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Original Article

Investigating Crescentic Glomerulonephritis in Children: Clinical Spectrum and Predictors of Renal Survival

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ABSTRACT

Background and Aim: This study aims to evaluate the clinical and histopathological profile in children with crescentic glomerulonephritis (CGN) and determine the predictors of renal outcome.

Methods: In this retrospective study, we reviewed all native kidney biopsies performed in patients <18 years over 9 years (2011-2019). Individuals with ≥20% crescents with follow-up for at least 1 year were enrolled.

Results: This study included 34 patients. The most common variety was immune-complex glomerulonephritis (GN) (type II CGN) (n=21; 62%), including patients with Henoch-Schonlein purpura (n=6), lupus nephritis (n=6), post-infectious GN (n=3), C3GN (n=3), and dense deposit disease (n=3). The second most common was pauci-immune GN (type III CGN; n=12; 35%) followed by anti-glomerular basement membrane disease (type I CGN; n=1; 3%). Hypertension (88%), hematuria (84.2%), and oliguria (64%) were the most common presenting features. The outcome predictors for poor renal survival were the presence of oliguria (HR-5.11, P=0.035), severe hypertension (HR-11.51, P=0.019), estimated glomerular filtration rate <15 mL/min/1.73 m^2 at presentation (HR-5.05, P=0.007), percentage of crescents (HR-10.66, P=0.001), presence of fibrous crescents (HR-6.34, P=0.001), and interstitial fibrosis and tubular atrophy (HR-8.88, P=0.0046). The overall outcome of the study revealed complete recovery (n=12), partial recovery (n=6), chronic kidney disease (n=3), and end-stage renal disease (n=13). The renal survival in patients with ≥50% crescents was poor (P=0.037) as compared to subjects with <50% crescents.

Conclusion: Renal survival can be predicted by the severity of presenting features and histopathological markers. Two-thirds of patients had type II CGN with renal survival outcomes similar to type III CGN. The percentage of crescents is the most important predictor of renal survival.

Keywords: Child, Crescentic glomerulonephritis, End stage renal disease, Survival

Introduction

Crescents are formed by the proliferation of activated parietal epithelial cells and infiltration of macrophages in the Bowman's capsule. The crescent formation leads to a reduced single nephron glomerular filtration rate (GFR) and represents the histopathological correlation of severe glomerular injury. Cellular crescents can be reversible; however, when they transform into fibrous crescents, the recovery of GFR becomes irreversible [1, 2].

The literature on pediatric crescentic glomerulonephritis (CGN) is quite limited as the overall incidence of this condition is low [3-6]. The clinical presentation is variable and includes a combination of renal and extra-renal features depending on the etiology. Many patients exhibit a rapidly progressive course while others may have an insidious onset disease causing a delay in the diagnosis. The duration of symptoms and timing of initiation of therapy are important determinants of outcome. The studies published on this subject are difficult to compare as different cut-off percentages of crescents have been adopted to define CGN by researchers. While many early studies used the criterion of ≥50% to >75% crescents to define CGN, others considered a less stringent >10% cut-off and the most recent ones even included patients with a single crescent [7-10]. Studies also differ in the relative importance of the etiology of crescents versus the percentage of crescents as the most important prognostic marker [11-14]. Considering all these factors, this study focuses on CGN using 20% crescents as the defining criterion and compared if adopting a 20% cut-off crescents instead of 50% significantly affected the outcome.

The aims of this study are as follows: 1) To study the demographic, clinical, and histopathological profile of children with CGN, 2) To determine the predictive factors of end-stage renal diseases (ESRD), 3) To compare the clinical, biochemical, and histopathological features and outcome of patients with 20% to 50% crescents with those having >50% crescents in renal biopsy.

Materials and Methods

This was a retrospective cohort study performed in a pediatric nephrology division of a tertiary hospital in Western India. Reports of all native kidney biopsies performed in the preceding 9 years (January 2011 to January 2019) in children less than 18 years of age were screened. The patients with histopathological findings of ≥20% crescents and a minimum follow-up duration of 1 year were enrolled in the study. Approval from the institutional Ethics Committee was obtained. The data on demography, clinical presentation, biopsy findings, received treatment, and the immediate outcome was recorded in a predesigned proforma. The patients with a follow-up duration of less than 1 year were excluded from the outcome analysis (Figure 1).

The clinical and biochemical definitions adopted in the current study were as follows:

Microscopic hematuria defined as the presence of 5 RBC/hpf in the centrifuged sample;

The nephrotic range proteinuria is defined as 24 h urine albumin >40 mg/m^2/h;

Hypertension is defined following the American Academy of Pediatrics clinical practice guidelines as blood pressure >95 thpercentile+12 mmHg for age, gender, and height [15];

Hypertension considered severe if 3 or more antihypertensives were required;

Complete remission defined as estimated glomerular filtration rate (eGFR) >60 mL/min/1.73 m^2, the absence of proteinuria, hematuria, and requirement of none or single antihypertensive;

Chronic kidney Disease was eGFR between 60 mL/1.73 m^2 and 15 mL/1.73 m^2 of body surface area;

The patients not satisfying either of the above two criteria were considered to have partial remission;

ESRD defined as eGFR of <15 mL/min/1.73 m^2 or the requirement of maintenance dialysis for more than 3 months;

Duration of disease was defined as the time from onset of symptom to initiation of treatment.

