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# HISTOPATHOLOGICAL SPECTRUM OF PAEDIATRIC RENAL DISEASE IN AND AROUND VARANASI

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## ABSTRACT

Morphological pattern of renal disease varies from place to place and is governed by mostly environmental factors and to some extent by genetic factor. Aim of the study was to find out morphological spectrum of renal diseases in children and its correlation with clinico-pathological features. Histopathological study of renal biopsies showed that mesangioproliferative(glomerulonephritis(GN) were the most common GN in children seen in 31.10% cases. Next common GN was FSGS(Focal segmental glomerolosclerosis,18.8%)followed by minimal change disease GN, Membranoproliferative GN (11.29%) each, Diffuse proliferative GN (9.67%),Cresentic GN (4.30%), membranous GNand end stage renal disease(2.15%)each. Among Secondary GN, lupus nephritis (LN)was the most common. Hyaline thrombi in glomerular capillaries were most common in membranous GN(50%) followed by LN(20.17%) and Minimal change GN (14.28%) and FSGS each. Microscopic hematuria was

most common in CGN (62.50%). Granuloma in interstitium were found in more than 1/3rd cases of MPGN (38%), followed by cresentic GN (37.50%), Lupus nephritis (13.33%) and diffuse mesangioproliferative GN (12%). Thrombi in medium sized blood vessels was seen in 50% cases of MGN, 16.66% cases of diffuse proliferative GN, 14.28% cases of FSGS & 50% cases of pyelonephritis. Thus our study concludes that Mesangio proliferative GN is the most common in paediatric GN. Presence of more venulitis, vasculitis & thrombosis in blood vessels of MGN suggest that probably vascular lesion due to both cell mediated immunity (venulitis) and humoral immunity (vasculitis) leading to thrombosis may be involved in pathogenesis of MGN.

**KEYWORDS:** Renal lesions in pediatric age group, glomerulonephritis, membranousGN, Lupusnephritis, membranoproliferative GN, mesangioproliferative GN.

## INTRODUCTION

The prevalence of childhood diseases varies from one area to another area in the same country.[1,2,3] It varies due to genetic and environmental factor and advances in technology.[4] Kidney biopsy in paediatric age group is performed to identify children with chronic glomerular diseases[5], recurrent nephrotic syndrome and steroid resistant nephrotic syndrome.

Kidney biopsy not only help in diagnosis but also has a role in treatment of disease[6] with the combined help oflight microscopy ,immunofluorescence and electron microscopy, renal pathologist is able to classify the glomerulonephritis(GN) and provides better clinic-pathological correlation. Kidney biopsy also help in knowing the pathogenesis.[7] There are only few studies of spectrum of renal diseases in children in India.Main aim of study was to describe the histopathological pattern of childhood glomerulonephritis.

## MATERIAL AND METHODS

Total 186 renal biopsy were analysed between period of January 2010 to October 2016.All cases were taken from outdoor and indoor of department of nephrology and department of paediatrics of Sir Sunderlal Hospital ofInstitute of Medical Sciences, Banaras Hindu University. This was the retrospective analysis of biopsy was performed by Tru cut needle in USG guidance. Formalin fixed tissue were processed & paraffin blocks were prepared. Sections were stained with haematoxylin & Eosin, PAS and, Acid fuschin orange G(AFOG) stain[8] to see the deposits in kidney biopsy. Direct immunofluorescence test was done in

paraffin embedded section after treatment with proteinase K.[9] Briefly method is as following Tris EDTA buffer of pH 8,0.05 M was made after dissolving Tris buffer 6.06 gm, EDTA,0.37 gm in 1liter of distilled water .pHof 9.00 was adjusted by concentrated HCl.

