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Association between uric acid/high-density lipoprotein (HDL) cholesterol ratio and the presence of diabetic retinopathy

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ABSTRACT

Aim: To evaluate the hypothesis that the uric acid–to–high-density lipoprotein cholesterol ratio (UHR) may serve as a potential biomarker for predicting diabetic retinopathy (DR) in patients with Type 2 diabetes mellitus (T2DM).

Methods: A total of 175 patient files were retrospectively analyzed between January 2023 and June 2024 at the Ophthalmology Clinic of Bolu Abant Izzet Baysal University. The study consisted of 100 diabetic patient files with diabetic retinopathy and 75 diabetic patient files without diabetic retinopathy. The parameters were recorded, and the calculated parameter was the Uric Acid/HDL Ratio (UHR).

Results: There were marked differences between the DR and control groups in a number of hematological and biochemical variables like Hemoglobin (HGB), High Density Lipoprotein Cholesterol (HDL-C), Lymphocyte Count (LYM), Red Blood Cell Distribution Width (RDW), Platelet Distribution Width (PDW), Alanine Transaminase (ALT), Serum Albumin, UHR. UHR correlated positively with RDW and C-reactive protein (CRP) and negatively with HGB, LYM and albumin. In ROC analysis, a UHR >0.1156 showed 86.1% sensitivity and 82.4% specificity in predicting DR. In the multivariate model, UHR was independently associated with diabetic retinopathy, retaining statistical significance ($p < 0.001$).

Conclusion: The association between UHR and chronic inflammatory parameters emphasizes the importance of inflammation in the pathogenesis of DR and supports its use as a suitable biomarker to determine the likelihood and severity of DR in T2DM patients. Longer-term research is needed to confirm the usefulness of UHR in clinical practice, which has the potential to supplement standard glycemic measurements.

Keywords: Type 2 diabetes mellitus, diabetic retinopathy, uric acid/HDL ratio, metabolic disorder, inflammation.

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1. Introduction

Type 2 diabetes mellitus (T2DM) is a complex and increasingly prevalent global health condition characterized by chronic hyperglycemia, which may result in substantial long-term complications [1]. Diabetic retinopathy (DR), one of the most severe microvascular complications of T2DM, represents a major cause of preventable blindness worldwide. Persistent hyperglycemia leads to damage of the retinal and choroidal vasculature, giving rise to pathological alterations including microaneurysms, hemorrhages, and neovascularization [2,3].

The glycemic control of type 2 diabetes mellitus (T2DM) has been traditionally assessed by the concentration of the glycated hemoglobin (HbA1c) form of hemoglobin. HbA1c reflects the mean values of blood glucose concentrations of the previous two to three months. Even so, HbA1c fails to be an excellent proxy marker of the complex processes of microvascular complications of diabetes mellitus [4]. Moreover, the relative lack of responsiveness of HbA1c to changes of short-term glycemic control indicates the need of other novel biological indicators.

Recently, the uric acid to high-density lipoprotein cholesterol ratio (UHR) has received attention as a new biomarker for metabolic and inflammatory conditions purine pathway and its final metabolite, uric acid, is a compound that has been often found to have a higher level in individuals suffering from type 2 diabetes mellitus (T2DM) [5,6]. Low HDL cholesterol, together with elevated LDL cholesterol and triglycerides, is a well-recognized risk factor for chronic inflammatory conditions such as T2DM, metabolic syndrome, and cardiovascular disease. HDL cholesterol levels are inversely associated with oxidative stress and systemic inflammation [7,8].

Elevated UHR levels may therefore reflect systemic inflammation and endothelial dysfunction, both of which can contribute to the development and progression of DR [9]. Given the chronic metabolic–inflammatory milieu involving uric acid and HDL cholesterol, UHR may represent a useful composite marker for estimating the risk and severity of DR.

The aim of the present study was to evaluate the utility of UHR recently recognized as a novel inflammatory biomarker as a predictor of diabetic retinopathy. By investigating the relationship between UHR and the presence of DR, as well as its association with inflammatory and metabolic parameters, we sought to gain a better understanding of how metabolic dysregulation contributes to retinal vascular damage.

