Cukurova Medical Journal
ÇUKUROVA ÜNİVERSİTESİ TIP FAKÜLTESİ

Cukurova Med J 2025;50(3):586-593
DOI: 10.17826/cumj.1625502

RESEARCH

Triglyceride-glucose index as a potential biomarker in acute central serous chorioretinopathy

Akut santral seröz koryoretinopatide potansiyel bir belirteç olarak trigliserit-glikoz indeksi

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Abstract

Purpose: The aim of this study is to compare triglyceride-glucose index (TGI) levels in patients with acute central serous chorioretinopathy (CSCR), chronic CSCR, and healthy controls, and to the value of TGI in indicating the risk of acute CSCR.

Materials and Methods: This retrospective study included 20 patients with acute CSCR, 20 patients with chronic CSCR, and 40 healthy controls. The TGI was calculated using fasting plasma triglyceride and glucose levels, and values were compared between groups. A receiver operating characteristic (ROC) curve was used to calculate the optimal TGI cut-off value, sensitivity, and specificity between the acute CSCR and control groups.

Results: There were no statistically significant differences between the groups in terms of age and sex. The TGI value was 8.48±0.29 in the control group, 8.91±0.34 in the acute CSCR group, and 8.59±0.40 in the chronic CSCR group ($p<0.001$ for control vs. acute CSCR; $p=0.703$ for control vs. chronic CSCR; $p=0.011$ for acute vs. chronic CSCR). The optimal ROC cut-off value of TGI between the control and acute CSCR groups was calculated as 8.74 with 75% sensitivity and 85% specificity (AUC: 0.835, $p<0.001$, 95% CI 0.719-0.951).

Conclusion: Caution should be exercised in patients with acute CSCR regarding the presence of underlying cardiovascular disease. Additionally, this biomarker, which can be easily measured through routine blood testing, may serve as a potential indicator associated with the acute form of CSCR.

Keywords:. Central serous chorioretinopathy, triglyceride, glucose, triglyceride-glucose index, blood-lipid profile

Öz

Amaç: Bu çalışmada akut santral seröz koryoretinopatili (SSKR) hastalarda, kronik SSKR'de ve sağlıklı kontrollerde trigliserid-glukoz indeksi (TGI) düzeylerini karşılaştırmak ve TGI'nin akut SSKR riskini göstermedeki değerini değerlendirmek amaçlanmıştır.

Gereç ve Yöntem: Bu retrospektif çalışmaya 20 akut SSKR hastası, 20 kronik SSKR hastası ve 40 sağlıklı kontrol dahil edildi. TGİ, açlık plazma trigliseridler düzeyi ve açlık plazma glukozu kullanılarak hesaplandı ve değerler gruplar arasında karşılaştırıldı. Akut SSKR ve kontrol grupları arasında optimal TGI cut-off değerininin ve bu değerin sensitivite ve spesifitesinin hesaplanması için ROC eğrisi kullanıldı.

Bulgular: Gruplar arasında yaş ve cinsiyet açısından istatistiksel olarak anlamlı farklılık tespit edilmedi. TGI değeri kontrol grubunda 8.48±0.29, akut SSKR grubunda 8.91±0.34, kronik SSKR grubunda 8.59±0.40 olarak bulundu. (akut SSKR grubu ile kontrol grubu için $p<0,001$; kronik SSKR grubuna ile kontrol grubu için $p=0,703$; kronik SSKR grubuna ile akut SSKR grubu için $p=0,011$) Kontrol ve akut SSKR grupları arasında TGI için optimum ROC cutoff değeri %75 duyarlılık ve %85 özgüllük ile 8.74 olarak hesaplanmıştır. (AUC: 0.835, $p<0.001$, %95CI 0.719-0.951)

Sonuç: Akut SSKR hastalarında kardiyovasküler hastalıklar açısından dikkatli olunması gerektiğini göstermektedir. Ayrıca rutin kan analizi ile kolayca ölçülebilen bu biyobelirteç, SSKR'nin akut formuyla ilişkili potansiyel bir gösterge olabilir.

