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Evaluation of the triglyceride-glucose index in coronary slow flow patients

Aim	Triglyceride glucose index (TyG index) is a surrogate marker for insulin resistance. No studies have evaluated the TyG index in patients with coronary slow flow phenomenon (CSFP). We investigated TyG index values in CSFP and evaluated whether it had a predictive value for the diagnosis of CSFP.
Material and Methods	132 CSFP patients and 148 subjects with normal coronary arteries were included in the study. Thrombolysis in myocardial infarction frame count (TFC) of each patient was calculated. Demographic, clinical features, information regarding medication use and biochemical variables of the patients were obtained from hospital records.
Results	TyG index of patients with CSFP and normal coronary flow were 9.02 (8.65–9.42) and 8.69 (8.39–9.18), respectively (p<0.001). Mean TFC showed positive correlation with the TyG index, glucose, triglyceride, and hemoglobin concentration (r=0.207, r=0.138, r=0.183, r=0.179 and p<0.001, p=0.020, p=0.002, p=0.003, respectively) and negative correlation with high density lipoprotein-cholesterol (HDL–C) level (r=–0.292, p<0.001). Receiver operating characteristic curve analysis of TyG index demonstrated that the value of 8.68 predicted CSFP curve analysis of TyG index demonstrated that the value of 8.68 predicted CSFP with sensitivity of 74.2% and specificity of 58.6%. In multivariate logistic regression analysis, HDL–C, hemoglobin and the TyG index were the independent predictors of CSFP.
Conclusions	Our findings supported the hypothesis that insulin resistance play role in CSFP.
Keywords	Triglyceride-glucose index; coronary slow flow; insulin resistance
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Introduction

Coronary slow flow phenomenon (CSFP) is an angiographically diagnosed entity that is characterized by delay in the progression of contrast material in the coronary tree without significant coronary artery narrowing. CSFP has been found in between 1 to 7% of the patients who underwent coronary angiography, and it has been reported in up to 34% of patients who have anatomically normal coronary arteries [1, 2]. These patients are more likely males, smokers, obese, and exhibit features of metabolic syndrome [3]. CSFP can lead to clinical manifestations of myocardial ischemia that might negatively impact patients' quality of life. Current guidelines do not classify CSFP into any kind of chronic coronary syndromes [4].

Pathophysiologic pathways underpinning CSFP still remain poorly understood. Histopathological fibromuscular hyperplasia, endothelial cell degeneration and medial hypertrophy of small resistance vessels have been encountered in CSFP patients [3, 5]. Moreover, endothelial dysfunction, subclinical atherosclerosis, inflammation, increased oxidative stress and rheological abnormalities with resultant disturbed coronary flow support the hypothesis that CSFP is a generalized process [6–8].

Endothelial dysfunction has been widely recognized as a contributor to hypertension, obesity, metabolic syndrome and atherosclerosis, which are also linked to insulin resistance (IR) syndrome. In addition to its metabolic effects, insulin stimulates the production of nitic oxide and regulates the secretion of endothelin-1 from vascular endothelial cells [9]. IR, the reduced response to the metabolic actions of insulin, starts a vicious cycle of metabolic and cardiovascular diseases. CSFP has been found to be correlated with the presence of IR and impaired glucose tolerance; thus pointing to a common pathophysiological mechanism between coronary vascular dysfunction and CSFP [10]. In clinical practice, the homeostasis model assessment of insulin resistance (HOMA-IR) method is the most popular method for assessing of insulin resistance [11]. However HOMA-IR is relatively expensive and requires measuring both serum insulin and glucose concentrations. This has led researchers to search for alternative methods.

The triglyceride glucose index (TyG index) is based on serum triglyceride (TG) and glucose concentrations, and is an easily obtainable and reliable marker for IR. The prognostic value of the TyG index has been shown in heart failure, acute coronary syndromes, atherosclerosis and hypertension [12–

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14]. Starting from the point that endothelial dysfunction is one of the mechanism underlying the pathogenesis of CSFP, we hypothesized that the TyG index should be increased in CSFP patients. As such, we investigated TyG index values in patients with CSFP and evaluated whether it had a predictive value for the diagnosis of CSFP.

