CLINICO PATHOLOGICAL STUDY OF NEPHROTIC SYNDROME IN ADULTS

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ABSTRACT: CONTEXT: Etiology of nephrotic syndrome (NS) in adults varies depending on the geographical location and is poorly studied in the Indian subcontinent.

AIMS: To study the clinical features, biochemical profile and histopathological pattern of various types of glomerulonephritis in adult patients with nephrotic syndrome.

METHODS AND MATERIAL: Patients (≥15 years old) with nephrotic syndrome presenting to our center and undergoing a kidney biopsy from May 2009 to August 2011 were included for this study. All biopsies were subjected to light microscopy.The histopathological spectrum was analyzed according to the various clinical parameters.

STATISTICAL ANALYSIS USED: Analysis was performed using SPSS software version 19.Measures obtained included percentages,medians,correlation coefficients and chi square tests.

RESULTS: A total of 50 kidney biopsies were included in analysis. Twenty six(52%) patients were male and twenty four(48%) patients were female. The average age at presentation was 15-24 years. Among the patients, 22(44%) were diagnosed with primary glomerular diseases (PGD) and 4(8%) with secondary glomerular diseases (SGD). The most common histological lesions was membranous nephropathy(24.4%) followed by minimal change disease (MCD) (17%) and membranous nephropathy (MN) (17%). The most common form of SGD was lupus nephritis (LN) (10%). Membranous nephropathy and focal segmental glomerulosclerosis were the commonest lesions in males.Among females,membranous nephropathy was the commonest. 31(62%) patients were in the age group of 15-34 yrs, 17(34%) were in the age group 34-54yrs and only 2(4%) were aged above 55yrs. Among the patients, 6(12%) had serum creatinine ≥1.5 mg/dL and 12(24%) had either macroscopic or microscopic hematuria.

CONCLUSIONS: Membranous nephropathy is still the commonest type of nephrotic syndrome in adults followed closely by focal segmental glomerulosclerosis and minimal change disease as per this study.

KEYWORDS: Nephrotic Syndrome, Kidney Biopsy, Light Microscopy.

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INTRODUCTION: Nephrotic syndrome is one of the principal presentation of glomerular disease. It may be caused by primary renal disease or by a variety of secondary causes. Patients present with marked edema, proteinuria, hypoalbuminemia, and often hyperlipidemia. In adults, diabetes mellitus is the most common secondary cause, and focal segmental glomerulosclerosis and membranous nephropathy are the most common primary causes. Proper diagnosis is critical to differentiate among the various nephrotic etiologies Renal biopsy is strongly recommended for diagnosis in adults. Treatment of most patients should include fluid and sodium restriction, oral or intravenous diuretics, and angiotensin-converting enzyme inhibitors. Some adults with nephrotic syndrome may benefit from corticosteroid treatment, particularly in nephrotic syndrome due to primary causes.

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The etiology of nephrotic syndrome in adults varies depending on the geographical location and is poorly studied in india.The present study was conducted to ascertain the histologic spectrum of nephrotic syndrome in adults at our institute.

SUBJECTS AND METHODS: Patients above 15 years presenting edema of pitting type,massive proteinuria (>3.5gms/day/1.73sq.m), hypoalbunemia, hypercholesterolemia. Patients presenting with a combination of nephritic and nephrotic syndrome (abrupt onset,smoky urine, oliguria, edema, hypertension, RBC/RBCS casts in urine with features of nephrotic syndrome as mentioned above)were also inlcuded in the study.

50 adult patients who have fulfilled the inclusion criteria were taken for clinical,biochemical and histopathological evaluation. Clinical evaluation included duration of illness, mode of onset, presence or absence of complications and possible etiological factors. Biochemical evaluation included 24 hour urinary protein,serum total protein and A/G ratio,lipid profile,blood urea,serum creatinine,collagen profile,fasting and 2 hours postprandial blood sugar. Histopathological evaluation included percutaneous renal biopsy which was performed under

ultrasonic guidance and the prepared slides were examined under light microscope.

RESULTS: A total of fifty patients were studied in this institute who presented with nephrotic syndrome. Maximum incidence of nephrotic syndrome was seen in age group 15-24 years.Age distribution and sex prevalence is given in tables (1) and (2). Edema was observed in all patient s.Oliguria was obsereved in 17(34%) patients of which 12(24%) are primary and 5(10%) were secondary cases of nephrotic syndrome. Acute nephritic -nephrotic syndrome was seen in 8 patients(16%). Hamaturia was present in 12(24%) patients of which 8(16%) were primary and 4(8%) were secondary.Hypertension was seen in 21(42%) of patients of which 13(26%) had primary glomerular disese and 8(16%) had secondary glomerular disease. Biochemical details are given in table (3), (4), (5) and(6). Percutaneous renal biopsy results are given in table (7).Out of 50 cases of adult glomeruonephritis,41 cases(82%) are primary and 9(18%)cases are secondary glomerulonepritis.

