Received: 27 Apr 2025 | **Accepted:** 04 Sep 2025 **Genel Tıp Derg. Volume** 35/**Issue** 5 (October),954-961
DOI: 10.54005/geneltip.1683730

ORIGINAL ARTICLE

Triglyceride-Glucose Index in Central Retinal Artery Occlusion: A Metabolic Risk Factor?

Santral Retinal Arter Tıkanıklığında Trigliserit-Glikoz İndeksi: Metabolik Bir Risk Faktörü Mü?

[1]Ali Osman GÜNDOĞAN, [1]Refik OLTULU, [1]Selman BELVİRANLI, [1]Ali TEZCAN, [1]Enver MİRZA, [1]Mehmet ADAM, [1]Mehmet OKKA

[1]Department of Ophthalmology, Meram Faculty of Medicine, Necmettin Erbakan University, Konya, Turkey,

Correspondence

Ali Osman GÜNDOĞAN,
at Department of Ophthalmology, Meram Faculty of Medicine, Necmettin Erbakan University, Konya, Turkey

E-Mail: aogundogan@hotmail.com

How to cite ?

Gündoğan A. O., Oltulu R., Belviranlı S., Tezcan A., Mirza E., Adam M., Okka M., Triglyceride-Glucose Index in Central Retinal Artery Occlusion: A Metabolic Risk Factor?, Genel Tıp Derg. 2025;35(5):954-961

ABSTRACT

Aim: To evaluate the triglyceride-glucose index (TGI) in patients with central retinal artery occlusion (CRAO) compared with controls and to determine its prognostic significance.
Methods: A total of 54 CRAO patients and 54 healthy controls participated in this retrospective study. The TGI was calculated based on fasting plasma triglyceride and glucose levels and the results were evaluated between the different groups. To determine the optimal TGI threshold, ROC curve analysis was performed and its sensitivity and specificity were determined between the CRAO and control groups.
Results: No statistically meaningful difference across the groups in terms of age and sex ($p = 0.547$ and $p = 1.000$, respectively). The TGI was identified as 8.46 [8.37, 8.59] in the control group and 8.86 [8.55, 9.10] in the CRAO group ($p<0.001$). The optimal ROC cut-off value for TGI distinguishing CRAO and control groups was determined as 8.68, with 63% sensitivity and 98% specificity (AUC: 0.865, $p<0.001$, 95% CI 0.800–0.931).
Conclusion: Higher levels of TGI appear to be associated with the occurrence of CRAO. TGI may be a potential marker for CRAO. Given the association of elevated TGI with increased metabolic and cardiovascular risk, patients with CRAO should be carefully evaluated.

Keywords: blood lipid profile, central retinal artery occlusion, glucose, triglyceride, triglyceride-glucose index

ÖZ

Amaç: Santral retinal arter tıkanıklığı (SRAT) olan hastalarda trigliserid glukoz indeksini (TGI) kontrol grubuyla karşılaştırmalı olarak değerlendirmek ve prognostik önemini belirlemek.
Gereç ve Yöntemler: Bu retrospektif çalışmaya toplam 54 SRAT hastası ve 54 sağlıklı kontrol katıldı. TGI, açlık plazma trigliseritleri ve glukoz seviyeleri kullanılarak belirlenmiş ve sonuçlar gruplar arasında karşılaştırılmıştır. ROC eğrisi, SRAT ve kontrol grupları arasındaki duyarlılık ve özgüllüğün yanı sıra optimum TGI cut-off değerini belirlemek için uygulandı.
Bulgular: Cinsiyet ve yaş bakımından gruplar karşılaştırıldığında anlamlı bir farklılık saptanmadı (sırasıyla $p = 0.547$ ve $p = 1.000$). TGI değeri kontrol grubunda 8.46 [8.37, 8.59] ve SRAT grubunda 8.86 [8.55, 9.10] olarak tespit edilmiştir ($p<0.001$). SRAT ve kontrol gruplarını ayırt eden TGI için optimal ROC cutoff değeri %63 duyarlılık ve %98 özgüllük ile 8.68 olarak hesaplanmıştır (AUC: 0.865, $p<0.001$, %95 CI 0.800-0.931).
Sonuçlar: Daha yüksek TGI seviyeleri SRAT oluşumu ile ilişkili görünmektedir. TGI, SRAT için potansiyel bir belirteç olabilir. Yüksek TGI'nın artmış metabolik ve kardiyovasküler risk ile ilişkisi nedeniyle, SRAT'lı hastalar dikkatle değerlendirilmelidir.