Anahtar kelimeler: Santral seröz koryoretinopati, trigliserid, glukoz, trigliserid-glukoz indeksi, kan-lipid profili

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Received: 25.01.2025 Accepted: 30.06.2025

INTRODUCTION

Central serous chorioretinopathy (CSCR) is a retinal disorder that involves fluid leakage from the choroidal capillaries, leading to detachment of the neurosensory retina. This condition can result in various symptoms, such as blurred vision, central blind spots, distorted vision, micropsia, dyschromatopsia and a decrease in visual contrast[1]. The disease most commonly affects young and middle-aged males[2]. Approximately 30% of patients with CSCR experience recurrence, and these patients are prone to develop recurrence and permanent vision loss[3].

CSCR can occur in three forms: acute, chronic or recurrent. Acute cases usually resolve spontaneously within 3 months and often don't require medical intervention. Chronic CSCR is characterised by persistent symptoms and clinical signs lasting 4 to 6 months or longer.[4] In recurrent cases, long-term effects such as retinal pigment epithelium (RPE) damage, choroidal neovascular membrane (CNVM) development and fibrin accumulation may be seen as sequelae of CSCR[5].

Although the pathogenesis of CSCR is not fully clarified, male gender, type A personality, psychological stress, high levels of exogenous or endogenous cortisol, pregnancy, and smoking are well-known risk factors[6-8]. Apart from these, the relationship between CSCR and cardiovascular and atherosclerotic diseases is a frequently investigated topic in the literature. Recent studies have indicated that CSCR may be linked to an increased risk of cardiovascular disease and stroke, particularly among men. Elevated cortisol, a key risk factor for CSCR, has been shown to contribute to endothelial dysfunction and hypercoagulability[11]. It is well known that endothelial dysfunction is the main trigger of atherosclerotic changes and the main etiological factor in many vascular diseases, including stroke.

The triglyceride-glucose index (TGI), first proposed as a marker of insulin resistance[12], later entered the literature as an atherogenic indicator, a marker of vascular damage. Recent studies have shown an association between TGI and various cardiovascular diseases, including coronary artery calcification, carotid atherosclerosis, as well as an association with ocular vascular diseases.[13-16] However, to our knowledge, no studies in the existing literature have investigated the relationship between TGI and CSCR. The aim of this study is to compare TGI levels in patients with acute CSCR with those with chronic CSCR and a control group and to determine its predictive value.

MATERIALS AND METHODS

Sample

This retrospective study took place at the Retina Section of the Department of Ophthalmology, Necmettin Erbakan University Faculty of Medicine, Data was collected from medical records and laboratory results of newly diagnosed or regularly followed-up acute and chronic CSCR patients between January 2023 and March 2024. These records were retrieved from the hospital information system to obtain the intended data. to obtain the intended data. Approval was obtained from the Ethics Committee of Necmettin Erbakan University (No: 2024/5050) before the research, which was conducted following the Declaration of Helsinki. The study included 20 patients over 18 years of age diagnosed with acute CSCR (Acute CSCR group), 20 patients followed up with a diagnosis of chronic CSCR (Chronic CSCR group), and 40 age- and sex-matched participants who met the inclusion criteria (Control Group).

The study included three groups: acute CSCR, chronic CSCR, and a control group. The acute CSCR group consisted of untreated patients with symptoms like central scotoma, metamorphopsia, and micropsia, along with localized serous retinal detachment (SRD) on fundus exam and OCT. These patients also had focal leaks and smokestack or ink blot leakage on fluorescein angiography (FFA). The chronic CSCR group included patients with symptoms lasting more than 6 months, showing widespread SRD, retinal pigment epithelial changes, and multifocal leakage points on FFA. The control group was made up of healthy individuals without CSCR, matched by age and sex. Diagnosis of both acute and chronic CSCR was confirmed by a retinal specialist.

