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Original Article

Causes of Nondiabetic Nephropathy in Patients with Type 2 Diabetes Mellitus

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ABSTRACT

Objective: The aim of this study is to evaluate the contribution of kidney biopsy performed with an appropriate indication to diagnosis and treatment in diabetic patients with nephropathy with a single-center experience.

Methods: In our study, 32 patients with type 2 diabetes who underwent kidney biopsy in our hospital between 2012 and 2019 were included. Kidney biopsy indications were determined as patients with diabetes without diabetic retinopathy and with proteinuria above 1 g/day.

Results: Diabetic nephropathy (DN) and nondiabetic nephropathy (NDN)were diagnosed with renal biopsy. In 14 of 32 patients, NDN was reported in histopathological evaluation. Membranous nephropathy was detected in 4 of these patients, focal segmental glomerulosclerosis (FSGS) in other 4 patients, light chain disease in 2 patients, IgA nephropathy in 2 patients, minimal change nephropathy in 1 patient, and finally AA amyloid in 1 patient. Nondiabetic renal disease superimposed on DN (DN + interstitial nephritis and DN + FSGS) was observed in two patients. In 16 diabetic patients, DN was detected by renal biopsy.

Conclusion: We believe that for diabetic patients, it may be important to distinguish nondiabetic kidney disease from diabetic kidney nephropathy, to choose proper treatment methods and determine kidney prognosis.

INTRODUCTION

Diabetes is a global epidemic disease and the number of patients worldwide is growing rapidly. Currently, there are about 500 million people affected by diabetes mellitus worldwide. It is expected that by the year 2045, this number will have increased to about 693 million. Diabetic nephropathy (DN) is one of the major complications of diabetes.[1] In addition, DN is the most common cause of chronic kidney failure and end-stage kidney disease in the world. Despite improvements in the follow-up of diabetic patients, the frequency of DN has not decreased in the last 30 years.[2]

Early developing proteinuria (<5 years), rapid impairment of kidney function, impaired kidney function without distinct proteinuria, and detection of active urine sediment incompatible with the nature of DN should suggest the presence of nondiabetic nephropathy (NDN). In addition, age, absence of retinopathy, microhematuria and subnephrotic proteinuria, and short-term history of diabetes suggest the possibility of NDN in type II diabetics, but their effects have varied in various studies.[3,4] Although kidney biopsy is a gold standard for diagnosis, biopsy is not performed in most patients as the diagnosis is based on clinical, end organ damage (retinopathy, neuropathy, and proteinuria), and laboratory findings with an ongoing traditional approach. However, although this approach is adequate for type I diabetes, it is not clear for type II. Diabetic kidney disease is known to be clinically and pathologically heterogeneous in these patients. The nature and prevalence of NDN differ in studies. On the other hand, while changes in DN are irreversible, some nondiabetic nephropathies, such as interstitial nephritis, membranous nephropathy, or minimal change disease, can often be treated. Therefore, it is essential to detect NDN in diabetic patients.[5,6]

Classical diabetic glomerulopathy is characterized by glomerular basement membrane thickening, endothelial damage, mesangial enlargement and the presence of mesangial nodules,and loss of podocytes. However, besides classical glomerulopathy, glomerular lesions and tubulointerstitial disease can also be detected in diabetes. In type I diabetes with albuminuria for 5 years or more, the cause of diabetic kidney disease is most likely DN while variety is higher in type 2 diabetics due to the possibility of superimposed or de novo nondiabetic kidney disease. A definitive diagnosis can only be made by kidney biopsy. As a result of many studies, it has been determined that nondiabetic kidney disease is seen between 27% and 79% in diabetic patients.[7–10]