Anahtar Kelimeler: Glukoz, kan lipid profili, santral retinal arter tıkanıklığı, trigliserid, trigliserid-glukoz indeksi

Peer-Review: Double anonymized - Two External
Plagiarism Checks: Yes - intihal.net
Complaints: geneltip@selcuk.edu.tr

Selcuk University Press
Genel Tıp Dergisi | e-ISSN: 2602-3741
https://dergipark.org.tr/tr/pub/geneltip
https://yayinevi.selcuk.edu.tr/

INTRODUCTION

The central retinal artery (CRA), originating from the ophthalmic artery—a primary branch of the internal carotid artery—plays a critical role in ocular blood supply. It provides nourishment to the outer sheath of the optic disc and the inner retinal layers through its bifurcated branches (1).

Retinal artery occlusion (RAO) is a severe ophthalmologic condition that commonly leads to abrupt and substantial vision loss, with an estimated occurrence of 1 per 100,000 individuals. Based on the presence of giant cell arteritis, RAO is classified as arteritic or non-arteritic. Non-arteritic RAO encompasses central retinal artery occlusion (CRAO), which constitutes 56% of cases, branch retinal artery occlusion (BRAO) at 38%, and the rare cilioretinal artery occlusion at 5% (2,3).

RAO arises from disrupted retinal blood flow, often caused by embolism, retinal vasculitis, atherosclerosis, or vascular trauma. Furthermore, stenosis in the internal carotid artery or retinal arterioles can critically compromise ocular perfusion, resulting in retinal ischemia (4). Notably, systemic diseases such as ischemic stroke, atrial fibrillation, and coronary artery disease are recognized risk factors in CRAO (5,6). Research indicates that the likelihood of ischemic stroke increases substantially in the weeks following CRAO onset (7). Diabetes mellitus (DM) affects multiple organ systems, with the eye being particularly vulnerable. Although diabetic retinopathy is the most recognized ocular complication of DM, retinal vascular occlusions are also significant (8). Diabetic patients often present with microvascular abnormalities, including arteriolar narrowing and arteriovenous notching, which are typical findings in CRAO (9-11).

The triglyceride-glucose index (TGI), initially proposed by Simmental-Mendra et al. serves as a marker of insulin resistance (12). Recent studies have associated TGI with cardiovascular conditions such as ischemic stroke, coronary artery calcification, hypertension, and carotid atherosclerosis. Therefore, TGI is closely linked to both insulin resistance and early atherosclerosis, both of which are key factors in the development of cardiovascular and cerebrovascular diseases. It is also a predictive tool for type 2 diabetes (13,14). However, no studies to date have investigated the relationship between TGI and CRAO. This study was designed to evaluate the predictive value of TGI by comparing its levels in CRAO patients with those in healthy controls.

MATERIALS and METHODS

This retrospective study was carried out at the ophthalmology clinic of Necmettin Erbakan University Faculty of Medicine Hospital. The intended data were obtained by scanning the medical records and laboratory results of patients newly diagnosed with CRAO between January 2021 and June 2023. Ethical permission for the study was obtained from the local ethics commission. (Decision No: 2023/4695), and the tenets of the Declaration of Helsinki were observed in the study. The analysis included 54 CRAO patients (CRAO group) aged 18 and above, along with 54 age- and gender-matched healthy controls (control group).