Histologically, crescents were defined as the presence of two or more layers of parietal epithelial cells in the Bowman's space. These were classified as cellular if >50% of the proliferation was occupied by cells, fibro cellular crescent if <50% of the lesion was occupied by cells, and <90% by matrix and fibrous if >90% of the lesion was occupied by matrix. Besides crescents, other major histological findings, namely the presence of neutrophilic infiltration, fibrinoid necrosis, sclerosis, mesangial and endocapillary proliferation, interstitial fibrosis, and tubular atrophy (IFTA), and vascular changes were also recorded.

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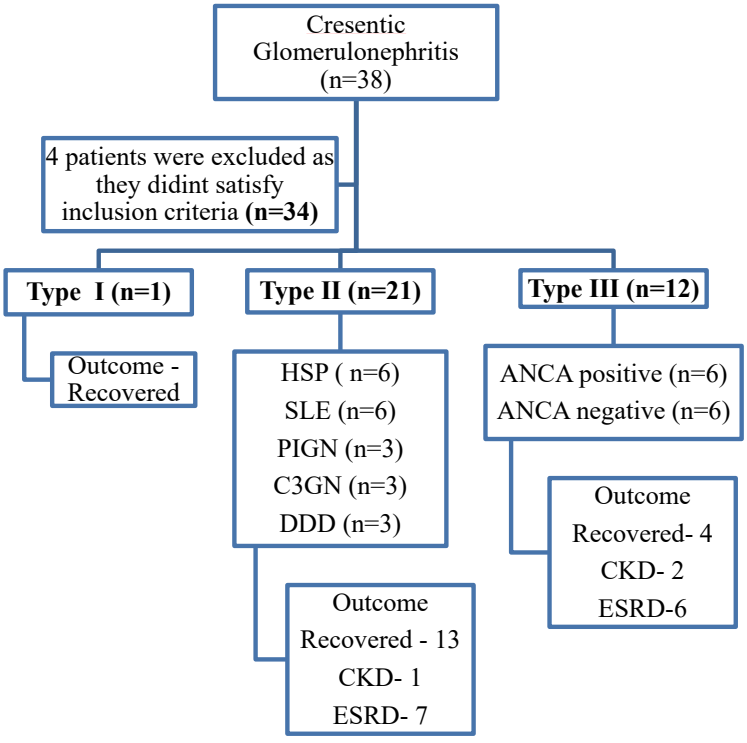

Figure 1. Distribution of etiology and types of glomerulonephritis and final outcome

CGN were classified into 3 types based on light microscopy in case of staining pattern and serology: Type I: Anti-glomerular basement membrane disease; type II: Immune complex disease, including systemic lupus erythematosus (SLE), IgA nephropathy, post-infectious GN (PIGN), C3GN, Henoch-Schonlein purpura (HSP); type III: Pauci-immune CGN.

All patients with pauci-immune GN were treated with methylprednisolone 30 mg/kg/day for 5 days and intravenous cyclophosphamide 6 doses 500 mg/m^2 fortnightly for induction, followed by long-term maintenance therapy with mycophenolate mofetil or azathioprine. Patients who were poorly responsive to first-line immunosuppression were also given rituximab and intravenous immunoglobulin (IVIG). Plasmapheresis was additionally done in patients showing unsatisfactory renal recovery after induction with methylprednisolone or in individuals having anti-GBM disease, anti-neutrophil cytoplasmic antibodies associated with vasculitis, and life-threatening pulmonary hemorrhage. Renal replacement therapy (hemodialysis or peritoneal dialysis) was offered as required in patients with renal failure. The patients were monthly followed up with clinical details, blood pressure, 24 h urine albumin, urine routine, and serum creatinine to monitor the response to maintenance therapy and relapses.

Statistical analysis

Data were analyzed using SPSS software, version 15. The data were given as Mean±SD, and N for continuous data and as number and percentage for categorical data. The comparison of the means of the 2 groups was carried out by student unpaired t-test for numerical normal data. Fisher exact probability tests were applied to compare percentages for categorical data between 2 groups. The chi-square test was applied to compare the percentages of more than 2 groups. The relationship between covariates and ESRD was assessed using the Cox proportional hazard analysis. To determine renal survival Kaplan-Meier analyses were done with $P<0.05$ as statistically significant.