Material and Methods

Clinical and angiographic data of patients who underwent angiography between January 2017 and January 2022 were retrospectively reviewed. Patients who had CSFP and were between the ages of 18 and 85 yrs were considered appropriate for the study. Patients with acute coronary syndrome, coronary plaques, coronary ectasia, coronary calcification, previous percutaneous coronary intervention/coronary artery bypass graft surgery, cardiomyopathies, heart failure, acute infection, systemic inflammatory or rheumatological diseases, hematological diseases, malignancy, hepatic and/or renal dysfunction, thyroid abnormalities, triglyceride-lowering drugs use (fenofibrate, omega-3 fatty acids), or congenital heart disease were excluded from the study. A total of 5 812 coronary angiography recordings were screened. 1255 patients with acute coronary syndrome, 1127 patients with coronary artery disease who were suggested to undergo percutaneous coronary intervention, 237 patients with previous coronary artery bypass graft operation, and 633 patients with previous percutaneous intervention were excluded from the study. Among the remaining 2 560 patients, 169 patients had CSFP. One hundred thirty-two CSFP patients and an additional 148 age and sex-matched subjects with normal coronary arteries were included in the study.

A normal coronary artery was defined as complete absence of luminal narrowing at coronary angiography. Clinical indication for coronary angiography was the presence of an abnormal exercise stress test and/or results of myocardial perfusion scanning. We tried to exclude any form of coronary artery disease by excluding patients who had coronary plaques and obstructive coronary artery disease and by including only patients with CSFP and completely normal coronary angiography results. We also specifically assessed the patients' complaints. Patients who had typical angina pectoris and/or angina equivalents were also excluded so as to exclude cardiac syndrome x. In this way, we tried to include patients who had false positive results on cardiac stress tests or myocardial perfusion scans. We believe that the control group of CSFP negative patients was composed of patients who had false-positive results of cardiac exercise stress tests or myocardial perfusion scans. Demographic, clinical features, information regarding medication use and biochemical variables of the patients were obtained from hospital records. The local ethical committee was approved the study and it was performed in conformity with the declaration of Helsinki. All patients gave informed consent before study enrollment.

After an overnight fast, blood samples were collected from antecubital fossa by venipuncture. Biochemical data including urea, creatinine, fasting glucose, total cholesterol (TC), high density lipoprotein cholesterol (HDL–C), low density lipoprotein cholesterol (LDL–C), TG, C-reactive protein (CRP) and complete blood count parameters were recorded. TyG index was calculated as ln (fasting triglyceride × fasting glucose/2) [15]. Diabetes mellitus (DM) was described as fasting glucose ≥126 mg/dl or taking antidiabetic medication. Hypertension (HT) was diagnosed when a patient's systolic and/or diastolic blood pressures were greater than 140 and 90 mmHg, respectively or use of antihypertensives. Hyperlipidemia (HL) was described as TC ≥200 mg/dl or taking anti-lipidemic medication.