Patients in the CRAO group presented to the emergency department with sudden, painless vision loss, subsequently confirmed to have CRAO following consultation. The diagnosis of CRAO was established

based on clinical presentation—sudden, painless, unilateral vision loss—along with fundoscopic signs such as retinal pallor and a cherry-red spot at the fovea. In all cases, fluorescein angiography (FFA) confirmed the diagnosis by showing delayed or absent filling of the central retinal artery and prolonged arteriovenous transit time. To ensure age and ocular health comparability, controls were chosen from patients scheduled for elective cataract surgery who had no history of retinal or systemic disease. For the control group, routine blood test results obtained before cataract surgery were analyzed. The control group consisted of individuals scheduled for uncomplicated cataract surgery, with no history of systemic diseases such as diabetes mellitus, hypertension, or cardiovascular disorders, and no ocular vascular pathology upon fundus examination and OCT imaging. These participants were selected due to their availability for both ophthalmologic and systemic evaluations under standardized preoperative conditions. For the CRAO group, blood test results taken within 24 hours of diagnosis were evaluated. Data from blood samples collected in the morning after 12 hours of fasting were recorded for all participants.

Exclusion criteria included the following: 1) systemic illnesses other than hypertension, such as diabetes, cardiovascular diseases (e.g., coronary artery disease, valve disorders, congestive heart failure), stroke, renal or hepatic dysfunction, 2) lipid disorders, including familial dyslipidemia, 3) active smoking or alcohol use, 4) prior COVID-19 infection, 5) malignancies or vasculitides, 6) use of medications such as steroids or lipid-lowering agents, with the exception of antihypertensives, 7) any history of intraocular surgery, and 8) eye conditions other than CRAO (e.g., glaucoma, uveitis, ocular trauma, corneal abnormalities, or infections). It should be noted that diabetic patients were excluded from the study to avoid the confounding effects of diabetes-related macrovascular and microvascular complications on TGI levels. However, this exclusion criterion may limit the generalizability of the findings to real-world clinical populations, where diabetes is a common comorbidity. Future studies including diabetic individuals may provide more comprehensive insight into the utility of TGI across broader patient groups.

To qualify for the study, participants' detailed medical records, including best-corrected visual acuity, intraocular pressure readings, a thorough ophthalmologic examination (encompassing both anterior segment and fundus evaluations), optical coherence tomography (OCT) results, fluorescein angiography data, and simultaneous fasting plasma glucose and triglyceride measurements, were required. The triglyceride-glucose index (TGI) was computed using the formula: Ln [fasting triglycerides (mg/dL) × fasting plasma glucose (mg/dL)/2], which was applied using Microsoft Excel software.

Statistical analysis was performed using IBM SPSS Statistics version 20.0 (IBM Corp., Armonk, NY, USA). The Kolmogorov-Smirnov test was applied to assess the normality of continuous data. Normally distributed variables are presented as mean ± standard deviation (SD), while non-normally distributed data are shown as median [25th, 75th percentiles]. Categorical variables are expressed as counts (n) and percentages

(%). Group comparisons were made using the independent samples t-test for normally distributed continuous variables and the Mann-Whitney U test for non-normally distributed ones. Pearson's chi-squared test was employed for categorical variables. The receiver operating characteristic (ROC) curve was used to identify the optimal TGI cut-off point and assess its sensitivity and specificity in distinguishing between the CRAO and control groups. A p-value of <0.05 was considered statistically significant.

RESULTS

Among the 54 subjects in both the control and CRAO groups, 35 (64.8%) were male and 19 (35.2%) were female. The mean age in the control group was 61.57 (11.65) years, compared to 63.11 (14.63) years in the CRAO group. There were no significant differences in age or sex across the groups. (p = 0.547 and p = 1.000, respectively) (Table 1).

When laboratory parameters were analyzed, the LDL levels in the control group had a mean 106.10±14.71 mg/dL, while the CRAO group had a mean of 113.09±33.37 mg/dL, with no significant difference observed between the two groups (p = 0.180). The level of HDL was significantly reduced in the CRAO group (42.80±9.49 mg/dL) compared to the control group (48.40±7.43 mg/dL) (p=0.001).

In terms of other lipid parameters, VLDL levels were 24.31±5.25 mg/dL in the control group and 28.18±8.91 mg/dL in the CRAO group, while total cholesterol levels were 175.34±14.16 mg/dL in the control group and 187.07±40.03 mg/dL in the CRAO group. Both of these blood parameters were found to be significantly increased in the CRAO group. (p=0.032 and p=0.045, respectively).