In general, in the presence of long-standing diabetes, especially if there is retinopathy, it is assumed that the cause of chronic kidney failure is DN.[11] While the absence of retinopathy in kidney biopsy studies supports possible NDN, the presence of severe proliferative diabetic retinopathy supports DN.[12–15] However, there were still many conditions that DN was not associated with diabetic retinopathy (DR), and the incidence of fundus lesions was inconsistent in different studies.[16,17] Although the purpose of kidney biopsy is diagnostic, prognostic information could be obtained through the evaluation of the class of glomerular disease and the degree of interstitial fibrosis. In a large study, results showed that most of the patients had significant renal dysfunction, with median creatinine of 2.5 mg/dL (interquartile range [IQR], 1.6–4.4) and estimated glomerular filtration rate (eGFR) of 29.1 mL/min per 1.73 m^2 (IQR, 14.5–54.5) at the time of biopsy; just over half of the patients had eGFR <30 mL/min per 1.73 m^2. Moreover, the median proteinuria for the entire cohort was in the nephrotic range. NDN was identified in >60% of biopsies: 220 patients with NDN alone and 164 patients with NDN and superimposed renal disease.[18]

The aim of this study was to determine the prevalence and independent determinants of NDN in follow-up type II diabetic patients in our center and determine the effect on prognosis in patients with type II diabetes.

MATERIALS AND METHODS

The study was a retrospective case-controlled study of type II diabetic patients treated at our Hospital. The study protocol was approved by the ethical committee our hospital (approval no: 2020.514.172.1 approval date: February 26, 2020).

Thirty-two type 2 diabetic patients who underwent kidney biopsy in our hospital between 2012 and 2019 were included in the study Type 2 diabetes patients with proteinuria over 1 g per day and without diabetic retinopathy were included in the study. Exclusion criteria were as follow: insufficient medical data, unqualified biopsy material, presence of diabetic retinopathy without any signs of superimposed glomerular disease (rapid increase in creatinine level, anti-neutrophil cytoplasmic antibody seropositivity, and persistent hematuria), stage 4 or 5 kidney failure, and patients with a kidney transplant.

Demographic data of patients, clinical information (duration of diabetes, accompanying diseases such as hypertension and coronary artery disease,and type of antidiabetic medication), and laboratory test results (level of proteinuria in 24 h urine, presence of hematuria in urine, blood urea, creatinine, albumin, and hemoglobin A1c levels) were gathered. Diabetic retinopathy was assessed by a specialist ophthalmologist.

All renal biopsy samples were evaluated by a nephropathologist with standard light microscopy and immunofluorescence. Electron microscopy is not routine in our center, thus it is not used for diagnosis. The pathological diagnostic criteria of DN were thickening of the glomerular basement membrane (>395 nm in women and >430 nm in men) and mesangial enlargement with or without nodular glomerulosclerosis.

Statistical analysis

Groups'gender, application complaint, age of diabetes, insulin requirement, additional diseases, amount of proteinuria, presence of hematuria, urea creatinine, albumin, hemoglobin A1c values were presented as numerical data mean±standard deviation, median (minimum–maximum), and categorical data number (frequency percentage). Patients were divided into two groups as NDN (group 1) and DN (group 2) according to kidney biopsy results. The distribution of each group was checked with the Kolmogorov–Smirnov test and histogram. Normally distributed numerical data were compared with Student's t-test and non-normally distributed data were compared with the Mann-Whitney U test. Categorical data were compared with Pearson's Chi-squared and Fisher's Exact test. $P<0.05$ was considered statistically significant. All statistical analyses were performed by The Jamovi Project (2020) made with jamovi (Version 1.2).[19]

RESULTS

In our study, the number of men and women was equal. The mean age of the patients was 52 years (range, 41–67 years), the duration of diabetes was 10 years (range, 3–16 years), and the mean creatinine values were 1.6 mg/dL (range, 0.64–3.4 mg/dL). In the groups, 81% (n=26) of our patients were using insulin and 34.4% (n=11) had accompanying coronary artery disease.