Coronary angiographic imaging of the patients were performed using a Siemens Axiom Artis Zee Cath Lab (Munich Germany) system. The right common femoral arterial access was preferred, and a 6F catheter was inserted into the coronary system with the Judkins technique. Multiplane images of each coronary artery were recorded. Thrombolysis in myocardial infarction (TIMI) flow grading, a semiquantitative grading system ranging from 0 (no flow) to 3 (normal flow), was used for the assessment of epicardial coronary flow. TIMI flow grade 2 patients, who had complete but slow/sluggish filling of distal coronary bed, were diagnosed with CSFP. Since the TIMI flow grading system was subjected to inter-/intra-observer variability, corrected TIMI frame count (TFC) was also calculated for each of the patients [16]. It was calculated from the number of frames necessary to fill the distal coronary landmarks of relevant coronary arteries. The point at which radiopaque material extends across not less than 70% of the arterial lumen with antegrade motion was defined as first frame, whereas the last frame was the point at which radiopaque material enters the standard distal mark of the artery. Distal coronary marks for left anterior descending artery (LAD) and circumflex artery (Cx) were the distal bifurcation of the LAD and the obtuse marginal branch or main Cx, respectively. The distal coronary mark of the right coronary artery (RCA) was defined as the first branch of the posterolateral artery. The TFC of the LAD was corrected by dividing the acquired result by 1.7. Previous studies stated that the normal TFC of the LAD, Cx and RCA were 36.3±2.6, 22.3±4.1 and 20.5±3.0, respectively [16]. Diagnosis of CSFP was made when the TFC of an artery had a value of greater than 2 standard deviations (SD) than the normal value. Mean TFC was calculated from adding the TFC of the LAD, Cx and RCA and dividing the result by 3.

Statistical analysis

Normality of data was evaluated with the Kolmogorov-Smirnov test. Data with normal or non-normal distribution

Table 1. Comparisons of clinical and biochemical variables of the CSFP (+) and CSFP (–) patients

Variable	CSFP (+)	CSFP (–)	p
Age (yrs)	57 (48–64)	60 (51–66)	0.071
Gender, n	–	–	0.320
Male	80 (60.3)	75 (65.2)	–
Female	52 (39.7)	73 (34.8)	–
Smoking	92 (69.7)	75 (50.7)	0.001
Diabetes mellitus	30 (22.7)	47 (31.8)	0.091
Hypertension	83 (62.9)	80 (54.1)	0.135
Hyperlipidemia	56 (42.4)	51 (34.5)	0.171
Body mas index (kg/m²)	29.38 (26.13–33.01)	30.26 (27.24–34.54)	0.087
HgbA1c	5.7 (5.4–6.3)	5.6 (5.2–6.2)	0.080
Glucose (mg/dl)	107.5 (97.00–125.00)	99 (91–117)	0.005
GFR (ml/min/1.73 m²)	94 (81.5–103)	96 (87–107)	0.211
LDL–C (mg/dl)	126 (93–150)	131 (97.5–152.5)	0.531
Triglyceride (mg/dl)	144 (105–205)	118 (89–165)	<0.001
HDL–C (mg/dl)	45 (38-52)	54 (46-59)	<0.001
Albumin (g/dl)	4.40 (4.10–4.60)	4.30 (4.00–4.47)	0.126
C-reactive protein (mg/l)	0.34 (0.18–0.52)	0.30 (0.20–0.50)	0.349
Hemoglobin (g/dl)	14.10 (13.00–15.00)	13.5 (12.35–14.20)	<0.001
Neutrophil (10^3/μL)	4.46±1.34	4.27±1.17	0.226
Platelet (10^9/l)	236.45±58.63	243.51±67.07	0.351
Lymhocyte (10^3/μL)	2.21 (1.812.77)	2.23 (1.71–2.82)	0.469
Monocyte (10^3/μL)	0.58±0.19	0.56±0.15	0.017
Red cell distribution width (%)	13.45 (12.9–14.3)	13.5 (13.0–14.4)	0.487
Platelet distribution width (fL)	13.0 (11.6–15.45)	12.1 (11.0–14.6)	0.038
Men platelet volume (fL)	10.4 (9.8–11.2)	10.4 (9.7–11.2)	0.850
LAD-TFC	35 (26–38)	21.5 (20–22)	<0.001
CX-TFC	24 (22–33)	20 (18–21)	<0.001
RCA-TFC	32 (23–35)	21 (19–23)	<0.001
Mean TFC	20.66 (19.66–21.66)	29.00 (26.75–32.25)	<0.001
TyG index	9.02 (8.65–9.42)	8.69 (8.39–9.18)	<0.001

