

# KSN 2016 Abstract Submission

## *Glomerular disease*

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### **Circulating CD89-IgA complex does not predict progression of IgA nephropathy**

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**Background:** Pathogenesis of IgA nephropathy (IgAN) is a complicated multi-step process involving generation of galactose-deficient IgA1 and autoantibodies against the misglycated IgA1, deposition of immune complex within the mesangium, and complement activation. CD89 is a soluble receptor for circulating IgA and CD89-IgA facilitates the formation of immune-complex. However, there is lack of evidence supporting circulating levels of CD89-IgA complex is associated with disease progression. Thus, this study aimed to delineate whether circulating CD89-IgA levels can predict the future renal outcome in patients with IgAN.

**Methods:** A total of 344 patients with biopsy-proven IgAN between 2005 and 2014 were included in this study. Patients with estimated glomerular filtration rate (eGFR) <30 ml/min/1.73m<sup>2</sup> were excluded from the analysis. Demographic and laboratory data were recruited from the Glomerulonephritis Registry of Yonsei University Health System. Sera of these patients were obtained at the time of biopsy and stored at -80°C. Circulating CD89-IgA complex levels were determined by sandwich ELISA method. The study outcome was a 30% decrease of eGFR during the follow up.

**Results:** The median value of CD89-IgA complex was 7.20 ng/ml (inter-quartile range 4.25 to 12.98). Patients were categorized into 3 groups by tertiles of circulating CD89-IgA complex levels. There were no significant differences in baseline eGFR and proteinuria among the 3 groups. In addition, circulating CD89-IgA complex levels were not correlated with eGFR at the time of biopsy and did not differ among chronic kidney disease stages. During follow-up, 23 (34.3%), 25 (37.3%), and 19 (28.4%) patients in the lowest, middle, and highest tertiles reached the study endpoint, respectively (P=0.59). In a multiple Cox model adjusted for age, sex, mean arterial pressure, IgA levels, eGFR, proteinuria, and circulating CD89-IgA complex levels were not associated with developing a 30% decrease in eGFR (lowest versus middle, hazard ratio [HR] 0.95, 95% confidence interval [CI] 0.44-2.07, P=0.89 and lowest versus highest, HR 1.14, 95% CI 0.49-2.61, P=0.76). A receiver operating curve analysis showed that area under the curve for CD89-IgA complex was 0.55.

**Conclusion:** Although CD89-IgA complex mediates formation of immune complex, our findings suggest that its circulating level is not a predictor of adverse renal outcome in IgAN.

**Keywords:** CD89, IgA complex, IgA nephropathy