2. Materials and methods

This retrospective, observational study was approved by the Ethics Committee of Bolu Abant Izzet Baysal University (Approval No: 2023/401) and it was carried out according to the principles of the Declaration of Helsinki, which was revised in 2000. Written informed consent was obtained from all participants prior to inclusion.

Patients with a diagnosis of type 2 diabetes mellitus who were evaluated at the Ophthalmology Department of Bolu Abant Izzet Baysal University between January 2023 - June 2024 were consecutively screened. A total of 175 individuals who met the predefined inclusion and exclusion criteria were enrolled. A total of 100 patients suffering from diabetic retinopathy were in the study group, while the control group had 75 diabetic individuals who did not show any funduscopic signs of retinopathy.

Comprehensive and standardized ocular tests were carried out for all the participants, which included the best correction vision parameters assessment, slit-lamp biomicroscopy of anterior segment, applanation Goldmann tonometry to measure intraocular pressure, and funduscopic examination after pupil dilation. Macular morphology was measured with a spectral-domain optical coherence tomography (SD-OCT; Heidelberg Spectralis®, v6.3.3.0, Heidelberg Engineering Inc., Heidelberg, Germany). Fundus fluorescein angiography (FFA; Topcon TRC-50DX, Topcon Corporation, Tokyo, Japan) was done when clinically suggested. International Clinical Diabetic Retinopathy Disease Severity

Scale was used for the diagnosis and categorization of diabetic retinopathy.

The information regarding demographic characteristics was obtained from medical records. A venous blood sample was used to collect the blood after fasting for at least 8 hours, and the biochemical tests to be done in the hospital's biochemistry laboratory using an automated analyzer (ARCHITECT c16000, Abbott Laboratories, IL, and USA). The tested parameters include glucose, HbA1c, creatinine, urea, and uric acid, levels of HDL, LDL and total cholesterol, as well as triglycerides, CRP, AST, and ALT. The uric acid/HDL cholesterol ratio (UHR) was calculated using the formula: UHR = uric acid/serum HDL cholesterol.

The G*Power 3.1 software was utilized for the purpose of sample size estimation. As per an expected effect size of Cohen's d = 1.30 which was referenced from similar studies, a total of 22 participants as the minimum required for each group in order to achieve 95% power at a 5% significance level. The sample size of the present study exceeds this requirement substantially, ensuring adequate statistical power.(10)

Exclusion criteria included a history of malignancy, acute systemic infection, hypertension, coronary artery or cerebrovascular disease, autoimmune, hepatic, hematologic, or renal disorders, use of medications affecting uric acid metabolism, previous ocular surgery or trauma, coexisting retinal pathologies unrelated to diabetes, history of vitreoretinal surgery, and the presence of media opacities that could interfere with fundus imaging.

2.1. Statistics

The analyzed data were processed using SPSS Statistical Software (IBM SPSS Statistics version 23; IBM Inc., Chicago, IL, USA). Data normality was assessed using the Kolmogorov–Smirnov test. For normally distributed variables, the Student's t-test was applied and results were reported as mean ± standard deviation. For non-normally distributed variables, the Mann–Whitney U test was performed and data were presented as median (minimum–maximum). Receiver operating characteristic (ROC) analysis was conducted to evaluate the association between UHR and diabetic retinopathy. Pearson correlation analysis was used to assess relationships between UHR and HGB, LYM, RDW, glucose, creatinine, HbA1c, CRP, and albumin. A *p* value <0.05 was accepted as statistically significant.

In addition, a multivariate logistic regression analysis was performed to identify independent predictors of diabetic retinopathy. Variables found to be statistically significant in the univariate analysis, or those of clinical interest (e.g., age, diabetes duration, HbA1c, glucose, creatinine, and urate to hematocrit ratio (UHR)), were then included in the regression analysis. The odds ratios (OR), with 95% confidence intervals (CI), were derived, and a P-value of < 0.05 was used to define statistical significance.

