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

Lecture Code : BR02-S1
Session Name : Basic Research 2
Session Topic : Basic Research 2
Date & Time, Place : June 12 (Fri) / 08:30-10:10 / Room 1 (GBR 101), 1F

Pathogenic Mechanisms of IgAN: Focusing on Mucosal B-Cell Responses

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IgA nephropathy (IgAN) is the most common primary glomerulonephritis in the world, particularly in East Asia. Untreated, approximately 40-50% of cases progress to end-stage kidney disease or death within 20 years. Over half a century has passed since the disease was first described, and its pathogenesis is gradually clarified. In IgA nephropathy, after developing upper respiratory infection or enteritis, macroscopic hematuria and increased proteinuria may occur indicating that dysregulated mucosal immunity plays a central role in its pathogenesis. In particular, plasmacytoid dendritic cells and abnormal activation and differentiation of mucosal B cells contribute to the overproduction of galactose-deficient IgA1 (Gd-IgA1), a key pathogenic molecule in IgAN. These aberrant immune responses, driven by interactions among mucosal antigens, the microbiota, and cytokines such as BAFF and APRIL, promote the formation of nephritogenic immune complexes that ultimately deposit in the glomerular mesangium. Activation of mesangial cells and complement activation by the deposited IgA1 immune complexes induces nephritis, which progresses to podocyte and tubular damage. This session focuses on the mechanisms by which mucosal B-cell responses contribute to the development and progression of IgAN and discusses the therapeutic implications of targeting mucosal immune pathways in IgAN.

Keywords: IgA nephropathy, Gd-IgA1, Immune complex, BAFF, APRIL