Triglyceride levels were also elevated in the CRAO group, with a median value of 143.50 mg/dL [101.75–180.00], compared to 98.50 mg/dL [89.50–108.25] in the control group (p < 0.001). Similarly, blood glucose levels were higher in the CRAO group, with a median value of 99.95 mg/dL [95.00–110.00] compared to 97.50 mg/dL [91.75–103.00] in the control group (p = 0.005).

The TGI values showed a marked difference between the groups, with the CRAO group having a median TGI of 8.86 [8.55–9.10], significantly higher than the control group's median of 8.46 [8.37–8.59] (p < 0.001) (Table 2). Using ROC curve analysis, the optimal TGI cut-off for distinguishing between CRAO and control groups was calculated as 8.68, with a sensitivity of 63% and specificity of 98% (AUC: 0.865, p < 0.001, 95% CI: 0.800–0.931) (Figure-1).

Table 1. Demographical and clinical characteristics of the participants in the CRAO and control groups.

		CRAO Group (n=54)	Control Group (n=54)	p value
Age, years, mean±SD		63.11±14.63	61.57±11.65	0.547*
Gender	**Male (n, %)**	35, 64.8%	35, 64.8%	1.000**
	Female (n, %)	19, 35.2%	19, 35.2%	

* Statistical significance in the Independent samples – t test

** Statistical significance in the Pearson's Chi – squared test

Table 2. Comparison of the laboratory findings and Triglyceride-Glucose Index (TGI) between the CRAO and control groups

	CRAO Group (n=54)	Control Group (n=54)	p value
Glucose, mg/dl, median [25th, 75th]	99.95 [95.00, 110.00]	97.50 [91.75, 103.00]	**0.005***
Triglyceride, mg/dl, median [25th, 75th]	143.50 [101.75, 180.00]	98.50 [89.50, 108.25]	**<0.001***
HDL, mg/dl, mean±SD	42.80±9.49	48.40±7.43	**0.001****
LDL, mg/dl, mean±SD	113.09±33.37	106.10±14.71	**0.180****
VLDL, mg/dl, mean±SD	28.18±8.91	24.31±5.25	**0.032****
Total Cholesterol, mg/dl, mean±SD	187.07±40.03	175.34±14.16	**0.045****
TGI, median [25th, 75th]	8.86 [8.55, 9.10]	8.46 [8.37, 8.59]	**<0.001***

* Statistical significance in the Mann Whitney-U test

** Statistical significance in the Independent samples – t test

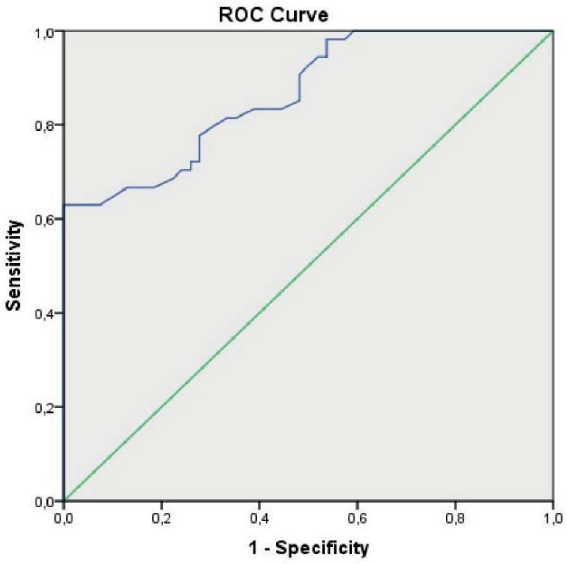

Figure 1. Receiver operating characteristics (ROC) curve analysis of Triglyceride-Glucose Index (TGI) between the CRAO and control groups (Optimal ROC cut-off value: 8.68 with 63% sensitivity and 98% specificity, AUC: 0.865, p<0.001, 95%CI 0.800-0.931)

DISCUSSION

The findings of the present study demonstrated a significant increase in TGI levels in patients diagnosed with CRAO compared to control groups. The cut-off value for TGI was determined as 8.68 with 63% sensitivity and 98% specificity in differentiating CRAO and control groups. The high specificity of the TGI (98%) for diagnosing central retinal artery occlusion (CRAO) suggests that the index is effective at distinguishing between CRAO patients and healthy individuals, with a low false positive rate. However, its relatively low sensitivity (63%) suggests that it may be unable to detect all CRAO cases. Therefore, TGI may not be suitable as a standalone screening tool. Instead, it may be more effective as a supportive biomarker in cases with clinical suspicion.