3. Results

A total of 175 patients were included in this study. Among them, 100 patients with diabetic retinopathy were analyzed as the study group, while 75 patients with diabetes mellitus but without any signs of retinopathy were analyzed as the control group. The mean ages of the study and control groups were 67.5 (21-85) and 69 (29-93) years, respectively. Of all patients included in the study, 91 were male (52%) and 84 were female (48%). In the study group, 53% (n=53) were male and 47% (n=47) were female, while 50.7% (n=38) were male and 49.3%

(n=37) were female in the control group. There was no significant difference between the 2 groups in terms of gender (p=0.76).

When Student-T test results were analyzed, HGB (p: 0.021) and HDL (p: 0.032) values were statistically significant. Total cholesterol values were similar between the groups (p=0.31).

According to Mann-Whitney U test results; LYM (p: 0.015), RDW (p<0.001), PDW (p: 0.024), ALT (p<0.010), Albumin (p<0.001) and UHR (p<0.001) (Figure 1) levels statistically significant. There was no difference between age, MCV, PLT, WBC, NEU, MON, EOS, MPV, glucose, urea, creatinine, AST, sodium, potassium, HbA1c, uric acid, LDL, TG, CRP levels.

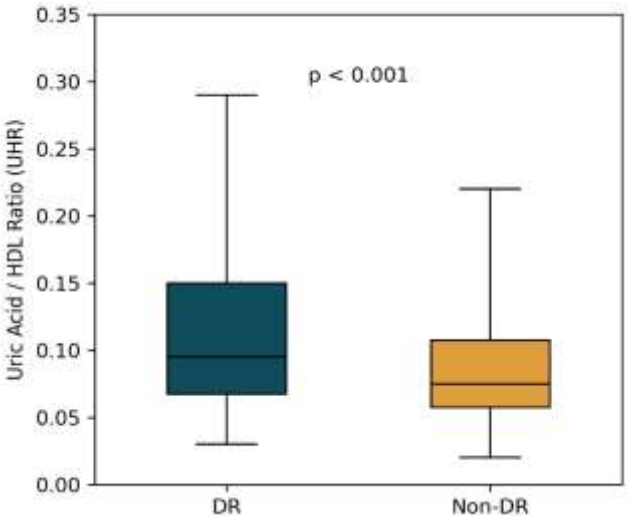

Figure 1. Comparison of UHR levels of DR and control group.

Table 1 displays the general characteristics of both groups as well as the results of tests performed.

The statistical analysis which was conducted using Pearson's correlation showed that UHR was positively correlated significantly with RDW (r=0.237, p: 0.016) and CRP (r=0.382, p<0.01). On the other hand, UHR was significantly and inversely correlated with HGB (r= -0.26, p: 0.02), LYM (r=, -0.32, p: 0.008), Albumin (r= -0.278, p<0.001). There was no correlation UHR with glucose, serum creatinine and HbA1c levels. Correlation of UHR levels with study parameters are shown in Table 2.

In multivariate analysis, UHR showed the strongest association with diabetic retinopathy independently of other hematological and biochemical variables (aOR: 2.78; 95% CI: 1.62–4.75). In the model, RDW and PDW showed a positive association, while albumin and lymphocyte levels were identified as independent negative predictors indicating a protective effect, as shown in Table 3.

UHR levels higher than 11.56% had 86.1% sensitivity and 82.4% specificity in predicting diabetic retinopathy, according to the ROC analysis. Figure 2 shows the ROC curve of UHR in predicting diabetic retinopathy.

4. Discussion

The findings of the present study suggest that the uric acid to high-density lipoprotein cholesterol ratio (UHR) may serve as a potential predictor of diabetic retinopathy in individuals with T2DM. Significantly elevated UHR levels identified in patients with DR indicate that this measure could be applied to estimate retinopathy risk within diabetic populations. With DR still being one of the main causes of vision problems that could have been prevented on the global scale, identifying high-risk patients at an early stage is essential for summary intervention and enhancement of visual outcomes [11].

Past research has scrutinized the correlation among uric acid, HDL cholesterol, and vascular structures of the retina. According to Venkatachalam et al., patients with DR exhibited elevated serum uric acid levels and

Table 1. General characteristics and laboratory data of study population.

