

Research Article

Article Info:

Received on: 27/01/2016
Accepted on: 25/02/2016



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ABSTRACT :

Nephrotic syndrome is commonest renal diseases in developing country. The nephrotic syndrome is a clinical syndrome complex characterized by a number of renal and extra renal features, the most prominent of which are proteinuria $>3.5\text{g}/1.73\text{m}^3/24\text{h}$ (in practice $>3.0\text{-}3.5\text{g}/24\text{h}$), hyperlipidemia, lipiduria, and hyper coagulability.

The present study was planned in North Indian Hospital. The total 20 patients referred to Out-Patient Department (OPD) and in-patient department (IPD). All the patients were subjected to renal biopsy.

The Swelling of feet and puffiness of face is found in about 8 patients out of 20. Weakness, loss of appetite, and generalized bodyache is seen in 4 patients. Distension of abdomen and difficulty in breathing is observed in 3 patients. Pallor and dizziness is seen in 2 cases and Decreased urinary volume is observed in 3 patients.

The most common primary glomerular diseases causing nephrotic syndrome in children and adults are minimal change diseases and membranous glomerulonephritis, respectively.

Key-words: Nephrotic syndrome, Histological study.

INTRODUCTION:

Nephrotic syndrome is a group of symptoms that include protein in the urine, low blood protein levels, high cholesterol levels, high triglyceride levels, and swelling. Nephrotic syndrome is caused by different disorders that damage the kidneys. This damage leads to the release of too much protein in the urine.

Nephrotic syndrome is not a disease in itself; rather, it is a group of symptoms that indicate kidney damage—particularly damage to the glomeruli, the tiny units within the kidney where blood is filtered result in the release of too much protein from the body into the urine. When the kidneys are damaged, the protein albumin, normally found in the blood, will leak into the urine. Proteins are large, complex molecules that perform a number of important functions in the body [1].

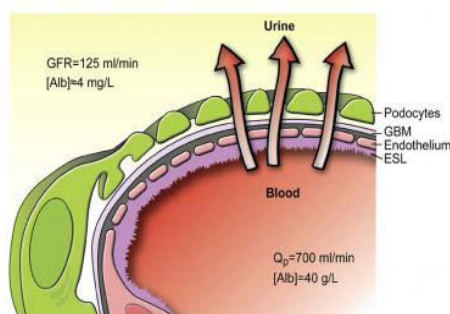


Figure 1 : protein in the urine through glomerular barrier

In order to establish the presence of nephrotic syndrome, laboratory tests should confirm the existence of nephrotic-range proteinuria, hypoalbuminemia,

and hyperlipidemia. Therefore, initial laboratory testing should include the following:

- Urinalysis
- Urine protein quantification
- Serum albumin
- Lipid panel

The following tests should be performed to determine whether the nephrotic syndrome is idiopathic or secondary and, if INS has been determined, whether signs of chronic kidney disease, kidney insufficiency, or other signs exclude the possibility of minimal change nephrotic syndrome (MCNS):

- Complete blood count (CBC)
- Metabolic panel - levels of serum electrolytes, calcium, phosphorus, and ionized calcium, as well as of blood urea nitrogen (BUN) and creatinine
- Testing for HIV
- Testing for hepatitis B and C
- Complement studies (C3, C4)
- Antinuclear antibody (ANA), anti-double-stranded DNA antibody (in selected patients)

Other tests and procedures in selected patients can include the following [2]:

- Genetic studies
- Kidney ultrasonography
- Chest radiography

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Conflict of interest: Authors reported none



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- Mantoux test
- Kidney biopsy

Some degree of correlation between lipids and serum albumin as suggested by Thomas et al and between lipidemia and edema by Peters et al generally, when edema regresses. Lipid levels fall but in some cases, it may continue to persist even after the edema has disappeared. Hyperlipidemia usually observed during the active phase of the disease and disappears with resolution of proteinuria. Hyperlipidemia may contribute to renal injury and Experimental studies demonstrated that reduction of plasma lipids level slows progression of Glomerular and Tubulointerstitial disease [3,4].

Hyperlipidemia is usually observed during the active phase of the disease and disappears with resolution of proteinuria. However, it may persist in some cases, leading to increased risk of atherosclerosis in later life. Hence, close monitoring of lipid levels during remission of nephrotic syndrome is necessary to select high-risk patients⁵.

Lipoproteins play an important role in the transport of plasma lipids; their increase or alteration in various fractions may be responsible for hypercholesterolemia, in nephrotic syndrome. There is increased total cholesterol, LDL cholesterol, VLDL cholesterol and triglycerides and normal or low HDL cholesterol⁶.

