

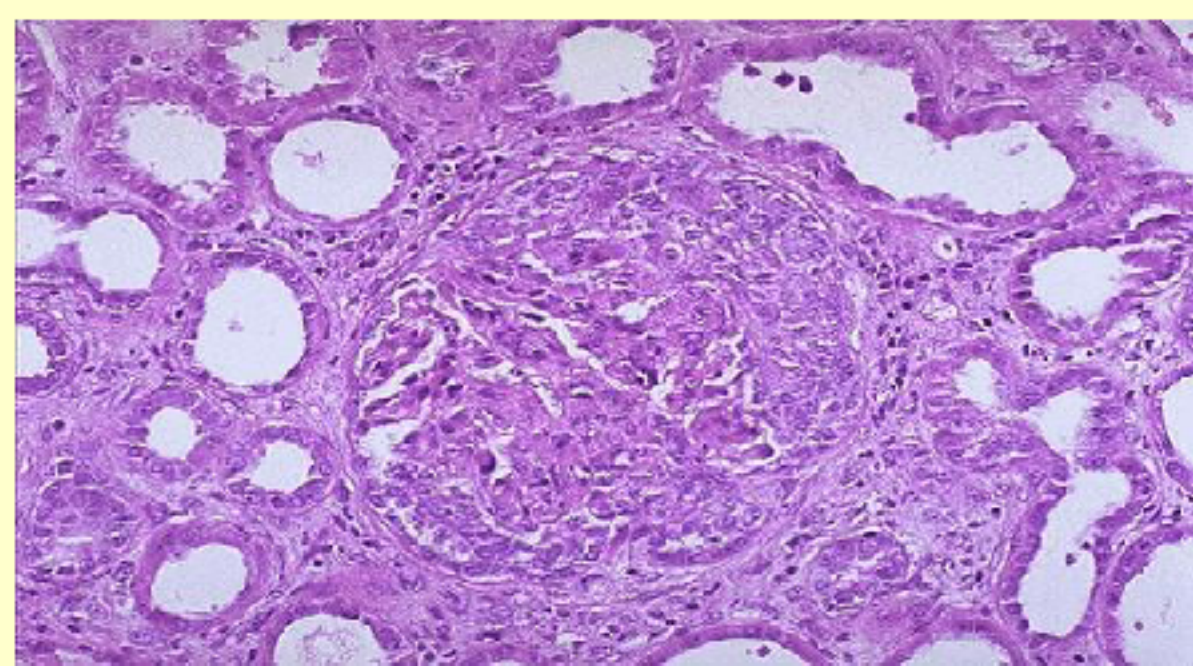
CLINICAL COURSE OF RAPIDLY PROGRESSIVE GLOMERULONEPHRITIS – A CROATIAN REFFERAL CENTRE EXPERIENCE

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OBJECTIVES

Rapidly progressive glomerulonephritis (RPGN) represents a clinical and pathological entity of various causes characterised by rapid deterioration of kidney function and loss of normal renal architecture with extracapillary crescent formations.

