Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care
KSN SEOUL, KOREA 2026
The 46th Annual Meeting of the Korean Society of Nephrology
THE KOREAN SOCIETY OF NEPHROLOGY
Jun 11 (Thu) – 14 (Sun), 2026
COEX, Seoul, Korea

Abstract Type : Poster exhibition
Abstract Submission No.: A-0676
Abstract Topic : Glomerular and Tubulointerstitial Disorders

Spatial Transcriptomic Profiling Reveals Molecular Signatures of Endocapillary Hypercellularity in IgA Nephropathy

Jin Sug Kim[1], Hyeon Do Jeon[3], Kyung Hwan Jeong[1], Hyeon Seok Hwang[1], Ji-Youn Sung[2], Kyungsik Ha[4], Woochang Choi[4], Hwajin Lee[5]
[1]Department of Internal Medicine-Nephrology, Kyung Hee University Medical Center, Korea, Republic of
[2]Department of Precision Medicine, Kyung Hee University College of Medicine, Korea, Republic of
[3]Department of Pathology, Kyung Hee University Medical Center, Korea, Republic of
[4]Department of UPPThera, inc., UPPThera, inc., Korea, Republic of
[5]Department of Biochemistry and Molecular Biology, Kyung Hee University College of Medicine, Korea, Republic of

Objectives : Immunoglobulin A nephropathy (IgAN) is a leading cause of chronic kidney disease, marked by mesangial IgA1 immune-complex deposition and a multi-hit pathogenesis. The Oxford classification grades histopathological lesions such as endocapillary hypercellularity (E0 and E1), which are associated with disease severity and prognosis. Despite advances in single-cell and spatial transcriptomics, the molecular basis of endocapillary hypercellularity in human IgAN remains unclear.
Methods : Spatial transcriptomic data obtained using the GeoMx DSP platform from kidney biopsy specimens of East Asian patients were analyzed to compare gene expression profiles across normal, E0, and E1 groups. Differentially expressed genes (DEGs) were identified, and subsequent gene ontology and protein–protein interaction network analyses were performed to investigate functional pathways and molecular interactions.
Results : Analysis identified 50 DEGs between E0 and controls, with more extensive changes in E1 compared with both E0 and controls (342 and 223 DEGs, respectively). E1 lesions displayed distinct molecular signatures, including the novel downregulation of amyloid beta precursor protein, cluster of differentiation 47, and integrin alpha V, along with well-known interactions among involving dual-specificity phosphatase 1, early growth response 1, and Fos proto-oncogene. Functional enrichment highlighted pathways related to ion response and mitogen-activated protein kinase/extracellular signal-regulated kinase signaling.
Conclusions : This study provides new insights into the molecular changes associated with endocapillary hypercellularity in IgAN progression. The identified gene signatures and pathways may serve as potential biomarkers of disease activity and targets for therapeutic intervention.