Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care
KSN 2026 SEOUL, KOREA
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 : Oral presentation
Abstract Submission No.: A-0609
Abstract Topic : Glomerular and Tubulointerstitial Disorders

Plasma Proteomic Profiling of Minimal Change Disease Reveals Immune Signatures with Impaired Mer-Associated Efferocytosis

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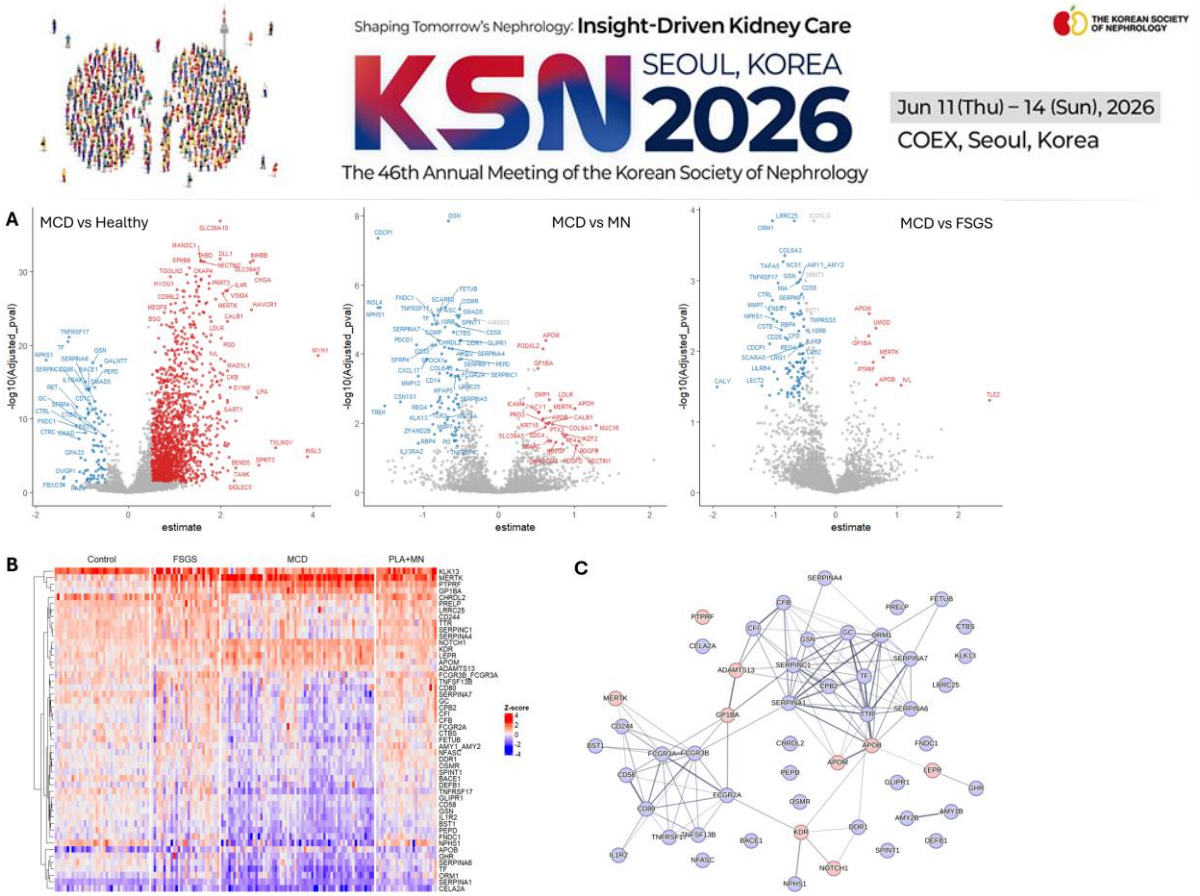
Objectives : Minimal change disease (MCD) is a major cause of nephrotic syndrome. Recent studies suggest autoantibodies targeting podocyte components such as nephrin contribute to pathogenesis. However, the circulating molecular landscape of MCD and its pathophysiologic significance remain incompletely characterized.
Methods : Plasma proteomes of 63 MCD cases, including 26 paired remission samples, and 94 controls (40 healthy, 28 focal segmental glomerulosclerosis, 26 membranous nephropathy) were profiled using the Olink Explore HT platform. Differential expression and functional enrichment analysis were complemented by single-cell RNA sequencing (scRNA-seq) analysis of peripheral blood mononuclear cells (PBMCs) including 29,713 MCD and 21,436 healthy control cells. ELISA validation was performed for plasma Mer and anti-nephrin autoantibodies. Patient-derived macrophages (5 MCD, 5 healthy controls) were assessed in a pHrodo-based efferocytosis assay using TNF (tumor necrosis factor)-α–induced apoptotic podocytes with or without Mer shedding inhibition by TAPI.
Results : Of 5,438 proteins profiled, 50 were consistently differentially expressed in MCD compared with all three control groups (false discovery rate < 0.05). Functional enrichment analysis highlighted complement and coagulation processes, serine-type endopeptidase inhibitors, and growth factors. Mer tyrosine kinase (+2.04) and nephrin (−1.78) showed the largest differences relative to healthy controls. As Mer is primarily a macrophage receptor promoting efferocytosis, monocyte populations were examined in the scRNA-seq dataset, where MCD was associated with lymphocyte activation, TNF pathway signals, and elevated ADAM17, which cleaves Mer. Consistently, soluble Mer and anti-nephrin autoantibody levels were significantly higher at baseline than in remission or other controls. MCD-derived macrophages exhibited impaired efferocytosis of apoptotic podocytes compared with healthy controls ($p = 0.004$) that improved after Mer shedding inhibition ($p = 0.001$).
Conclusions : The plasma proteome of MCD reveals prominent immune-related alterations. Increased Mer shedding in MCD may impair efferocytosis and thereby promote autoantibody formation, suggesting a previously unrecognized pathophysiologic mechanism.

Fig1.png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A
MCD vs Healthy
MCD vs MN
MCD vs FSGS
-log10(Adjusted_pval)
estimate
B
Control
FSGS
MCD
PLA+MN
Z-score
C

Fig1.png

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