

Acute Glomerulonephritis: A Study of Histo-Clinical Correlation of Patients at Tertiary Care Centre

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Abstract: Acute glomerulonephritis is defined as the sudden onset of hematuria, proteinuria and red blood cell casts. This clinical picture is often accompanied by hypertension, edema and impaired renal function. Acute glomerulonephritis can be due to a primary renal disease or secondary to systemic diseases. **Aim:** To study the clinical and histopathological correlation of acute glomerulonephritis in hospitalized patients. **Materials and Methods:** This is a prospective study for a period of two years from Jan.2011 to Jan. 2013 performed in GMC Jammu. **Results:** A total of 80 patients were included in this study. Out of 80 patients admitted as clinically suspected cases of acute glomerulonephritis (AGN), 9 patients responded to medical treatment. 56 patients underwent renal biopsy. 10 patients diagnosed on renal biopsies as chronic glomerulonephritis and excluded on further analysis. 46 patients diagnosed on renal biopsies as acute glomerulonephritis. **Conclusion:** PSAGN is the most common entity.

Keywords: Acute glomerulonephritis, renal biosy, hematuria

1. Introduction

Acute glomerulonephritis refers to a specific set of renal diseases in which an immunologic mechanism triggers inflammation and proliferation of glomerular tissue that can result in damage to the basement membrane, mesangium, or capillary endothelium. The exact triggers for inflammation are unknown.

Acute glomerulonephritis is defined as the sudden onset of hematuria, proteinuria and red blood cell casts. This clinical picture is often accompanied by hypertension, edema and impaired renal function (Rodrigues-Iturbe 1984)¹. Acute glomerulonephritis can be due to a primary renal disease or secondary to systemic diseases.

2. Aim

To study the clinical and histopathological correlation of acute glomerulonephritis in hospitalized patients.

3. Materials and Methods

This is a prospective study for a period of two years from Jan. 2011 to Jan.2013 performed in GMC Jammu. All patients of acute glomerulonephritis were subjected to detail history, clinical examination and laboratory investigations. Renal biopsies were performed on these patients and sent to department of pathology for histopathological examination.

4. Results

A total of 80 patients were included in this study. Out of 80 patients admitted as clinically suspected cases of acute glomerulonephritis (AGN), 9 patients responded to medical treatment. 56 patients underwent renal biosy. 10 patients diagnosed on renal biopsies as chronic glomerulonephritis

and excluded on further analysis. 46 patients diagnosed on renal biopsies as acute glomerulonephritis.

Table 1: Showing distribution of cases into adults and children (<14 years)

Cases	No. of patients	Percentage (%)
Children	8	12.3
Adult	57	87.7
Total	65	100.0

Table 2: Showing age and sex distribution of patients under study

Age group (in years)	Male No.	Female No.	Total No.	Percentage (%)
<15	5	3	8	12.3
16 – 30	20	15	35	53.9
31 – 45	9	5	14	21.5
46 – 60	5	2	7	10.8
>60	1	0	1	1.5
Total	40	25	65	100.0

Table 3: Showing distribution of patients according to the duration of illness

Duration of illness	No. of patients	Percentage (%)
<1 week	12	18.5
1 – 3 weeks	31	47.7
4 – 8 weeks	22	33.8
Total	65	100.0

Table 4: Showing cases under study with hypertension

Blood pressure range SBP/DBP mmHg	Children (n=8) No. (%)	Adult (n=57) No. (%)	Total (n=65) No. (%)
120-139/80-89	2 (25.0)	13 (22.8)	15 (23.1)
140-159/90-99	4 (50.0)	28 (49.1)	32 (49.2)
≥160/≥100	2 (25.0)	16 (28.1)	21 (27.7)
Total	8 (100.0)	57 (100.0)	65 (100.0)

Table 5: Showing distribution of patients under study with edema

Edema	No. of patients	Percentage (%)
Generalized	55	84.6
Periorbital	6	9.2
Pedal	4	6.2
Total	65	100.0

Table 6: Showing distribution of cases according to clinical presentation of sore throat, gross hematuria and nephritic range proteinuria

	Sore throat		Gross hematuria		Nephrotic range	
	Yes (%)	No (%)	Yes (%)	No (%)	>3gm/24hr (%)	<3gm/24hr (%)
Children (n=8)	7 (87.55)	1 (12.5)	4 (50)	4 (50)	6 (75)	2 (25)
Adult (n=57)	35 (61.4)	22 (38.6)	41 (71.9)	16 (28.1)	43 (75.4)	14 (24.6)
Total (n=65)	42 (64.6)	23 (35.4)	45 (69.2)	20 (30.8)	49 (75.4)	16 (24.6)

Table 7: Showing distribution of cases under study according to the various lesions seen on biopsy

Lesion	Children No.	Adult No.	Total (n=56) No. (%)
PSAGN	6	10	16 (28.6)
RPGN	1	5	6 (10.7)
DPGN	2	2	4 (7.1)
FSGS	0	2	2 (3.6)
MGN	1	7	8 (14.3)
Lupus nephritis	0	6	6 (10.7)
Idiopathic MPGN	0	14	14 (25.0)
Total	10 (17.9)	46 (82.1)	56 (100.0)

