Triglyceride-Glucose Index Could be Used to Predict Severity of Coronary Artery Ectasia

Ersan Oflar[1](İD), İsmail Ünğan[2](İD), Cennet Yıldız[1](İD), Güngör İlayda Bostancı Alp[1](İD), Büşra Mavi[1](İD), Fatma Nihan Turhan Çağlar[1](İD), Demet Erciyes[3](İD), Alparslan Şahin[1](İD)

[1] Clinic of Cardiology, Bakırköy Dr. Sadi Konuk Training and Research Hospital, İstanbul, Türkiye
[2] Clinic of Cardiology, Yalova State Hospital, Yalova, Türkiye
[3] Department of Cardiology, Demiroğlu Science University Faculty of Medicine, İstanbul, Türkiye

ABSTRACT

Introduction: Coronary artery ectasia (CAE) refers to a 1.5 fold increase in coronary artery diameter compared to the normal segment. Although the underlying pathophysiological mechanism is not fully elucidated, coronary atherosclerosis is thought to play a role in more than half of the cases. Triglyceride glucose (TyG) index is an indicator of insulin resistance. In the present study, aimed to evaluated TyG index values in CAE cases and find if a predictive value of TyG index existed.

Patients and Methods: Sixty-one patients with CAE (study group) and 64 patients with normal coronary arteries (control group) were included in the study. TyG index was calculated as ln (fasting triglyceride × fasting glucose/2).

Results: There were no differences between the two groups with respect to age, sex, diabetes mellitus prevalence, and biochemical parameters including, white blood cell, platelet, lymphocyte, monocyte counts, hemoglobin, hematocrit, urea, glucose, total cholesterol, high density lipoprotein-cholesterol, triglyceride levels and TyG index value. Twenty-five patients (41.0%) had one-vessel involvement, 24 (39.3%) patients had two-vessel involvement and 12 (19.7%) patients had three-vessel involvement. Right coronary artery was the most commonly affected vessel (11, 44%), followed by left anterior descending artery (8, 35%), left main coronary artery (4, 16%) and circumflex artery (1, 4%). TyG index was statistically significantly higher in patients who had two/three vessel involvement (8.80 ± 0.48 vs 9.14 ± 0.62, p= 0.027). TyG index had statistically significant correlation with the number of ectatic vessels (r= 0.275, p= 0.032). ROC curve analysis showed that TyG index was useful for the prediction of ectasia severity (AUC= 0.653, 95% CI= 0.515-0.792, p= 0.043). TyG index value of 9.16 had 41.7% sensitivity and 84% specificity for prediction of CAE severity.

Conclusion: TyG index, which is easily calculated from biochemical parameters, might be used for the assessment of CAE severity.

Key Words: Triglyceride; glucose; index; coronary arteries; ectasia

Trigliserid Glukoz İndeksi Koroner Arter Ektazi Ciddiyetinin Değerlendirilmesinde Kullanılabilir Bir Ölçektir

ÖZET

Giriş: Koroner arter ektazisi (KAE), normal segmente göre koroner arter çapında 1.5 kat artışı ifade eder. Altta yatan patofizyolojik mekanizma tam olarak aydınlatılmış olmasa bile vakaların yarısından fazlasında koroner aterosklerozun rol oynadığı düşünülmektedir. Trigliserid glukoz (TyG) indeksi insülin direncini gösteren bir belirteçtir. Bu çalışmada KAE olan hastalarda TyG indeks değerlerinin araştırılması amaçlandı.

Hastalar ve Yöntem: KAE olan 61 hasta ile normal koroner arteri olan 64 kontrol çalışmaya alındı. TyG indeksi ln (açlık trigliserid × açlık glukoz/2) formülü kullanılarak hesaplandı.

Bulgular: Her iki grup arasında yaş, cinsiyet, diyabetes mellitus prevalansı, lökosit, trombosit, lenfosit, monosit sayıları, hemoglobin, hematokrit, üre, glukoz, total kolesterol, yüksek yoğunluklu lipoprotein kolesterol, trigliserid seviyeleri ve TyG indeksi değeri açısından fark gözlenmedi. En sık etkilenen damar sağ koroner arterdi (%11, 44), bunu sol ön inen arter (%8, 35), sol ana koroner arter (%4, 16) ve sirkumfleks arter (%1.4) izledi. İki ve üç damar tutulumu gösteren hastaların TyG indeks değerleri kontrol grubuna göre anlamlı olarak yüksekti (8.80 ± 0.48, 9.14 ± 0.62, p= 0.027). TyG indeksinin ektazi gösteren damar sayısı ile korelasyon gösterdiği saptandı (r= 0.275, p= 0.032). ROC eğrisi analizi TyG indeks 9.16 değerinin KAE'sinin %41.7 duyarlılık ve %84 özgüllük ile predikte ettiğini gösterdi. (Eğri altında kalan alan (AUC)= 0.653, %95 güven aralığı= 0.515-0.792, p= 0.043).

