A Study of Biochemical Status of Serum and Urine in Patients of Nephrotic Syndrome

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Abstract

Introduction: Nephrotic syndrome (NS) is caused by increased permeability of the glomerular capillary wall for proteins. *Aims and Objectives*: To Study biochemical status of serum and urine in patients of Nephrotic Syndrome. *Methodology*: The present study was carried out in department of Pathology, ACPM Medical College Dhule. The period of study was one year. Diagnosed cases of nephrotic syndrome of different age and sex in the hospital during one year period were included in study. Diagnosis of nephrotic syndrome was recommended by international study of kidney disease for diagnosis. *Result:* Age and sex wise distribution of 84 cases of nephrotic syndrome is tabulated in table. Maximum number of patient 54(64.28%) were-observed in 0-10 years age group prevalence of nephrotic syndrome decreases as age advanceThe various Biochemical serum values were (Mean ±SD) i.e. Total proteins (g/dl) were 3.32 ± 2.6 and Albumin (g/dl) was 1.52 ± 0.9 ; Sr. Cholesterol (mg/dl) was 235 ± 1.2 and Total antioxidants (mmol/L) were 1.77 ± 0.92 ; Homocysteine (umol/L) was 18.11 ± 4.12 and Vit.C (mg/L) was 0.52 ± 0.42 . The majority of the Patients were having Urine Protein values i.e. Total urine protein g/24 hrs. in 2-2.9 range were 33(39.28%) followed by 3-3.9 were 33. (39.28%); 4-6 were 17(20.23%) and >6 were 1(1.19%). *Conclusion:* In Nephrotic Syndrome, various serums biochemical values like total Proteins, Total antioxidants and Vit.C levels were decreased while Homocysteine level was increased and in urine there was proteinuria.

Keywords: Total Proteins; Total Antioxidants; Homocysteine; Proteinuria.

Introduction

Nephrotic syndrome (NS) is caused by increased permeability of the glomerular capillary wall for proteins [1]. Nephrotic syndrome is the common chronic disorder characterized by alteration of permeability of the glomerular capillary wall, resulting in its inability to restrict the urinary loss of proteins [2]. Nephrotic syndrome is a stressful condition for children where oxidative damage would also influence the response of these patients to therapy [3]. The production of free radicals can cause renal injury and play an important role in the pathogenesis of Nephrotic syndrome [4]. Excessive generation of reactive oxygen species is one of

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the incriminated mechanisms in the pathogenesis of progressive renal injury. The role of oxidant stress in acute and chronic glomerular diseases has been investigated through experimental and clinical studies [5]. Nephrotic syndrome is characterized by heavy proteinuria and hypoalbuminemia. Reactive oxygen species (ROS) seem to play an important role in the etiopathogenesis of proteinuria in Nephrotic Syndrome. The potential role of reactive oxygen species in pathogenesis of Nephrotic Syndrome by estimating the levels of oxidants and antioxidants in Nephrotic Syndrome [6]. The atherothrombotic risk pattern of the Nephrotic syndrome resembles that of Hyperhomo-cysteinemia [7]. Proteinuria and Hyperhomocysteinaemia are independently associated with increased risk of atherosclerosis and cardiovascular disease [8].

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Aims and Objectives

To Study biochemical status of serum and urine in patients of Nephrotic Syndrome.

Methodology

The present study was carried out in department of Pathology, ACPM medical ,Dhule. The period of study was one year. Diagnosed cases of nephrotic syndrome of different age and sex in the hospital during one year period were included in study. Diagnosis of nephrotic syndrome was recommended by international study of kidney disease used was as follows(Serum Proteins more than 3 gm in 24 hrs/ 1.73 m²/more than 40 mg/hr/m²) (serum cholesterol more than 200 mg/dl)(hypoalbuminaemia-serum albumin less than 2.5 g/dl).

Result

Age and sex wise distribution of 84 cases of nephrotic syndrome is tabulated in above table.

Maximum number of patient 54(64.28%) wereobserved in 0-10 years age group. Of this 54 patients, 28(51.85%) were male and 26 (48.14%) were female. So male female ratio is 1.07:1

The prevalence of nephrotic syndrome decreases as age advance. This is evident from above table number 1.13(15.4%) patients were seen in 11-20 years' age group of which, 7 patients were male and 6 patients were female. So male female ratio is 1.16:1. In age group of 21-30 years, 10(11.90%) patients were observed, of which 6 patients were male 4 patients were female. So male female ratio is 1.5:1 minimum number of patients i.e. only 7(8.33%) were noted in age group 31-60 years with same male female ratio.Out of overall 84 patients, 45 patients were male and 39 patients were female. So male female ratio is 1.15:1.

The various Biochemical serum values were (Mean \pm SD) i.e. Total proteins (g/dl) were 3.32 ± 2.6 and Albumin (g/dl) was 1.52 ± 0.9 ; Sr.Cholesterol (mg/dl) was 235 ± 1.2 and Total antioxidants (mmol/L) were 1.77 ± 0.92 ; Homocysteine (umol/L) was 18.11 ± 4.12 and Vit.C (mg/L) was 0.52 ± 0.42 .