Table 8: Showing distribution of pathology of glomerulopathies observed in the present study

Disease	No. of cases	Light microscopy
PSAGN	16	Hypercellularity of mesangial and endothelial cells, neutrophilic infiltration
RPGN	6	Crescents on the inside of Bowman's capsule, inflammatory cells in interstitium
DPGN	4	Diffuse proliferation
FSGS	2	Focal and segmental sclerosis
MGN	8	Basement membrane thickening
Idiopathic MPGN	14	Mesangial proliferation, basement membrane thickening

5. Discussion

In the present study, out of 65 patients, 40 (61.5%) were male and 25 (38.5%) were female. These results are in accordance to studies by **Francis D. Murphy et al.²** and **Parag et al.³**

The interval from latent of infection to onset of nephritis in majority of patients *i.e.* 31 out of 65 (47.7%) showed a duration of 1 to 3 weeks in the present study. These results are similar to findings of **M.S.R Hutt et al.⁴**, **Anthony et al.⁵** and **Ralph Goldman et al.⁶**

Our patients were divided into three groups according to the distribution of fluid retention or edema and 55 out of 65 (84.6%) patients showed generalized edema involving both

upper and lower extremity. **Frederic Gerald Burke et al.⁷** found that 84.4% had fluid retention. So, both the studies were consistent with each other. Similarly, in a study by **Ralph Goldman et al.⁶**. The percentage was 79%.

In our study, majority of patients *i.e.* 42 out of 65 (64.6%) gave history of previous sore throat infections. In a study by **Roy and Stapleton⁸**, PSAGN results usually from upper respiratory and skin infections.

Eighteen patients out of 65 (27.7%) in the present study had history of gross hematuria. In a study by **Murphy et al.²** percentage of gross hematuria was 38% and in study by **Burke et al.⁷** percentage was 25.6%.

In the present study, out of 65 patients, 60 (92.3%) had history of decreased urine output and out of them 41.7% had oliguria. Study by **Goldman et al.⁶** showed the percentage of oliguria as 42% in acute nephritis.

We observed that out of 65 cases, 50 (76.9%) patients had hypertension *i.e.* SBP \geq 140 mmHg and DBP \geq 90 and 15 (23.1%) patients were normotensive or pre-hypertensive. **Murphy et al.²** in their study reported hypertension in 78.7% patients, which is in accordance with the present study. However, in a study by **Goldman et al.⁶** percentage of hypertension was 84%.

Out of 56 biopsies, 10 (17.8%) patients showed lesions of chronic glomerulonephritis and were excluded for further analysis from the study group. Rest 46 (82.1%) patients had acute glomerulonephritis with lesions as follows:

16 (34.7%) were diagnosed as PSAGN, 6 (13.1%) were diagnosed as RPGN, 4 (8.7%) were diagnosed as DPGN, 6 (13.1%) were diagnosed as lupus nephritis and 14 (30.4%) were diagnosed as idiopathic MPGN.

Study by **Deshpande GU et al.⁹** reported 22.5% cases of membranoproliferative glomerulonephritis on renal biopsy which is in accordance to our study.

6. Conclusion

From our study on acute glomerulonephritis, it is concluded that the patients with clinical presentations like edema, hematuria and hypertension have more evidence of acute glomerulonephritis on histopathological examination. PSAGN is the most common entity among the others.

References

- [1] **Rodriguez-Iturbe et al.(1984)**. Epidemic post-streptococcal glomerulonephritis (nephrology forum). *Kidney Inter.* **25**: 129-136.
- [2] **Francis D Murphy et al. (1938)**. Acute glomerulonephritis with special reference to the course and prognosis as study of 150 cases. *JAMA* **111**(8): 668-674.
- [3] **Parag et al.(1988)**. Profile of Crescentic glomerulonephritis in Natal – A clinicopathological assessment. *An International Journal of Medicine* **68**(2); 1 August :629-636

- [4] **M.S.R, HUTT. et al. (1964).** A clinico-pathological study of acute glomerulonephritis in East African Children. *Arch.Dis.Childh.* **39**:313
- [5] **Anthony et al. (1969).** Attack rates of acute nephritis after type 49. Streptococcal infections of the skin and of the respiratory tract. *J Clin Invest.* **48**: 1697-1704.
- [6] **Ralph Goldman et.al. (1962).** *Sporadic acute glomerulonephritis in Adults. California Medicine.***96**(6.)
- [7] **Frederic Gerald Burke et al.(1947).** *Acute glomerulonephritis- a study of ninety cases. Journal of Pediatrics ;30*(2):157-170.
- [8] **Roy and Stapleton (1990).** Changing perspectives in children hospitalized with post-streptococcal glomerulonephritis. *Pediatr Nephrol.* **4**: 585-588.
- [9] **Deshpande GU et al. (2000).** Spectrum of nephropathies with special reference to primary glomerulonephritis. *Medical J Armed Forces, India.* **56**: 125-129.