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Membranous glomerulonephritis

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Membranous nephropathy (MN) is a common cause of adult nephrotic syndrome, and frequently diagnosed by renal biopsy practice.

Heymann nephritis, a rat model of experimental MN, reveals the pathogenesis of MN being an antibody-mediated autoimmune disease. However, the auto-antigen of MN remained elusive for over 50 years. The auto-antigen of MN revealed only in cases of BSA-related MN, neonatal (anti-NEP) MN and some secondary causes. In 2009, Beck and colleagues discovered that autoantigen is mainly a M-type trans-membrane phospholipase A2 receptor (PLA2R) located on podocytes, and autoantibody is mainly an IgG4 subclass. With these new findings idiopathic MN should no longer be considered as an idiopathic disease. However, the triggers of autoantibody production and mechanisms of its action are still unknown and investigations for other likely antigens are in progress.

Membranous nephropathy is characterized by thickened glomerular capillary walls in light microscopic examination. The earliest stages may have no alterations visible by light microscopy. The early lesion consists only of rigid-appearing capillary walls without visible deposits. In well prepared tangential sections, a pinpoint "pore" or mottled appearance can be observed on Jones silver stain. Perpendicular extension of glomerular basement membrane matrix to the deposits results in a "spike". With progressing, the matrix encircles the deposits resulting in an irregular thickening and a chain-like appearance with or without resorption of deposits. Segmental sclerosis, tubular atrophy and interstitial fibrosis may be present and known to associate with a poor renal survival. By immunofluorescence, diffuse and global, finely granular staining along the capillary walls for IgG (often IgG4 dominant in primary MN) and C3 is characteristics. By electron microscopy, glomerular basement membrane is thickened by subepithelial electron dense deposits with varying degree of matrix reactions and diffuse foot process effacement.

Minimal changes disease, focal segmental glomerulosclerosis, membranoproliferative glomerulonephritis, diabetic nephropathy, and amyloidosis can be considered as differential diagnosis, however, diagnosis of MN can be made based on immunofluorescence and electron microscopic findings. Secondary MN may have additional features, including endocapillary/mesangial proliferation, mesangial/subendothelial deposits, tubuloreticular structure, more deposits than IgG/C3, and negative IgG4 staining.