



RENAL FUNCTION IS ASSOCIATED WITH CATARACT DEVELOPMENT IN PATIENTS WITH TYPE 2 DIABETES

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SUMMARY – In this study, we investigated the impact of renal function, metabolic risk factors, and duration of diabetes on cataract development in patients with type 2 diabetes (T2DM). This study was cross-sectional and included 107 T2DM (67 male/40 female). Renal function was estimated with a creatinine-based formula (eGFR) and with albumin/creatinine (A/C) ratio. Patients were divided into three groups according to the LOCSIII classification: group 1 represents patients with clear crystalline lens (n=16), group 2 represent patients with initial cataract (n=74), and group 3 represents patients with immature cataract (n=17). Compared to group 1, group 3 had significantly longer diabetes duration (17.12±6.38 vs. 10.81±4.09 years; p=0.004) and marginally higher HbA_{1c} (7.11±1.41 vs. 6.38±0.83%; p=0.057). Diastolic blood pressure (DBP) was also significantly higher (90.94±15.41 vs. 76.47±6.32 mmHg; p=0.002) while eGFR was significantly lower (53 ± 18 vs. 72 ± 12 ml/min⁻¹1.73m⁻²; p=0.014). In logistic regression analysis, DBP (AOR=1.06, 95%CI 1.00-1.12, p=0.039) and eGFR (AOR=3.02, 95%CI 1.07-8.49, p=0.034) had a significant influence on cataract development even after adjustment for well-known risk factors HbA_{1c} and duration of diabetes. The results of the study suggest a connection between renal function and cataract development in T2DM.

Key words: *Cataract; Type 2 diabetes; Blood pressure; Renal function*

Introduction

Diabetes prevalence is rapidly increasing worldwide, affecting almost 500 million people in 2019¹. Besides the risk of mortality (the seventh leading cause of death worldwide), chronic complications of diabetes lead to invalidity and reduction in quality of life and are also a burden on the health system². Diabetic reti-

nopathy (DR) is the most important preventable cause of vision loss in diabetes and nowadays the leading cause of blindness in the working-age populations³. Risk factors for DR are long duration of diabetes, inadequate glycemic control, hypertension, and diabetic nephropathy classified as reduced glomerular filtration rate (GFR) and/or increased albumin excretion rate (AER). A possible explanation for this retinal-renal association is that similar structural changes and histological findings are seen in both chronic diabetic complications⁴.

A cataract is also a frequent cause of blindness worldwide that can be prevented⁵. The characteristic of

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patients with diabetes is that cataract occurs earlier in life and also progresses faster than senile cataract, which causes earlier cataract surgery and risk of poorer vision outcome, especially if it is complicated with active proliferative DR and/or preexisting diabetic macular edema^{6,7}. The association between diabetes and cataract formation has been shown in basic researches as well as in clinical and epidemiological studies^{8,9}. Due to the increasing prevalence of diabetes worldwide, the incidence of diabetic cataracts progressively rises and the clarification of pathogenesis and mechanisms for preventing or delaying the development of cataracts in diabetic patients remains a challenge. Specific pathogenic mechanisms linking diabetes and lens opacification is not fully understood and probably involves complications of diabetes and metabolic risk factors. A recently published study suggested for the first time that cataract become more common as renal function worsens¹⁰. Besides, chronic kidney disease and major eye diseases, including cataracts, share common metabolic and vascular risk factors¹¹. In this study, we investigated the impact of renal function, metabolic risk factors, and duration of diabetes on cataract development in patients with type 2 diabetes (T2DM).

Patients and Methods

This cross-sectional study included 107 T2DM. After an overnight fast, venous blood samples were collected in order to determine metabolic risk factors: glycated hemoglobin (HbA_{1c}), and serum lipids (total, HDL, LDL cholesterol, and triglycerides). Triglycerides and cholesterol in serum were measured by an enzymatic colorimetric method, while HbA_{1c} was measured spectrophotometrically by turbidimetric immuno-inhibition (Olympus AU600, Beckman-Coulter, USA).

Blood pressure was measured minimally two times after a resting period of 10 minutes with a mercury sphygmomanometer. Renal function was determined using serum creatinine with a creatinine-based formula (Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula) and with albumin/creatinine (A/C) ratio^{12,13}. A random urine sample was collected for assessment of the A/C ratio. Urine albumin and creatinine levels were determined by turbidimetric immunoassay and photometric assays. A/C ratio less than 3.0 mg/mmol was considered normal, micro-albuminuria was defined as any value between

3.0 to 30.0 mg/mmol, and albuminuria more than 30.0 mg/mmol was considered as macro-albuminuria. Patients had normal urine sediment without elements of urinary tract infections.

