

A review of the “MEST-C” score in IgAN

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Background:

IgAN is the most common glomerular pathology worldwide. In addition to BP, proteinuria and eGFR at presentation, the MEST criteria were published in 2009 to assist with histological classification and disease prognostication. The IgAN Nephropathy Classification Working Group added a fifth parameter; “C” crescent score. It is well accepted that BP control with renin-angiotensin system inhibition (RASi) is the cornerstone of IgAN management. The appropriate use of immunosuppression remains an area of debate. The aim of this retrospective study, was to review those biopsies with positive “C” scores.

Methods:

A list of all renal biopsies performed in our centre over a twenty-year period (January 2000 – January 2020) was collated. From a total of 3555 biopsies performed, 525 had a diagnosis of IgAN. Each of these biopsies were reviewed including their MEST scores and baseline demographic and laboratory data; therapeutic strategies and outcomes were analysed.

Results:

A total of 34 biopsies had a positive “C” score (C1 or C1). These 34 biopsies were done in 32 patients. (2 of the patients had 2 biopsies). All 34 were native kidney biopsies. Males accounted for 59% of the cohort. The mean age of biopsy was 46 years (ranging from 18-90years). Indication for biopsy included: 38% for Haemato-proteinuria and renal impairment; 29% were for HSP-type presentations; 14% had nephrotic syndrome at presentation. 70% had C1 scores; 30% had C2 scores. The mean number of glomeruli in each biopsy was 15 (ranging from 5-38).

With regard to treatment strategies, 78% of patients received RASi and 85% received immunosuppressive therapy. These treatment strategies included intravenous cyclophosphamide, prednisolone monotherapy, MMF and prednisolone in combination, and azathioprine in conjunction with steroids. A total of 62% of patients received dual therapy in the form of both RASi and immunosuppression.

At the time of biopsy, the mean creatinine was 297 $\mu\text{mol/l}$ (ranging from 47-880) and mean UCPR was 587g/l (ranging from 70- 1863). 40% of the cohort reached ESKD during follow-up. There was a 28% mortality within the cohort. There was an 18% 1-year mortality; 2 deaths were attributable to sepsis; one death was attributable to unrelated pulmonary fibrosis; the cause of death in the remaining three patients were unknown.

Conclusion:

Crescentic IgAN is defined as >50% crescentic glomeruli on kidney biopsy. It is well documented that crescentic IgAN, a rare phenotype, has a poor prognosis; often presenting as rapidly progressive glomerulonephritis. Little is known about those patients which have evidence of crescents on their biopsies, but that do not meet criteria for “crescentic IgAN”. Our small study gives real-world outcomes in those that have evidence of crescents on their biopsies. This data confirms that this cohort warrants close surveillance.