Results

A total of 34 patients fulfilled the study criteria and were included in the final analysis as shown in Figure 1. The mean age of presentation for the entire cohort was 7.08±2.06 years. Type II CGN was diagnosed in 21 patients and type III in 12 out of 34 patients. Type I variety was found only in 1 patient during the study. The etiology of type II included Henoch-Schonlein purpura (n=6), lupus nephritis (n=6), post-infectious GN (n=3), C3GN (n=3), and dense deposit disease (n=3). Among type III patients 50% (6 out of 12) were antineutrophilic cytoplasmic anti-

body positive. There was no statistically significant difference in age or gender among the various types of CGN. The most common features at presentation were hypertension (88%), gross hematuria (84.2%), and oliguria (64%) (Table 1). All patients with type III CGN were hypertensive at presentation with 75% exhibiting severe hypertension. Extrarenal manifestations were seen in both groups. Type II patients had significant arthralgia (P=0.014) and a higher frequency of rash (33% vs 16.7%), whereas hemoptysis was exclusively seen in four antineutrophilic cytoplasmic antibody-positive patients in type III CGN. Another notable extra-renal manifestation in patients with type III CGN was the presence of fever in 50% as compared to 33% in type II which was attributed to vasculitis syndrome in the absence of fever. Nephrotic syndrome was seen in 20% of patients with the majority belonging to type II CGN (Table 1).

The mean duration of illness at the time of presentation was 47.4±42.34 days for the entire cohort. This was significantly shorter in patients with type III CGN as compared to type II. A total of 58% (7 out of 12) of patients with type III initiated the treatment within 30 days of the onset of symptoms as compared to only 23% (5 out of 21) of type II CGN. Further, patients with type III CGN had a lower eGFR at admission (mean=18±15.6 mL/min/1.73m^2; P=0.027)) and at discharge (mean eGFR=40.03±25.64 mL/min/1.73 m^2, P=0.01) as compared to patients with type II CGN. Low serum C3 level was seen in 14 out of 34 patients, including all patients with degenerative disc disease and lupus, in addition to 2 out of 12 patients with type III CGN.

Table 1. Demographic, clinical, and laboratory parameters of patients

Parameters		No. (%)/Mean±SD/(%)				P
		Total (n=34)	Type I* (n=1)	Type II (n=21)	Type III (n=12)	
Sex	Male	18(54.5)	0	14(66.7)	4(33.3)	0.083
	Female	15(45.5)	1	7(33.3)	8(66.7)	
Age (y)		8.41±2.50	9±0	8.14±2.7	8.8±2.25	0.55
Duration of disease (days)		47.4±42.34	10±0	56.67±46.13	31.2±29.90	0.04
<30 days		12(35.3)	1(100.0)	4(19)	7(58.3)	0.03
≥30 days		22(64.7)	0(0.0)	17(81)	5(41.7)	
Fever		44.1	100	38.1	50	0.5
Hematuria		84.2	100	81	83.3	1
Oliguria		64.7	100	57.1	75.0	0.45
Hypertension		88.23	100	81	100	0.3
Arthralgia		26.5	100	38.1	0.0	0.03
Nephrotic syndrome		20.6	-	23.8	16.7	0.8
Proteinuria	On admission	122.15±79.4	83±0	122.15±79.4	122.15±79.40	0.16
	At discharge	50.82±60.34	55±0	48.19±25.29	55.82±39.56	0.8
	After 1 year	16.82±19.8	16±0	18.95±22.84	11.33±12.29	0.3
eGFR (mL/min)	On admission	37.79±35.76	10.8±0	50.39±39.16	18±15.86	0.027
	At discharge	64.51±38.61	95±0	73.62±33.56	40.03±25.64	0.01
	After 1 year	78.23±50.97	138±0	87.65±50.16	57.54±47.53	0.1

*Patient with type I CGN was excluded from the statistical calculation.

CGN: Crescentic glomerulonephritis; eGFR: Estimated glomerular filtration rate.

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Table 2. Histopathological characteristics of patients

Histopathological Characteristics		No. (%)/Mean±SD/(%)				P
		All Types (n=34)	Type I* (n=1)	Type II (n=21)	Type III (n=12)	
No of crescents (%)		54.02±24.2	78.00±0	45.4±24.8	67.3±16.3	0.014
Cellular		29(85.3)	100	20(95)	8(66)	0.02
Fibrocellular		18(52.9)	0.0	11(52.38)	75(58.3)	0.74
Fibrous		7(20.5)	0.0	1(4.8)	6(50)	0.005
Glomerular lesions	Sclerosis	14 (41.2)	0.0	9(42.9)	5(41.7)	0.85
	Mesangial proliferation	27(79.4)	0.0	19(90.5)	8(66.7)	0.15
	Endocapillary proliferation	20(58.8)	0.0	13(61.9)	7(58.3)	0.84
	Neutrophilic infiltration	13(41.2)	100	7(33.3)	6(50)	0.31
	Fibrinoid necrosis	4(11.7)	0	2(9.5)	2(16.7)	0.61
	IFTA	15(44.1)	0.0	6(28.6)	9(75)	0.014
	Vascular changes	8(23.5)	0.0	5(23.8)	3(27.3)	0.6

*Patient with type I CGN was excluded from the statistical calculation.