DR was determined according to the EURODIAB retinal photography methodology with a trained ophthalmologist. Eye examination of both eyes included best corrected visual acuity (BCVA), biomicroscopy of the lens, funduscopy, and color fundus photography¹⁴. According to the Lens Opacity Classification System version III (LOCSIII), patients were divided into three groups¹⁵.

Local ethics committees have approved the study protocol, and the study has been conducted according to the Declaration of Helsinki and local institutional guidelines.

Statistical analysis was performed by Statistica software package version 13.3¹⁶. The Kolmogorov-Smirnov test was used to test the normality of data distribution and the Leven test to test homogeneity of variance. Results of descriptive analyses were expressed as means \pm SD for continuous variables and as percentages for categorical variables. One-way ANOVA was used to test differences between continuous data, while the Bonferroni post hoc test was used where needed. The Chi-square test was used to evaluate differences among categorical data. We used binary logistic regression analysis to investigate the associations between cataract and the other evaluated parameters. In all analyses, the p value of less than 0.05 was set as statistically significant.

Results

The study included 107 T2DM (67M/40F, aged 66 years with a mean duration of diabetes of 15 years). In accordance with LOCSIII grading scales, we divided patients into three groups: group 1 represent patients with clear crystalline lens (n=16), group 2 represent patients with initial cataract (n=74), and group 3 represent patients with immature cataract (n=17) (Table 1). Comparing those three groups, patients have similar ages and a similar treatment for diabetes. Patients also had similar metabolic risk factors like systolic blood pressure (SBP) and all serum lipids. In the post hoc analyses, diabetes duration was significantly shorter in group 1 compared to groups 2 and 3 (p=0.007; p=0.004), while HbA_{1c} was marginally lower in groups 1 and 2 compared to group 3 (p=0.053; p=0.057). Post

Table 1. Basic characteristics, metabolic risk factors, blood pressure, diabetic retinopathy, and renal function parameters in patients with type 2 diabetes (n=107) divided into Group 1-3 according to the cataract status.

	Group 1 (n=16)	Group 2 (n=74)	Group 3 (n=17)	P
Age (years)*	66.31 ± 8.31	68.47 ± 7.11	66.52 ± 7.98	0.583 ^a
Sex (f/m)**	31.3 / 68.75	39.2 / 60.81	35.3 / 64.7	1.000 ^b
Diabetes duration (years)*	10.81 ± 4.09	15.49 ± 5.43	17.12 ± 6.38	<0.001 ^a
Diabetes treatment (OHA/insulin)**	7 / 9	25 / 49	8 / 9	1.000 ^b
HbA _{1c} (%)*	6.38 ± 0.83	6.38 ± 1.06	7.11 ± 1.41	0.052 ^a
Total cholesterol (mmol/L)*	4.87 ± 1.28	4.88 ± 0.83	5.43 ± 1.14	0.117 ^a
HDL cholesterol (mmol/L)*	1.37 ± 0.19	1.36 ± 0.35	1.29 ± 0.35	0.691 ^a
LDL cholesterol (mmol/L)*	2.71 ± 1.09	2.62 ± 0.80	3.03 ± 0.98	0.245 ^a
Triglycerides (mmol/L)*	1.89 ± 1.02	2.23 ± 1.41	2.91 ± 0.23	0.540 ^a
Systolic blood pressure (mmHg)*	134.06 ± 21.15	143.65 ± 22.58	144.41 ± 20.38	0.297 ^a
Diastolic blood pressure (mmHg)*	76.47 ± 6.32	80.00 ± 11.96	90.94 ± 15.41	0.001 ^a
Serum creatinine (umol/L)*	95.25 ± 7.28	109.28 ± 19.53	122.41 ± 41.43	0.249 ^a
eGFR (ml/min ⁻¹ .73m ⁻²)*	72 ± 12	60 ± 11	53 ± 18	0.017 ^a
A/C ratio (mg/mmol)*	10.04 ± 17.01	14.45 ± 34.61	15.44 ± 16.39	0.850 ^a
BCVA (decimal)*	0.97 ± 0.10	0.91 ± 0.19	0.84 ± 0.34	0.207 ^a
Diabetic retinopathy (DR)**	81.2 / 6.3 / 12.5	62.2 / 17.6 / 20.2	35.3 / 29.4 / 35.3	0.023 ^b

* mean ± SD ** percentages ^a one-way ANOVA ^b Chi-square test

Abbreviations: OHA: oral hypoglycemic agent; HbA_{1c}: glycated hemoglobin value; eGFR estimated glomerular filtration rate; A/C ratio: albumin/ creatinine ratio; BCVA: best corrected visual acuity; DR: no retinopathy / mild-moderate nonproliferative DR / severe NPDR-proliferative DR.

hoc analyses also showed that diastolic blood pressure (DBP) was significantly higher (p=0.002), DR significantly more severe (p=0.047), and eGFR significantly lower (p=0.014) in group 3 compared to group 1. Significant positive associations were observed between cataract and duration of diabetes, A/C ratio, DR, and DBP (p=0.002-0.009 for all) (Table 2). Significant positive associations were also observed between DR and duration of diabetes, A/C ratio, triglycerides, and HbA_{1c} (p<0.04 for all) (data not shown).