In 14 of 32 patients, NDN was reported in histopathological evaluation. Membranous nephropathy was detected in 4 of these patients, focal segmental glomerulosclerosis (FSGS) in other 4 patients, IgA nephropathy in 2 patients, light chain disease in 2 patients, minimal change nephropathy in 1 patient, and finally AA amyloid in 1 patient. Nondiabetic renal disease (NDRD) superimposed on DN (DN + interstitial nephritis and DN + FSGS) was observed in 2 patients. These 2 patients were included in the NDN group during statistical analysis. In our study, 16 of 32 patients were shown to have NDN (NDN group). The rest of the 16 patients were named as DN group.

Demographic and clinical and laboratory features according to the groups are given in Table 1. Among the parameters evaluated, HbA1c levels ($p<0.015$) and the duration of diabetes ($p<0.01$) were higher in the DN group. Also,the mean serum albumin level was lower in the DN group (0.027).There was no statistically significant difference between the groups according to the other data ($p>0.05$).

Both groups were subgrouped as nephrotic and nonnephrotic proteinuria and evaluated separately (Table 2). The longest duration of diabetes and the highest creatinine values were found in the nephrotic DN group. Contrary to this, the mean HbA1c level was higher in nonnephrotic

Table 1. Clinical and laboratory characteristics of nephrotic and nonnephrotic groups

	Nondiabetic nephropathy		Diabetic nephropathy		p
	Nephrotic (n=9)	Nonnephrotic (n=7)	Nephrotic (n=11)	Nonnephrotic (n=5)	
Age	52.33 (49–67)	53.57 (41–62)	50.63 (42–63)	48.2 (44–53)	0.589
Duration of diabetes (years)	7.2 (5–15)	7.71 (4–15)	13.18 (8–16)	12.4 (10–15)	0.003
HbA1c (%)	7.07 (5.9–9.5)	7.9 (7.9–10)	7.8 (5.7–8.8)	8.6 (6.6–11)	0.133
Creatinine (mg/dL)	1.32 (0.6–2.6)	1.72 (0.8–1.2)	1.9 (0.9–3.1)	1.78 (0.7–2.4)	0.246
Albumin (g/L)	3.81 (3.1–4.4)	3.91 (3.5–4.6)	3.51 (3.2–3.8)	3.76 (3.4–4.1)	0.089

Student'st-test and the Mann–Whitney U test were used to compare the groups.

Table 2. Clinical and laboratory characteristics of diabetic and nondiabetic nephropathy groups

	Total	Diabetic	Nondiabetic	p
Age	51.375±6.676	50.294±5.966	52.60±7.613	0.345
Gender (male), n (%)	16 (50)	10 (62.5)	6 (37.5)	0.157
Duration of diabetes (years)	10.19±4.295	13.5 (8–16)	6 (3–15)	<0.001
Insulin use, n (%)	17 (53.1)	10 (62.5)	7 (43.8)	0.288
Oral antidiabetic drug use, n (%)	15 (46.9)	7 (41.2)	8 (53.3)	0.78
HbA1c (%)	7.775±1.284	8.282±1.195	7.2±1.172	0.015
Coronary artery disease, n (%)	11 (34.4)	7 (43.8)	4 (25)	0.264
Thyroid dysfunction, n (%)	11 (34.4)	7 (43.8)	4 (25)	0.264
Dialysis need, n (%)	7 (21.9)	5 (31.3)	2 (12.5)	0.394
Proteinuria (g/24 h)	4.334±1.883	4.500±2.193	4.186±1.615	0.645
Hematuria, n (%)	26 (81.3)	12 (75)	14 (87.5)	0.654
Creatinine (mg/dL)	1.682±0.734	1.800±0.730	1.549±0.739	0.343
Total protein (g/L)	6.834±0.335	6.859±0.264	6.807±0.409	0.668
Albumin (g/L)	3.725±0.377	3.588±0.254	3.880±0.439	0.027

Kruskal–Wallis test was used to calculate p values.

DN group. The lowest duration of diabetes, the lowest mean HbA1c values, and the lowest mean creatinine values were found to be in the nephrotic NDN group.