Stock proteinase K solution 0.04% was prepared by dissolving 4 mg proteinase K in 10 ml of Tris EDTA buffer pH 8.0. Stock solution was stored in small solution aliquots (60ml). At the time of test Stock solution was diluted 1:20 by dissolving 0.04% Proteinase K solution into distilled water (DW) (100µpK 0.4 %+ 1900ml DW). Slides were heated in incubator at $37^0$c overnight--Next morning itwas kept in xylene for 45 minutes-Slides were blotted & put in descending graded alcohol then brought to running water -Slides were dried & about 100µl of working solution of Proteinase K was poured over the section & kept it in moist chamber at $40^0$c for 30minutes-Slides were washed with several changes of DW and then kept in phosphate buffered saline (PBS pH7.2.0.01 M) for 10 minutes. Slides were dried in air, about 100µl of FITC conjugate anti human IgG, IgM, IgA, C3 & C4 were diluted in 1:40 dilution in PBS & were poured over the biopsy tissue which was kept in moist chamber for 30 minutes in dark. After 30 minutes slides were washed twice in PBS in dark and mounted in PBS: glycerine (1:9) media, Edges of cover slips were sealed by nail polish. Biopsy slides were seen in Nikon upright fluorescent trinocular microscope. In positive area apple green fluorescence, Biopsy was reported as per World Health Organization classification.[10]

Clinical features were recorded 1995 in all cases. Grosshematuria was defined when urine appeared red by naked eye, microscopic haematuria was defined when more than 5 red blood cells were present per high power field. Nephrotic syndrome was called when more than 40mg/$m^2$/hrprotein excretion was there along with oedema and hypoalbuminemia (less than 2.5 g/dl) Resistance to steroid was defined as persistent proteinuria after4 week treatment with prednisolone. Steroid dependence was defined when relapse of nephrotic syndrome occurred within 2 weeks after stopping prednisolone therapy .Systemic lupus erythematosus was diagnosed with criteria laid down by American College of Rheumatology 1997. Diagnosis of Amyloidosis was confirmed by Congo red staining.

**RESULT**

Study included total 186 cases in which 109 cases (58.60%) were males and 77(41. 39%) were females. In histological types, mesangioproliferative GN formed the maximum number of cases (30.10%). It was splitted into two groups Focal mesangial proliferative GN(16.66%) & Diffuse mesangial proliferative GN (13.44%). In focal MesP GN, predominant picture was

of minimal change GN but 25% glomeruli showed segmental proliferation of Mesangial cells while in diffuse variety more than 60% glomeruli showed diffuse proliferation of mesangialcells. Next common GN was FSGS which was found in18.8% cases. Minimal change GN and Membrano proliferative GN formed 11.29% cases each. Both groups presented with nephroticsyndrome. Diffuse proliferative GN was found in 9.6% cases &cresentic GN was seen in 4.30% cases. Membranous GN & ESRD were notice in only 4 cases (2.15%)each. Amongsecondary GN, lupus nephritis formed 8.06% followed by pyelonephritis (1.07%) & one each of amyloid nephropathy& diffuse mesangiosclerosis. Analysis of GN according to gender, showed marked male predomince. All cases of membranous GN(100%),77.4% patients of Diffuse mesangioproliferative GN, 72% Patients of diffuse mesangioproliferative GN & 52.3% patients of minimal change GN were males. Female predominance was found in pyelonephritis (100%), Cresentic GN (62%) & Lupus nephritis (60%) Table I.

Oedema feet and or face was the main manifestations in all cases of renal patients. Fever was more common in lupus nephritis (46.66%),followed by membranous GN (25%), membranoproliferative GN(14.28). Oliguria was the commonest manifestation of End stage kidney (100%) & cresentiic GN (37.50%). About 9.67% cases of focal mesangioproliferative GN & 8.57% cases of FSGS also showedhematuria. Rashes (26.66%) & oral ulcer (33.33%) were seen in SLE. One case of minimal change GN & one case of focal mesangio proliferative GN also showed rashes. Amenorrhoea was found only in mesangioproliferative GN (19%) & one case of FSGS. Arthritis was again more common in SLE (33.33%) Table II. Histological hyaline thrombi in glomerular capillaries were more common in membranous GN (50%), Lupus nephritis (20%) & minimal change GN (14.28%) while thrombi in arcuate blood vessels and or venules were seen in all cases pyelonephritis, 50%cases of MGN,16.6% cases of diffuse Proliferative GN,14.2% cases of FSGS,17.6% cases of mesangioproliferative GN & 13.3% cases of LN. Foam cells in tubules were seen in 57% cases of MPGN, 14.28% cases of FSGS &minimal change GN & 11% cases mesangioproliferative GN. Vasculitis of small blood vessels (capillaries and venules) was seen in 50% cases of membranous GN, 28.5% cases of MPGN,41.76% cases of mesangioproliferative GN & 26.6% patients of lupus nephritis. Focal tubular necrosis was seen in all cases of CGN, 71.42 % of MPGN & 6.0% cases of diffuse proliferative GN & LN. RBC in tubules were found in 62.5% cases of CGN,53.38% Patients of Lupus nephritis 42.85% patients of FSGS, 28% patients of minimal change GN & mesangioproliferative GN. Leucocytoturiawas seen in all cases of cresentic

GN, pyelonephritis, 38.09% cases of MPGN, 25% Mesangioproliferative GN& 50% cases of ESRD. About 20% cases of FSGS, Diffuse proliferative GN, Lupus nephritis also had Leucocytoturia.