IFTA: Interstitial fibrosis, and tubular atrophy.

The most common histological feature was cellular crescents (85%) while fibrocellular crescents and fibrous were 55% and 20%, respectively. Other histopathological findings are detailed in Table 2. The subgroup analysis showed that type III CGN patients had a significantly greater percentage of crescents (mean=62.75%±17.11%) with a higher proportion of fibrous crescents (50%) and interstitial fibrosis and tubular atrophy (75%). Plasmapheresis was performed in 7 out of 34 patients. This included 5 patients of type III and 2 from type II CGN. A total of 22(64%) patients required dialysis during admission.

Study outcome

The mean follow-up duration of the patients was 2.18±1.64 years. One-third of patients followed up for ≥3 years. Overall, 18 patients (52.9%) were in remission (12 complete and 6 partial) at the last follow-up. A total of 16 patients had impaired renal functions, 3 patients were in chronic kidney disease stage 2-4, and 13 had progressed to ESRD (Figure 1). Patients with type II CGN showed statistically significant recovery (P=0.04). Although renal survival at the end of 1 year and 3 year is low in type

Table 3. Outcome predictors of end-stage renal disease

Variables	Hazard Ratio (95% CI)	P
Oliguria	5.11 (1.12, 23.33)	0.03
Severe hypertension	11.51 (1.48, 89.4)	0.01
eGFR <15	5.05 (1.55, 16.49)	0.007
eGFR at onset	0.95 (0.91, 0.99)	0.01
Fibrocellular crescents	5.45 (1.19, 24.91)	0.02
Fibrous crescents	6.34 (2, 20.12)	0.001
IFTA	8.88 (1.96, 40.24)	0.004

IFTA: Interstitial fibrosis, and tubular atrophy; eGFR: Estimated glomerular filtration rate.

Table 4. Comparison between patients having 20-50 % crescents and >50% crescents on biopsy

Parameters		No. (%)/Mean±SD/(%)		P
		20% to 50% Crescents (n=15)	>50% Crescents (n=19)	
Type of CGN	Type 1	0(0.0)	1(100)	0.028
	Type 2	13(61.9)	8(38.1)	
	Type 3	2(16.7)	10(52.6)	
Clinical presentation	Age of onset	8.33±3.09	8.47±2.03	0.9
	Duration of diagnosis	66.00±50.71	30.47±26.38	0.013
	eGFR at diagnosis	50.85±31.15	27.48±36.54	0.057
	eGFR at discharge	79.79±30.02	52.46±41.04	0.038
	Proteinuria at diagnosis	141.60±91.73	106.79±66.69	0.2
	Severe hypertension	45.5	73.7	0.2
Histopathology	Cellular crescents	86.6	84.2	0.9
	Fibrocellular crescents	53.3	57.7	0.9
	Fibrous crescents	6	31.5	0.17
	IFTA	26.6	57.8	0.14
Treatment outcome	Dialysis	46.7	78.9	0.08
	Plasmapheresis	6.7	8(42.1)	0.023
	Duration of hospitalization	28.53±14.89	39.95±21.58	0.1
	Complete recovery	73.33	42	1
	Deaths	6.7	21.1	0.36
	ESRD	20	52.6	0.03

Abbreviations: CGN: Crescentic glomerulonephritis; eGFR: Estimated glomerular filtration rate; IFTA: Interstitial fibrosis, and tubular atrophy; ERSD: End-stage renal disease.

III CGN (66.7% and 50%, respectively) as compared to type II CGN (76.2% and 71.4%, respectively), statistical significance could not be attained (Figure 2 and 3). Among various subtypes of type II, all the patients of HSP recovered completely while patients with degenerative disc disease exhibited the worst renal outcome.

The outcome predictors of renal survival were the presence of oliguria (HR-5.11, P=0.035), severe hypertension (HR-11.51, P=0.019), eGFR <15 at presentation (HR-5.05, P=0.007), the occurrence of the fibrous crescent (HR-6.34, P=0.001), and interstitial fibrosis and tubular atrophy (HR-8.88, P=0.0046) (Table 3). The percentage of crescents (HR-10.66) also had a positive correlation with progression to ESRD with P=0.001 (Figure 2).

Comparison in clinical profile of ≥50% and <50% crescents

The criterion of ≥50% crescents on histopathology examination was fulfilled by 83% of patients with type III and 17% of patients with type II CGN (Table 4). The duration of symptoms at admission was longer in patients with <50% crescents with P<0.05. The eGFR at diagnosis was lower in the >50% group (mean=27.48±36.54) in comparison to the <50% group (50.85±31.15). Regarding the other histopathological findings, the need for dialysis was not significantly different but the requirement of plasmapheresis was more in a patient with ≥50% crescents. Renal survival was significantly worse in patients with >50% patients (P=0.03) (Figure 4).

