



## Study of Etiology and Clinical Assessment of Crescentic Glomerulonephritis

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### Abstract

*Rapidly progressive glomerulonephritis (RPGN) is a condition of the kidney that is considered by a rapid loss of renal function. The study had prearranged with the purpose to designate the etiological profile and clinical course in patients with crescentic GN. The study was attended in the Indira Gandhi Institute of Medical Sciences, Patna for the period of the 1 year in the patients identified with the crescentic glomerulonephritis. The clinical, biochemical and histopathological constraints and primary consequences were allied between immune-complex glomerulonephritis (ICGN) and pauci-immune glomerulonephritis (PauciGN).*

*The total number of glomeruli, crescents, Fibrocellular crescents, Tubular atrophy, Interstitial fibrosis are comprehended more in immune-complex glomerulonephritis. The Hypertension is perceived in the both the glomerulonephritis conditions. Edema is seen more in the PauciGN than in ICGN group. There is for emostalteration in the urine output in ICGN and PauciGN. Total 77% of the Immune-complex glomerulonephritis cases were observed. 27% of the cases also showed the IgA nephropathy as a major cause of the crescentic glomerulonephritis. Lupus nephritis is found in 20% of the study group.*

*From the above study as well as the already published literature it can be determined that Immune-complex glomerulonephritis is the most communal reason of Crescentic Glomerulonephritis in the selected study group patients with IgA nephropathy existence the principal disease.*

**Keyword:** Crescentic glomerulonephritis, immune-complex glomerulonephritis, pauci-immune glomerulonephritis.

### Introduction

Rapidly progressive glomerulonephritis (RPGN) is a condition of the kidney that is measured by a rapid injury of renal function <sup>[1][2]</sup>. It is mainly seen as a 50% decline in the glomerular filtration rate (GFR) within 3 months <sup>[2]</sup>. Also glomerular

crescent development seen in at least 50% <sup>[2]</sup> or 75% <sup>[1]</sup> of glomeruli seen on kidney biopsies. If these conditions are not treated then it rapidly progresses into acute renal failure <sup>[3]</sup> and death within months. In the majority of 50% patients RPGN is linked with an primary disease such as

Goodpasture syndrome, systemic lupus erythematosus or granulomatosis with polyangiitis. The remaining cases are idiopathic conditions. RPGN includes severe injury to the kidneys' glomeruli. The numerous of the glomeruli comprises distinguishing glomerular crescents.

Furthermost types of RPGN are categorized by modest and quick loss of kidney function presenting simple hematuria (blood in the urine), red blood cell casts in the urine, and proteinuria (protein in the urine). In some cases exceeding 3 g protein/24 h, a range related with nephrotic syndrome. Some patients also showed hypertension (high blood pressure) and edema. Severe disease is considered by marked oliguria or anuria, which signifies a deprived prognosis.<sup>[4]</sup>

The rapidly progressive glomerulonephritis conditions pathophysiology displayed the antineutrophil cytoplasmic antibodies (ANCA) interrelate with antigens in cytoplasm of neutrophils. It is supposed that ANCA grounds an early degranulation providing way to issue of lytic enzymes at site of injury. ANCA are connected to the pathogenesis of glomerulonephritis, antineutrophil cytoplasmic antibodies specificity is determined via (ELISA), with pANCA (antibody) directed against MPO<sup>[5]</sup>.

Serum analysis frequently supports in the identification of a specific original disease. The occurrence of anti-Glomerular basement membrane (GBM) antibodies suggests type I RPGN; antinuclear antibodies (ANA) may support a diagnosis of systemic lupus erythematosus and type II RPGN; and type III and idiopathic RPGN are frequently associated with anti-neutrophil cytoplasmic antibodies (ANCA)-positive serum.<sup>[4]</sup>

The Impaired renal function in an individual with 3 months or less of the condition is an indication of RPGN. An ultrasonographic investigation of the abdomen should also be done. Upon urine examination, urinary sediment (proteinuria) can indicate proliferative glomerulonephritis, many cases of rapidly progressive glomerulonephritis need a renal biopsy to make a diagnosis.<sup>[6]</sup>

### Classification

RPGN can be classified into three types, based upon the immunofluorescence patterns:<sup>[7]</sup>

#### Type I

This type is responsible for approximately 20% of RPGN, type I RPGN is considered by the occurrence of autoantibodies focussed in contradiction of the glomerular basement membrane (GBM). It is also called anti-GBM glomerulonephritis. The antibodies are focussed against a particular protein found in the GBM, type IV collagen, precisely the non collagenous region of its  $\alpha 3$  chain.<sup>[4]</sup> In addition to the anti-GBM antibodies, some cases of type I RPGN are also connected with antibodies directed beside the basement membrane of lung alveoli, producing Goodpasture syndrome. The majority of type I disease, however, features anti-GBM antibodies alone; these cases are measured idiopathic.<sup>[4]</sup>

#### Type II

The type II RPGN produced by the deposition of immune complexes. This accounts for 25% of RPGN and is classified as type II. Thus any immune complex disease that involves the glomerulus may progress to RPGN if severe enough. These diseases include systemic lupus erythematosus, acute proliferative glomerulonephritis, Henoch-Schönleinpurpura and IgA nephropathy.<sup>[4]</sup>

#### Type III

The Type III condition is also known as pauci-immune RPGN. The type III RPGN accounts for 55% of RPGN and designates neither immune complex deposition nor anti-GBM antibodies. As an alternative, the glomeruli are injured in an indeterminate way, possibly over the initiation of neutrophils in reply to ANCA. Type III RPGN may be quarantined to the glomerulus (primary, or idiopathic) or linked with a systemic disease (secondary). In utmost cases of the latter, the systemic disease is an ANCA-associated vasculitis such as granulomatosis with polyangiitis, microscopic polyangiitis or eosinophilic-granulomatosis with polyangiitis.<sup>[4]</sup>

The study had planned with the aim to describe the etiological profile and clinical course in patients with crescentic GN.