Those with any systemic disease such as hypertension, diabetes, cardiovascular disease (congestive heart failure, coronary artery disease), stroke, renal failure, hepatic disorders; those with lipid metabolism disorders such as familial dyslipidemia; those with smoking and alcohol use;

those with any retinal disease other than CSCR (age-related macular degeneration, diabetic retinopathy, retinal vein occlusion) and other ocular pathologies (such as glaucoma, uveitis, history of ocular trauma, ocular infection and corneal pathologies); those using any medication other than chronic CSCR treatment, including steroid-containing or lipid-lowering drugs; those with a history of Covid-19; those with a history of malignancy and vasculitis; and those with a history of any intraocular surgery were excluded from this study.

Procedure

Participants were selected on the basis of a comprehensive ophthalmological examination, including measurement of best corrected visual acuity, intraocular pressure, and detailed anterior segment and fundus examinations. Optical coherence tomography (OCT) and fluorescein angiography findings were reviewed as part of the inclusion criteria.

Laboratory data including fasting plasma glucose and triglyceride levels were also required. The triglyceride-glucose index (TGI) was determined by following formula: Ln[fasting triglycerides (mg/dL) × fasting plasma glucose (mg/dL) / 2], with calculations performed in Microsoft Excel.[12] Data from blood samples collected in the morning after 12 hours of fasting were recorded for all participants.

Statistical analysis

IBM SPSS statistics software version 29.0 (IBM Corp., Armonk, NY, USA) was used for statistical analysis of the data. Normality was checked for each continuous variable using the Kolmogorov–Smirnov test. Normally distributed continuous variables were expressed as mean ± standard deviation (SD), non-normally distributed continuous variables were expressed as median [25th, 75th percentiles], and categorical variables were expressed as count (n) and percent (%). Categorical variables were compared with Pearson's Chi-squared test. The Kruskal Wallis test was used to compare non-normally distributed continuous variables, and post hoc analysis was performed with Tamhane's T2 correction for multiple comparisons. One-way ANOVA test was used to compare normally distributed continuous variables, and post hoc analysis was performed with Bonferroni correction for multiple comparisons. The receiver operating characteristic (ROC) curve was used to calculate the optimal TGI cut-off value and the sensitivity and specificity of this value between control and acute CSCR groups. A two-sided p value <0.05 was considered statistically significant.

RESULTS

The mean age for the participants in the acute CSCR group was 48.4±7.7 years, in the chronic CSCR group it was 49.6±10.0 years, and in the control group it was 46.3±11.9 years. In terms of gender, 12 out of 20 participants (60%) in both the acute and chronic CSCR groups were male, while 8 (40%) were female. In the control group, 22 of 40 participants (55%) were male and 18 (45%) were female. No significant differences were found between the groups regarding age (p=0.486) or gender (p=0.903) (Table 1).

Table 1. Demographical characteristics of the participants in the control, acute CSCR and chronic CSCR groups.

Variable	Control (n=40)	Acute CSCR (n=20)	Chronic CSCR (n=20)	*P value*
Age, years, mean±SD	46.3±11.9	48.4±7.7	49.6±10.0	0.486*
Gender				
Male (n, %)	22 (55%)	12 (60%)	12 (60%)	0.903**
Female (n, %)	18 (45%)	8 (40%)	8 (40%)	

***** Tested using one-way ANOVA test.
****** Tested using Pearson's Chi – squared test

A subsequent investigation of the laboratory parameters revealed that there were no statistically significant variations between the groups with regard to glucose, HDL, and LDL levels (p=0.884, p=0.582, p=0.449). However, triglyceride levels were significantly different: 98.50 [92.00, 114.75] mg/dl in the control group, 152.50 [119.25, 191.25] mg/dl in the acute CSCR group, and 106.00 [80.25, 153.50] mg/dl in the chronic CSCR group (p<0.001). Post-hoc analysis revealed a significant increase in triglyceride levels in the acute CSCR group compared to the control group (p<0.001). VLDL levels were