While patients in the isolated DN group received conservative treatment, patients in the NDN group were treated according to the underlying disease. During the follow-up period, 5 patients in the DN group and 2 patients in the NDN group needed dialysis. Two patients in the NDN group and 1 patient in the DN group passed away. Follow-up of other patients continues in the nephrology outpatient clinic.

DISCUSSION

Information on NDN development mechanisms is inadequate and speculative. Recent information suggests that hyperglycemia, glycolysis end products, immune complexes, and biochemical changes in diabetes activate kidney cells by causing increased cell adhesion molecules and proinflammatory cytokines through protein kinase.[20] Some proteins that have been altered in diabetes have the potential to trigger inflammation such as oxidized LDL. Immune complexes and glomerular IgG deposits (especially proinflammatory IgG1 and IgG3) were detected in experimental models of diabetes. Enhanced exposure of antigenic cellular components that triggers immune responses and glomerular changes may cause an immune reaction in the subepithelial are.[21] However, some authors found no difference in the frequency of NDN in patients with and without diabetes and argued that glomerulonephritis detected in the diabetic kidney is only a coincidence.[9]

Aclinical diagnosis of DNwas performed when diabetic patients have retinopathy and proteinuria. Therefore, diabetic patients did not receive renal biopsy until they were suspected to have NDN. Unfortunately, there is no available guideline on which diabetic patient should receive kidney biopsy. Although DN is generally considered to exist during the development of microalbuminuria in patients with type I diabetes, the probability of having NDN or mixed glomerulopathy should be considered in patients with type II diabetes. Many studies have found a strong relationship between diabetic retinopathy and nephropathy.[22] The presence of DN in 44%–70% of diabetic patients without retinopathy indicates that the likelihood of DN should not be ignored in the absence of retinopathy, but the absence of retinopathy may be a strong indicator of

NDN.[23] In our study, patients without diabetic retinopathy were selected, and it was noteworthy that the kidney biopsy results in the group with a long duration of diabetes were related to DN.

In most regression studies, NDN was found to be associated with the absence of retinopathy and the short duration of diabetes. Therefore, it will be appropriate to perform kidney biopsy in this group of patients in order not to skip an underlying nondiabetic glomerular disease. In our study, the detection of 43.75% NDN and 6.25% mixed nephropathy in the biopsy results of type II diabetic patients without diabetic retinopathy supports the importance of biopsy in this group of patients.

The specificity of microscopic hematuria and active urinary sediment for the diagnosis of NDN in the diabetic patient group is 93.1%–100%, and the positive predictive value is 81%–100%. Some studies have suggested that in typical diabetic glomerulopathy, hematuria can be detected at a rate of 35%–78%, so it is not useful for the diagnosis of NDN.[5] Dysmorphic RBCs in the urine sediment may be more useful than microhematuria for indicating NDRD.[24] In our study, hematuria was found in 12 (75%) of 16 patients in the group with DN and in 14 (87.5%) patients in the NDN group. In addition, the duration and severity of hyperglycemia, hyperlipidemia, hypertension, and proteinuria are also known risk factors for DN. In a single-center study,a diagnostic model valuable to physicianswas developed based on logistic regression featuring six variables (i.e., anemia, eGFR levels, DR, proteinuria, hypertension, and DM) which can effectively discriminate between DN and NDRD with 93.2% sensitivity and 82.6% specificity.[5]

After all, with the latest evidence, the traditional clinical course of diabetes is changing. Studies show that the development of proteinuria and the reduction in eGFR may have independent pathogenesis rather than a consequence. This phenomenon may be caused by the widespread use of drugs that block the renin-angiotensin systems and develop glycemic control.[25] As the traditional clinical course of diabetes continues to change, the prevalence of isolated DN patients with severe proteinuria will decrease, while those with NDN will increase proportionally. As a result, renal biopsy will be considered more intensively in this group. The prognosis of diabetic patients with NDN is significantly better than that of patients with diabetes-proven DN. In patients with isolated DN, the risk of progression to end-stage renal disease is between 30% and 60% within 3 years of pathological diagnosis. The risk is less than 10% in NDN cases, whereas it is similar to DN in mixed cases.[7] Since there are no globally accepted diagnostic guidelines, the most accurate approach in this patient group would be to perform intermittent reevaluation and renal biopsy when necessary.[23,26]