	Primsec		Total
	Primary	Secondary	
15-24	16(32.0%)	2(4.0%)	18(36.0%)
25-34	11(22.0%)	2(4.0%)	13(26.0%)
AGE 35-44	8(16.0%)	2(4.0%)	10(20.0%)
45-44	5(10.0%)	2(4.0%)	7(14.0%)
55 +	1(2.0%)	1(2.0%)	2(4.0%)
Total	**41(82.0%)**	**9(18.0%)**	**50(100.0%)**

Table 1: Age Distribution

	Primsec		Total
	Primary	Secondary	
SEX			
Male	22(44.0%)	4(8.0%)	26(52.0%)
Female	19(38.0%)	5(10.0%)	24(48.0%)
Total	41(82.0%)	9(18.0%)	50(100.0%)

Table 2: Sex prevalence

Sl. No	Pattern	Avg. Proteinuria gm/day	Haematuria
1.	MCD	4.1	0
2.	Membranous GN	4.7	0
3.	MPGN	4.26	1
4.	FSGS	4.51	1
5.	Mesangio proliferative GN	4.76	0
6.	DPGN	5	1
7.	Crescentic GN	4.56	2
8.	IgA nephropathy	3.53	3

Table 3: Urine Examination In Primary GN

Sl. No	Pattern	Avg. Proteinuria gm/day	Haematuria
	Lupus Nephritis	4.2	4
	Diabetic Nephropathy	6.6	0
	HBV associated GN	6.1	0

Table 4: Urine Examination In Secondary GN

Sl. No.	Glomerular Pattern	Hypoalbum -inemia	Hypercholester -olemia
1.	MCD	3(42.8%)	5 (71.4%)
2.	Membranous GN	6(60%)	8(80%)
3.	MPGN	3(100%)	1(33.3%)
4.	FSGS	6(85%)	5(71%)
5.	Mesangio Proliferative GN	3(60%)	4(80%)
6.	DPGN	0	1(100%)
7.	Crescentic GN	1(50%)	2(100%)
8.	IgA nephropathy	2(66%)	1(33.3%)

Table 5: Biochemical Profile Primary GN

Sl. No.	Glomerular Pattern	Hypoalbum -inemia	Hypercholest -erolemia
1.	Lupus nephritis	3(60%)	3(60%)
2.	HBV associated GN	1(100%)	1(100%)
3.	Diabetic Nephropathy	2(66.6%)	3(100%)

Table 6: Biochemical Profile Secondary GN

Sl.No	GN	Cases	%
1.	MCN	7	17%
2.	MGN	10	24.4%
3.	MPGN	3	7.3%
4.	FSGS	7	17%
5.	Mesangioproliferative GN	5	12.1%
6.	DPGN	1	2.45%
7.	Crescentic GN	2	4.9%
8.	Chronic GN	2	4.9%
9.	Mesangial sclerosis	1	2.45%
10.	IgA nephropathy	3	7.3%

Table 7: Pattern Primary GN (41 cases)

DISCUSSION: Nephrotic syndrome describes the association of heavy proteinuria,peripheral oedema, hyploalbuminaemia, and hypercholesterolaemia.Nephrotic syndrome has an incidence of around three cases per 100 000 each year. It is an uncommon manisfetation compared with reduced kidney function as a complication of systemic

diseasses.[1] Dabetes mellitus is the commonest secondary cause and focal segmental glomerulosclerosis and membranous nephropathy are the most common primary causes. Imaging studies are are rarely required. Renal biopsy is required to confirm an underlying disease and identify idiopathic disease that is more likely to respond to corticosteroid management. Treatment includes fluid and sodium restriction, diuretics, and ACE inhibitors. Corticosteroid treatment may be beneficial in some patients.[2] The prevalence of MCD within India is less than 12% in Vellore in the Southern part of the country to approximately 33% in Haryana. FSGS is the second most common cause of nephrotic syndrome with a high incidence of hematuria, hypertension and renal impairment.[3]

Glomerular diseases are an important cause of end-stage renal disease. Pathological spectrum s varies according to age and geography. During the last five decades there is a 5-fold increase in the frequency of FSGS and 3-fold increase in the frequency of MGN. Globally in the incidence of FSGS is increasing.[4] Studies from Delhi found MCD to be the cause for more than one third of nephrotic syndrome. The studies done at vellore found that the incidence of FSGS had increased and it became the most common etiology for primary nephrotic syndrome. In Kolkata, Golay et al., found that FSGS was the most common one and membranous nephropathy was third most common (25%).[5,6]

While studies from Pakistan and Nepal have shown that IgA Nephropathy is an infrequent cause of nephrotic syndrome with figures of around 2% the ones from China have found it to be very common. Chang et al., observed that IgA Nephropathy was responsible for 28% of nephrotic syndrome making it the most common cause in Korea. Zhou et al., found IgA Nephropathy to be the second most common cause of nephrotic syndrome after membranous nephropathy accounting for 20% of cases in China. Studies from Pakistan have found FSGS to be the most common cause (40%) of their cases, followed by MGN (26.6%) and MCD (14.8%). Amerciacan studies have demonstrated an increase in incidence of FSGS in African-Americans making it the most common cause of nephrotic syndrome in their adult population.[7,8] FSGS as cause of end-stage renal disease in USA has increased in the last two decades. Various Studies done from other parts of the world have shown FSGS to be the most common cause of adult nephrotic syndrome. Incidence in children is also increasing and in Indian pediatric patients FSGS is the most common cause in adolescents as compared with minimal change disease in younger patients. European data do not agree with this trend. Italian studies have shown membranous nephropathy as the most common cause of adult nephrotic syndrome while those from Denmark have shown minimal change disease, IgA nephropathy and MPGN respectively to be the most common lesions.

Women with MGN and FSGS progress slowly and have better survival.[9] There has been great progress in our understanding of the pathogenesis of a number of glomerular diseases. Novel diagnostic and prognostic tests are being developed and newer therapeutic agents are being evaluated.[10,11]

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