Sonuç: TyG indeks KAE ciddiyetini göstermede yararlı olabilecek basit bir biyokimyasal parametredir.

Anahtar Kelimeler: Trigliserid; glukoz; indeks; koroner arter; ektazi

Cite this article as: Oflar E, Ünğan İ, Yıldız C, Bostancı Alp Gİ, Mavi B, Turhan Çağlar FN, et al. Triglyceride-glucose index could be used to predict severity of coronary artery ectasia. Koşuyolu Heart J 2023;26(1):34-39.

Correspondence

Cennet Yıldız

E-mail: cennet_yildiz@live.com
Submitted: 27.10.2022
Accepted: 06.02.2023
Available Online Date: 21.03.2023

INTRODUCTION

Coronary artery ectasia (CAE) is described as 1.5 fold dilatation of coronary artery segment relative to adjacent normal coronary artery[1]. Its prevalence differs according to the diagnostic modality used. Estimated angiographic prevalence of CAE ranges between 1 to 5%; however, an Indian study has reported 10% incidence in patients with ischemic heart disease[2,3]. CAE has occurred with a frequency of 9.9% in Turkish patients having undergone cardiac catheterization. Although pathophysiological mechanisms underlying CAE have not been fully elucidated, almost half of cases has been attributed to coronary atherosclerosis. Other etiologies for CAE include inflammatory and connective tissue diseases, congenital diseases and percutaneous interventions[4-6]. Markis et al. have shown that histopathological changes underlying CAE have similarities with atherosclerosis, suggesting a common mechanism[7]. Atherosclerosis usually begins as vascular thickening without causing stenosis (positive remodeling). Enlargement of the artery mainly occurs towards external elastic lamina giving a false impression of normal luminal diameter[8]. In this regard, CAE could be considered as a form of positive remodeling. Chronic exposure to herbicides, vasodilator substances, anabolic steroid use, alterations of angiotensin converting enzyme gene, abnormal vessel metabolism are other probable causes of CAE[9-11]. Right coronary artery (RCA) is the most commonly affected artery, followed by left anterior descending artery (LAD) and circumflex artery (Cx)[2]. Concomitant coronary artery stenosis is usually present in 80% to 90% of CAE cases, and if it occurs without coronary artery disease, it is called isolated CAE[12,13]. According to its morphology, CAE can be classified into two forms: saccular (longitudinal diameter is less than transverse diameter) or fusiform (longitudinal diameter is greater than transverse diameter). Sluggish flow in the ectatic segments may cause angina pectoris, acute coronary syndrome, thrombus formation, embolization, and congestive heart failure[14,15].

There is ample scientific evidence demonstrating increased risk of atherosclerosis in patients with insulin resistance (IR) even without presence of hyperglycemia[16]. Proinflammatory cytokine secretion, impaired insulin signaling in peripheral tissues, dyslipidemia with resultant endothelial dysfunction could promote formation of atherosclerotic lesions[17]. Since measurement of IR usually requires sophisticated techniques, simpler methods have been sought in clinical practice. Most commonly used method for quantification of IR is homeostasis model assessment -estimated insulin resistance (HOMA-IR), which is relatively expensive and requires measurement of both serum glucose and insulin levels. Hyperglycemia and hypertriglyceridemia are the predominant laboratory abnormalities in insulin resistant patients. Triglyceride glucose index (TyG index), which is calculated from fasting triglyceride and glucose concentrations, has been proposed as a simple and reliable marker for the screening of IR. Its predictive value has been shown in various diseases including acute myocardial infarction, atherosclerosis, hypertension (HT) and diabetes mellitus (DM)[18-21]. To the best of our knowledge, no study has investigated the value TyG index in patients with CAE. Since atherosclerosis plays role in pathogenesis of CAE, we hypothesize that TyG index is increased in patients with CAE. Therefore, the aim of the present study was to evaluate TyG index values in isolated CAE cases and to find if a predictive value of TyG index existed.