Data are number (%), mean±SD, or median (IQR). GFR, Glomerular filtration rate; LDL–C, Low density lipoprotein cholesterol; HDL–C, High density lipoprotein cholesterol; LAD, Left anterior descendant coronary artery; CX, Circumflex coronary artery; RCA, Right coronary artery; TFC, TIMI frame count; TyG index; Triglyceride glucose index..

are expressed as mean±SD or median IQR, respectively. Categorical data are expressed as number and percentages. Comparison of patients who had CSFP with patients who had normal coronary arteries was conducted by independent samples-t test or Mann-Whitney U test. Categorical comparisons were made by the Chi-square test. The correlation of mean TFC with other parameters was examined by Spearman correlation analysis. Receiver operating characteristic (ROC) curve analysis was conducted to determine cut-off value of the TyG index for predicting CSFP. Predictors of CSFP were identified by logistic regression analysis. Quade's ANCOVA test was conducted to compare the TyG index between groups where smoking served as covariate.

Results

There were no differences between two CSFP (+) and CSFP (–) patient groups with respect to age, gender, body mass index, prevalence of DM, HT and HL. Likewise, antihypertensive, anti-diabetic and statin use did not differ between the two groups. Smoking percentage of CSFP patients was significantly higher than that of controls. When the analysis was conducted with smoking as a covariate, the TyG index was still significantly higher in the CSFP patients (F=10.921; p=0.001). Among laboratory parameters, glucose, TG, CRP, hemoglobin concentrations and monocyte count were significantly higher in CSFP patients. As expected, TFC of patients with CSFP were increased in comparison to patients with normal coronary flow. TyG index of patients with CSFP and normal coronaries were 9.02 (8.65–9.42) and 8.69 (8.39–9.18), respectively (p<0.001).

Tables 1 and 2 show the clinical and biochemical variables and medical treatment of the two groups, Mean TFC

Table 2. Comparison of medical treatment of the CSFP (+) and CSFP (–) patients

Medical Treatment	CSFP (+)	CSFP (–)	p
ACEI/ARB	72 (54.5)	71 (48.0)	0.272
Beta blocker	65 (49.2)	62 (41.9)	0.217
Ca channel blocker	38 (28.8)	43 (29.1)	0.961
Diuretic use	38 (28.8)	98 (26.4)	0.649
Statin	58 (43.9)	52 (35.4)	0.144
Acetylcalycilic acid	61 (46.2)	79 (53.4)	0.231
Oral anticoagulant	18 (13.6)	6 (4.1)	0.004
Antidiabetic	28 (21.2)	34 (23.0)	0.051

Data are number (%), ACEI, Angiotensin converting enzyme inhibitor; ARB, Angiotensin receptor blocker.

Table 3. Correlation of mean TFC with other variables

Variable	r	p
TyG index	0.407	<0.001
Age	-0.100	0.094
Glucose	0.138	0.020
HgA1c	0.104	0.084
Hemoglobin	0.179	0.003
LDL-C	0.054	0.370
Triglyceride	0.183	0.002
HDL-C	-0.292	<0.001
C-reactive protein	0.119	0.062
Neutrophil	0.084	0.161
Glomerular filtration rate	-0.074	0.219

Abbreviations were same as Table 1.

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Table 4. Univariate and multifvariate logistic regression for prediciton of CSFP

Variable	Univariate logistic regression			Multivariate logistic regression		
	OR	95% CI	p	OR	95% CI	p
Age	0.980	0.959–1.003	0.094	–	–	–
Body mass index	0.966	0.922–1.012	0.147	–	–	–
HgA1c	1.055	0.894–1.244	0.528	–	–	–
Hemoglobin	1.375	1.160–1.630	<0.001	1.258	1.052–1.505	0.012
Glucose	1.003	0.998–1.008	0.242	–	–	–
GFR	0.990	0.977–1.003	0.128	–	–	–
Triglyceride	1.005	1.002–1.008	0.001	–	–	–
LDL–C	0.999	0.993–1.005	0.684	–	–	–
HDL–C	0.959	0.938–0.980	<0.001	0.957	0.946–0.988	0.002
TyG index	2.180	1.447–3.286	<0.001	1.857	1.207–2.858	0.005

Abbreviations were same as Table 1.

showed weak positive correlation with hemoglobin, glucose, and triglyceride concentrations, it had a moderate positive correlation with TyG index and a weak negative correlation with HDL–C level. It did not show any correlation with age, HgA1c, LDL–C, CRP concentrations, glomerular filtration rate and neutrophil count (Table 3).