Central retinal artery occlusion (CRAO) is a medical emergency characterized by the obstruction of retinal blood flow due to a thrombus in the central retinal artery, leading to sudden vision loss. This condition is considered a form of acute ischemic stroke and a potential warning sign for future cerebrovascular and cardiovascular complications. Studies suggest that the mechanism of CRAO involves thrombotic or embolic causes similar to those in ischemic stroke and heart disease, often originating from carotid artery plaques or cardiac valve abnormalities (15). Current evidence indicates that CRAO typically results from thrombus formation near or within the lamina cribrosa (16). The shared pathophysiology between CRAO, cerebrovascular, and cardiovascular diseases has been linked to atherosclerosis. Additionally, serotonin

secreted during platelet aggregation on atherosclerotic plaques in the carotid artery has been implicated in CRAO pathogenesis by triggering retinal artery vasospasms and reduced blood flow (17). Song et al. highlighted that carotid intima-media thickness and total area of plaque were notably higher in individuals with retinal artery occlusion than in the general population (18).

Although patients with diabetes mellitus were excluded from the current study to eliminate potential confounding factors, it is important to acknowledge that diabetes is a well-established risk factor for both macrovascular and microvascular complications, including atherosclerosis and endothelial dysfunction. These mechanisms are also central to the development of CRAO. It has been determined that the main pathological mechanism present in the arterial wall of diabetic subject is atherosclerotic changes that may lead to arterial stiffening and atherothrombotic disorders (19–21).

The primary shared mechanism between DM and CRAO involves macrovascular changes, including arterial stiffness and atherosclerosis (22). Another shared feature is microvascular retinopathy, characterized by narrowing of the retinal arterioles. The retina's high oxygen demand is met by the ophthalmic artery, and prolonged hyperglycemia leads to collagen accumulation in arterioles and basement membrane thickening (23). Studies suggest that DM contributes significantly to microvascular changes, such as arteriolar narrowing and arteriovenous nicking (24,25). Other factors causing vascular alterations in DM include oxidative stress due to hyperglycemia, overproduction of advanced glycation end-products, insulin resistance, and endothelial dysfunction (26,27).

The connection between CRAO and lipid profiles is not well-documented. Risimic et al. reported no meaningful differences in triglyceride values or BMI among CRAO patients and controls, though LDL and HDL levels were significantly higher in the CRAO group (28). While our study supports the finding of elevated LDL in CRAO patients, we observed lower HDL levels. Stojakovic et al. emphasized the role of dyslipidemia in retinal vascular diseases and identified increased LDL-C and LP(a) levels as predictive factors for retinal artery and vein occlusions (29). Hwang et al. further identified low HDL levels as an independent risk factor for retinal artery occlusion, a finding consistent with our study (30).

The triglyceride-glucose index (TGI) has recently garnered attention for its association with cardiovascular and atherosclerotic diseases. Alessandra et al. demonstrated a positive relationship between TGI and symptomatic coronary artery disease, supporting its utility as a marker for atherosclerosis (31). Similarly, Irace et al. established a strong and consistent link between TGI and carotid atherosclerosis across two cohorts (32). Kim MK et al. identified TGI as an independent prognostic marker for coronary artery calcification (33).

TGI has also been studied in the context of insulin resistance. Low et al. found TGI to be an effective tool for tracking type 2 diabetes progression in adults (34). While Yoon et al. demonstrated that TGI correlated with the HOMA-IR index in identifying type 2 diabetes in children and adolescents.

They noted that TGI outperformed HOMA-IR as a predictive marker for the disease, highlighting its potential in monitoring type 2 diabetes development (35).

This study has some limitations. It was a single-center study with a relatively small sample size, which may limit the generalizability of the findings. Its retrospective design and the absence of long-term follow-up also limit evaluation of the prognostic value of TGI in CRAO. In addition, the lack of HbA1c or formal prediabetes screening may be a limitation as undiagnosed diabetes or insulin resistance may have influenced TGI levels in some participants. Also including CRAO patients under the age of 50 may have introduced some variability, as younger individuals may have distinct underlying mechanisms. Future studies may benefit from age-stratified analysis to improve population homogeneity.