Results of univariate and multiple logistic regression analyses revealed that major risk factors for cataract development in T2DM are hyperglycemia (OR=1.65, 95%CI 1.07-2.53, p=0.022), duration of diabetes (OR=1.23, 95%CI 1.07-1.41, p=0.003), DBP (OR=1.08, 95%CI 1.03-1.13, p=0.002) and eGFR (OR=3.31, 95%CI 1.34-8.22, p=0.008) (Table 3). After further adjustment for HbA_{1c} and duration of diabetes, DBP (AOR=1.06, 95%CI 1.00-1.12, p=0.039) and eGFR (AOR=3.02, 95%CI 1.07-8.49, p=0.034) still stayed significantly associated with cataract development.

Table 2. Correlation between cataract and diabetes duration, albumin/creatinine ratio, diastolic blood pressure, and retinopathy in type 2 diabetes (n=107).

	Cataract		
	Spearman R	t(N-2)	p
Diabetes duration (years)	0.299	3.219	0.002
Diastolic blood pressure (mmHg)	0.298	3.203	0.002
A/C ratio (mg/mmol)	0.196	2.049	0.042
Diabetic retinopathy	0.249	2.640	0.009

Abbreviation: A/C ratio: albumin/creatinine ratio

Discussion

The results of this study suggest that the duration of diabetes, hyperglycemia, blood pressure, decreased renal function, and the presence of DR are involved in cataract development in T2DM. In addition, in logistic regression analyses after adjustment for HbA_{1c} and duration of diabetes, effects of DBP and renal function

Table 3. Risk factors and predictors for the development of cataract by means of univariate and multiple logistic regression analyses.

Variable	OR (95% CI)	P	AOR (95% CI)*	P*
Diabetes duration	1.23 (1.07-1.41)	0.003	/	/
HbA _{1c}	1.65 (1.07-2.53)	0.022	/	/
Systolic blood pressure	1.02 (0.99-1.04)	0.122	1.02 (0.99-1.05)	0.204
Diastolic blood pressure	1.08 (1.03-1.13)	0.002	1.06 (1.00-1.12)	0.039
Serum creatinine	1.02 (0.99-1.04)	0.151	1.00 (0.98-1.04)	0.457
eGFR	3.31 (1.34-8.22)	0.008	3.02 (1.07-8.49)	0.034
A/C ratio	1.00 (0.98-1.03)	0.580	1.01 (0.98-1.03)	0.864

* OR after adjustment for diabetes duration and HbA_{1c}

Abbreviation: HbA_{1c}: glycated hemoglobin value; eGFR: estimated glomerular filtration rate; A/C ratio: albumin/creatinine ratio.

presented by eGFR on cataract development was still significant. In patients with diabetes relationship between ocular and renal disease are more frequent in T2DM than type 1 diabetes¹⁷. Visual impairment is often associated with the progression of renal disease because there are several pathogenic mechanisms underlying both chronic kidney and eye disease^{11,18}. In addition, the structure of the kidney and the eye are similar, as well as developmental processes with the same transcription factors and epithelial cells (retinal or glomerular) overlying a basement membrane of collagen IV and capillary¹⁹.

Cortical, nuclear, and posterior subcapsular cataracts are morphologically three main cataract types, whereas etiologically, the most common is the age-related cataract. The pathophysiology of cataract is not wholly understood and include age, genetic, nutritional, environmental, ultraviolet light exposure, and systemic factors²⁰. The results of previous studies investigating the association between chronic kidney disease and cataract formation or progression vary considerably. The first large population-based study published in 1998 and investigating the 5-year incidence of cataract and renal function concluded that renal function is not associated with cataract after adjusting for gender and age²¹. However, after the extension of the same study over a 15-year interval, investigators found a significant association between renal function (measured with cystatin C, serum urea, and creatinine) with an incidence of posterior subcapsular and cortical cataract²². It should be stressed that serum cystatin C is a more sensitive marker of glomerular filtration compared to serum creatinine in patients with mildly im-

paired renal function, and patients in our study had normal or mildly impaired renal function²³. Another population-based prospective study found that patients with eGFR below 60 ml/min⁻¹1.73m⁻² had a higher incidence of cataract surgery compared to patients with eGFR over 60 ml/min⁻¹1.73m⁻²²⁴. A case-control study based on the Health insurance database found an increased incidence of cataract in patients with nephropathy²⁵.

