagnoses in adults, despite its more frequent observation in pediatric patients.[3,4]Despite significant progress in healthcare, glomerular disease remains a prominent cause of renal failure, leading to significant morbidity and mortality.[3]

Nephrotic syndrome arises from heightened permeability due to damage in the renal glomerulus' basement membrane, often triggered by factors like infections or thromboembolic events.[2]It arises from an irregularity in the permeability of the glomeruli, which could stem from a primary condition within the kidneys or secondary causes like congenital infections, diabetes mellitus, systemic lupus erythematosus, neoplasia, or specific medication usage.[2,4]Among adults, diabetes mellitus stands as the most prevalent secondary cause, while focal segmental glomerulosclerosis (FSGS) and

membranous nephropathy emerge as the primary causes most frequently observed.[5]

Proper diagnosis holds utmost importance in distinguishing between the diverse causes of nephrotic syndrome. The American Academy of Family Physicians recommends employing spot urine protein-to-creatinine ratio as an alternative to the 24-hour urine collection method for confirming nephrotic-range proteinuria .[4]In addition to this, conducting a biopsy becomes essential for both diagnosing the condition and estimating the prognosis for the patients.[6]

The appropriate treatment in patients with nephrotic syndrome can involve fluid and sodium restriction, loop diuretics, therapy with angiotensin-converting enzyme inhibitors or angiotensin receptor blockers, and meticulous evaluation to identify potential disease complications. Immunotherapy, such as corticosteroids, is frequently employed in nephritic syndrome, despite the lack of substantial evidence.[4,5]

The present study aimed to explore the clinical profile of nephrotic syndrome and to assess the clinical response to the given treatment.

METHODS

Study design

This prospective hospital-based study was conducted at the King George Hospital, Andhra Medical College, Visakhapatnam,between December 2014 and December 2016.The Ethical approval was obtained from Institutional Ethical Committee prior to commencement of the study. A written informed consent was obtained from each patient or their caretakers.

Inclusion and exclusion criteria

Patients aged between 18 and 60 years diagnosed with proteinuria (3.5>g/24hours), hypoalbuminemia (<3.0g/dL), generalized edema and hyperlipidemia having nephrotic syndrome were included in this study. Patients with diabetes mellitus, clinical/biochemical evidence of diabetic nephropathy, and pregnant women were excluded.

Data collection

Data related to age, sex, locality, tribe and detailed review of clinical history, social, and family history were collected.Clinical examinations of weight, blood pressureand systemic examinations were done.Various investigations including complete urine analysis, 24hrs urine protein estimation, urine forculture and sensitivity, blood samples for biochemical and hematological analysis, Mantoux test, ascitic fluid for culture and sensitivity when indicated, ultrasound, and chest X-ray were conducted. Renal biopsies were performed and analyzed using Bard fine needle aspiration cytology Renal Biopsy (gun No: 18) with real time ultrasound sonography guidance. Other investigations were also conducted for atypical cases, including tests such as antistreptolysin O titre, venereal disease research laboratory, antinuclear antibody, complements, and double-strand deoxyribonucleic acid analysis etc.

Follow-up

Patients were advised to attend monthly nephrology outpatient visits and undergo specified investigations. During the follow-uppatients were monitored for complications, treatment response, drug side effects, and short-term outcomes. Patients were also advised to immediately visit nephrology ward upon developing complications and were admitted to the wards if necessary.

Sample size calculation

The sample size was calculated according to the advice of statistician using the equation

$N = Z^2 \times P \times Q / D^2$

Where:

N (sample size)

Z (statistical certainty)

P (probability)

Q (1-P)

D (desired margin of error)

Statistical analysis

Data were analyzed using statistical package for the social sciences (SPSS). Descriptive statistics were used to describe categorical variables, such as frequency and percentages, and continuous variables,such as the mean and standard deviation (SD).