Patients with advanced-stage renal failure without diabetic retinopathy were not included in this study. This group of patients is perhaps the most unlucky group who lost the chance of treatment due to the possibility that the diagnosis of NDN was missed. Therefore, a biopsy could be reevaluated either periodically or when clinical condition changes (e.g., increasing urinary RBC count). Another limitation of our study is the low number of patients. Studies with a broader and larger number of patients may change the traditional approach to diabetic kidney patients in the future.

CONCLUSION

Patients with diabetes subjected to renal biopsy may have DN, DN with superimposed NDN, or NDN alone. There is no available guideline on which patient should receive a kidney biopsy. In our study, it has been shown that NDN (alone or superimposed with DN) is detected in 50% of 32 diabetic patients without diabetic retinopathy. Classical ACE or ARB inhibitor therapy may not be sufficient for NDN patients diagnosing NDN is especially important as it may lead to a specific change in therapy. The nephrologist should consider if NDN is potentially present in diabetic patients and the risk/benefit ratio of biopsy.

Ethics Committee Approval

This study approved by the Kartal Dr. Lutfi Kirdar Training and Research Hospital Clinical Research Ethics Committee (Date: 26.02.2020, Decision No: 2020/514/172/1).

Informed Consent

Retrospective study.

Peer-review

Internally peer-reviewed.

Authorship Contributions

Concept: M.M.; Design: E.P.; Supervision: M.M.; Fundings: S.Y.; Materials: S.Y.; Data: S.Y.; Analysis: M.M.; Literature search: E.P.; Writing: M.M.; Critical revision: S.Y.

Conflict of Interest

None declared.

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Tip 2 Diyabet Mellituslu Hastalarda Non Diyabetik Nefropati Nedenleri

Amaç: Bu çalışmanın amacı nefropatili diyabetik hastalarda uygun endikasyonla yapılan böbrek biyopsisinin tanı ve tedaviye katkısını tek merkez deneyimi ile değerlendirmektir.

Gereç ve Yöntem: Çalışmamıza 2012–2019 yılları arasında hastanemizde böbrek biyopsisi yapılan 32 tip 2 diyabet hastası dahil edildi. Böbrek biyopsisi endikasyonları diyabetik retinopatisi olmayan diyabetli ve proteinürisi 1 g/gün'ün üzerinde olan hastalar olarak belirlendi.

Bulgular: Diyabetik ve diyabetik olmayan nefropati tanısı böbrek biyopsisi ile konuldu. Histopatolojik değerlendirmede 32 hastanın 14'ünde NDN rapor edildi. Bu hastaların dördünde membranöz nefropati, diğer dört hastada fokal segmental glomerüloskleroz (FSGS), iki hastada hafif zincir hastalığı, iki hastada IgA nefropatisi, diğer hastada minimal değişiklik nefropatisi ve son olarak bir hastada AA amiloid saptandı. DN üzerine bindirilmiş görülen NDRD (DN + interstisyel nefrit ve DN + FSGS) iki hastada gözlendi. On altı diyabetik hastada böbrek biyopsisi ile diyabetik nefropati tespit edildi.

Sonuç: Diyabetik hastalarda diyabetik olmayan böbrek hastalığını diyabetik böbrek nefropatisinden ayırt etmenin, uygun tedavi yöntemlerini seçmenin ve böbrek prognozunu belirlemenin önemli olabileceğine inanıyoruz.

Anahtar Sözcükler: Diyabet mellitus; nefropati; proteinüri.