	DR group	Non-DR group	*p*
Parameters	***Mean ± standard deviation***		
HGB (gr/dL)	13.11 ± 1.89	12.42 ± 2.17	0.021
HDL cholesterol (mg/dL)	45.05 ± 9.82	48.98 ± 10.63	0.032
Total Cholesterol (mg/dL)	184.69 ± 42.66	191.09 ± 40.87	0.31
	Median (Min–Max)		
Age (year)	67.5 (21-85)	69 (29-93)	0.99
MCV (fL)	86.3 (64-98)	86.5 (68.8-105.9)	0.95
PLT (10^3/uL)	242 (119-598)	243 (90-501)	0.68
WBC (10^3/uL)	7.2 (3.68-12.90)	7.36 (3.60-19.73)	0.97
NEU (10^3/uL)	4.24 (1.68-8.20)	4.47 (1.77-19.35)	0.37
LYM (10^3/uL)	2.08 (0.19-5.47)	1.71 (0.20-4.20)	0.015
MON (10^3/uL)	0.60 (0.20-8.70)	0.57 (0.12-1.22)	0.49
EOS (10^3/uL)	0.15 (0.01-1.60)	0.12 (0.01-1.11)	0.12
RDW (fL)	13.60 (10.9-20.1)	14.5 (12.1-44.7)	<0.001
MPV (fL)	10.2 (7-33.8)	9.90 (5.7-14.60)	0.08
PDW (fL)	13.05 (8.7-19.4)	15.20 (9.8-22.80)	0.024
Glucose (mg/dL)	157 (64-437)	161 (63-469)	0.61
Serum Urea (mg/dL)	39 (18-184)	45 (5-265)	0.47
Serum Creatinine (mg/dL)	0.95 (0.59-11.10)	1.02 (0.40-7.42)	0.16
AST (IU/L)	20 (8-528)	19 (5-116)	0.63
ALT (IU/L)	20 (6-449)	16 (6-80)	<0.001
Sodium (mEq/L)	138 (131-146)	138 (128-158)	0.67
Potassium (mEq/L)	4.60 (4-6)	4.5 (3-6)	0.06
HbA1c (%)	9 (5.6-14.2)	8.4 (5.1-14.1)	0.07
Serum Uric Acid (mg/dL)	5.7 (2.7-10.2)	5.4 (2.8-9.6)	0.10
LDL (mg/dL)	103.4 (34.5-190.2)	108 (47-148)	0.35
TG (mg/dL)	134.5 (37-609)	140 (35-420)	0.87
CRP (mg/dL)	2.5 (0.1-20)	3.41 (0.10-27.6)	0.68
Albumin (mg/dL)	43 (29-54)	42 (25-52)	<0.001
UHR (%)	0.1291 (0.06-0.29)	0.875 (0.05-0.24)	<0.001

HGB: Hemoglobin; HDL: High-density lipoprotein; MCV: Mean corpuscular volume; PLT: Platelet count; WBC: White blood cell; NEU: Neutrophil; LYM: Lymphocyte; MON: Monocyte; EOS: Eosinophil; RDW: Red cell distribution width; MPV: Mean platelet volume; PDW: Platelet distribution width; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; HbA1c: Hemoglobin A1c; LDL: Low-density lipoprotein; TG: Triglyceride; CRP: C-reactive protein; UHR: Uric acid/HDL cholesterol ratio; DR: Diabetic retinopathy.

Table 2. Correlation o UHR levels with study parameters.

	RDW	CRP	HGB	LYM	Albumin
UHR					
r	**0.237**	**0.382**	**-0.26**	**-0.32**	**-0.278**
p	**0.016**	**<0.001**	**0.02**	**0.008**	**<0.001**

UHR – Uric acid/HDL cholesterol ratio; RDW – Red cell distribution width; CRP – C-reactive protein; HGB – Hemoglobin; LYM – Lymphocyte;

Table 3. Multivariate logistic regression analysis for predictors of diabetic retinopathy.