Definition

Patients were treated according to the 2012 Kidney Disease Improving Global Outcomes (KDIGO) Clinical Practice Guideline for glomerulonephritis.[7]

Nephrotic syndrome is defined as a tetrad of proteinuria (nephrotic range), hypoalbuminemia (serum albumin <2.5gm/dL), edema and hyperlipidemia (serum cholesterol >200mg/dL). 1 Nephrotic-range proteinuria in the adult population is defined as protein excretion of >3.5 gm/day in a timed urine collection.

RESULTS

Age and sex distribution of patients

A total of 100 patients were included, of which 59 patients were males and 41 patients were female. The majority of female (51.2%) and male (45.8%) patients belonged to the age group of 18-30 years followed by the age group of 31-40 years (26.8% of female and 22.0% of male) (Table 1).

Table 1: Age and sex distribution of patients

Age (Years)	Male (n=59)	Female (n=41)
18-30	27 (45.8)	21 (51.2)
31-40	13 (22.0)	11 (26.8)
41-50	9 (15.3)	5 (12.2)
51-60	10 (16.9)	4 (9.8)
Data presents as n (%), unless otherwise specified.		

Demographic characteristics of patients

Out of 100 patients, 88 patients had primary glomerular disease and 12 patients had secondary glomerular disease. The FSGS was the most common primary glomerular lesion observed in 24 patients followed by membranous glomerulonephritis (MGN) (n=22). Among the secondary glomerular diseases, 41.7% patients had lupus nephritis, while 16.7% of patients had HCV associated membranoproliferative glomerulonephritis (MPGN) and 16.7% of patients had hepatitis B surface antigen (HBsAg) associated MGN.

The mean age of patients with diffuse proliferative glomerulonephritis (DPGN) was 36.5 years with male to female ratio of 1:1.Among primary glomerular disease, all patients withimmunoglobulin A (IgA) nephropathy (100%) were in the <40 years of age group, followed by those with minimal change disease (MCD; 84.2%).Among secondary glomerular disease,83.3% of patients were in the <40 years age group. The majority of patients with immunoglobulin M (IgM) nephropathy (50.0%) and DPGN (50.0%) belonged to the age group of > 40 years. In primary and secondary glomerular diseases group, edema (100%) was present in all patients. Oliguriawas present in all patients with DPGN (100%), and in 58.3% of patients with secondary glomerular disease. All patients in both the primary and secondary glomerular diseases groups exhibited pedal edema (100%). Hypertension was prevalent in patients with DPGN (100%), IgA nephropathy (100%) and MPGN (83.3%) (Table 2).

Table 2: Demographic characteristics

Parameter	Primary (n=88)								Secondary (n=12)
	MCD (n=19)	FSGS (n=24)	MGN (n=22)	MPGN (n=12)	IgA nephropathy (n=3)	IgM nephropathy (n=2)	C1q nephropathy (n=4)	DPGN (n=2)	
Age (years), mean(range)	30.2 (18-60)	35.8 (18-58)	36.1 (19-58)	31.5 (18-56)	24.0 (18-35)	32.5 (22-43)	32.3 (18-50)	36.5 (28-45)	32.7 (19-59)
Male female ratio	13:6	16:9	11:11	6:6	3:0	2:0	3:1	1:1	7:5
Distribution among age groups									
<40 years	16 (84.2)	15 (62.5)	14 (63.6)	9 (75.0)	3 (100)	1 (50.0)	3 (75.0)	1 (50.0)	10 (83.3)
>40 years	3 (15.8)	9 (37.5)	8 (36.4)	3 (25.0)	0	1 (50.0)	1 (25.0)	1 (50.0)	2 (16.7)
Symptoms									
Edema	19 (100)	24 (100)	22 (100)	12 (100)	3 (100)	2 (100)	4 (100)	2 (100)	12 (100)
Oliguria	2 (10.5)	5 (20.8)	4 (18.2)	3 (25.0)	1 (33.3)	1 (50.0)	0	2 (100)	7 (58.3)
Fever	1 (5.2)	4 (16.6)	3 (13.6)	4 (33.3)	1 (33.3)	0	1 (25.0)	2 (100)	1 (8.3)
Anorexia vomiting	0	2 (8.3)	1 (4.5)	1 (8.3)	0	0	0	0	1 (8.3)
Dyspnoea	0	2 (8.3)	1 (4.5)	1 (8.3)	0	0	0	0	1 (8.3)
Physical examination									
Pedal edema	19 (100)	24(100)	22 (100)	12 (100)	3 (100)	2 (100)	4 (100)	2 (100)	12 (100)
Hypertension	1 (5.3)	11 (45.8)	11 (50.0)	10 (83.3)	3 (100)	1 (50.0)	2 (50.0)	2 (100)	1 (8.3)
Pleural effusion	9 (47.4)	8 (33.3)	11 (50.0)	4 (33.3)	0	2 (100)	1 (25.0)	0	4 (33.3)