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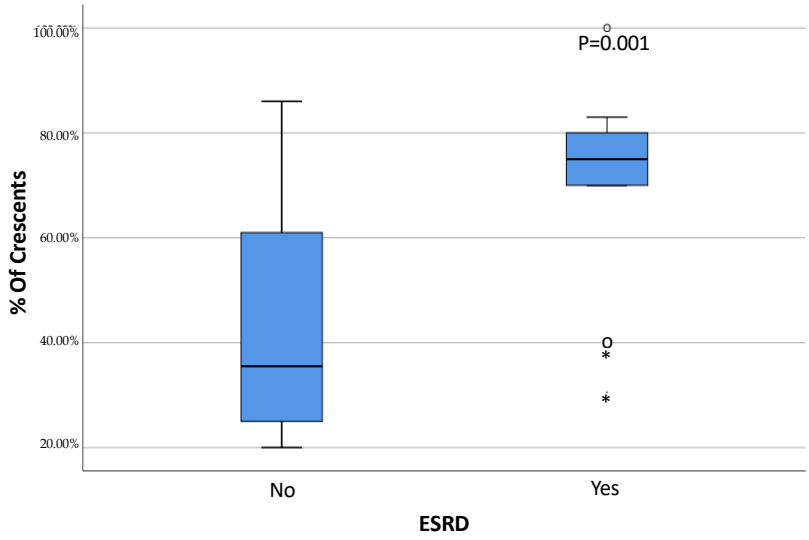

Figure 2. Correlation of percentage of crescents in biopsy with progression to end-stage renal disease

Discussion

CGN is a rare form of glomerulonephritis in children with a grave prognosis. Although the universally accepted definition for CGN is the presence of at least 50% crescents on light microscopy, many recent publications have used a much lower percentage criterion of even up to 10% crescents. The adoption of such liberal criteria may be partly driven by the need to increase the sample size for a very rare disorder but significantly reflects the researchers' conviction that the prognosis of CGN remains unchanged irrespective of the percentage of crescents. A study from South Arabia included children with >20% crescents and 36.4% developed chronic kid-

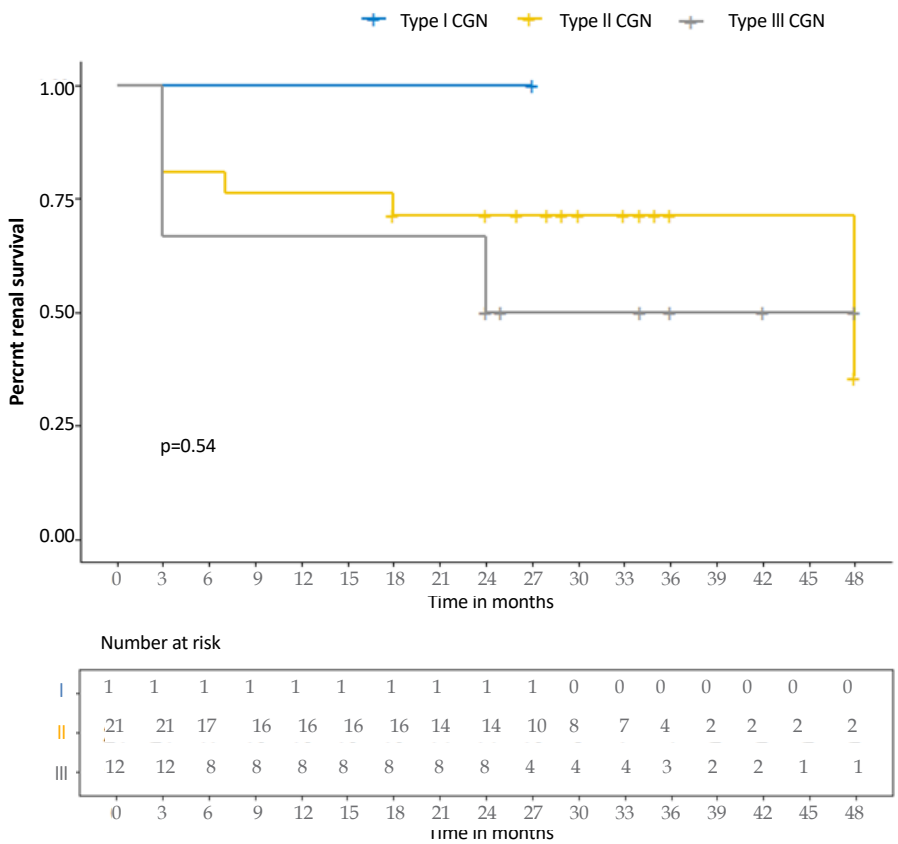

Figure 3. Kaplan-Meier estimate for renal survival curves for types of crescentic glomerulonephritis

