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Review of histological variants and outcomes in IgA Nephropathy

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Introduction
IgA nephropathy (IgAN) is the most common cause of primary glomerular disease worldwide. It has a wide spectrum of clinical presentations, varying from isolated haematuria to anuric renal failure. The risk of progression is often evaluated in routine clinical practice by thorough evaluation of proteinuria, blood pressure and eGFR at the time of presentation. In addition, the OXFORD MEST score gives consensus criterion for the pathological classification of this common renal diagnosis. This enables accurate prediction of disease progression and individual prognostication. The aim of our study was to investigate the association of baseline clinical data and histological variants upon renal outcomes in our cohort of patients with IgA nephropathy. Since end of 2013, our renal centre cohorts patients with known glomerulonephritis diagnoses into a bespoke "Complex Glomerulonephritis" (CGN) clinic.

Methods
All available patients with biopsy-proven IgAN in our centre between January 2014 and January 2020 were included in this retrospective observational study. Baseline data at the time of biopsy included patient demographics and laboratory variants. Renal biopsies were reviewed and histopathological variants were collected according to their MEST scores. Follow up data on renal function and mortality data was collected, which included death, loss to follow up and ESKD.

Results
A total of 1165 renal biopsies were performed at our centre in this 6 year period; 182 of these biopsies revealed a diagnosis of IgAN. The median age of the cohort at diagnosis was 46 years, ranging from 17 to 86 years. Males accounted for 66% of the cohort. 91% of these biopsies were of native kidneys, with the remaining 9% being transplanted kidneys. 9% of the cohort died during the follow up. The main indications for biopsy were: renal impairment and haematoproteinuria (36%); known IgAN with progressive CKD and/or proteinuria (20%), nephrotic syndrome (12%) and HSP-type presentation (10%). Median creatinine at biopsy was 152umol (ranging from 46-2045). Median UPCR was 212 (ranging from 5-3083). 24% required some form of RRT. 46% were treated with RASi alone following the biopsy result; 28% received some form of immunosuppressive therapy (prednisolone monotherapy, MMF and prednisolone, IV cyclophosphamide and prednisolone). 18% had crescents on their biopsies (+C score). Those with crescents on their biopsies had an average 8% increase in their creatinine during follow-up, compared to a 10% decrease in creatinine for those with no crescents.

Conclusion
Ongoing evaluation of the predictive utility of these scoring systems is recommended for early effective treatment intervention. Despite considerable improvements in histological characterisation to predict progression in IgAN, the optimal therapeutic management remains debatable. RASi is essential in management of BP control and proteinuria reduction. Additional immunosuppression is of benefit in those at risk for a progressive disease course.
Mesangial hypercellularity (M-score) is considered a sensitive predictor of disease progression. 53% of our cohort had an M1" score. Our CGN clinic allows us to appropriately cater for these individuals with a more rapid annual decline in eGFR.