

A short note on how podocyte injury impacts diabetic nephropathy

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Introduction

Podocytes are highly specialized glomerular epithelial cells that form the GFB together with the fenestrated endothelium and Glomerular Basement Membrane (GBM). The podocyte soma arches the urethra and gives rise to long primary processes that branch into the foot processes and envelop the glomerular capillaries. Her FPs of adjacent podocytes ligate, leaving a long filtering slit between them bridged by a junction called the Slit Diaphragm (SD). These interactions are essential for maintaining the highly ordered structure of FPs. SD is considered the major restriction site for protein filtration. However, the negatively charged sialoglycoproteins lining the GBM-facing outer tube surface of podocytes also contribute to GFB by shedding plasma anionic proteins. Moreover, compression of her GBM against her FP *via* flow alters the physical properties of the GBM, thereby increasing her GBM permselectivity. Finally, Vascular Endothelial Growth Factor (VEGF) secretion by podocytes influences glomerular endothelium permeability.

Description

This simplified architecture is termed FP loss and is associated with proteinuria even in the absence of podocyte loss. Initial FP loss is reversible, but if the underlying injury is not resolved, FP loss progresses until the FP-deprived podocyte binds to the GMB only through the cell body. Podocyte loss, a key feature of progressive proteinuria glomerulopathy, is due to apoptosis or podocyte detachment. Although dedifferentiation may protect podocytes from death, it may alter podocyte function/structure and lead to GBM detachment. In addition, injury leads to acti-

vation of integrin $\alpha\beta3$, which promotes podocyte detachment. In glomerulopathies characterized by glomerular hypertrophy involving DN, podocyte hypertrophy overlying the increased GBM surface area. This could reduce podocyte density without altering the number of podocytes. Podocyte injury is associated with proteinuria glomerulopathy, regardless of the initiating cause of podocyte injury (genetic, immune, infectious, toxic, metabolic, and hemodynamic) and the presence of other associated histological abnormalities. Therefore, these diseases have recently been renamed podocytopathies.

Podocytes are highly specialized cells of the kidney glomerulus that are present around bowman's capsule. The term Diabetic Kidney Disease (DKD) includes all types of kidney damage that occurs in people with diabetes. The classic albuminuria form of DKD is primarily due to glomerular/podocyte damage and is characterized by both increased glomerular permeability to proteins and a persistent decline in renal function. DKD can occur in the absence of albuminuria, and non-albuminuria is now the dominant phenotype in his type 2 diabetic patients. However, the non-albuminuria phenotype appears to be primarily associated with atypical vascular or tubulointerstitial lesions rather than podocyte injury.

Conclusion

Over the past decades, numerous studies on podocytes exposed to high-glucose environments have shown that hyperglycemia can alter podocyte phenotype by inducing loss of nephrin and alterations in the production/degradation of extracellular matrix components proved to be viable. A decrease in nephron number due to low nephron mass/nephron loss results in a compensatory increase in glomerular capillary pressure/filtration of the remaining single nephron. This leaves the total glomerular filtration rate unchanged for long periods, but causes podocyte damage, representing an important mechanism in CKD progression. Also, in obesity and diabetes, both glomerular capillary hypertension and single-nephron hyperfiltration are early events that precede nephron loss.

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