Non caseating granuloma was found in 38% patients of MPGN & 32% Patients of CGN &12 to 13% patients ofmesangioproliferative GN & Lupus nephritis. Mononuclear cell infiltrates were seen in all cases of ESRD, pyelonephritis, CGN, MPGN and 77.14% cases of MPGN. About 28.5% cases of minimal change GN also had mild degree of infiltration in interstitium.(Table III).

**Table I: Showing age and sex wise distribution of patients of various renal diseases.**

| **Groups of Renal disease** | **Male n=109 no. %** | **Female n=77 no. %** | **Total no of cases= 186 No.** | **Percentage %** |
|---|---|---|---|---|
| FSGS(35) | 21.0<br>60.0 | 14.0<br>40.0 | 35 | 18.81 |
| Focal mesangioproliferative GN(24) | 24.0<br>77.41 | 7.0<br>22.58 | 24 | 16.66 |
| Diffuse mesangioproliferative GN(25) | 18.0<br>72.0 | 7.0<br>28.0 | 25 | 13.44 |
| Minimal change GN(21) | 11.0<br>52.38 | 10.0<br>47.61 | 28 | 11.29 |
| Membranoproliferative GN(21) | 10.0<br>47.61 | 11.0<br>52.38 | 21 | 11.29 |
| Diffuse proliferative GN(18) | 8.0<br>44.44 | 10.0<br>55.55 | 18 | 9.17 |
| Lupus nephritis (15) | 6.0<br>40.0 | 9.0<br>60.0 | 15 | 8.06 |
| Cresentric GN(8) | 3.0<br>37.5 | 5.0<br>62.5 | 8 | 4.30 |
| Membranous GN(4) | 4.0<br>100.0 | 0.0<br>00 | 4 | 2.15 |
| End stage renal disease(4) | 2.0<br>50.0 | 2.0<br>50.0 | 4 | 2.15 |
| Pyelonephritis(2) | 0.0<br>00 | 2.0<br>100.0 | 2 | 1.07 |
| Amyloidosis(1) | 1.0<br>100.0 | 0.0<br>00 | 1 | .537 |
| Diffuse mesangio sclerosis(1) | 1.0<br>100.0 | 0.0<br>00 | 1 | .537 |
| Total =186 | 109.0 | 77.0<br>41.39 | 186 | |