ROC curve analysis of TyG index demonstrated that the value of 8.68 predicted CSFP with sensitivity of 74.2% and specificity of 58.6%. When the analysis was done according to the TyG index cut-off value, mean TFC was significantly lower in patients with low values of the TyG index compared to patients who had higher values of TyG index (p=0.023, Figure 1). Figure 2 shows the ROC curve analysis of TyG index for prediction of CSFP (AUC: 0.631, p<0.001, CI 95% 0.566–0.696).

Univariate logistic regression showed that TG, HDL–C, hemoglobin and TyG index were the independent predictors of CSFP. Age, body mass index, HgA1c, glucose and LDL–C did not have any power in predicting the presence of CSFP. Since TyG index and TG level had strong correlation, TG was not included in multivariate logistic regression analysis. In multivariate logistic regression analysis HDL–C, hemoglobin and TyG index were the independent predictors of CSFP (Table 4).

Figure 1. Mean TFC according to TyG index

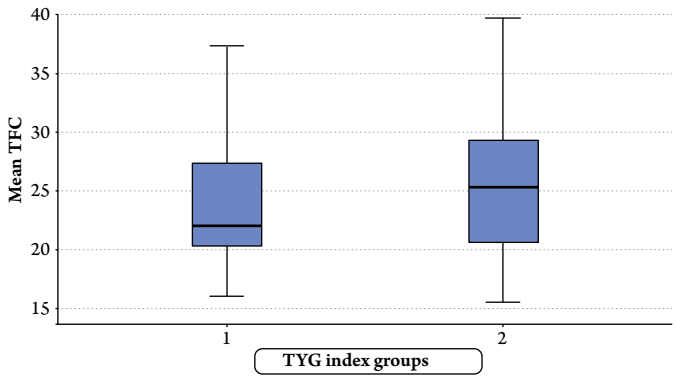
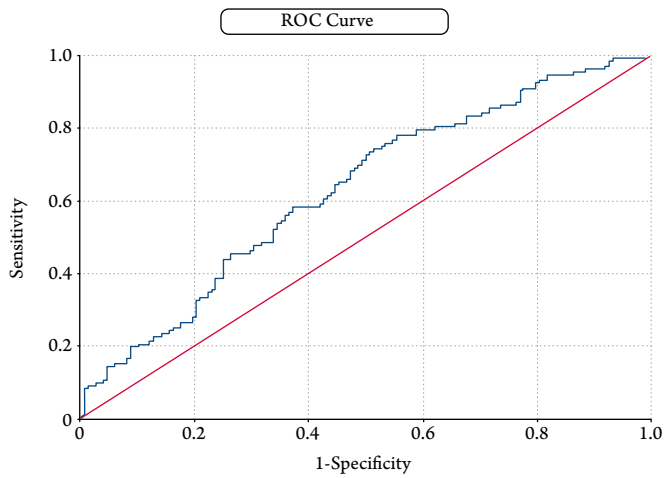


Figure 2. ROC curve of TyG index for prediction of CSFP



Diagonal segmets are produced by ties.

Discussion

The present study demonstrated that the TyG index was increased in CSFP, and it had a moderate positive correlation with mean TFC. In addition, besides hemoglobin and HDL–C, the TyG index was an independent predictor for the presence of CSFP.