The majority of the Patients were having Urine

Table 1: Distribution of nephrotic syndrome according to age and sex

Age (in yrs.)	Male Patients	Female Patients	Total	M/F Ratio
0-10 yrs.	28(48.14%)	26(48.14%)	54(64.28%)	1.07:1
11-20 yrs.	7(53.84%)	6(46.15%)	13(15.47%)	1.16:1
21-30 yrs.	6(60.00%)	4(40.00%)	10(11.90%)	1.5:1
31-60 yrs.	4(57.14%)	3(42.85%)	7(8.33%)	1.3:1
Total	45(53.57%)	39(46.42%)	84(100%)	1.15:1

Serum Values	Mean ±SD (n=84)	
Total proteins (g/dl)	3.32 ± 2.6	
Albumin (g/dl)	1.52 ± 0.9	
Sr.Cholesterol (mg/dl)	235 ± 1.2	
Total antioxidants (mmol/L)	1.77 ± 0.92	
Homocysteine (umol/L)	18.11 ± 4.12	
Vit.C (mg/L)	0.52 ± 0.42	

Table 3: Distribution of total 24 hours urine protein

Total urine protein g/24 hrs.	No. of cases (%)
2-2.9	33(39.28%)
3-3.9	33.(39.28%)
4-6	17(20.23%)
>6	1(1.19%)
Total	84 (100%)

Protein values i.e. Total urine protein g/24 hrs. in 2-2.9 range were 33(39.28%) followed by 3-3.9 were 33. (39.28%); 4-6 were 17(20.23%) and >6 were 1(1.19%).

Discussion

Nephrotic syndrome is a syndrome comprising signs of nephrosis, chiefly proteinuria, hypoalbuminemia, and edema [13,14]. It is a component of glomerulonephrosis, in which different degrees of proteinuria occur. Essentially, loss of protein through the kidneys (proteinuria) leads to low protein levels in the blood (hypoproteinemia including hypoalbuminemia), which causes water to be drawn into soft tissues (edema). Very low hypoalbuminemia can also cause a variety of secondary problems, such as water in the abdominal cavity (ascites), around the heart or lung (pericardial effusion, pleural effusion), high cholesterol (hence hyperlipidemia), loss of molecules regulating coagulation (hence increased risk of thrombosis)[13].

In our study The various Biochemical serum values were (Mean \pm SD) i.e. Total proteins (g/dl) were 3.32 \pm 2.6 and Albumin (g/dl) was 1.52 \pm 0.9 and Sr. Cholesterol (mg/dl) was235 \pm 1.2 these findings are similar to study of Jyoti Dwivedi [9].

Total antioxidants (mmol/L) were 1.77 ± 0.92 ;Disturbances in oxidant and antioxidant status were observed by many other studies, which was in agreement of our study Warwick et al [10].

Homocysteine (umol/L) was 18.11 ± 4.12 and Vit.C (mg/L) was 0.52 ± 0.42 these findings are as in the study of Sydow et al., Van Guldener et al. [11,12]. Hyperhomocysteinemia persists in the majority of patients. Primary (fasting) Hyperhomocysteinemia can be treated with folic acid (0.5-5 mg/day).

Conclusion

In Nephrotic Syndrome , various biochemicaql values like Total Proteins, Total antioxidants and Vitamin C levels were decreased while Homocysteine levels was increased and in urine there was proteinuria.

References

- Tesar V, Zima T. Recent progress in the pathogenesis of nephrotic proteinuria. Crit Rev Clin Lab Sci. 2008; 45(2):139-220.
- 2. Ghodake SR, Suryakar AN. Role of reactive oxygen species in pathogenesis of nephrotic syndrome. Indian J ClinBiochem. 2010 Jan; 25(1):82-5.

- 3. Kamireddy R, Kavuri S, Devi S, Vemula H, Chandana D, Harinarayanan S, James R, Rao A. Oxidative stress in pediatric nephrotic syndrome. ClinChimActa. 2002 Nov; 325(1-2):147-50.
- 4. Mishra OP, Gupta AK, Prasad R, Ali Z, Upadhyay RS, Mishra SP, Tiwary NK, Schaefer FS. Antioxidant status of children with idiopathic nephrotic syndrome. PediatrNephrol. 2011 Feb; 26(2):251-6.
- 5. Bulucu F, Vural A, Aydin A, Sayal A. Oxidative stress status in adults with nephrotic syndrome. ClinNephrol. 2000 Mar; 53(3):169-73.
- Ghodake SR, Suryakar AN, Ankush RD, Katkam RV, Shaikh K, Katta AV. Role of free radicals and antioxidant status in childhood nephrotic syndrome. Indian J Nephrol. 2011 Jan; 21(1):37-40.
- 7. Arnadottir M, Hultberg B, Berg AL. Plasma total homocysteine concentration in nephrotic patients with idiopathic membranous nephropathy. Nephrol Dial Transplant. 2001 Jan; 16(1):45-7.
- Aminzadeh MA, Gollapudi P, Vaziri ND. Effect of nephrotic syndrome on homocysteine metabolism. Nephrol Dial Transplant. 2011 Apr; 26(4):1244-7.
- 9. JyotiDwivedi, Purnima Dey Sarkar. The Study of Oxidant and Antioxidant Status with Homocysteine, Total Protein and Albumin in Nephrotic Syndrome. Int J Med Health Sci. 2014 Jan; 3(1):18-21.
- 10. Warwick GL, Waller H, Ferns GA. Antioxidant vitamin concentration and LDL oxidation in nephrotic syndrome. Ann ClinBiochem 2000; 37(pt4):488-491.
- 11. Sydow K, Boger RH Homocysteine, endothelial dysfunction and cardiovascular risk: Pathomechanism and therapeutic options. Z. Kardiol 2001; 90(1):1-11
- 12. Van GC, Stehonwer CD. Homocysteine lowering treatment: an overview. Expect opin Pharmacotherapy 2001; 2(9):1449-1460.
- Dunphy, Lynne M; Winland-Brown, Jill; Porter, Brian; Thomas, Debera (19 February 2015). Primary Care. F.A. Davis. p. 634. ISBN 9780803644946.
- 14. http://wordnetweb.princeton.edu/perl/ webwn?s=nephrotic%20syndrome