17.50 [14.75, 26.75] mg/dl in the control group, 30.00 [23.00, 39.00] mg/dl in the acute CSCR group and 20.50 [13.25, 30.00] mg/dl in the chronic CSCR group (p=0.003). VLDL levels were significantly elevated in the acute CSCR group than in the control group (p=0.003), but there were no significant differences between the chronic CSCR group and either of the other two groups (p=0.901 and p=0.055, respectively). Total cholesterol levels were 179.88±20.39 mg/dl in the control group, 199.05±33.78 mg/dl in the acute CSCR group, and 185.95±31.28 mg/dl in the chronic CSCR group. The acute CSCR group exhibited significantly elevated total cholesterol levels in comparison with the control group (p=0.035), but no significant differences were found between the chronic CSCR group and either the control or acute CSCR groups (p=1.000 and p=0.391, respectively).

The TGI values were 8.48±0.29 in the control group, 8.91±0.34 in the acute CSCR group, and 8.59±0.40 in the chronic CSCR group. Multiple comparisons revealed that the TGI in the acute CSCR group was significantly higher than both the chronic CSCR and control groups, but no significant difference was observed between the chronic CSCR and control groups (p<0.001 for control vs. acute CSCR; p=0.703 for control vs. chronic CSCR; p=0.011 for acute vs. chronic CSCR) (Table 2). The optimal TGI cut-off value for distinguishing between the control and acute CSCR groups was 8.74, with 75% sensitivity and 85% specificity (AUC: 0.835, p<0.001, 95% CI 0.719-0.951) (Figure 1).

Table 2. Comparison of the laboratory findings and Triglyceride-Glucose Index (TGI) between the control, acute CSCR and chronic CSCR groups.

	Control	**Acute CSCR**	**Chronic CSCR**	***P value***	***P Value (Multiple Comparisons)***		
					Control *vs* Acute CSCR	Control *vs* Chronic CSCR	Acute *vs* Chronic CSCR
Glucose, mg/dl, median [25th, 75th]	94.00 [91.00, 100.75]	96.00 [87.25, 103.75]	93.50 [89.25, 102.00]	0.884*			
Triglyceride, mg/dl, median [25th, 75th]	98.50 [92.00, 114.75]	152.50 [119.25, 191.25]	106.00 [80.25, 153.50]	<0.001*	<0.001†	0.548†	0.055†
HDL, mg/dl, median [25th, 75th]	45.00 [41.00, 56.00]	42.50 [39.25, 52.75]	48.50 [36.50, 53.00]	0.582*			
LDL, mg/dl, median [25th, 75th]	112.50 [104.50, 122.00]	118.00 [94.50, 156.75]	106.50 [97.25, 130.00]	0.449*			
VLDL, mg/dl, median [25th, 75th]	17.50 [14.75, 26.75]	30.00 [23.00, 39.00]	20.50 [13.25, 30.00]	0.003*	0.003†	0.901†	0.055†
Total Cholesterol, mg/dl, mean±SD	179.88±20.39	199.05±33.78	185.95±31.28	0.041**	0.035††	1.000††	0.391††
TGI, mean±SD	8.48±0.29	8.91±0.34	8.59±0.40	<0.001**	<0.001††	0.703††	0.011††

* Statistical significance in the Kruskal Wallis test
** Statistical significance in the one-way ANOVA test
† Multiple comparisons with Tamhane's T2 adjustment
†† Multiple comparisons with Bonferroni adjustment
Bold: statistically significant results