**Table II: Showing clinical manifestations of pediatric GN.**

| Clinical features | FSGS (35) | Focal mesp GN (24) | MCGN (21) | Diffuse mesp GN(25) | MPGN (21) | DPGN (18) | LN (15) | CGN (8) | MGN (8) |
|---|---|---|---|---|---|---|---|---|---|
| Fever | 1 2.8% | 4 12.90% | 1 4.76% | 3 12% | 3 14.28% | 0 | 7 46.66% | 1 12.5% | 1 25% |
| Odema | 35(100%) | 25(80.64%) | 21(100%) | 20(80%) | 21(100%) | 18(100%) | 13(86.66%) | 8(100%) | 3(75%) |
| Oliguria | 10 28.57% | 8 25.80% | 0 | 1 4.76% | 0 | 3 16.66% | 1 6.66% | 3 37.52% | 0 |
| Smoky urine | 3 8.57% | 3 9.67% | 0 | 1 4.76% | 0 | 2 11.10% | 0 | 3 37.50% | 0 |
| Rashes | 0 | 1 3.22% | 1 4.76% | | 0 | 0 | 4 26.66% | 0 | 0 |
| Oral ulcer | 0 | 0 | 0 | | 0 | 0 | 5 33.33% | 0 | 0 |
| hair fall | 0 | 0 | 0 | 1 4.76% | 0 | 0 | 0 | 0 | 0 |
| Hypertension | 5(14.2%) | 1(3.22%) | 3(14.28%) | | 0 | 0 | 1(6.66%) | 0 | 0 |
| Nausea/vomit | 2(5.7%) | 2(6.44%) | 2(9.5%) | 1(4.76%) | 1 (4.76%) | 0 | 0 | 1 (6.66%) | 0 |
| Amenorrhoea | 1(2.85%) | 0 | 0 | | 4(19.04%) | 0 | 0 | 0 | 0 |
| Breathlessness | 0 | 0 | 0 | | 1 4.76% | 0 | 1 6.66% | 0 | 0 |
| Urinary comp | 0 | 0 | 1(3.22%) | 0 | 1(4.76%) | 0 | 1(6.66%) | 0 | 0 |
| Pain in abdomen | 0 | 0 | 0 | 1 4.76% | 0 | 1 5.55% | 0 | 0 | 0 |
| Cough | 10(28.5%) | 0 | 0 | 0 | 0 | 1(5.55%) | 0 | 1(12.5%) | 0 |
| Arthritis | 1(2.85%) | 1(3.22%) | 0 | 0 | 0 | 0 | 5(33.33%) | 1(12.5%) | 0 |
| Seizure | 0 | 0 | 0 | 0 | 0 | 0 | 1(6.66%) | 0 | 0 |
| Flank pain | 1 2.85% | 0 | 0 | 0 | 0 | 1 5.55% | | 0 | 0 |

**Table III: Histopathological findings in paediatric renal diseases.**

| | FSGS | Focal | Diffuse | MCGN | MPGN | DPGN | LN | CGN | MGN |
|---|---|---|---|---|---|---|---|---|---|
| Renal lesions | 35 | mesp GN 31 | mesp GN 25 | 21 | 21 | 18 | 15 | 8 | 4 |
| Hyaline thrombi in glomerular capsule | 2 5.71% | 0 | 0 | 3 14.28% | 2 9.52% | 0 | 3 20.0% | 0 | 2 50.0% |
| Nuclear clumping | 1 2.85% | 0 | 0 | 0 | 0 | 1 5.55% | 2 12.32% | 1 12.50% | 0 |
| Focal tubular necrosis | 15 42.85% | 13 41.93% | 11 44% | 8 38.09% | 15 71.42% | 11 61.11% | 9 60.0% | 8 100% | 0 |
| Focal tubular atrophy | 16 45.71% | 2 6.45% | 3 12% | 0 | 8 38.09% | 1 5.55% | 2 13.33% | 6 75% | 1 25% |
| RBC in tubules | 15 42.85% | 6 19.35% | 7 28.01% | 6 28.57% | 8 38.09% | 4 22.22% | 8 53.08% | 5 62.50% | 2 50.0% |
| WBC in tubules | 7 20.05 | 3 9.67% | 4 16% | 2 6.44% | 8 38.09% | 3 20.0% | 3 20.0% | 8 100% | 0 |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Granuloma in interstitium | 1<br>2.85% | 2<br>6.45% | 3<br>12% | 0 | 8<br>38.09% | 0 | 2<br>13.33% | 3<br>37.50% | 0 |
| Lymphoid follicles in interstitium | 0 | 0 | 1<br>4% | 0 | 0 | 0 | 1<br>6.66% | 1<br>12.51% | 0 |
| venulitis | 4<br>11.4% | 8<br>16.12% | 4<br>16% | 1<br>4.76% | 3<br>14.28% | 1<br>5.55% | 4<br>26.66% | 1<br>12.51% | 1<br>25.0% |
| Vasculitis of capillaries & blood vessels | 3<br>8.57% | 3<br>9.67% | 3<br>12% | 1<br>4.76% | 5<br>23.8% | 1<br>5.55% | 3<br>20.0% | 1<br>12.51% | 1<br>25.0% |
| Thrombi in medium sized blood vessels | 5<br>14.28% | 3<br>9.67% | 2<br>8.01% | 1<br>4.76% | 0 | 3<br>20.0% | 2<br>13.33% | 0 | 2<br>50.0% |
| Foam cells in tubules | 5<br>14.28% | 1<br>3.22% | 2<br>8.01% | 3<br>14.28% | 12<br>57.12% | 0 | 0 | 0 | 0 |
| Mononuclear cells in interstitium | 27<br>77.14% | 8<br>25.80% | 9<br>36% | 6<br>28.5% | 21<br>100% | 0 | 8<br>53.33% | 8<br>100% | 1<br>25% |

## DISCUSSION

Prevalence of childhood GN varies in different series depending upon the use of immunoflorescenttest, electronmicroscopy, geneticmake up of patient, environmental factors and awareness of public.[4,12,13,14]

In present series mesangioproliferative GN was the most common GN found in 30% cases. out of these 16.66% cases were of focal mesangioproliferative type while 13.44% cases were of diffuse mesangioproliferative GN.