CONCLUSION

In conclusion, this study identified a significant correlation between elevated TGI levels and CRAO, marking it as the first to demonstrate this relationship. TGI may serve as a potential biomarker for CRAO. Given the link between high TGI levels and increased metabolic and cardiovascular risks, CRAO patients should be evaluated for these associated conditions. Larger prospective research is necessary to explore the predictive and prognostic value of TGI in CRAO.

Conflict of Interest: The authors declared no conflicts of interest concerning the authorship and/or publication of this article.

Financial Support: The authors received no financial support for the research, authorship, and/or publication of this article.

REFERENCES

1. Singh S, Dass R. The central artery of the retina. I. Origin and course. Br J Ophthalmol 1960;44:193-212

2. Hayreh SS. Acute retinal arterial occlusive disorders. Prog Retin Eye Res 2011;30:359-394

3. Leavitt JA, Larson TA, Hodge DO, Gullerud RE. The incidence of central retinal artery occlusion in Olmsted County, Minnesota. Am J Ophthalmol 2011;152:820-3.e2

4. Hayreh SS, Podhajsky PA, Zimmerman MB. Retinal artery occlusion: associated systemic and ophthalmic abnormalities. Ophthalmology 2009;116:1928-1936

5. Park SJ, Choi NK, Yang BR, Park KH, Lee J, Jung SY, et al. Risk and Risk Periods for Stroke and Acute Myocardial Infarction in Patients with Central Retinal Artery Occlusion. Ophthalmology 2015;122:2336-2343.e2

6. Chang YS, Chu CC, Weng SF, Chang C, Wang JJ, Jan RL. The risk of acute coronary syndrome after retinal artery occlusion: a population-based cohort study. Br J Ophthalmol 2015;99:227-231

7. Chodnicki KD, Pulido JS, Hodge DO, Klaas JP, Chen JJ. Stroke Risk Before and After Central Retinal Artery Occlusion in a US Cohort. Mayo Clin Proc 2019;94:236-241

8. Khan A, Petropoulos IN, Ponirakis G, Malik RA. Visual complications in diabetes mellitus: beyond retinopathy. Diabet Med 2017;34:478-484

9. Klein R, Klein BE, Moss SE, Wong TY. Retinal vessel caliber and microvascular and macrovascular disease in type 2 diabetes: XXI: the Wisconsin Epidemiologic Study of Diabetic Retinopathy. Ophthalmology 2007;114:1884-1892

10. Haymore JG, Mejico LJ. Retinal vascular occlusion syndromes. Int Ophthalmol Clin 2009;49:63-79

11. Petzold A, Islam N, Hu HH, Plant GT. Embolic and nonembolic transient monocular visual field loss: a clinicopathologic review. Surv Ophthalmol 2013;58:42-62

12. Simental-Mendía LE, Rodríguez-Morán M, Guerrero-Romero F. The product of fasting glucose and triglycerides as surrogate for identifying insulin resistance in apparently healthy subjects. Metab Syndr Relat Disord 2008;6:299-304

13. Hong S, Han K, Park CY. The triglyceride glucose index is a simple and low-cost marker associated with atherosclerotic cardiovascular disease: a population-based study. BMC Med 2020;18:361

14. Chamroonkiadtikun P, Ananchaisarp T, Wanichanon W. The triglyceride-glucose index, a predictor of type 2 diabetes development: A retrospective cohort study. Prim Care Diabetes 2020;14:161-167

15. Mac Grory B, Schrag M, Biousse V, Furie KL, Gerhard-Herman M, Lavin PJ, et al. Management of Central Retinal

Artery Occlusion: A Scientific Statement From the American Heart Association [published correction appears in Stroke. 2021 Jun;52(6):e309]. Stroke 2021;52:e309

16. Duker JS, Sivalingam A, Brown GC, Reber R. A prospective study of acute central retinal artery obstruction. The incidence of secondary ocular neovascularization. Arch Ophthalmol 1991;109:339-342