More recently it has been expressed that hyperlipidemia may contribute to renal injury [5]. And experimental studies demonstrated that reduction of plasma lipid levels slows progression of glomerular and tubulointerstitial disease [6]. A great deal of evidence is now available to show that the incidence of Nephrotic Syndrome varies from place to place due to changes in food habits, climate, type of work and ethnic origin. It has also been noted that certain factors like diet, malnutrition, genetic traits etc., are known to alter the frequency and severity of lipid pattern. The Indian patient has a different dietary, constitutional and genetic background.

Hence, we undertook a study to determine the clinicopathological correlation in primary nephrotic syndrome patients.

MATERIALS AND METHODS [7]:-

The present study was planned in North Indian Hospital. The total 20 patients referred to Out-Patient Department (OPD) and in-patient department (IPD). The aim and the objective of the study were conveyed to patients. All the patients were subjected to renal biopsy.

Inclusion Criteria:

- Patients having nephrotic syndrome
- Hyper proteinuria

Exclusion Criteria:

- Parents having other diseases
- Patients on Diuretic therapy
- Diabetes, renal failure and urinary tract infection.

RESULTS AND DISCUSSION:-

The data obtained from the 20 enrolled patients is presented as below.

Table 1: Age & number of patients:

Age in yrs	Group I
21-30 yrs	9
31-40 yrs	5
41-50 yrs	3
51-60 yrs	3
Total	20

Table 2: Observed Symptoms

Age in yrs	Group I
Swelling of feet and puffiness of face	8
Distension of abdomen and difficulty in breathing	3
Weakness, loss of appetite, and generalized bodyache	4
Pallor and dizziness	2
Decreased urinary volume	3
Total	20

The Swelling of feet and puffiness of face is found in about 8 patients out of 20. Weakness, loss of appetite, and generalized bodyache is seen in 4 patients. Distension of abdomen and difficulty in breathing is observed in 3 patients. Pallor and dizziness is seen in 2 cases and Decreased urinary volume is observed in 3 patients.

Table 3: Histopathology of renal biopsy

Histopathological Observation	Number of Observation
Minimal change glomerulonephritis	11
Membranoproliferative glomerulonephritis	2
Focal segmental glomerulosclerosis	1
Membranous glomerulonephritis	3
Diffuse proliferative glomerulonephritis	2

The most common primary glomerular diseases causing nephrotic syndrome in children and adults are minimal change diseases and membranous glomerulonephritis, respectively [8]. Recently it has been found that Focal segmental glomerulosclerosis is most common cause of primary nephrotic syndrome in children and adults[9].

Control of proteinuria is therefore essential to prevent the progression of renal diseases to chronic renal failure [10].

The most common cause of primary nephrotic syndrome is minimal change diseases, edema being the

commonest presenting sign.

CONCLUSION:-

Establishing the type of glomerular diseases is essential from the therapeutic point of view and for predicting the prognosis. Therefore, renal biopsy should be performed particularly in adult patients of nephrotic syndrome.

REFERENCES:-

1. <http://www.niddk.nih.gov/health-information/health-topics/kidney-disease/nephrotic-syndrome-in-children/Pages/index.aspx>
2. <http://emedicine.medscape.com/article/982920-overview>.
3. J.P Peters, E.B Man, The inter relationship of Serum lipids in patients with diseases of kidneys, J Clin Invest, 22, 1943,721.
4. Bhandari, Mandowara. Lipoprotein profile in nephrotic syndrome. Indian Paediatrics 1980 17:416-9.
5. Moorhead JF, Chan MK, Nahas AM, Varghese Z (1982). Lipid nephrotoxicity in chronic progressive glomerular and tubulo interstitial disease. Lancet; 2: 1309-11.
6. Keane WF, Peter JV, Kasiske BL, Kim Y(1991). The role of altered lipid metabolism in the progression of renal disease. Am J Kidney Dis; 17: 38-42.
7. Singh GK, Singh SN, Niroula D, Singh IY, Debnath K, Sharma RL. Clinicopathological correlation in primary nephrotic syndrome. J Med Soc 2014;28:180-4.
8. Broyer M, Meyrier A, Naiduet P. Minimal changes and focal segmental glomerulosclerosis. In: Davidson AM, Cameron JS, Grunfeld JP, Kerr NS, Ritz E, Winearls CG. Editors. Oxford textbook of clinical nephrology. 2nd ed., vol.1. London: Oxford University press; 1988. p. 493-535.
9. Cameron JS, Turner DR, Ogg CS, Chantler C, Williams DS. The long term prognosis of patients with focal segmental glomerulosclerosis. Clin Nephrol 1978;10:213-8.
10. Burton C, Harris KP. The role of proteinuria in the progression of chronic renal failure. Am J Kidney Dis 1996;27:765-75.