CSFP is among one of the causes of ischemic symptoms and its pathogenesis is still not well understood. CSFP could be thought of as a specific syndrome with specific characteristics and treatment modalities. Vascular endothelial dysfunction and subclinical atherosclerosis have been shown to be related to CSFP [7]. Basically, coronary arterial endothelial dysfunction has been considered as an early form of atherosclerosis.

CSFP patients had decreased adiponectin concentrations and paraoxonase activities, as well as decreased flow mediated dilatation of the brachial artery, pointing to impairment of endothelial function [17]. Since insulin resistance is central to pathogenesis of endothelial dysfunction, evaluation

of insulin resistance has attracted the attention of many researchers. A recent study that measure HOMA-IR in patients with CSFP found a positive correlation of TFC with HOMA-IR [10]. Binak et al. performed oral glucose tolerance tests on patients with CSFP and found higher oral glucose test values in these patients [18]. On the other hand, Yazici et al. did not find any association between serum glucose and insulin concentrations and CSFP. However, they only measured baseline insulin concentration and IR was not investigated [19].

The TyG index and HOMA-IR are interrelated parameters that are representative of IR; they reflect IR in muscle and in liver, respectively [20, 21]. A Korean study showed that the TyG index was a better predictor of arterial stiffness than HOMA-IR [22]. Hence, Irace et al. suggested that the TyG index might be a more useful marker of coronary and carotid atherosclerosis [23]. In the current study, the TyG index was significantly elevated in patients with CSFP, and it was an independent predictor of CSFP.

Other findings of the present study merit some comment. Almost two thirds of the CSFP patients were males, indicating a male predominance. This result was in accordance with previous reports that also showed increased prevalence of CSFP in males [2]. Although the pathophysiological mechanisms in which male gender and CSFP are interrelated have not been determined; hormonal changes and stress factors might be some of the underlying factors behind this relationship.

Data regarding the biochemical parameters in CSFP patients are controversial. Some studies found significant differences in biochemical parameters, whereas others did not find any differences [24]. Although mean values of hemoglobin, monocyte count and platelet distribution width were within the normal range in both the CSFP (+) and CSFP (–) groups, these variables were significantly higher in patients with CSFP. In the present study, neutrophil count and CRP were higher and glomerular filtration rate lower in the CSFP group than in the control group, but these differences did not reach statistical significance. Altun et al. showed increased creatinine and hemoglobin concentrations in patients with slow coronary flow, but they did not find any differences in other hematological parameters [25].

Platelet distribution width, a parameter increased during platelet activation, provides information about inflammatory status. Like our results, Seyyed-Mohammadzad reported higher platelet distribution width values in CSFP [26]. We also found significantly higher monocyte counts and lower concentrations of HDL–C in CSFP patients. Monocytes are among the cells that are involved in inflammatory reactions. Activated monocytes produces proinflammatory cytokines and interact with endothelium, after which they differentiate into macrophages and play a role in the atherosclerotic process [27]. Contrary to this, HDL–C exerts its anti-inflammatory activity by counteracting monocyte activation, migration of macrophages and enhancing nitric oxide synthase expression [28] Last but not least, increased hemoglobin concentration, by increasing blood viscosity and flow resistance, might lead in to endothelial dysfunction and CSFP [29].

In conclusion, our findings support the hypothesis that insulin resistance and inflammatory activity play role in CSFP. Moreover, the TyG index was one of the independent predictors for the presence of CSFP that could be used to assess the severity of CSFP. Calculation of the TyG index is simple and could be used easily in clinical practice.

Limitations

This was a single center study with a relatively small sample size. There was no long term follow-up or prognostic evaluation of the patients. Nutritional and exertional habits, which effect the TyG index values were not evaluated.