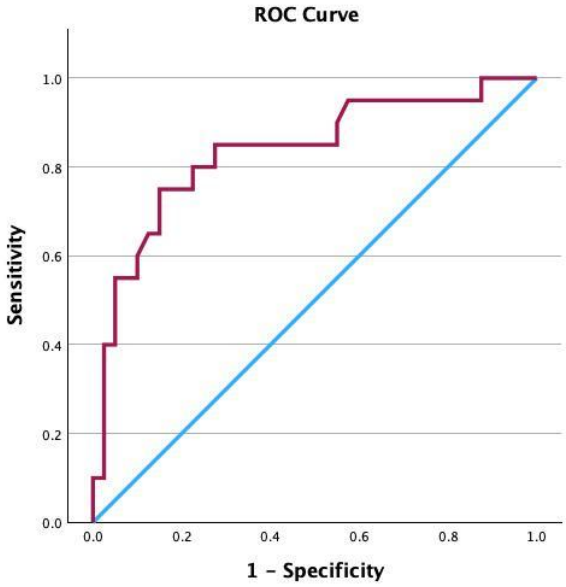

Figure 1. Receiver operating characteristics (ROC) curve analysis of Triglyceride-Glucose Index (TGI) between acute CSCR and control groups (Optimal ROC cutoff value: 8.74 with 75% sensitivity and 85% specificity, AUC: 0.835, p<0.001, 95%CI 0.719-0.951).

DISCUSSION

The findings of the present study demonstrated that there was a significant increase in TGI levels in patients diagnosed with acute CSCR when compared to both the chronic CSCR and control groups. The interesting aspect of our study is that, unlike acute CSCR, this index did not increase significantly in the chronic form of the disease compared to the control group. The cut-off value for TGI was determined as 8.74 with 75% sensitivity and 85% specificity in the differentiation of acute CSCR and control groups. This means that approximately 75% of patients diagnosed with acute CSCR had a TGI value above 8.74, whereas only 15% of the control group exceeded this threshold. These results suggest that TGI, which is an atherogenic index, is particularly associated with the acute form of CSCR. The results we obtained from our study suggest that acute CSCR may be linked to cardiovascular disease and that TGI may be a potential indicator for acute CSCR. These results suggest that TGI, an atherogenic index, is strongly associated with the acute form of CSCR. Our findings also indicate that acute CSCR may be linked to cardiovascular disease, with TGI potentially serving as a predictive marker for this condition. To the best of our knowledge, our study is the first to investigate the association between CSCR and TGI.

The exact mechanisms behind CSCR are not fully understood, but it is generally accepted that increased permeability of the choroidal capillaries plays a central role in the disease. This hyperpermeability causes dysfunction of the retinal pigment epithelium and results in serous retinal detachment[6]. In addition, psychological stress and personality traits are other factors that contribute to the development of CSCR. A cross-sectional study by Yannuzzi found that Type A behaviour was more common in patients with CSCR than in those with other eye diseases. It is well known that psychological stress can lead to increased blood pressure, heart rate, increased levels of free fatty acids and lipids, increased platelet aggregation, changes in blood clotting, increased cortisol secretion and activation of the sympathetic nervous system[17]. These mechanisms cause hypercortisolism and impaired fibrinolysis, creating a predisposition to microthrombus formation, and ultimately may have a negative effect on the cerebrovascular system, as well as accelerate choroidal microcirculatory disturbances in CSCR[18]. Haimovici et al. found that about half of patients with active acute CSCR had elevated 24-hour urinary cortisol levels. In addition, plasma cortisol

levels were found to be more than 50% higher on average in CSCR patients[19]. Elevated glucocorticoid levels can lead to platelet aggregation, vasoconstriction and an increased tendency to clot[20].

Numerous studies have explored the link between CSCR and cardiovascular or atherosclerotic diseases. Hsu et al. found that CSCR is associated with an increased risk of cardiovascular disease in middle-aged men and recommended routine cardiovascular screenings for this group[21]. Tittl et al compared 230 CSCR patients with 230 controls and showed that hypertension is one of the independent risk factors for the development of CSCR, and it has been reported that hypertension is associated with CSCR[22]. Huang et al found that the incidence of CSCR development was increased in patients with heart failure and that heart failure was an independent indicator for CSCR[23].

