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

Abstract
If diabetic nephropathy is well known by clinicians and is systematically sought after some duration of diabetic disease nevertheless, kidneys in diabetic patients diabetics kidney may also be affected by other conditions than diabetes. Non diabetic nephropathies is a heterogeneous set of pathologies which mechanism 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 that can be estimated by new biological and morphological tools thanks to current scientific progress.

Keywords: kidneys, kidney biopsy, etiological diagnosis, renal damage, glomerular lesions, glycemic imbalance, interstitial, vascular, tubular lesions, diabetic retinopathy

Introduction
Modern diabetology has undergone a continual change ranging from the biological definition of different clinical diagnoses to anatomo-pathological classifications, going through panoply of radiological examinations. It is in this sense that introduced a new histopathological classification based on the different components of nephrons (not only glomerular lesions but also interstitial, vascular and tubular lesions). All this has been accompanied by a change in the concept and denomination of the pathology from diabetic nephropathy to diabetic kidney disease. If the mechanism explaining the aggravation of Diabetic kidney disease in cases of glycemic imbalance or association with high blood pressure (hyperactivity of the cotransporter SGLT2 as well as hyperfiltration which exhausts the nephron in the long course). 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 my knowledge no study has been done until to now on the impact of non-diabetic nephropathy on progression of coexisting diabetic nephropathy) because contrary to what some people think the effect is not necessarily an aggravation, which can be simulated to the influence of refractory vices on Diabetic retinopathy (myopia protects against Diabetic retinopathy (DR) and hyperopia aggravates it).

However, unlike Diabetic Retinopathy, the impact of refractory defects is explained by a pressure game on the retina. This effect not yet proven of the group of non-diabetic nephropathies on 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 area involved in the biopsy remains small (source of false positive such as a sampling area made on a superimposed glomerulonephritis or false negative if it is a sampling concerning healthy area). Some clinical contexts require the realization of a renal biopsy to establish the etiological diagnosis such as: Nephrotic Proteinuria without retinopathy, kidney function involvement without retinopathy, Nephrotic Proteinuria and diabetes duration less than years, nephrotic Proteinuria discordant with normal clearance (because it must be the first altered), Unexplained hematuria and rapid degradation of kidney function. The result of this histopathological analysis should be interpreted according to the age of the patient and should be avoided if atrophy of vascular origin.

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, systolic blood pressure, albuminuria, diabetes duration, patient’s age and sex. Precisely to avoid this aggressive character of this exploration and to allow the reproducibility of this examination, recent years have seen innovations in exploration, whether biological or morphological.

I. Biological markers
These are glycoproteins therefore variable already according to the glycemic balance. Most of them are dosable in the urine so their thresholds are dependent on glomerular filtration example: Ngal KIM1 L-FABP. A new marker has recently been discovered to be BMP-7 measurable in anti-fibrotic blood by inhibiting TGF-B1 pro fibrosating factor. Studies have shown that treatment of patients with BMP-7 attenuates progression of stage 4 diabetic kidney disease characterized by high TGF-B1 to low BMP7 stage 5 end stage renal failure.

II. Morphological markers:
Several exams such as:
Kidney elastography: correlation between renal cortical thickness and degree of albuminuria And also correlation existing between BMP-7 as well as kidney disease progression proved by kidney elastography.
Renal MRI: New sequences have been described for kidney imaging. In my opinion this sequence makes it possible to determine among diabetic patients stage 4 of diabetic nephropathy who can benefit from treatment by BMP7 which makes it possible to lengthen...
the time of kidney transplant as well as the overall survival of these patients and even classify patients on transplant schedules.

Conclusion

The prolongation of the survival in diabetic patients could be obtained thanks to nephroprotection after the advent of the different types of insulin allowing a better glycemic control but also thanks to the therapeutic interventions of the final stages of kidney dysfunction such as dialysis and graft kidney. However, acting at an earlier stage will be safer and less invasive, in addition to glycemic control, which remains an effective barrier to the progression of diabetic kidney disease, innovation in the field of biological and morphological exploration will allow target abnormalities by knowing the physiopathological cascades at best, which will lead to etiological treatment.

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Conflicts of interest

The author declares there is no conflict of interest.

References


