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Clinical spectrum of crescentic glomerulonephritis - A single centre experience

Gaurav S Sharma, D Agrawal and Alok K Pandey
SMS Medical College & Hospital, India

Background: Rapidly Progressive Glomerulonephritis (RPGN), the most common cause of rapidly progressive renal failure (RPRF), usually has extensive crescent formation. Hence, RPGN is also called Crescentic Glomerulonephritis (CrGN). CrGN is a histological term, defined as the presence of crescents in >50% of glomeruli. There is limited data on the etiology, clinical and histopathological spectrum of Crescentic Glomerulonephritis (CrGN) in adult Indian population.

Objective: To study the clinical and histopathological profile of patients with CrGN.

Design & Methods: A retrospective descriptive study was conducted at Department of Nephrology, SMS Medical College & Hospital, Jaipur. Patients aged >18 years, with CrGN (defined as the presence of crescents in >50% of glomeruli) biopsied in 2015 & 2016 were included in the study.

Results: Out of 323 renal biopsies done at our centre in the years 2015 & 2016, 32 patients had CrGN. Immune-complex glomerulonephritis (ICGN) was the most common etiology (n=26; 81.25%) found followed by pauci-immune glomerulonephritis (Pauci GN; n=4; 12.50%). The most common etiology of ICGN was IgA Nephropathy (n=7, 21.80%). This was followed by C3 glomerulopathy (n=6, 18.75), Lupus Nephritis (LN; n=4; 12.50%), Post-infectious glomerulonephritis (PIGN; n=4; 12.50%), unclassified (n=4; 12.50%) followed by MPGN (n=1; 3.12%). Two cases of anti-GBM disease (n=2; 6.25%) were detected, one of which was also pANCA positive. Histopathological correlation was done with clinical parameters. The mean age was 32 years (range-9 yrs to 74 yrs). The mean duration of symptoms at the time of presentation was 32 days. Amongst the ICGN group, 57.69% of patients and 25% patients with Pauci GN were hypertensive at presentation. Gross Hematuria was present in 30.76% of patients with ICGN and in 25% of patients with Pauci GN. Anuria was found in 34.6% of patients with ICGN and in 50% of patients with Pauci GN. RRT was required in 65.38% and in 75% of patients with ICGN and Pauci GN, respectively. Mean S. Creatinine level at presentation was 5.06 mg/dl & 7.17mg/dl for ICGN and Pauci GN, respectively. The difference between the percentages of total crescents amongst the two groups was not found to be statistically significant. However, the percentage of cellular crescents was significantly higher in the ICGN group as compared to the Pauci GN (32.93% vs. 8.57%; p=0.0150). On the other hand, the percentage of fibrous crescents was significantly higher in the Pauci GN group than in the ICGN group (36.22% vs. 10.08; p=0.0050). It was also found that the percentage of interstitial fibrosis/tubular atrophy was significantly higher in the Pauci GN group than in the ICGN group (43.33% vs. 17.95; p=0.0052).

Conclusion: The most common cause of Cr GN in our centre was found to be ICGN, out of which IgA nephropathy was the most common etiology, followed by C3 glomerulopathy. The patients with ICGN had a higher proportion of cellular crescents than those with Pauci GN.

shekhargaurav2012@gmail.com