KSN 2020
FULLY VIRTUAL MEETING

Abstract Type : Oral
Abstract Submission No. : OR-1054

Foot process effacement induced by mesangial proliferation leads to proteinuria in IgA neprhopathy

Jin Young Yu[1], Ji-Hye Lee[2], Nam-Jun Cho[1], Eun Young Lee[1], Hyo-Wook Gil[1], Samel Park[1]
[1]Department of Internal Medicine-Nephrology, Soonchunhyang University Cheonan Hospital, Korea, Republic of
[2]Department of Pathology, Soonchunhyang University Cheonan Hospital, Korea, Republic of

Objectives: Proteinuria is a significant risk factor of the progression of IgA nephropathy (IgAN), and has a positive correlation with the severity of foot process effacement (FPE). We evaluated the relation between FPE, proteinuria, and histologic characteristics, including the Oxford classification.

Methods: Patients who performed a renal biopsy and were diagnosed with IgAN at a single center were retrospectively reviewed. Young patients with age under 18 years old and the possibility of secondary causes were excluded. Then, we evaluated the association between the degree of proteinuria, the severity of FPE, and histologic characteristics, including the Oxford classification and other immunofluorescence stains.

Results: total of 805 cases of the renal biopsy performed at our institution, 327 patients were diagnosed with IgAN. Among them, 57 patients were excluded. The severity of FPE had an impact on the degree of proteinuria. Notably, the group with FPE ≥ 90% had more than about 2 g/day of urine protein compared to those with FPE < 10%. Among the histologic characteristics, M1 score and immune deposition of IgG affected the severity of FPE [hazard ratios (95% CI), 1.93 (1.15 – 3.24), and 3.86 (1.78 – 8.36), respectively] (Table 1).

Conclusions: The severity of FPE had an impact on the degree of proteinuria, and the severity of FPE was associated with the pathogenesis of IgA nephropathy.

Table 1. The histologic characteristics associated with the severity of foot process e

Variables	Odds ratios (95% CI)	*p*-value
The Oxford classification		
M1 (vs. M0)	**1.93 (1.15 - 3.24)**	**0.013**
E1 (vs. E0)	1.23 (0.73 - 2.05)	0.437
S1 (vs. S0)	0.96 (0.49 - 1.88)	0.914
T1 (vs. T0)	1.49 (0.81 - 2.75)	0.197
T2 (vs. T0)	2.66 (0.83 - 8.58)	0.101
C1 (vs. C0)	0.78 (0.45 - 1.35)	0.372
C2 (vs. C0)	0.27 (0.02 - 4.81)	0.376
Immunoflourescence		
IgA		
1+	Reference	
2+	0.89 (0.50 - 1.59)	0.693
≥ 3+	0.63 (0.26 - 1.53)	0.305
IgG		
Negative	Reference	
Positive	**3.86 (1.78 - 8.36)**	< **0.001**
C3		
Negative	Reference	
Trace	2.92 (0.37 - 22.81)	0.307
1+	0.91 (0.13 - 6.26)	0.922
≥ 2+	1.24 (0.16 - 9.51)	0.834
Age-adjusted glomerulosclerosis		
GSG normal for age	Reference	
GSG abnormal for age	1.5 (0.87 - 2.56)	0.142

Note: Multivariable ordinary logistic analysis was used.

Abbreviations: M, mesangial hypercellularity; E, endocapillary hypercellularity; S glomerulosclerosis; T, tubular atrophy/interstitial fibrosis; C, crescents, GSG, globa