Variable	Adjusted OR	95% CI	p
UHR	2.78	1.62–4.75	**<0.001**
RDW (fL)	1.17	1.05–1.30	0.003
PDW (fL)	1.08	1.01–1.16	0.029
Lymphocyte count (10^3/µL)	0.76	0.58–0.99	0.044
ALT (IU/L)	1.01	1.00–1.02	0.021
Albumin (g/L)	0.90	0.83–0.97	0.006
HDL cholesterol (mg/dL)	0.98	0.95–1.01	0.11
Hemoglobin (g/dL)	1.09	0.94–1.27	0.24
Age (years)	1.00	0.98–1.02	0.92

*ALT, alanine aminotransferase; CI, confidence interval; DR, diabetic retinopathy; HDL, high-density lipoprotein; HGB, hemoglobin; LDL, low-density lipoprotein; OR, odds ratio; PDW, platelet distribution width; RDW, red cell distribution width; TG, triglycerides; UHR, uric acid/high-density lipoprotein cholesterol ratio. *Variables with p < 0.10 in univariate analysis and those considered clinically relevant were included in the multivariate logistic regression model.*

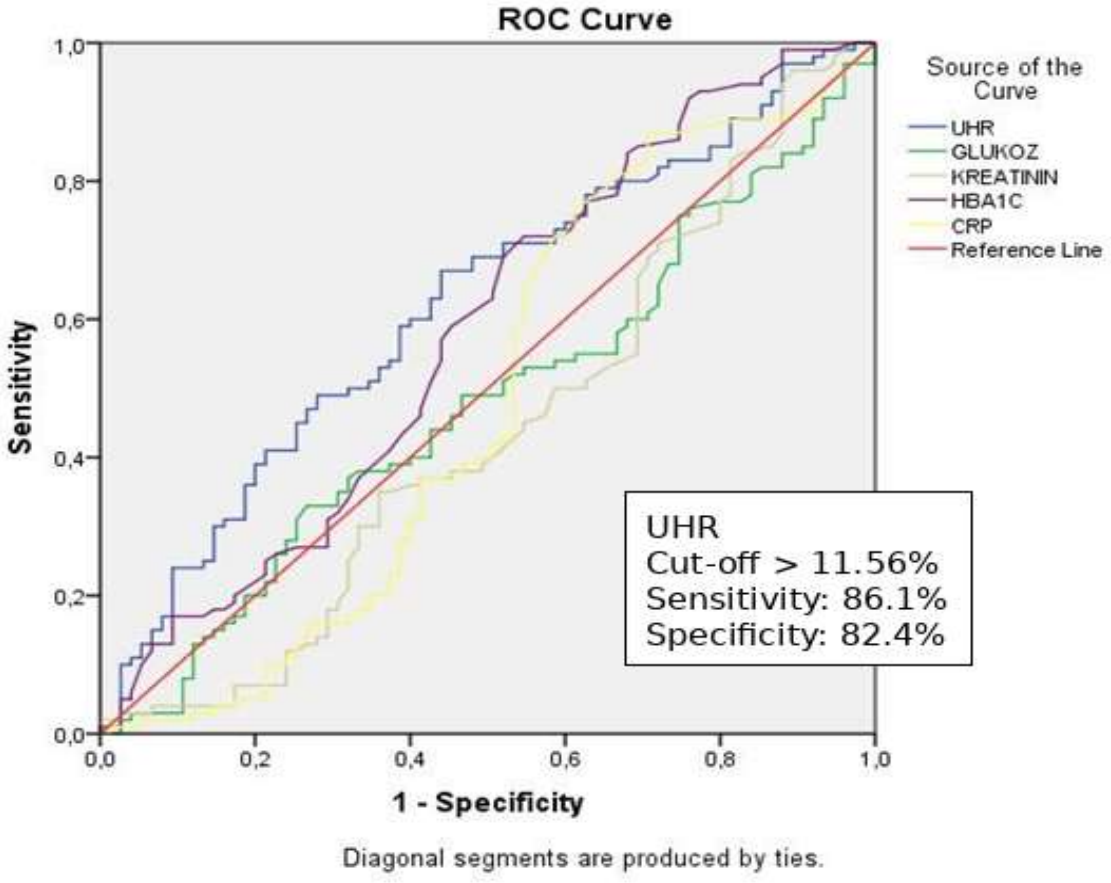

Figure 2. ROC curve of UHR in predicting diabetic retinopathy.

decreased HDL cholesterol [10]. Lee et al. likewise proved that serum uric acid levels were the most important predictor of DR [12]. Similarly, Yang et al. found that high uric acid levels were found to be associated with lower density of retinal capillary plexus vessels [13]. Our results are consistent with the above studies and we support that UHR can be a useful way in predicting the development of DR.