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REFERENCES

1. Alvarez C, Siu H. Coronary Slow-Flow Phenomenon as an Underrecognized and Treatable Source of Chest Pain: Case Series and Literature Review. Journal of Investigative Medicine High Impact Case Reports. 2018;6:232470961878919. DOI: 10.1177/2324709618789194
2. Sanghvi S, Mathur R, Baroopal A, Kumar A. Clinical, demographic, risk factor and angiographic profile of coronary slow flow phenomenon: A single centre experience. Indian Heart Journal. 2018;70(Suppl 3):S290–4. DOI: 10.1016/j.ihj.2018.06.001
3. Chalikias G, Tziakas D. Slow Coronary Flow: Pathophysiology, Clinical Implications, and Therapeutic Management. Angiology. 2021;72(9):808–18. DOI: 10.1177/00033197211004390
4. Knuuti J, Wijns W, Saraste A, Capodanno D, Barbato E, Funck-Brentano C et al. 2019 ESC Guidelines for the diagnosis and management of chronic coronary syndromes. European Heart Journal. 2020;41(3):407–77. DOI: 10.1093/eurheartj/ehz425
5. Wybraniec MT, Bańka P, Bochenek T, Roleder T, Mizia-Stec K. Small vessel coronary artery disease: How small can we go with myocardial revascularization? Cardiology Journal. 2021;28(5):767–78. DOI: 10.5603/CJ.a2020.0127
6. Tanriverdi H, Evrengul H, Enli Y, Kuru O, Seleci D, Tanriverdi S et al. Effect of Homocysteine-Induced Oxidative Stress on Endothelial Function in Coronary Slow-Flow. Cardiology. 2007;107(4):313–20. DOI: 10.1159/000099068
7. Sezgin AT, Sigirci A, Barutcu I, Topal E, Sezgin N, Ozdemir R et al. Vascular endothelial function in patients with slow coronary flow. Coronary Artery Disease. 2003;14(2):155–61. DOI: 10.1097/00019501-200304000-00008