17. Hayreh SS, Piegors DJ, Heistad DD. Serotonin-induced constriction of ocular arteries in atherosclerotic monkeys. Implications for ischemic disorders of the retina and optic nerve head. Arch Ophthalmol 1997;115:220-228

18. Song YJ, Cho KI, Kim SM, Jang HD, Park JM, Kim SS, et al. The predictive value of retinal vascular findings for carotid artery atherosclerosis: are further recommendations with regard to carotid atherosclerosis screening needed? Heart Vessels 2013;28:369-376

19. Shi Y, Vanhoutte PM. Macro- and microvascular endothelial dysfunction in diabetes. J Diabetes 2017;9:434-449

20. Kozakova M, Palombo C. Diabetes Mellitus, Arterial Wall, and Cardiovascular Risk Assessment. Int J Environ Res Public Health 2016;13:201

21. Beckman JA, Creager MA, Libby P. Diabetes and atherosclerosis: epidemiology, pathophysiology, and management. JAMA 2002;287:2570-2581

22. Booth GL, Kapral MK, Fung K, Tu JV. Relation between age and cardiovascular disease in men and women with diabetes compared with non-diabetic people: a population-based retrospective cohort study Lancet 2006;368:29-36

23. Davidson JA, Ciulla TA, McGill JB, Kles KA, Anderson PW. How the diabetic eye loses vision. Endocrine 2007;32:107-116

24. Wong TY, Klein R, Sharrett AR, Schmidt MI, Pankow JS, Couper DJ, et al; ARIC Investigators. Retinal arteriolar narrowing and risk of diabetes mellitus in middle-aged persons. JAMA 2002;287:2528-2533

25. Carlson EC. Scanning and transmission electron microscopic studies of normal and diabetic acellular glomerular and retinal microvessel basement membranes. Microsc Res Tech 1994;28:165-177

26. Maritim AC, Sanders RA, Watkins JB 3rd. Diabetes, oxidative stress, and antioxidants: a review. J Biochem Mol Toxicol 2003;17:24-38

27. Nigro J, Osman N, Dart AM, Little PJ. Insulin resistance and atherosclerosis. Endocr Rev 2006;27:242-259

28. Risimić D, Nikolić D, Jaksić V, Simeunović D, Milenković S, Stefanović I, et al. Evaluation of body mass index and lipid fractions levels in patients with retinal artery occlusion. Vojnosanit Pregl 2011;68:231-234

29. Stojakovic T, Scharnagl H, März W, Winkelmann BR, Boehm BO, Schmut O. Low density lipoprotein triglycerides and lipoprotein(a) are risk factors for retinal vascular occlusion. Clin Chim Acta 2007;382:77-81

30. Hwang S, Kang SW, Choi KJ, Son KY, Lim DH, Shin DW, et al. High-density Lipoprotein Cholesterol and the Risk of Future Retinal Artery Occlusion Development: A Nationwide Cohort Study. Am J Ophthalmol 2022;235:188-196.

31. da Silva A, Caldas APS, Hermsdorff HHM, Bersch-Ferreira ÂC, Torreglosa CR, Weber B, et al. Triglyceride-glucose index is associated with symptomatic coronary artery disease in patients in secondary care. Cardiovasc Diabetol 2019;18:89

32. Irace C, Carallo C, Scavelli FB, De Franceschi MS, Esposito T, Tripolino C, et al. Markers of insulin resistance and carotid atherosclerosis. A comparison of the homeostasis model assessment and triglyceride glucose index. Int J Clin Pract 2013;67:665-672

33. Kim MK, Ahn CW, Kang S, Nam JS, Kim KR, Park JS. Relationship between the triglyceride glucose index and coronary artery calcification in Korean adults. Cardiovasc Diabetol 2017;16:108

34. Low S, Khoo KCJ, Irwan B, Sum CF, Subramaniam T, Lim SC, et al. The role of triglyceride glucose index in development of Type 2 diabetes mellitus. Diabetes Res Clin Pract 2018;143:43-49

35. Yoon JS, Lee HJ, Jeong HR, Shim YS, Kang MJ, Hwang IT. Triglyceride glucose index is superior biomarker for predicting type 2 diabetes mellitus in children and adolescents. Endocr J 2022;69:559-565