

GLOMERULAR LESIONS CAUSING NEPHROTIC SYNDROME (LIGHT MICROSCOPIC FINDINGS) IN 75 ADULT PATIENTS

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This study includes seventy-five adult patients of Nephrotic syndrome who underwent successful Renal Biopsy in order to determine the Glomerular pathology on light microscopy. More than 90% patients had primary glomerular lesions and the rest had secondary glomerular involvement.

Focal and segmental proliferative glomerulonephritis (38%) and membranous glomerulonephritis (30%) were found to be the commonest lesions. Diabetes was the commonest (5%) cause of secondary glomerular lesions.

INTRODUCTION

Nephrotic syndrome is defined as a clinical state characterized by massive proteinuria (more than 3.5/24 hrs/1.73 m²) and hypoalbuminemia (30 Gm/L).¹

It results from diseases primarily involving the glomeruli or it occurs as feature of a systemic disease secondarily involving the kidney. The primary glomerular lesions causing nephrotic syndrome (idiopathic nephrotic syndrome) are divided into some well-defined groups based mainly on light microscopic appearance and is supplemented by immunofluorescence and electron microscopic studies and it includes the following:^{2,3}

- i. Minimal light microscopy changes.
- ii. Focal proliferative glomerulonephritis.
- iii. Focal and segmental glomerulosclerosis.
- iv. Membranous glomerulopathy.
- v. Diffuse mesangial proliferative glomerulonephritis (IgA and IgM)
- vi. Diffuse endocapillary proliferative glomerulonephritis.
- vii. Mesangiocapillary glomerulonephritis (type I and II).
- viii. Crescentic glomerulonephritis.

MATERIALS AND METHODS

This study was carried out in Hayat Shaheed Teaching Hospital, Peshawar. All adult patients (>15 years) with the established diagnosis of nephrotic syndrome and no contraindications to renal biopsy were included in the study.

History and physical examination of the patients was undertaken and questions related to use of medicine, chronic illness and allergy were asked. Examination was aimed at assessing the severity of disease and looking for signs leading to aetiology.

Investigations included Haemoglobin estimation, white cell count (total and differential), x-ray Chest, Blood Urea & Sugar, Urine routine examination, 24 hours' urinary protein estimation, total

serum proteins, serum albumin, creatinine, triglycerides and cholesterol was also done. In selected cases antinuclear factor and anti-double strand DNA were ordered.

Renal biopsy was attempted on all patients considered fit for the procedure. Before performing renal biopsy each patient had intravenous urogram⁴, coagulation profile study and platelet count. Informed verbal consent was taken from each patient. All the renal biopsies were performed with Tru-Cut biopsy needle of appropriate size after marking kidney surface anatomy with the help of intravenous urogram. The lower pole of left kidney was biopsied in all cases. After the biopsy patients were observed in the ward for at least 24 hours for possible complications. The specimen was fixed in 10% formalin and was processed as for paraffin embedding. The paraffin embedded tissue was sectioned serially at 2-3-micron thickness and stained with Hematoxylin and Eosin (H and E), Periodic Acid Schiff (PAS), and, Congo Red Stains. The stained slides were then examined under the light microscope.

RESULTS

The study consisted of seventy-five adult patients of nephrotic syndrome with age ranging between 15 to 56 years.

A variety of primary glomerular lesions were found in patients of nephrotic syndrome. (Table-1).

Focal and Segmental proliferative glomerulonephritis (38%) and membranous nephropathy (30%) were found to be the commonest primary glomerular lesions. Other primary glomerular lesions included Mesangiocapillary glomerulonephritis (10%), focal glomerular sclerosis (6%), minimal change lesion (4%), mesangial proliferative glomerulonephritis (2%), and chronic progressive glomerulonephritis (2%).

Seven patients had nephrotic syndrome due to systemic diseases secondarily involving the kidney (Table 2). Four of them had diabetic nephropathy causing nephrotic syndrome. One female patient had systemic lupus erythematosus with membranous nephropathy on renal biopsy. Two patients having chronic pulmonary tuberculosis had changes of amyloidosis on renal biopsy.

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Majority of patients in our study were male (n = 48) and belonged to the age group of 15 to 35 years, with mean age of 27.42 years (range 15 to 56 years).

TABLE-1: PRIMARY GLOMERULAR LESION CAUSING NEPHROTIC SYNDROME

Histological Type of Lesions	No. of Patients	Percentage of Total (n=75)
Focal and segmental proliferative glomerulonephritis	28	38
Membranous glomerulonephritis	23	30.5
Focal Glomerular sclerosis	4	6
Mesangiocapillary glomerulonephritis	7	10
Minimal change disease	3	4
Mesangial proliferative glomerulonephritis	1	0.75
Chronic progressive glomerulonephritis	2	2.5

TABLE-2: SECONDARY GLOMERULAR LESION CAUSING NEPHROTIC SYNDROME

Histological lesions	No. of Patients	Percentage of Total (n=75)
Diabetic nephropathy	4	5
Lupus nephritis (Membranous)	1	0.75
Renal amyloidosis	2	2.5

The age distribution of patients was as follows:

Age (years)	No of patients
15-24	36
25 - 35	30
> 35	09

Mortality was nil in our study. Most of the patients complained of pain at biopsy site and required mild analgesics. Four patients had Macroscopic haematuria which subsided spontaneously in 24 - 48 hours' period. One patient developed unexplained fever within 6 hours of the biopsy and was treated successfully with antibiotics.

DISCUSSION

Nephrotic Syndrome is a common disease and is due to a variety of causes. Apart from primary glomerular diseases, at least a hundred specific aetiological agents or disease association have been described³. Majority of our patients had primary glomerular lesions and only about 8% (n=7) had secondary nephrotic syndrome.

Our study showed preponderance of male patients as reported by others⁶.

Majority of our patients had proliferative glomerulonephritis (38%). An earlier study from Karachi by Sadiq et al have reported primary

glomerulonephritis to be the cause of nephrotic syndrome in 32% of their patients⁷. This is an expected finding due to increased prevalence of infections in our country and is supported by studies from other developing countries⁸.

Membranous nephropathy was the next common glomerular pathology (30%). This is a high figure as compared to that reported by Haq *et al.*,⁹ which may be due to the difference in the studied population, as the later included patients of nephrotic syndrome as well as acute glomerulonephritis, while in the present study only patients of nephrotic syndrome were included. There are many causes of membranous nephropathy including Hepatitis B Virus infection, carcinoma and heavy metal ingestion. The former may be the commonest association, as hepatitis B is very common in this community and 50% of our admitted patients of hepatitis are positive for hepatitis B surface antigen¹⁰.

Four of our patients had nephrotic syndrome due to diabetic nephropathy, two patients had renal amyloidosis and another had lupus nephritis causing nephrotic syndrome. This comprises about 8% of our patients having secondary glomerular disease which is low as compared to other studies^{5,11,12}. This was probably due to involvement of our patients at a younger age without having other disorders which could cause nephrotic syndrome. Most of our patients had age between 20 and 35 years, and had proliferative glomerulonephritis on renal biopsy. This is alarming because of poor prognosis of this type of glomerulonephritis and the prime age of the patients.

Although our study is a useful beginning in finding out the glomerular lesions causing nephrotic syndrome, but it consists of small number of patients and only light microscopy was used for histopathological examination. More extensive studies with immunofluorescence and electron microscopy would be required to thoroughly address the problem.

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