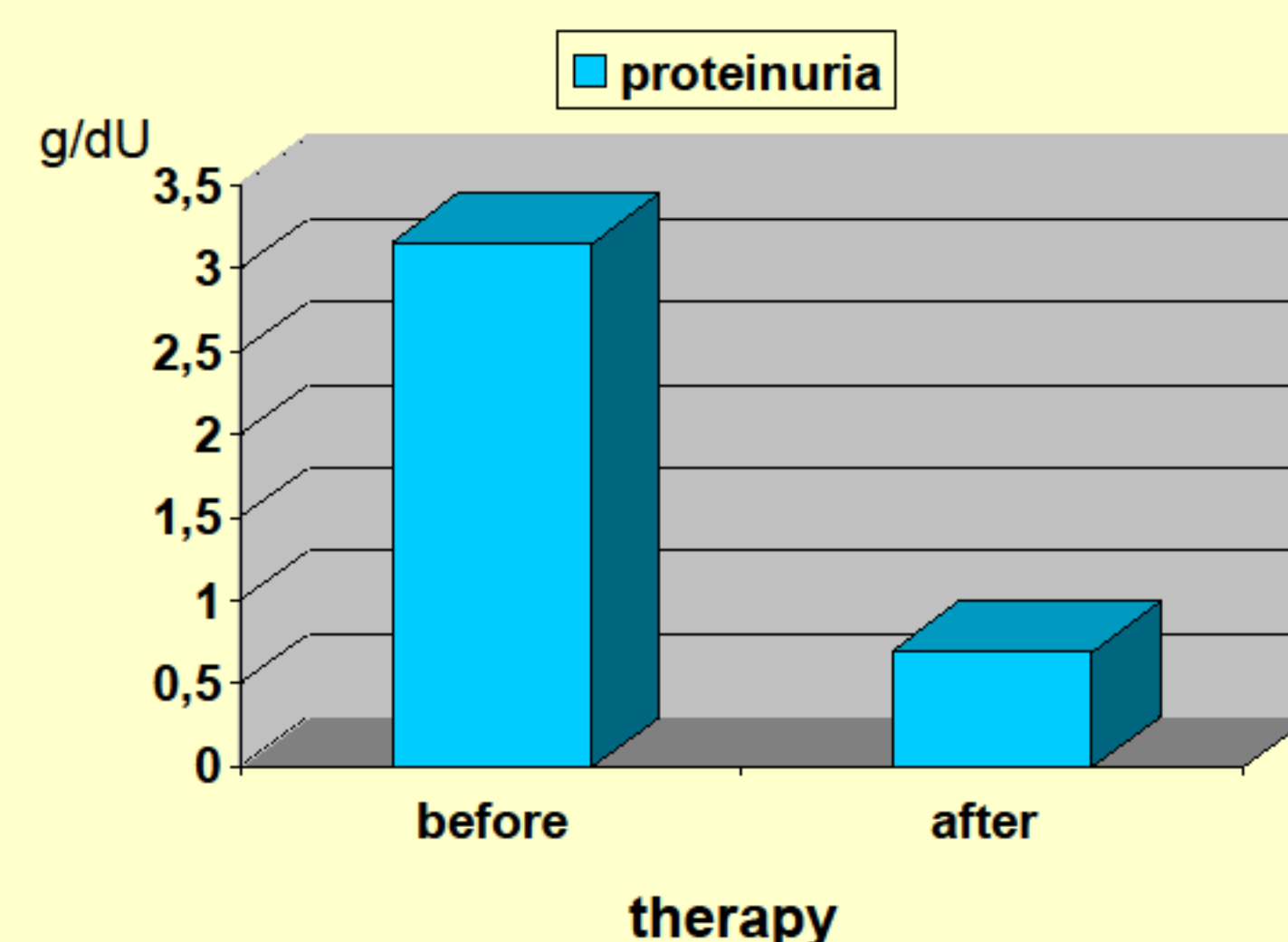
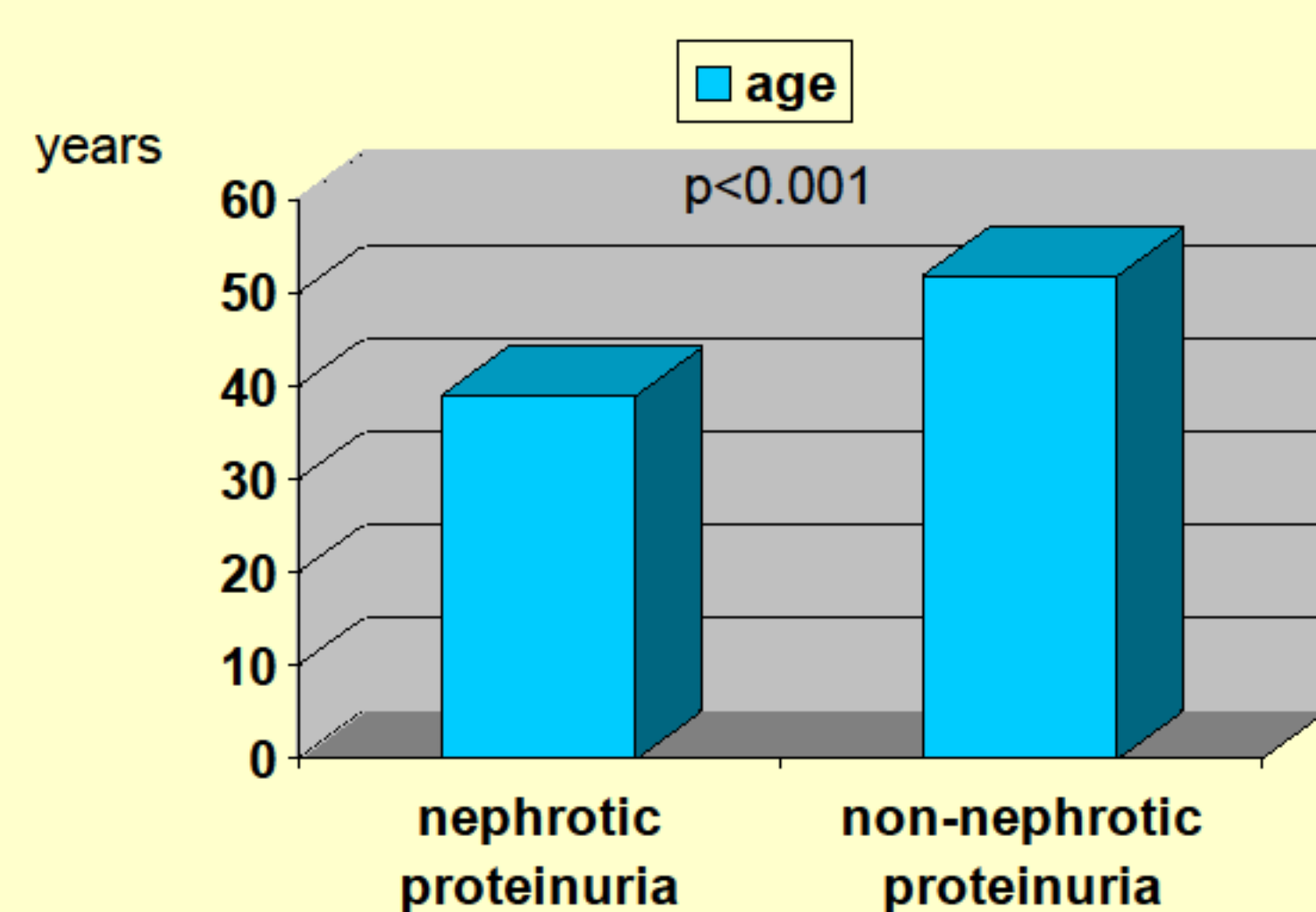