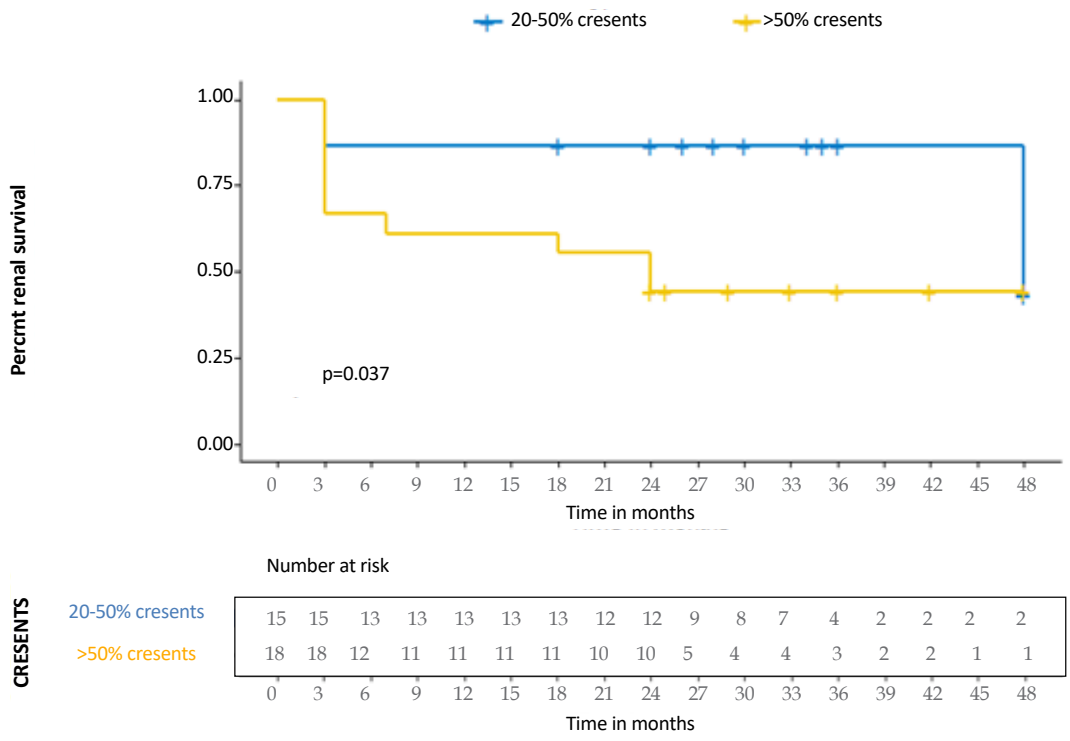

Figure 4. Kaplan-Meier estimate for renal survival curve for 20% to 50% and >50% crescents on biopsy

ney disease, whereas a pediatric cohort from Thailand defined CGN as those with >10% crescent, and a quarter of patients developed ESRD [8, 9]. These results seem to indicate that the adoption of less stringent criteria of percentage of crescents as compared to the previous criterion of 50% does not significantly alter the outcome; however, the etiological profile of CGN is variable worldwide and the results of studies in one ethnic region may not be valid for another geo-ethnic group. Hence, this retrospective study was conducted to analyze the clinical and etiological profile of CGN patients with CGN from Western India and also to ascertain if adoption of more than 20% crescents versus 50% crescents significantly altered the outcome.

The most common type of CGN in our cohort was immune complex GN which corroborates with previously reported studies [3, 10]. However, the etiology of immune complex CGN found in our study was at variance with the previous literature. Historically, PIGN has been reported as the most frequent etiology of immune complex GN. This study found HSP and lupus nephritis as the predominant cause of immune complex CGN. The rates of CGN due to PIGN seem to have declined in children from Western India probably because of timely and appropriate medical treatment of streptococcal infections. Our study cohort comprised of younger children than previously reported [4, 6]. There was no age or gender predilection among the different categories of CGN. The most common clinical presentation was hypertension which was severe in over half of the patients. On the long-term follow-up, severe hypertension served as an important predictor of ESRD. Other clinical parameters which predicted adverse long-term outcomes in our cohort were oliguria and eGFR <15 mL/min/1.73m^2 at presentation. Low eGFR at presentation has been reported previously as a significant risk factor for the progression to ESRD by most of the pediatric and adult studies [8, 16]. Quiroga et al. [17] described it as the strongest predictor for renal survival outcome in CGN. We did not find the presence of nephrotic range proteinuria and the need for dialysis at admission to be significant risk factors for renal loss as has been reported by other researchers [6, 18].

