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Clinical profile and treatment responses in C3 glomerulonephritis: a case series.

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INTRODUCTION

C3 glomerulonephritis (C3 GN) is a rare and heterogeneous disease caused by dysregulated activation of the alternative complement pathway*.The diversity in clinical presentation and pathogenesis are it's unique features. There is no approved treatment for C3 GN, and current recommendations are largely based on limited evidence. In this report, we describe a series of 10 cases of C3GN.

METHODOLOGY

We enrolled biopsy-proven cases of C3GN diagnosed between January 2021 and August 2024, at IGIMS, Patna.

Clinical data were obtained from patient records, and laboratory values were retrieved from the hospital information system and analysed.

RESULTS

- The study included 10 cases (mean age 39 years) with mean serum creatinine of 3.95 mg/dl and mean 24-hour urinary protein of 3.49 g/day. Five had hypertension, five had macroscopic hematuria, and seven had low serum C3 levels. Clinical syndromes included RPGN (3), nephrotic syndrome (3), CGN (3), and ANS (1).
- Renal histology showed mesangioproliferative (6) and MPGN (4) patterns. Of five with crescentic changes, four had fibro-cellular crescents (>40% of glomeruli), and one had cellular crescents (>10%). IFTA was mild (20-30%) in 2, moderate to severe (30-50%) in 2, and in rest 6 cases had only 15-20%. Electron-dense deposits were present in all regions of glomeruli.
- Treatments included cyclophosphamide with prednisolone (3), MMF with prednisolone (3), and prednisolone alone (3). One CGN case did not receive immunosuppression. Five remain on MMF and prednisolone, and three received only prednisolone for 6 months. All received ACEi/ARB.
- Three cases required hemodialysis at presentation; two became dialysis-independent by the third month, and one remains on dialysis. Three nephrotic syndrome cases achieved CR within 3-6 months. Two of the 3 with RPGN reached CR at 3 months, and the third patient had stabilization of serum creatinine and remission of proteinuria at 6 months. Two CGN cases achieved CR by 6 months, while one progressed to ESKD. The ANS case is still under treatment.

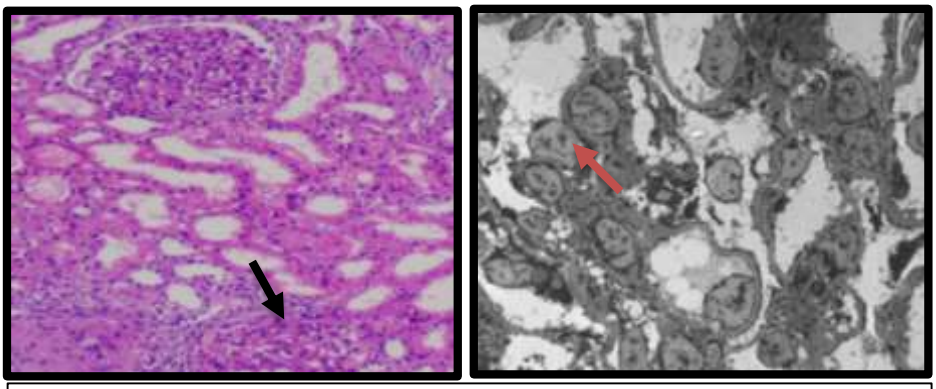

Figure-1 showing light microscopy and electron biopsy of patient with ANS
Black arrow showing crescent formation; red arrow shows multiple EDDs in Mesangial, subendothelial and subepithelial regions; (*Image courtesy – Dr. Lal path lab, India)*

AIM AND OBJECTIVES

AIM

To study the clinical profile and treatment response in biopsy proven cases of C3GN

OBJECTIVES

1. To analyse the various clinical profiles of biopsy proven cases of C3GN.
2. To analyse the response of immunosuppressive treatments and their clinical outcome at 3 and 6 months

CONCLUSION

The study highlights the heterogeneity in the clinical presentations of C3GN. Standard treatments led to clinical improvement in a substantial number of patients, with only one patient progressed to ESKD. Therefore, early and individualized treatment approaches are crucial, as they can significantly change the outcomes for patients with C3GN.

Table-1 Clinical characteristics, laboratory values and histopathology of C3GN Patients (n=10)

Case no.	Clinical syndrome	Macroscopic hematuria	Proteinuria (g/day)	Peak S. Creat. (mg/dl)	Serum C3 (mg/dl)	Outcome
1.	NS	ABSENT	6	0.98	155	CR by 3 months
2.	RPGN	PRESENT	2.8	7.6	46	CR by 3 months
3.	NS	ABSENT	9.6	2.5	151	CR by 6 months
4.	CGN	ABSENT	1.84	5.1	68	ESKD
5.	RPGN	PRESENT	2.3	7.2	51	Stabilization of serum creatinine and remission of proteinuria by 6 months
6.	ANS	PRESENT	1.8	1.37	36	Under treatment
7.	CGN	ABSENT	1.2	2.72	136	CR by 6 months
8.	NS	ABSENT	3.8	0.88	48	CR by 6 months
9.	CGN	PRESENT	2.4	3.72	39	CR by 6 months
10.	RPGN	PRESENT	3.2	7.5	55	CR by 3 months

C3GN- C3 Glomerulonephritis, NS- Nephrotic Syndrome, CGN- Chronic glomerulonephritis, CR- Complete Remission, RPGN- Rapidly progressive glomerulonephritis, ESKD- End stage kidney disease, IFTA- Interstitial fibrosis and tubular atrophy, MPGN-membranoproliferative, EDD-Electron dense deposits

References: * Sethi S, Fervenza FC, Zhang Y, Zand L, Vrana JA, Nasr SH, Theis JD, Dogan A, Smith RJ. C3 glomerulonephritis: clinicopathological findings, complement abnormalities, glomerular proteomic profile, treatment, and follow-up. Kidney Int. 2012 Aug;82(4):465-73. doi: 10.1038/ki.2012.212. PMID: 22673887; PMCID: PMC4438675.