More or less similar to our study, one group of worker from India.[6] Also found mesangioproliferative GN as most common GN in children. They reported very high incidence of 38%.

Contrary to us, report from western world 5 found mesangioproliferativeGNin only 6.9% cases. This discrepancy may be due to the fact that in western countries electron microscopy, immunofluorescent test are done as routine test in renal biopsy. Because of IF, EM Many cases of mesangioproliferative GN were diagnosed as IgA nephropathy Henoch' sschonlein purpura[15] or IgM nephropathy or early stage OF FSGS. Second common GN in our series was FSGS which was seen in 19% cases. Moreor less similar to our study other workers from

abroad[5] & India also found its frequency to be18.8% & 15%.A study from Saudi Arabia[17] reported its incidence in nephrotic syndrome to be 24%.Moorani andSherali[16] reported still higher prevalence of FSGS of 29.66% ,while Mubarak et al from Pakistan reported a very high frequency of FSGS in 38.14 % cases. Minimal change GN which was supposed to be commonest GN of Nephrotic syndrome in children, was found to be present in only 11.29% cases in present series. Similar to our study Saca et al(13.8%) and Garg et al (19%) also found low prevalence of MPGN. In some series still a high frequency of minimal change GN varying from 32.2%[16] to 40.7%[15] have been reported. Second common proliferative GN was MPGN which had same prevalence as that of MCGN (11.29%).Many reports from Saudi Arabia[17,18] reported prevalence of MPGN to be 10.2%which is very close to our observation. Contrary to it some workers[16] slightly higher frequency of PGN(17.2%).

Diffuse proliferative GN was found in 9.67 % cases while Saca et al 2004 found low incidence of DPGN (5.2%) butKhoo et al 2004[15] Found post infectious proliferative GN in 8% which is close to our observation .Cresentic GN was found in only 4.30% patients .One study from Malayasia 16 reported its prevalence to be 2.7% only while some of the studies (Garg et al[5]) Have not reported Cresentic GN intheir study.[5,6] Lupus nephritis was the most common secondary GN noted in 8.06 % cases Moorani&sherali 2010[16] found prevalence of LN in 9.32% cases which is close to our observation. In certain countries like Malayasia[15] very high prevalence of LN in children have been reported (23%). Sex wise distribution revealed male predominance in majority GN like FSGS, MesangioproliferativeGN, Minimal change GN, diffuse mesangial sclerosis and amyloidosis similar to our studysome of the other workers[4,5,20] also noticed male predominance in these GN. In some renal disease pyelonephritis 100%, Cresentic GN (62.5%), LN (60%), Diffuse proliferative GN (55.55%) female were more affected. There are several reports who found female predominance in lupus nephritis,[21] pyelonephritis[23,24] About 14.28% patients of MGN had microscopic haematuria and mild mononuclear cell infiltration in interstitium. Microscopic haematuria[25] have been reported to occur in about 36% cases of MCGN. Leucocytopenia was present in 57.14%cases of MCGN while in 28% cases mild degree of interstitial nephritis was noted. Leucocytopenia may be due to this associated interstitial nephritis or it may be due to vesicoureteral reflex[24] MPGN patient had more leucocytoturia (87.71%) microscopic haematuria (90.47%) & foam cells in tubular lumina (57.12%) syndrome like us other workers have also reported that majority patients of MPGN have nephrotic syndrome [26,27]

and microscopic haematuria[28,29] but lipid laden tubular foam cells have not been given importance in MPGN. Infact it is more emphasised in MCGN.

Thus our study concludes that mesangioproliferative GN is the commonest GN in childhood and lipoid nephrosis is more common in MPGN. Presence of granuloma in MPGN & lipid laden foam cells in tubules in MPGN suggest that low grade chronic inflammation is the probably cause of MPGN.

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