The statistically significant correlation of UHR with inflammatory markers of RDW and CRP in our cohort highlights inflammation as the primary factor in DR [14]. According to Aktas et al., UHR can be an indicator of bad glycemic control in men with T2DM [15]. Excluding the studies of association between UHR and DR, Xuan et al. disclosed the strong relationship of UHR and myocardial infarction which is a reoccurring condition in patients with T2DM that could be explained with the help of UHR [16]. UHR, in line with the previous research, negatively related with albumin, LYM, and HGB, while it was positively associated with RDW, as shown by our analysis. These trends are similar to those found in chronic inflammatory and metabolic states, frequently detected in obesity, hyperlipidemia, and T2DM [17,18].

The presence of elevated uric acid levels may also lead to the development of oxidative stress and endothelial dysfunction, which in turn would facilitate disease progress [19,20]. Kuwata et al. demonstrated that the elevated levels of uric acid is a major determining factor for the occurrence of retinal vascular disease in diabetic individuals, this result also supports the current study [21]. The advanced glycation end products (AGEs) are the main contributors to pericyte loss and vascular injury, which are the following damaged caused in the retina in patients with long-standing hyperglycemia [22,23]. The combination of all these makes the chronic inflammation the trigger for the development of the diabetic retinopathy. The connection between UHR and both inflammatory and metabolic indices noted in our study could, therefore, serve as an explanation of the mechanism through which the increase of UHR contributes to the DR risk. In addition, UHR has been connected with a variety of chronic inflammatory diseases, including non-alcoholic fatty liver disease, Hashimoto's thyroiditis, and diabetic kidney disease [24 – 26]. The given evidence tends to bolster the idea that UHR is a marker of systemic inflammation and metabolic dysregulation in diabetes.

The relation of UHR with DR in our analysis was additionally sustained by ROC findings, which showed extremely good sensitivity and specificity for UHR in detecting DR. Such a good diagnostic performance is in agreement with previous studies that highlight the importance of UHR for the prognosis of diabetic kidney disease and other microangiopathic complications [27].

The connection of UHR and lipid metabolism can be presumed to be significant. The low HDL levels that are components of UHR, lead to the cardiovascular disease and other inflammatory conditions. The decrease in HDL levels in diabetes can be more severe and thus reduce the protective effects of the vascular endothelium and the antioxidant capacity [28,29]. A number of research works have pointed to the relationship between low HDL cholesterol, metabolic syndrome and diabetes [30-32]. Here, it is particularly important to note the considerable differences in HDL levels found between the DR and the control groups in our study.

Despite the strengths of the research, there are limitations associated with this study based on its merits. Some of the significant strengths

of this study are its retrospective nature and small population base conducted at a single center. These aspects of the research might limit its universality and acceptance and the generalization of its results and outcomes. To further justify and substantiate the results of this research and explore this concept of ultrahigh risk (UHR) indicators in a detailed and broader manner for risk stratification of diabetic retinopathy (DR), further research needs to focus on multi-center and larger population studies with a prospective approach and forward-looking outcomes.

In conclusion, it is safe to say that the use of UHR for the purpose of predicting diabetic retinopathy in patients with T2DM is effective and reliable. Inflammatory and metabolic markers, together with the great sensitivity and specificity of UHR in distinguishing DR, recommend UHR as a key instrument in the early diagnosis and treatment of DR in everyday practice. Following the earlier observations that UHR is related to different metabolic and inflammatory diseases, the current study gives new evidence to support this relationship and also highlights the importance of exploring UHR in diabetic individuals more thoroughly.

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Ethical Statement: *The ethical committee of Bolu Abant İzzet Baysal University (ethics no:2023/401) granted approval for the research and before collecting patient data, the required approval was obtained. The individuals involved in the study consented to their participation in writing. Informed consent forms were obtained from all participants.*

Informed consent: *Informed consent was obtained from all participants prior to being included in the study.*

Data Availability: *The data used in this study can be made available upon reasonable request.*

Open Access Statement

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