8. Pekdemir H, Cin VG, Cicek D, Camsari A, Akkus N, Döven O et al. Slow coronary flow may be a sign o diffuse atherosclerosis. Acta Cardiologica. 2004;59(2):127–33. DOI: 10.2143/AC.59.2.2005166
9. Muniyappa R, Chen H, Montagnani M, Sherman A, Quon MJ. Endothelial dysfunction due to selective insulin resistance in vascular endothelium: insights from mechanistic modeling. American Journal of Physiology-Endocrinology and Metabolism. 2020;319(3):E629–46. DOI: 10.1152/ajpendo.00247.2020
10. Metwally YG, Sedrak HK, Shaltout IF. Coronary slow flow in patients with impaired glucose tolerance and insulin resistance. The Egyptian Journal of Internal Medicine. 2020;32(1):20. DOI: 10.1186/s43162-020-00020-1
11. Katsuki A, Sumida Y, Gabazza EC, Murashima S, Furuta M, Araki-Sasaki R et al. Homeostasis Model Assessment Is a Reliable Indicator of Insulin Resistance During Follow-up of Patients With Type 2 Diabetes. Diabetes Care. 2001;24(2):362–5. DOI: 10.2337/diacare.24.2.362
12. Akbar MR, Pranata R, Wibowo A, Irvan, Sihite TA, Martha JW. The association between triglyceride-glucose index and major adverse cardiovascular events in patients with acute coronary syndrome – dose–response meta-analysis. Nutrition, Metabolism and Cardiovascular Diseases. 2021;31(11):3024–30. DOI: 10.1016/j.numecd.2021.08.026
13. Huang R, Wang Z, Chen J, Bao X, Xu N, Guo S et al. Prognostic value of triglyceride glucose (TyG) index in patients with acute decompensated heart failure. Cardiovascular Diabetology. 2022;21(1):88. DOI: 10.1186/s12933-022-01507-7
14. Zheng R, Mao Y. Triglyceride and glucose (TyG) index as a predictor of incident hypertension: a 9-year longitudinal population-based study. Lipids in Health and Disease. 2017;16(1):175. DOI: 10.1186/s12944-017-0562-y
15. Hong S, Han K, Park C-Y. The triglyceride glucose index is a simple and low-cost marker associated with atherosclerotic cardiovascular disease: a population-based study. BMC Medicine. 2020;18(1):361. DOI: 10.1186/s12916-020-01824-2
16. Gibson CM, Cannon CP, Daley WL, Dodge JT, Alexander B, Marble SJ et al. TIMI Frame Count: A Quantitative Method of Assessing Coronary Artery Flow. Circulation. 1996;93(5):879–88. DOI: 10.1161/01.CIR.93.5.879
17. Selcuk H, Selcuk MT, Temizhan A, Maden O, Saydam GS, Ulupinar H et al. Decreased plasma concentrations of adiponectin in patients with slow coronary flow. Heart and Vessels. 2009;24(1):1–7. DOI: 10.1007/s00380-008-1074-5
18. Binak E, Gunduz H, Sahin M, Kurtoglu N, Dindar I. The relation between impaired glucose tolerance and slow coronary flow. International Journal of Cardiology. 2006;111(1):142–6. DOI: 10.1016/j.ijcard.2005.09.007
19. Yazici M, Demircan S, Aksakal E, Sahin M, Meriç M, Dursun I et al. Plasma insulin, glucose and lipid levels, and their relations with corrected TIMI frame count in patients with slow coronary flow. Anatolian journal of cardiology. 2003;3(3):222–6. PMID: 12967888
20. Riboldi BP, Luft VC, de Castilhos CD, de Cardoso LO, Schmidt MI, Barreto SM et al. Glucose and triglyceride excursions following a standardized meal in individuals with diabetes: ELSA-Brasil study. Cardiovascular Diabetology. 2015;14(1):21. DOI: 10.1186/s12933-015-0181-8
21. Tripathy D, Almgren P, Tuomi T, Groop L. Contribution of Insulin-Stimulated Glucose Uptake and Basal Hepatic Insulin Sensitivity to Surrogate Measures of Insulin Sensitivity. Diabetes Care. 2004;27(9):2204–10. DOI: 10.2337/diacare.27.9.2204
22. Lee SB, Ahn CW, Lee BK, Kang S, Nam JS, You JH et al. Association between triglyceride glucose index and arterial stiffness in Korean adults. Cardiovascular Diabetology. 2018;17(1):41. DOI: 10.1186/s12933-018-0692-1
23. 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. International Journal of Clinical Practice. 2013;67(7):665–72. DOI: 10.1111/ijcp.12124
24. Seyyed-Mohammadzad MH, Khademvatani K, Gardeshkhah S, Sedokani A. Echocardiographic and laboratory findings in coronary slow flow phenomenon: cross-sectional study and review. BMC Cardiovascular Disorders. 2021;21(1):230. DOI: 10.1186/s12872-021-02044-z
25. Altun I, Akin F, Kose N, Sahin C, Kirli I. Predictors of slow flow in angiographically normal coronary arteries. International Journal of Clinical and Experimental Medicine. 2015;8(8):13762–8. PMID: 26550323
26. Seyyed-Mohammadzad MH, Khademvatani K, Kerachian A, Eskandari R, Rezaei Y. Slow Coronary Flow Phenomenon and Increased Platelet Volume Indices. Korean Circulation Journal. 2014;44(6):400–5. DOI: 10.4070/kcj.2014.44.6.400
27. Kim K-W, Ivanov S, Williams JW. Monocyte Recruitment, Specification, and Function in Atherosclerosis. Cells. 2020;10(1):15. DOI: 10.3390/cells10010015
28. Bonacina F, Pirillo A, Catapano AL, Norata GD. HDL in Immune-Inflammatory Responses: Implications beyond Cardiovascular Diseases. Cells. 2021;10(5):1061. DOI: 10.3390/cells10051061
29. Huang Q, Zhang F, Chen S, Dong Z, Liu W, Zhou X. Clinical characteristics in patients with coronary slow flow phenomenon: A retrospective study. Medicine. 2021;100(6):e24643. DOI: 10.1097/MD.0000000000024643