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Urology & Nephrology Open Access Journal

Letter to Editor

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Diabetic kidney disease: diabetic and non-diabetic nephropathy, updates 2019

Volume 7 Issue 5 - 2019

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Received: July 07, 2019 | **Published:** October 15, 2019

Letter to editor

Diabetic nephropathy, well known to the clinicians, is systematically sought after some duration of diabetic disease; nevertheless, kidneys in diabetic patients may also be affected by other conditions than diabetes. Non-diabetic nephropathies are represented by heterogeneous set of conditions, which mechanisms of kidney structure as well as function alterations are much complex. The etiological diagnosis of renal damage is essentially based on kidney biopsy, which remains an invasive procedure. Sometimes the difference between these two entities can be observed only by a careful monitoring of the progression, which can be estimated by new biological and morphological tools, available due to current scientific progress.

Modern diabetology has undergone a continual change, ranging from the biological definition of different clinical diagnoses to pathology classifications, going through panoply of radiological examinations. It is in this sense that Tervaert et al in 2010 introduced a new histopathological classification, based on the different components of kidney tissue lesions (not only glomerular, but also interstitial, tubular and vascular).[1,2] All that has been accompanied by a change in the concept and denomination of the pathology from diabetic nephropathy to diabetic kidney disease.[3]

The mechanism, explaining the aggravation of diabetic kidney disease in cases of glycemic imbalance and/or in association with high blood pressure (hyperactivity of the cotransporter SGLT2, as well as hyperfiltration which exhausts the nephron in the long course) is well described.[4] This is not yet the case for the heterogeneous group of non-diabetic nephropathies for lack of histological evidence, based on invasive diagnostic tools, such as biopsy, which can be supplanted by the new biological and radiological tools. To our knowledge, no studies had been performed yet to investigate the impact of non-diabetic nephropathy on the prognosis of coexisting diabetic nephropathy. Contrary to what some investigators suggest, the effect is not necessarily an aggravation, which can be simulated by the influence of refractory vices on diabetic retinopathy (DR) - myopia protects against DR and hypermetropia aggravates it.[5] However, unlike DR, the impact of refractory defects is explained by a pressure game on the retina.

This effect not yet proven for the group of non-diabetic nephropathies, and in diabetic nephropathy can probably be explained by the involvement of cascades of different inflammatory voices, the activation of which may inhibit others. Kidney biopsy seems to be a reliable way to distinguish diabetic nephropathy from non-diabetic, but it lacks sensitivity because the sample size, obtained by the biopsy, remains small (false positivity, if the sample taken demonstrates a superimposed glomerulonephritis, or false negativity if sample taken from relatively preserved part of the kidney).[6] Some clinical contexts require performing of renal biopsy to establish the pathophysiological diagnosis such as: nephrotic syndrome without retinopathy, kidney function alteration without retinopathy, nephrotic syndrome with diabetes duration less than 5 years, nephrotic syndrome discordant with normal creatinine clearance (because it must be the first altered), unexplained hematuria, and rapid deterioration of kidney function. The result of this histopathological analysis should be interpreted according to the age of the patient and presence of vascular complications.[7]

Given the invasive nature of this exploration, a careful analysis of the patient's context should be made taking into account the classic predictors of progression of diabetic kidney disease such as hemoglobin A1C level, systolic blood pressure, albuminuria, diabetes duration, patient's age and sex.[8] To avoid the aggressive character of this exploration and to allow the reproducibility of the examination, last years some innovations in exploration had been proposed.

I. Biological markers: These are glycoproteins therefore variable already according to the glycemic balance. Most of them are measurable in the urine so their thresholds are dependent on glomerular filtration, for example ***N gal KIM1 L-FABP***.[9] A new marker, recently discovered, is ***BMP-7***, measurable in anti-fibrotic blood by inhibiting ***TGF-B1 pro fibrotic factor.*** Some studies showed that treatment of patients with BMP-7 attenuates progression of stage 4 diabetic kidney disease, characterized by high TGF-B1 to low BMP7 in stage 5/end stage renal failure.

II. Morphological markers: several examples such as:

Kidney elastography: correlation between renal cortical thickness and degree of albuminuria;[10] **a**nd also a correlation between BMP-7 and the kidney disease progression, proved by kidney elastography.[11] ***Kidney MRI:*** New sequences had been described for kidney imaging.[12] In our opinion this sequence makes possible to determine which patients among those with diabetic nephropathy stage 4 can benefit from treatment by BMP7, which makes possible to prolong the time to kidney replacement therapy, as well as to improve the overall survival of these patients.

The prolongation of the survival in diabetic patients could be obtained by the nephroprotection after the advent of the different types of insulin, allowing better glycemic control, but also by the therapeutic interventions on the final stages of kidney disease, such as dialysis and kidney transplantation. However, interventions at the earlier stages will be safer and less invasive, in addition to glycemic control, which remains an effective barrier to the progression of diabetic kidney

disease. Innovations in the field of biological and morphological exploration will allow target abnormalities by understanding of the pathophysiological cascades at best, which in turn will lead to the improvement of the treatment.

Acknowledgments

None.

Funding details

None.

Conflicts of interest

The author declares there is no conflict of interest.

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