The possible mechanism for cataract formation in chronic renal disease is the deposition of urea and water in the lens leading to the development of an osmotic cataract²⁶. Besides, in patients with diabetes, oxidative stress is increased in chronic kidney disease inducing cataract formation²⁷. Other mechanisms underlying cataracts in chronic kidney disease include endothelial dysfunction, atherosclerosis, inflammation, vitamin D deficiency, renin-angiotensin system dysfunction, anemia associated with nephropathy, and hypocalcemia^{11,28}. Moreover, nearly two-thirds of patients with diabetes have evidence of cataracts, mainly mixed cataracts⁶. Recently published retrospective analysis of data from a public health system of patients who had accessed cataract surgery confirmed previously suggested assumption that cataracts are more common as renal failure worsens¹⁰. Patients with chronic kidney disease had an almost two-fold higher risk of cataract development after correcting for comorbidities, and risk is further increased in patients with advanced kidney failure. Chronic kidney disease increases the risk of bilateral cortical and posterior subcapsular cataracts⁵. Although cataract is increased in some other conditions closely connected with

chronic kidney disease like diabetes, hypertension, smoking, obesity, and dyslipidemia, multivariate analyses confirmed that renal dysfunction is a strong and independent risk factor for cataract development¹¹. Besides hyperglycemia, albuminuria is also a risk factor for cataract development even in those with a short duration of diabetes and in those with newly diagnosed diabetes⁶. In those with prolonged diabetes, hyperglycemia induces modification of the lens proteins with the ACE formation or modification of the ATPase pumps with osmotic stress contributing to cataract formation²⁹.

This study has some potential limitations. First, the small number of patients with clear crystalline lenses and those with immature cataracts makes it difficult to generalize our results. Second, the results would be primarily referred to as a white European population. Third, selection bias is likely because our study was conducted in a hospital. Fourth, several risk factors involved in the development of cataract, such as sunlight exposure and nutritional history, were not considered.

In conclusion, this study's results suggest that renal function parameters, primarily A/C ratio and eGFR, are involved in cataract development in T2DM with traditional risk factors like duration of diabetes, HbA1c, and DBP. In order to prevent chronic kidney disease, cataract development, and disability, and surgery related to cataract, strict control of risk factors is needed.

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Conflict of interest: None to declare.

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Sažetak

BUBREŽNA FUNKCIJA JE POVEZANA S RAZVOJEM KATARAKTE U BOLESNIKA SA ŠEĆERNOM BOLEŠĆU TIPA 2

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Cilj istraživanja bio je istražiti utjecaj trajanja šećerne bolesti (ŠB), metaboličkih rizičnih čimbenika i bubrežne funkcije na razvoj katarakte u bolesnika sa šećernom bolešću tipa 2 (ŠB2). Ovo presječno istraživanje je uključilo 107 bolesnika sa ŠB2 (67 muškaraca/40 žena). Bubrežna funkcija je procijenjena određivanjem glomerularne filtracije (GF) i omjerom albumin/kreatinin (A/K) u urinu. Bolesnici su prema LOCS III bili podijeljeni u tri skupine: sk. 1 - bolesnici s prozirnom očnom lećom (n=16), sk. 2 - bolesnici s početnom kataraktom (n=74) i sk. 3 - bolesnici s nezrelom kataraktom (n=17). Bolesnici u sk. 3 imali su značajno duže trajanje ŠB (17.12±6.38 vs. 10.81±4.09 godina; p=0.004) i granično viši HbA_{1c} (7.11±1.41 vs. 6.38±0.83%; p=0.057) nego oni u sk. 1. Dijastolički krvni tlak (DKT) bio je značajno viši (90.94±15.41 vs. 76.47±6.32 mmHg; p=0.002), a procijenjena GF značajno niža (53±18 vs. 72±12 mlmin⁻¹.73m⁻²; p=0.017) u sk. 3 nego u sk. 1. Logistička regresija je utvrdila da su DKT (AOR=1.06, 95%CI 1.00-1.12, p=0.039) i GF (AOR=3.02, 95%CI 1.07-8.49, p=0.034) povezani s razvojem katarakte u ŠB2 čak i nakon standardizacije rezultata za trajanje ŠB i HbA_{1c}. Rezultati ovog istraživanja su pokazali da bubrežna funkcija ima važnu ulogu u razvoju katarakte u ŠB2.

Ključne riječi: *Katarakta; Šećerna bolest tipa 2; Krvni tlak; Bubrežna funkcija*