Asictes	14 (73.7)	14 (58.3)	13 (59.1)	6 (50.0)	2 (66.7)	2 (100)	3 (75.0)	1 (50.0)	7 (58.3)

Laboratory findings

The mean hemoglobin levels were lower in patients with IgA nephropathy (10.8 g/dL). The serum cholesterol levels were higher in patients with MCD (293.9 mg/dL), followed by IgM nephropathy (257.5 mg/dL) and focal segmental glomerulosclerosis (FSGS; 255.2 mg/dL). Out of 100 patients, the microscopic hematuria was observed in 12 patients. Renal failure was seen in 36 patients. Among primary glomerular disease, all patients with DPGN (100%) presented with glomerular filtration rate (GFR) of <60 ml/min/1.73 m^2 (Table 3).

Table 3: Laboratory findings

Parameter		Primary (n=88)								Secondary (n=12)
		MCD (n=19)	FSGS (n=24)	MGN (n=22)	MPGN (n=12)	IgA nephropathy (n=3)	IgM nephropathy (n=2)	C1q nephropathy (n=4)	DPGN (n=2)	
Laboratory investigations										
Mean haemoglobin (g/dL)		11.9	12.0	11.6	11.9	10.8	13.5	12.9	11.8	11.8
Serum cholesterol (mg/dL)		293.9	255.2	233.5	204	202.3	257.5	197.7	241	237.5
Hypoalbunemia (g/dL)		2.1	2.4	2.2	2.8	2.5	1.9	2.8	2.8	2.5
Microscopic hematuria		0	3 (12.5)	1 (4.5)	1 (8.3)	2 (66.7)	0	0	2 (100)	3 (25.0)
Renal failure										
Urea	<45 (mg/dL)	18 (94.7)	11 (45.8)	19 (86.4)	7 (58.3)	3 (100)	2 (100)	2 (50.0)	1 (50.0)	6 (50.0)
	>45 (mg/dL)	1 (5.3)	13 (54.2)	3 (13.6)	5 (41.7)	0	0	2 (50.0)	1 (50.0)	6 (50.0)
GFR	<60 (ml/min/1.73 m^2)	2 (10.5)	14 (58.3)	4 (18.2)	5 (41.7)	0	0	2 (50.0)	2 (100)	7 (58.3)
	GFR >60 (ml/min/1.73 m^2)	17 (89.5)	10 (41.7)	18 (81.8)	7 (58.3)	3 (100)	2 (100)	2 (50.0)	0	5 (41.7)

Data presents as n (%), unless otherwise specified.
DPGN, diffuse proliferative glomerulonephritis; FSGS, focal segmental glomerulosclerosis;GFR, glomerular filtration rate; IgA, immunoglobin A; IgM, immunoglobin M; MCD, minimal change disease; MGN, membranous glomerulonephritis;MPGN, membranoproliferative glomerulonephritis.

