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Mini Review

Non-Diabetic Nephropathy, When Should We Think About It?

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Abstract

Diabetic nephropathy is the result of pathophysiological mechanisms whose best knowledge has offered new therapeutic possibilities and a better life expectancy for both type 1 and type 2 diabetes. Nevertheless, the diabetic kidney may be the seat of other lesions. This is due to diabetics whose positive diagnosis is based on renal biopsy, which is certainly an invasive means but will allow etiological therapeutic management other than glycemic control. diabetic nephropathy is a chronic pathology which therefore requires non-invasive monitoring means that will allow us not only to determine the rate of progression of diabetic kidney disease but also to indicate screening for previously undiagnosed non-diabetic renal lesion. for this reason, biological and morphological means are necessary whose cost-benefit ratio is necessary before their standardization by targeting a certain diabetic population thus allowing to act at an earlier stage before the installation of irreversible lesions.

Introduction

In recent years we are witnessing a change in concept, diabetic nephropathy is now called diabetic kidney disease [1]. This is because clinical data have shown that the evolution from micro albuminuria to proteinuria that characterizes diabetic nephropathy is not always concomitant with degradation of renal function, all studies in this subject are observational and lacking Biopsy evidence .It is in this perspective that Tervaert et al in 2010 introduced a new histopathological classification based not only on glomerular lesions but also tubulo interstitial and vascular lesions. Some hypotheses assume that glomerular hyperfiltration is preceded by sodium and glucidic hyperabsorption at the tubular proximal level due to sodium loading and hyperglycemia common in diabetics as well as an increase in SGLT2 activity in the proximal hypertrophied tube. On the molecular scale hyperfiltration secondary to hyper reabsorption will lead to the passage of various molecules such as Albumin, fatty acids, IGF-1 and TGF-B1 which by binding to luminal receptors will lead to a reaction cascade involving mediators Type interleukins IL-8 TGF-B1 resulting in macrophage activation as well as chemotaxis of other cells such fibroblast in the interstitial tissue, this inflammatory reaction will be maintained by other factors such as VEGF and AGE aggravated by hypoxia secondary to pre-renal factors such as anemia and vasoconstriction .Other studies have investigated the mechanism explaining the vulnerability of the diabetic kidney and suggest that hyperglycemia activates the apoptotic mitochondrial pathway, P53, and that insulin therapy attenuates both apoptosis and the severity of renal insufficiency acute.

Kidney biopsy indication:

a) Nephrotic Proteinuria without retinopathy

b) kidney function involvement without retinopathy

c) Nephrotic Proteinuria and diabetes duration<5ans NDRD

d) Nephrotic Proteinuria discordant with normal clearance

e) Acute kidney injury unexplained

f) Unexplained hematuria.

g) Rapid degradation of kidney function

Taking into account the age of the kidney, never indicated if ischemia or atrophy of vascular origin. Nevertheless, several factors are predictive of the nature of nephropathy and will allow a diagnostic orientation but only the renal biopsy will allow a definitive diagnosis. It is in this sense that a study published in Int Urol Nephrol 2016 showed that a 500mg /day proteinuria is suggestive of a non-diabetic nephropathy whereas rather it is the abundant hematuria which evokes a non-diabetic origin knowing Than the microscopic appearance of the red blood cells in the

urine that best directs to the non-diabetic origin of nephropathy if it is an irregularly shaped red blood cell [2]. There are several predictive factors for the progression of diabetic kidney disease common to both type 1 and 2 diabetes, including: hemoglobin A1C, systolic blood pressure, albuminuria, diabetes duration, age and sex of the patient, and is their absence in the event of impaired renal function which must evoke a non-diabetic nephropathy. Studies have focused on the retinal kidney bond in diabetics and the results have shown a significant association between non-diabetic nephropathy and the absence of diabetic retinopathy, while its presence does not in any way eliminate a non-diabetic origin of nephropathy [3]. HTA was significantly related to diabetic nephropathy suggesting a synergistic interaction of hypertension and diabetes through endoplasmic reticulum stress. Nodular glomerulosclerosis has been described as the histological lesion characteristic of diabetic nephropathy but recently some cases have been reported in the literature on results of renal biopsy suggesting nodular glomerulosclerosis in non-diabetic subjects but they had some criteria's of the Metabolic syndrome such hypertension and dyslipidemia in addition to a history of heavy smoking but who do not even have a glucose intolerance suggesting non-specific diabetes of this lesion in the renal biopsy [4]. Analysis of the etiologies of non-diabetic nephropathy in diabetic subjects revealed that the most common etiologies are represented by membrane nephropathy and immunoglobulin A (Ig A) nephropathy, but the analysis also made it possible to highlight certain lesions that do not associate with diabetic nephropathy which can be explained by three hypotheses

a) Or it is a genetically nephro-protected subject

b) this is non-diabetic nephropathy which protects the kidney from developing another

c) or it is a biopsy made too early in a subject well balanced in terms of glycemic

To choose one of his hypotheses of the genetic studies as well as a biopsic control after treatment of the causal affection and a long-term follow-up of this category of patients by an invasive means like the kidney biopsy proves too heavy hence the interest of discovering non-invasive means for monitoring the progression of diabetic nephropathy and allowing us to establish more links between diabetic and non-diabetic nephropathy [5]. Among these non-invasive objective markers for monitoring the progression of diabetic kidney disease are two categories:

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: N gal KIM1 L-FABP [6]. 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.

Morphological Markers

a. Renal Elastography: correlation between renal cortical thickness and degree of albuminuria [7] Correlation between renal elastography and BMP-7 as well as progression of renal disease [7].

b. Renal MRI: New sequences have been described for kidney imaging [8]. This sequence makes it possible to determine among the patient's stage 4 who can benefit from TRT by BMP7 which makes it possible to lengthen the time of renal transplant as well as the overall survival of these patients.

Conclusion

The kidney of the diabetic can be affected by a group of heterogeneous groups of pathologies among which diabetes-related kidney disease is the most common form and the one that determines the renal prognosis the most. As we are currently witnessing tremendous progress in PEC as well as exploration of this pathology which constitutes a real public health problem. New therapeutic targets appeared in the image of BMP7 being tested in animals, for which markers of biological and morphological progression are being validated and will allow new therapeutic indications by all practitioners (Diabetologists, Nephrologists, kidney transplant surgeons ...)

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