IS IT POSSIBLE TO PREDICT PROGRESSION OF IGA NEPHROPATHY IN THE CAUCASIANS BY USING HISTOLOGICAL AND CLINICAL MARKERS?

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INTRODUCTION AND AIMS

Predictors of renal failure in IgA nephropathy (IgAN) have been assessed in several clinicopathologic studies and IgAN progression risk scores have been derived based on cohorts of Chinese patients. However, prospective validation of this risk scores especially in the Caucasians is lacking. This study aimed to evaluate IgAN progression and its histological and clinical correlates in a Caucasian cohort.

METHODS

A total of 123 IgAN patients (55.3% male, mean age:36±12 years, mean follow up of 39±29 months) who were being followed-up for more than six months were evaluated. Renal survival and the relationship between clinical parameters and composite kidney failure events (defined as end stage renal disease (ESRD) or a two-fold increase in serum creatinine level as compared to baseline) were assessed. The impact of histopathological lesions (glomerular sclerosis, intensity and pattern of staining for C3, C1Q, IgA, IgG, IgM) and clinical markers (age, gender, systolic blood pressure (SBP), eGFR, serum creatinine, hemoglobin (Hgb), albumin, proteinuria, hematuria) on progression were analyzed using Cox regression.

RESULTS

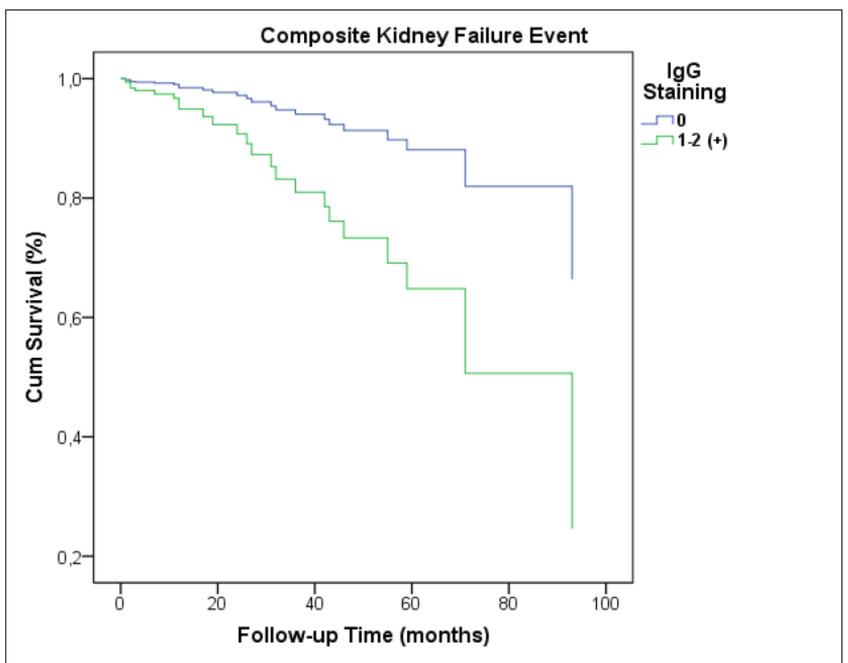
The mean eGFR of patients at baseline was 69 34 ml/min/1.73 m^2 and composite kidney failure events developed in 26 (21%) patients after a median of 31 months. Although the intensity of C3 staining and IgA were not associated with progression of IgAN, the presence of IgG [6/26 (23.1%) vs 6/97 (%6.2) (p=0.01)] and IgM staining [14/26 (53.8%) vs 31/97 (%32.2) (p=0.04)] were associated with kidney failure. In cox regression analysis, percentage of glomerulosclerosis (HR:1.028, p=0.021), presence of IgG staining (HR:3.326, p=0.027) as histopathological markers, proteinuria (g/24 hour) (HR:1.468, p=0.001), eGFR (HR:0.968, p=0.003) and Hgb (HR:0.598, p=0.006) levels at baseline as clinical markers predicted composite kidney failure events.

Figure 1: Cox-regression analysis of composite kidney failure events (defined as ESRD or a two-fold increase in serum creatinine level as compared to baseline) and variables in the equation.

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
Age	-,035	,027	1,741	1	,187	,966
IgG Staining	1,202	,543	4,899	1	,027	3,326
Proteinuria (baseline)	,384	,116	10,896	1	,001	1,468
eGFR (baseline)	-,032	,011	9,111	1	,003	,968
%Glomerulosclerosis	,027	,012	5,297	1	,021	1,028
Systolic BP (baseline)	,000	,013	,001	1	,980	1,000
S. Albumin (baseline)	-,256	,523	,240	1	,624	,774
Hgb (baseline)	-,515	,188	7,524	1	,006	,598
IgM Staining	-,090	,576	,024	1	,876	,914

Figure 2: Cox-regression analysis of composite kidney failure events according to IgG staining levels in biopsy specimens of patients (HR:3.326, p=0.027).



CONCLUSIONS

A new progression risk score for IgAN can be calculated based on these histopathological markers including percentage of glomerulosclerosis and presence of IgG staining, and clinical markers including proteinuria, eGFR and Hgb levels at baseline to predict the risk of progression to ESRD.

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