METHODS

In this report we present a clinical course of 70 patients treated for RPGN in Croatian Referral Centre for Glomerulonephritis in the period from 1987-2012. Median age of patients was 50 years (range 18-70), 60% were men. Definitions: renal insufficiency (RI) = serum creatinine (sCr) > 115 μmol/l; nephrotic proteinuria (NP) > 3.5 g/24h; arterial hypertension (AH) > 140/90 mmHg and/or administration of antihypertensive medication. Complete remission was defined as recovery of renal function, proteinuria < 0.25 g/dU and negative urine sediment. Partial remission was defined as proteinuria range from 0.25-3.5 g/dU, negative urine sediment and 50% reduction in the serum creatinine values. Data are presented as mean±SD for normally distributed and median, interquartile range (IQR) for not normally distributed variables, respectively.

RESULTS

Mean duration of renal disease was 6 (4-12) months prior to the kidney biopsy, 65 (93%) of patients presented with RI, 32 (46%) had NP, 52 (83.9%) hematuria and 52 (74%) had AH. Eleven patients (19%) were ANCA positive of which 7 (64%) had lung affection. Four patients (7%) were ANA positive. Patients with nephrotic syndrome were younger (39±17 vs. 52±16; p<0.001), while disease in patients older than the group median presented more commonly with RI (85% vs. 100%; p=0.020). Depending on the clinical course and histology findings, patients were treated with immunosuppressive drugs while in 12 patients (20.3%) plasmapheresis was performed. Forty-nine patients were continuously followed up. Mean time of follow up was 4 (2-30) months. During the follow up significant reduction in proteinuria was observed (3.15 (1.40-5.32) vs. 0.70 (0.23-2.57); p<0.001) with no difference in serum creatinine values (p=0.170) or blood pressure (p=0.573). At the end of follow up, 9 patients (18%) were in complete remission while 20 (36%) were in partial remission and 28 patients (45%) developed ESRD. Basal serum creatinine values and percentage of crescent formations were independent predictors of progression to ESRD (OR 1.029; CI 1.002-1.059; p=0.047, and OR 1.005; CI 1.001-1.008; p=0.005, respectively).



Independent predictors of progression to ESRD		
	OR (95% CI)	p
Creatinine (s) at presentation	1,029 (1,001-1,059)	0,047
Percentage of crescents	1,005 (1,001-1,008)	0,005

CONCLUSIONS

Our results confirmed that RPGN is severe and potentially life-threatening clinico-pathological entity despite aggressive therapy. Basal serum creatinine and histology findings determine the therapeutic approach and prognosis. Results of our centre are in concordance with the results of other authors.