Treatment, follow-up, and outcomes

Out of 100 patients, 86 patients with primary glomerular disease and 9 patients with secondary glomerular diseasewere followed up for>6 months. Steroid was given in all patients with MCD, FSGS, MPGN, IgM nephropathy, C1q nephropathy and DPGN.Approximately,77.8% of patients with secondary glomerular disease received steroids treatment. About 66.7% of patients with secondary glomerular disease, and 50.0% of patients with both IgM nephropathy and DPGN among primary glomerular diseases received second-line immunosuppressant therapy. Anti-hypertensive treatment was given in all patients with IgA nephropathy (100%) and 77.8% of patients with secondary glomerular disease. Approximately 70.6% of patients with MCD had complete remission followed by patients with C1q nephropathy (66.7 %). About 50% patients with IgM nephropathy had relapse. Partial remission rate was higher in patients with IgM nephropathy (50.0%) and DPGN (50.0%). Progressive renal failure occurred in 50.0% in patients with IgM nephropathy and 44.4 % of patients with secondary glomerular disease (Table 4).

Table 4: Treatment, follow-up and outcomes

Parameter	Primary (n=86)								Secondary (n=9)
	MCD (n=17)	FSGS (n=22)	MGN (n=17)	MPGN (n=11)	IgA nephropathy (n=3)	IgM nephropathy (n=2)	C1q nephropathy (n=3)	DPGN (n=2)	
Treatment and follow-up									
Mean duration	15.6	14.4	13.2	14.9	12.0	13.0	22.7	23.0	14.6
Steroids	17 (100)	22(100)	4 (23.5)	11 (100)	0	2 (100)	3 (100)	2 (100)	7 (77.8)
2nd line immunosuppressants	1 (5.9)	5 (22.7)	4 (23.5)	0	0	1 (50.0)	0	1 (50.0)	6 (66.7)
Anti-hypertensive	1 (5.9)	9 (40.9)	5 (29.4)	7 (63.6)	3 (100)	1 (50.0)	1 (33.3)	1 (50.0)	7 (77.8)
Clinical outcome									
Complete remission	12 (70.6)	12 (54.5)	7 (41.2)	5 (45.5)	1 (33.3)	0	2 (66.7)	0	1 (11.1)
Relapse	3 (17.6)	2 (9.1)	0	1 (9.1)	0	1 (50.0)	0	0	1 (11.1)
Partial remission	2 (11.7)	4 (18.2)	8 (47.1)	3 (27.3)	1 (33.3)	1 (50.0)	1 (33.3)	1 (50.0)	3 (33.3)
Progressive renal failure	0	4 (18.2)	2 (11.8)	2 (18.2)	1 (33.3)	0	0	1 (50.0)	4 (44.4)

Data presents as n (%), unless otherwise specified.
DPGN, diffuse proliferative glomerulonephritis; FSGS, focal segmental glomerulosclerosis; IgA, immunoglobin A; IgM, immunoglobin M; MCD, minimal change disease; MGN, membranous glomerulonephritis;MPGN, membranoproliferative glomerulonephritis.

Complications

Among the 86 patients, infections were observed in 7 patients.Of these, urinary tract infections were observed in 3.5% of patients.Deep vein thrombosis and proximal myopathy were seen in 2.3% each (Table 5).

Table 5: Complications

Complications	Number of patients (n=86)
Infections	**[n=7]**
UTI	3 (3.5)
SABP	1 (1.2)
PTB	1 (1.2)
Consolidation	1 (1.2)
Cellulitis	1 (1.2)
DVT	2 (2.3)
Proximal myopathy	2 (2.3)

Data presentsas n (%), unless otherwise specified.
DVT, deep vein thrombosis; PTB, pulmonary tuberculosis; SABP, sub-acute bacterial peritonitis; UTI, urinary tract infections.