Histopathological predictors of end-stage renal disease in our study were the percentage of crescents, fibro cellular crescents, fibrous crescents, interstitial fibrosis, and tubular atrophy. This is in line with other studies [8, 16, 19]. In the current study, we compared the outcome of patients with <50% crescents to subjects with ≥50% crescents. Although the prognosticating factors, such as the severity of hypertension, the presence of fibro cellular, fibrous crescents, and interstitial fibrosis and tubular

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atrophy were comparable among the two groups, renal survival was significantly worse in subjects with ≥50% crescents. This reinforces that the percentage of the crescent is the most important predictor of renal survival, thus demonstrating the need to adopt unifying criteria of ≥50% crescents while investigating and reporting CGN. A recent multicenter pediatric study from the US quantified the prognostic importance of crescents and revealed that for each 1% increase in crescents log odds of 1-year renal survival decreases by 3% [20].

Previously reported studies from India showed pauci-immune GN to be a significant risk factor for ESRD [8, 21]. The most striking finding was less time required from diagnosis to initiation of treatment in patients with type III CGN as they were referred immediately because of the severity of symptoms. However, renal survival could not be improved even with early aggressive immunosuppression. In this cohort, the majority (83%) of patients with type III CGN had >50% crescents in biopsy and presented with a rapidly progressing course. They had a significant number of crescents, fibrous variety, and the presence of interstitial fibrosis and tubular atrophy as compared to type II patients contributing to significantly low residual renal function at discharge and even on follow-up. Although half of the patients with type III developed ESRD at the end of 3 years, renal survival in patients with type II and type III were statistically comparable.

Amongst various etiologies of immune complex glomerulonephritis, the worst renal survival outcome was found in patients having degenerative disc disease as all the patients developed ESRD on follow-up despite treatment. Patients with HSP showed better outcomes in the form of complete recovery, despite the severe disease at presentation.

Being extremely rare in children, we had one patient belonging to type I CGN adding to the existing sparse literature from the past [22, 23]. Our patient has shown partial recovery in 1-year follow-up. We could not derive any valid conclusion about the same. The incidence of ESRD reported in our study was similar to the results reported in the existing pediatric literature.

Study limitations

The limitations of the study are its retrospective design and the small size of the cohort. The strengths include a detailed analysis of clinical, histopathological, and prognostic factors with a comparison between patients with 20% to 50% and >50% crescents not reported previously.

Conclusion

Immune complex glomerulonephritis remains the most common cause of CGN in children. CGN poses a high risk to develop ESRD in children that can be predicted by clinical and histopathological features. The difference in renal outcome between pauci-immune and immune complex glomerulonephritis was insignificant. The percentage of crescents was the most important predictor of renal survival. Further research is needed to formulate more aggressive treatment policies to improve renal as well as patient survival in the long-term.

Ethical Considerations

Compliance with ethical guidelines

This study was approved by Institutional Ethics Committee of Bai Jerbai Wadia Hospital for Children (No.: IEC-BJWHC/35/2018) on February 5, 2018.

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Authors' contributions

Conceptualization: Nikita Gupta, Alpana Ohri and Amish Udani; Methodology, data curation, formal analysis, drafting original article: Nikita Gupta; Supervision, critical revision and editing: Alpana Ohri, Amish Udani and Chintan Shah.

Conflict of interest

The authors declared no conflict of interest.

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References

[1] Jennette JC. Rapidly progressive crescentic glomerulonephritis. Kidney Int. 2003; 63:1164-77. [DOI:10.1046/j.1523-1755.2003.00843.x] [PMID]

[2] Bagga A, Menon B. Rapidly progressive glomerulonephritis. In: Geary DF, Schaefer F, editors. Comprehensive pediatric nephrology. Philadelphia: Mosby Elsevier. 2008: 319-327. [DOI:10.1016/B978-0-323-04883-5.50027-1]

[3] Jardim HM, Leake J, Risdon RA, Barratt TM, Dillon MJ. Crescentic glomerulonephritis in children. Pediatr Nephrol. 1992; 6(3):231-5. [DOI:10.1007/BF00878354] [PMID]

[4] Dewan D, Gulati S, Sharma RK, Prasad N, Jain M, Gupta A, et al. Clinical spectrum and outcome of crescentic glomerulonephritis in children in developing countries. Pediatr Nephrol. 2008; 23(3):389-94. [DOI:10.1007/s00467-007-0647-3] [PMID]

[5] Özlü SG, Çaltık A, Aydoğ Ö, Bülbül M, Demircin G, Çakıcı E, et al. Crescentic glomerulonephritis in children: A single centre experience. World J Pediatr. 2016; 12(2):225-30. [DOI:10.1007/s12519-015-0036-0] [PMID]

[6] Sinha A, Puri K, Hari P, Dinda AK, Bagga A. Etiology and outcome of crescentic glomerulonephritis. Indian Pediatr. 2013; 50(3):283-8. [DOI:10.1007/s13312-013-0095-z] [PMID]

[7] BaggaA. Crescentic glomerulonephritis CH 34. In: Avner E, Harmon W, Niaudet P, Yoshikawa N, editors. Pediatric Nephrology. Berlin: Springer Science & Business Media; 2009. [Link]