In addition, several studies have shown that the physiological fibrinolysis mechanism is disturbed in patients with CSCR. This explains the localized occlusion of the choroidal artery and choriocapillaris, which is one of the pathophysiological causes of CSCR. Ijima et al found elevated serum levels of plasminogen activator inhibitor-1 (PAI-1) in patients with CSCR[24]. High PAI-1 levels suggest that the imbalance between clotting and coagulation may increase the predisposition to coronary heart disease in patients with CSCR. The association between elevated serum PAI-1 levels and PAI-1 gene polymorphism with coronary heart disease (CHD) supports this notion[25]. Additionally, a recent study demonstrated that reducing serum PAI-1 levels through low-dose acetylsalicylic acid in CSCR patients led to quicker visual recovery and fewer recurrences compared to those who were not treated[16].

Mineralocorticoid receptor activation is another shared mechanism between CSCR and cardiovascular disease. In heart failure, reduced ventricular function leads to decreased blood flow, triggering the renin-angiotensin-aldosterone system. Aldosterone, a key mineralocorticoid, contributes to endothelial dysfunction in coronary vessels. Increased vascular permeability due to mineralocorticoid receptor activation leads to fluid accumulation, worsening organ congestion[26]. The mineralocorticoid receptor is also found in the neuroretina and choroid, and fluid accumulation associated with this receptor makes it important in the pathogenesis of CSCR[27].

TGI is a parameter that has been extensively studied in recent years for its association with cardiovascular and atherosclerotic disease. In particular, many studies have shown its association with acute cardiovascular events and ocular vascular disorders. Wang et al. demonstrated that TGI could serve as a useful marker in the evaluation of major cardiovascular adverse events in patients with acute coronary syndrome and diabetes[28]. Mao et al found that TGI may be an independent determinant of the severity of non-ST acute coronary artery disease and cardiovascular outcomes[13]. In another study by Sirakaya HA et al, it was stated that higher TGI index levels are linked with central retinal artery occlusion (CRAO) risk, making TGI a potential predictive biomarker for this condition[29]. Liu et al found that TGI was strongly associated with recurrence and stroke-related deaths in acute ischemic stroke patients with type 2 diabetes mellitus, and that this index may be a potential predictor of clinical outcome in these patients[30].

The limitations of this study are twofold. Firstly, the sample size is small. Because the strict exclusion criteria that eliminated patients with systemic diseases resulted in a study population consisting of healthy SSCR patients. Secondly, and perhaps more significantly, the study was conducted at a single centre. Furthermore, the retrospective design and lack of long-term follow-up for the participants are important factors to consider.

In conclusion, our research revealed that TGI levels were significantly elevated in individuals with acute CSCR compared to those with chronic CSCR and the control group. It is evident that TGI can be readily measured through routine blood tests, thus rendering it a potentially valuable indicator for acute CSCR. In clinical settings, it may be advantageous to screen patients with acute CSCR for underlying cardiovascular conditions. Further studies involving larger, prospective cohorts will be necessary to confirm these findings.

Author Contributions: Concept/Design : AOG, NT, SB; Data acquisition: MA, GS, RO; Data analysis and interpretation: MA, GŞ, RO; Drafting manuscript: AOG, NT, SB; Critical revision of manuscript: MA, GS, RO; Final approval and accountability: AOG, NT, SB, MA, GŞ, RO; Technical or material support: AOG; Supervision: SB; Securing funding (if available): n/a.
Ethical Approval: This study was approved by the Clinical Research Ethics Committee of the Necmettin Erbakan University (Decision No: 2023/4695).
Peer-review: Externally peer-reviewed.
Conflict of Interest: The authors declare that there is no conflict of interest.
Financial Disclosure: The work has been done without receiving any financial support from any third party.

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