DISCUSSION

Nephrotic syndrome is a well-recognized presentation of kidney disease in both adults and children, predominantly stems from primary kidney disease. [1]About one-third of primary nephrotic syndrome cases are attributed to Membranous nephropathy and FSGS each, with FSGS being the primary cause of idiopathic nephrotic syndrome in adults.[3]

In the present study, the majority of patients with nephrotic syndrome belonged to the age group of 18-40 years. In a previous study done by Sarkar A et al., the most common age group was 18 to 50 years.[3]In the current study, FSGS is the most common cause of nephrotic syndrome, followed by MGN and MCD. This finding aligns with the results reported in the previous study by JI Kazi et al.[8]Similarly, another study done by Rathi M et al., reported that among primary nephrotic diseases, FSGS was the most prevalent, followed by MGN.[9]

In current study, the secondary glomerular disease was observed in 12 patients. In previous study conducted by Rathi M et al., reported that 40 patients had secondary glomerular disease. [9] Similarly, CD De La Roque et al. reported a higher predominance of idiopathic MCD and FSGS in males compared to females, a trend observed in this present study as well.[10]

Edema, the predominant symptom of nephrotic syndrome,[11]was observed in 100% of patients in this study, consistent with the findings observed by by Sarkar A et al.[3]

In the current study, DPGN had a higher incidence of hematuria. In contrast, a study conducted by Reshi AR et al. found that FSGS had a higher incidence of hematuria.[12]

In present study, the steroid was given to all patients with primary nephritic syndrome. In previous study done by Rasic S et al., the 7 out of 13 patients were given steroids followed by IgA nephropathy (3/5) and MCD (5/6).[13]In the current study, second-line immunosuppressant treatment was administered to patients with IgM nephropathy, DPGN, MGN, FSGS, and MCD, as well as secondary nephritic syndrome. A study conducted by Rasic S et al., reported that patients with MGN were treated with cyclosporine.[13]

In present study, complete remission was observed in patients with MCD followed by C1q nephropathy, FSGS, MPGN, MGN and IgA nephropathy. Previous study conducted by Rasic S et al., reported that complete remission was observed in all patients with MCD.[13]Similarly, study by De La Roque CD et al., the complete remission was observed in majority of patients with idiopathic MCD.[10]In the present study relapse rate was higher in patients with IgM nephropathy. A previous study done by De La Roque CD et al., reported that the relapse rate was higher in patients with idiopathic FSGS.[10]In current study the particle remission rate was higher in IgM nephropathy and DPGN. A previous study done by Rasic S et al., reported that the partial remission was higher in IgA nephropathy, followed by MGN and DPGN.[13] A study conducted by Maria Faye et al. included 134 patients: 85.8% received steroids alone, 7.6% received cyclophosphamide, and 2.5% received azathioprine in combination with steroids. The outcomes showed favourable progression, with 57 patients achieving remission. Eight patients experienced relapse, while seven patients developed steroid dependence.[14]

Nephrotic syndrome often leads to a range of systemic complications, believed to stem from an excess production of hepatic proteins and the excretion of low-molecular-weight proteins in the urine.[4]In the present study, infectious complications were observed in 7 patients, with urinary tract infections noted in 3.5% of the patients. In a previous study conducted by Ali Taha A et al., among the 139 patients with nephrotic syndrome, 119 were found to have infections.[15]

LIMITATION

The study's limitations include a small sample size, potentially limiting result generalizability. Additionally, the study's duration might not adequately capture long-term outcomes. Practical challenges with patient compliance in attending follow-up clinics could introduce data gaps, impacting the study's comprehensiveness and reliability. Therefore further prospective studies with larger sample size are needed to validate the present study findings.

CONCLUSION

There is considerable variation in the etiology of adult nephrotic syndrome worldwide. The FSGS stands out as the most prevalent glomerular lesion among adults with nephrotic syndrome. Patients with MCD typically respond well to treatment, whereas those with DPGN often face a poor prognosis. Infections represent the most frequent complications observed in adults with nephrotic syndrome.

Conflict of Interest: The authors have no conflicts of interest to declare.

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Ethical approval: The Ethical approval was obtained from Institutional Ethical Committee prior to commencement of the study. A written informed consent was obtained from each patient or their caretakers.

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