[8] Rianthavorn P, Chacranon M. Long-term renal outcome in pediatric glomerulonephritis associated with crescent formation. Clin Exp Nephrol. 2018; 22(3):661-7. [DOI:10.1007/s10157-017-1498-2] [PMID]

[9] Alsaad K, Oudah N, Al Ameer A, Fakeeh K, Al Jomaih A, Al Sayyari A. Glomerulonephritis with crescents in children: Etiology and predictors of renal outcome. ISRN Pediatr. 2011; 2011:507298. [DOI:10.5402/2011/507298] [PMID] [PMCID]

[10] Mayer U, Schmitz J, Bräsen JH, Pape L. Crescentic glomerulonephritis in children. Pediatr Nephrol. 2020; 35(5):829-42. [DOI:10.1007/s00467-019-04436-y] [PMID] [PMCID]

[11] Jennette JC, Thomas DB. Crescentic glomerulonephritis. Nephrol Dial Transplant. 2001;16 Suppl 6:80-82. [DOI:10.1093/ndt/16.suppl_6.80] [PMID]

[12] Tao J, Wang H, Wang SX, Yu F, Zhao MH. The predictive value of crescents in the disease progression of lupus nephritis based on the 2018 International society of nephrology/renal pathology society revision system: A large cohort study from China. Ren Fail. 2020; 42(1):166-72. [DOI:10.1080/0886022X.2020.1726385] [PMID] [PMCID]

[13] Lee MJ, Kim SJ, Oh HJ, Ko KI, Koo HM, Kim CH, et al. Clinical implication of crescentic lesions in immunoglobulin A nephropathy. Nephrol Dial Transplant. 2014; 29(2):356-64. [DOI:10.1093/ndt/gft398] [PMID]

[14] Baikunje S, Vankalakunti M, Nikith A, Srivatsa A, Alva S, Kamath J. Post-infectious glomerulonephritis with crescents in adults: A retrospective study. Clin Kidney J. 2016; 9(2):222-6. [DOI:10.1093/ckj/sfv147] [PMID] [PMCID]

[15] Flynn J. T., Kaelber D. C., Baker-Smith C. M., Blowery D., Carroll A. E., Daniels S. R., et al. Clinical Practice Guideline for Screening and Management of High Blood Pressure in Children and Adolescents. Pediatrics. 2017; 140:pii: e20171904 [DOI:10.1542/peds.2017-3035] [PMID]

[16] Rampelli SK, Rajesh NG, Srinivas BH, Harichandra Kumar KT, Swaminathan RP, Priyamvada PS. Clinical spectrum and outcomes of crescentic glomerulonephritis: A single center experience. Indian J Nephrol. 2016; 26(4):252-6. [PMID] [PMCID]

[17] Quiroga B, Vega A, Rivera F, López-Gómez JM; Spanish Registry of Glomerulonephritis. Crescentic glomerulonephritis: Data from the Spanish glomerulonephritis registry. Intern Med J. 2015; 45(5):557-62. [DOI:10.1111/imj.12725] [PMID]

[18] El-Husseini AA, El-Agroudy AE, Moustafa FE, Fouda MA, Sobh MA. Impact of clinical and histopathological factors on outcome of Egyptian patients with crescentic glomerulonephritis. Int Urol Nephrol. 2003; 35(4):543-51. [DOI:10.1023/B:UROL.0000025607.41722.aa] [PMID]

[19] No author. A clinico-pathologic study of crescentic glomerulonephritis in 50 children. A report of the southwest pediatric nephrology study group. Kidney Int. 1985; 27(2):450-8. [DOI:10.1038/ki.1985.30] [PMID]

[20] Maliakkal JG, Hicks MJ, Michael M, Selewski DT, Twombley K, Rheault MN et al. Renal Survival in children with glomerulonephritis with crescents: A pediatric nephrology research consortium cohort study. J Clin Med. 2020; 9(8):2385. [DOI:10.3390/jcm9082385] [PMID] [PMCID]

[21] Alexander S, Yusuf S, Rajan G, Elias John E, Roy S, Annamalai VC, et al. Crescentic glomerulonephritis: What's different in South Asia? A single center observational cohort study. Wellcome Open Res. 2020; 5:164. [DOI:10.12688/wellcomeopenres.16071.1] [PMID] [PMCID]

[22] Williamson SR, Phillips CL, Andreoli SP, Nailescu C. A 25-year experience with pediatric anti-glomerular basement membrane disease. Pediatr Nephrol. 2011; 26(1):85-91. [DOI:10.1007/s00467-010-1663-2] [PMID]

[23] Chauhan R, Betts G, Long EL, et al. Anti-Gbm disease in children: Outcomes and association with systemic vasculitis. Rheumatol. 2017; 56(6):1-4. [DOI:10.1093/rheumatology/kex356.046]