

Biomarker Discovery in Diabetic Kidney Disease

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Brief Report

Although estimated glomerular filtration rate and albuminuria are well-established biomarkers of diabetic kidney disease (DKD), more biomarkers are needed, particularly in the early stages of the disease when both albuminuria and estimated glomerular filtration rate may still be within normal limits and are less useful for identifying those at risk of progression. Because there are few excellent early clinical end points for early DKD, traditional biomarker studies are difficult to conduct, and most rely on changes in existing imprecise biomarkers to determine the utility of novel biomarkers. However, there are well-defined changes in kidney structure that are closely linked with kidney function, invariably occur before the clinical manifestations of DKD, and can predict DKD progression at preclinical stages. As a result, these structural features could be used as therapeutically meaningful endpoints for finding new early DKD biomarkers. Furthermore, researchers are analysing tissue transcriptomic data to identify pathways involved in early DKD that may have associated candidate biomarkers measurable in blood or urine, and differentially expressed microRNAs and epigenetic modifications in kidney tissue are beginning to yield important observations that could aid in the discovery of new clinically useful biomarkers. The current literature on the use of kidney tissue in the development of biomarkers in DKD is examined in this review.

Diabetic kidney disease (DKD) is mostly clinically diagnosed based on proteinuria and reduced kidney function in the presence of diabetes. As a result, kidney biopsies are not a common element of DKD treatment. Kidney tissue, on the other hand, has shown to be essential in defining the structural abnormalities that underpin DKD and demonstrating how these structural changes connect to treatment outcomes. In normoalbuminuric persons with type 1 diabetes, the width of the glomerular basement membrane (GBM) predicts the development of microalbuminuria, proteinuria, ESRD, and cardiovascular mortality. Furthermore, glomerular structural parameters traditionally associated with DKD, such as increased GBM width, increased mesangial fractional volume, and reduced glomerular filtration surface, correlate strongly with albuminuria and kidney functional changes throughout much of the clinical natural history of DKD, and kidney interstitial changes are responsible for kidney function loss in the later stages of the disease, as per multivariate piece-wise regression models, and kidney interstitial changes are responsible for kidney function loss in the later stages of the disease.

Albuminuria and estimated glomerular filtration rate are the two main biomarkers currently utilised to predict DKD development (eGFR). However, not all cases of classical DKD are associated with an increase in albuminuria, lowering the utility of this biomarker, especially in early DKD. Furthermore, "chronic microalbuminuria," defined as two or more consecutive urine samples in the microalbuminuria range, frequently normalises or stabilises on its

own, reducing the relevance of this otherwise helpful biomarker. As a result, developing new indicators to supplement albuminuria is critical. The hunt for new DKD biomarkers has mostly focused on discovering analytes in urine and blood that can help predict later defined end goals, such as ESRD, a GFR loss of 40%, or mortality. There is also a pressing need to develop biomarkers of DKD in its early phases, when breakthroughs in treatment have the best possibility of slowing disease progression, but when eGFR is still in the normal range and before the emergence of albuminuria levels that are highly predictive of disease progression.

Much of what follows is based on the basic but crucial idea that the early preclinical lesions of diabetic nephropathy are necessary antecedents to the later, more severe lesions that underpin the loss of GFR that leads to ESRD. Kidney biopsy samples can help researchers find DKD biomarkers in a variety of ways. DKD-related structural abnormalities are quantifiable and always precede alterations in renal function. As a result, they could be employed as endpoints in biomarker research. Proteins, peptides, or metabolites found in the blood or urine that are reliably related with earlier structural damage or anticipate changes in kidney structure are now being investigated as potential biomarkers of early tissue injury and eventual clinical progression of DKD. Such markers are most likely coupled in extensive networks that can be disrupted by disease, resulting in changes in their concentrations in biological materials like urine or blood. The dynamic molecular disturbances underlying diabetic kidney structural injury may be reflected in gene expression patterns taken from sick kidney tissue [1-5].

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Received 29 January, 2022, Manuscript No. JNT-22-55595; **Editor Assigned:** 30 January, 2022, PreQC No. P-55595; **Reviewed:** 12 February, 2022, QC No. Q-55595; **Revised:** 17 February, 2022, Manuscript No. R-55595; **Published:** 24 February, 2022, DOI: 10.4172/2161-0959.1000375

How to cite this article: Emli, Jessica. "Biomarker Discovery in Diabetic Kidney Disease." *J Nephrol Ther* 12 (2022): 375.