Perspective
Volume 12:4, 2022

Journal of Nephrology & Therapeutics

ISSN: 2161-0959 Open Access

Diabetic Kidney Disease in Youths

William Fred*

Department of Medicine, Veterans Affairs Medical Center, The University of California, San Francisco, California

Introduction

Proteinuria and impaired kidney function in the presence of diabetes are the most common clinical signs of diabetic kidney disease (DKD). As a result, kidney biopsies are rarely used in the treatment of DKD. Kidney tissue, on the other hand, has been proven to be critical in characterising the structural abnormalities that underlying DKD and showing how these structural changes relate to treatment outcomes [1]. The width of the Glomerular Basement Membrane (GBM) predicts the development of microalbuminuria, proteinuria, ESRD, and cardiovascular mortality in normoalbuminuric people with type 1 diabetes. Furthermore, albuminuria and kidney functional alterations are substantially correlated with glomerular structural measures usually associated with DKD, such as increased GBM width, increased mesangial fractional volume, and reduced glomerular filtration surface [2].

Although estimated glomerular filtration rate and albuminuria are well-established biomarkers of Diabetic Kidney Disease (DKD), more biomarkers are needed, especially in the early stages of the disease, when both albuminuria and estimated glomerular filtration rate may still be within normal limits, making them less useful for identifying those at risk of progression. Traditional biomarker studies are difficult to undertake since there are few excellent early clinical end points for early DKD, therefore most rely on changes in existing imprecise biomarkers to assess the efficacy of novel biomarkers. There are, however, well-defined alterations in kidney structure that are intimately linked to kidney function, occur invariably before the clinical signs of DKD, and can predict DKD progression at preclinical stages [3].

Description

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 [4]. 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 [5]. 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.

Conclusion

The two main indicators used to predict DKD progression are albuminuria and estimated glomerular filtration rate (eGFR). However, not all cases of classical DKD are linked with an increase in albuminuria, making this biomarker less useful, particularly in early DKD. Furthermore, "chronic microalbuminuria," which is defined as two or more consecutive urine samples in the microalbuminuria range, frequently normalises or stabilises on its own, decreasing the use of this otherwise useful biomarker. As a result, it's necessary to create new indicators to supplement albuminuria. The search for new DKD biomarkers has been focused on identifying analytes in urine and blood that can aid in the prediction of later defined end goals, such as ESRD, a GFR loss of 40%, or mortality.

Acknowledgements

None.

Conflict of Interest

There are no conflicts of interest by author.

References

1. Niwa, Toshimitsu. "Biomarker discovery for kidney diseases by mass spectrometry." *J Chromatogr B* 870 (2008): 148-153.
2. Barreiro, Karina, Om Prakash Dwivedi, German Leparc and Marcel Rolser, et al. "Comparison of urinary extracellular vesicle isolation methods for transcriptomic biomarker research in diabetic kidney disease." *J Extracell Vesicles* 10 (2020): e12038.
3. Bringans, Scott D., Jun Ito, Thomas Stoll and Kaye Winfield, et al. "Comprehensive mass spectrometry based biomarker discovery and validation platform as applied to diabetic kidney disease." *EuPA Open Proteom* 14 (2017): 1-10.
4. Looker, Helen C., Michael Mauer, and Robert G. Nelson. "Role of kidney biopsies for biomarker discovery in diabetic kidney disease." *Adv Chronic Kidney Dis* 25 (2018): 192-201.
5. Wu, Jing, Yi-ding Chen and Wei Gu. "Urinary proteomics as a novel tool for biomarker discovery in kidney diseases." *J Zhejiang Univ Sci B* 11 (2010): 227-237.

***Address for Correspondence:** William Fred, Department of Medicine, Veterans Affairs Medical Center, The University of California, San Francisco, California; E-mail: fred.william69@hotmail.com*

Received: 28 March, 2022, Manuscript No. JNT-22-62519; **Editor Assigned:** 30 March, 2022, PreQC No. P-62519; **Reviewed:** 11 April, 2022, QC No. Q-62519; **Revised:** 16 April, 2022, Manuscript No.R-62519; **Published:** 23 April, 2022, DOI: 10.37421/2161-0959.2022.12.386

How to cite this article: Fred, William. "Diabetic Kidney Disease in Youths." J Nephrol Ther 12 (2022): 386.