**Methodology**

The study was conducted in the Indira Gandhi Institute of Medical Sciences, Patna. for the period of the 1 years. The patients diagnosed with the crescentic glomerulonephritis. Renal biopsy was measured acceptable if a minimum number of five glomeruli were present, with >50% crescents. Biopsy specimens were evaluated by light (LM) and immunofluorescence microscopy (IF). Patients were followed up with monthly serum creatinine, urine protein, urine microscopy and blood counts. Major conclusions were analyzed at the end of 3 months.

The clinical, biochemical and histopathological parameters and primary outcomes were compared between immune-complex glomerulonephritis (ICGN) and pauci-immune glomerulonephritis (PauciGN).

**Results and Discussion**

Total 250 kidney biopsies were performed in the study period. Out of the total biopsy done 30 patients were found with the crescentic glomerulonephritis.

**Table 1** Etiology of crescentic glomerulonephritis

Etiology	No of Cases	Percentage
Immune-complex glomerulonephritis (ICGN)	23	77
IgA nephropathy	8	27
Lupus nephritis	6	20
Postinfectious GN	5	17
Membranoproliferative	1	3
Unclassified	2	7
PauciGN	4	13
Anti-GBM disease	1	3

The table number 1 shows the data of the etiology of crescentic glomerulonephritis. Total 77% of the Immune-complex glomerulonephritis cases were observed. 27% of the cases also showed the IgA nephropathy as a major cause of the crescentic glomerulonephritis. Lupus nephritis is found in 20% of the study group.

**Table 2:** Clinical parameters at baseline

Parameter	Total		ICGN		PauciGN	
	30	Percentage	23	Percentage	7	Percentage
Hypertension	25	83	20	87	7	100
Edema	18	60	10	43	3	75
Breathlessness	12	40	7	30	2	50
Gross hematuria	7	23	6	26	1	25
Anuria	4	13	1	4	2	50
Need for RRT	22	73	15	65	3	75
Urine output ml/day	400-1200 ml		500 - 1600		400-1300	
Duration of symptoms for days	5-50		4-45		5-60	

Table 2 indicated the clinical parameters of the Immune-complex glomerulonephritis (ICGN) and Pauci-immuneglomerulonephritis (PauciGN). The Hypertension is seen in the both the glomerulonephritis conditions. Edema is seen more in the PauciGN than in ICGN group. There is major difference in the urine output in ICGN and PauciGN.

**Table 3 :** Histopathological parameters

	Total	ICGN	PauciGN
Number of glomeruli	8-15	9-15	5-13
Number of sclerosed glomeruli	0-3	1-3	0-1.5
Crescents %	54-92	54-90	52-96
Cellular crescents %	31-81	29-78	26-88
Fibrocellular crescents %	5-30	6-33	4-32
Interstitial inflammation %	10-30	20-30	10-34
Tubular atrophy %	10-30	10-30	6-14
Interstitial fibrosis %	6-14	6-15	5-11
Acute tubular necrosis %	1-10	0-10	5-15

In the data collected from the enrolled study group, histopathological findings were mentioned in table number 3. The total number of glomeruli, crescents, Fibrocellular crescents, Tubular atrophy, Interstitial fibrosis are seen more in immune-complex glomerulonephritis.

We observed that ICGN was the most common cause of CrGN. ICGN is the predominant cause of CrGN in the paediatric population, whereas PauciGN is common among adults<sup>[8,9]</sup>. The most common etiology of ICGN in the current study was IgA nephropathy followed by lupus and PIGN. A few studies from Asia have reported ICGN as the predominant etiology in adults<sup>[10-13]</sup>. This observation is not surprising considering the high prevalence of IgA nephropathy in Asian countries.<sup>[12]</sup> Another interesting observation is the higher proportion of patients with PIGN, a disease that is considered to be common in the paediatric population.<sup>[14]</sup> The high prevalence of PIGN in the current study might be due to the tropical location and poor socioeconomic conditions predisposing to infections.

Even though the age distribution of patients was similar to previous studies, comparison with the existing series might have limitations in view of the different etiologic spectrum.<sup>[9-11]</sup>

The histological parameters were comparable in the two groups except for the higher proportion of sclerosed glomeruli in ICGN. This might be secondary to the high proportion of patients with IgA nephropathy and lupus nephritis, which follows a more indolent course<sup>[12]</sup>. Two-third of PauciGN patients showed ANCA positivity, similar to what reported from previous series<sup>[8]</sup>. Renal limited vasculitis as the sole etiology of PauciGN in this series might be secondary to the local reference patterns. Interestingly, two patients with lupus nephritis had p-ANCA positivity, which is considered as an epiphenomenon<sup>[14]</sup>.

### Conclusion

From the above study as well as the previously published literature it can be resolved that Immune-complex glomerulonephritis is the further

most communal cause of Crescentic Glomerulonephritis in the selected study group patients with IgA nephropathy being the principal disease. The clinical appearance manifest by severe renal failure at presentation and comparatively lesser response rates, higher mortality.

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