PATIENTS and METHODS

In this single-center, retrospective study, we evaluated the coronary angiographies of patients who had undergone diagnostic coronary angiography in our clinic between January 2018 and January 2020. A total of 10.843 coronary angiographic recordings of the patients were screened, 545 patients had both CAE and coronary artery stenosis of more than 50%, 61 patients had isolated CAE. Demographic and clinical characteristics of the patients were obtained from electronic data files. Sixty-one patients with CAE and 64 age and sex-matched controls were included in the study. Study and control groups consisted of patients with CAE and normal coronary arteries, respectively. Since coronary artery stenosis more than 50%, acute coronary syndromes, congestive heart failure, valvular heart disease, malignancy, renal/hepatic failure, inflammatory diseases might have impact on TyG index, these patients were excluded from the study. Local ethics committee approved the study, and it was conducted in accordance with the declaration of Helsinki.

After an overnight fast, blood samples were collected from antecubital fossa using venipuncture method. Biochemical parameters including urea, creatinine, glomerular filtration rate (GFR), total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), triglyceride (TG), white blood cell count (WBC), hemoglobin (Hgb), hematocrit (Hct), platelet count (Plt), red cell distribution width (RDW), lymphocyte and monocyte counts were assessed. TyG index was calculated as ln (fasting triglyceride × fasting glucose/2)[22]. DM was described as fasting glucose ≥126 mg/dL or taking antidiabetic medication. HT was diagnosed when patient's systolic and/or diastolic blood pressures were greater than 140 and 90 mmHg, respectively or use of antihypertensives. Hyperlipidemia was described as TC ≥200 mg/dL or taking anti-lipidemic medication. Diagnosis of CAE was made by coronary angiography. Coronary angiographies of the study population were assessed by two experienced cardiologists blinded to the patient's data. CAE was described as more than 50% dilatation of a coronary segment relative to the normal segment. Mild involvement by CAE was described as diffuse or segmental ectasia of one vessel. If the patient had diffuse involvement of two/three vessels or diffuse involvement of one

vessel with segmental involvement of another, that was considered as severe involvement.

Primary outcome of the study was to evaluate if there was a difference in TyG index values in patients with CAE and normal coronary arteries. Secondary outcomes of the study were to find the incidence of CAE and its correlation with TyG index.

Statistical Analysis

Normality of the data was tested by Kolmogorov-Smirnov test. Parametric and non-parametric variables were expressed as mean ± SD and median (min-max), respectively. Based on previous studies results, we found that the effect size for this study was 0.75. In order to evaluate differences between the two groups with an alpha value of 0.05 and power of 80%, the required sample size was 23 subjects per group. For comparison of two groups, Mann-Whitney U test and independent samples t-test were used. Correlation of TyG index with the number of ectatic vessels was done by Spearman correlation analysis. Receiver operating characteristic (ROC) curve analysis was used to select optimal threshold of TyG index for the prediction of CAE. Independent predictors of CAE severity were assessed by univariate logistic regression analysis. All analyses were conducted by Statistical Package for Social Sciences (version 25, USA).

RESULTS

Mean ages of the study and control groups were 55.5 (28-77) and 54 (23-74) years, respectively (p= 0.538). There were no differences between the two groups with respect to sex, DM prevalence, biochemical parameters including, WBC, Plt, lymphocyte, monocyte counts, Hgb, Hct, urea, glucose, TC, HDL-C, TG levels, and TyG index value. The number of hypertensive patients was found to be higher in the study group as compared to the control group. In addition, RDW, GFR and creatinine levels were higher in the study group than those of the control group (Table 1).

Table 1. Clinical and biochemical characteristic of the groups

Parameter	Control Group (n= 59)	Study Group (n= 61)	p
Age (years)	54 (23-74)	55.5 (28-77)	0.538
Sex			0.331
Female (n, %)	22 (37.3)	17 (27.9)	
Male (n, %)	37 (62.7)	44 (72.1)	
HT (n, %)	22 (34.9)	41 (65.1)	0.001
DM (n, %)	11 (40.7)	16 (59.3)	0.343
Hgb (g/dL)	13.62 ± 1.66	13.49 ± 1.52	0.648
WBC ($10^3/\mu$L)	7.89 (4.11-18.51)	7.52 (3.6-14.4)	0.674
Hct (%)	40.57 ± 4.06	4.17 ± 4.32	0.436
Plt ($\times10^9$/l)	269 (86-529)	252 (160-437)	0.398
RDW (%)	13.2 (11.9-20.7)	13.95 (12.2-22.7)	0.000
Lymphocyte ($10^3/\mu$L)	2.49 (1.22-5.04)	2.11 (0.9-4.4)	0.131
Monocyte ($10^3/\mu$L)	0.57 (0.31-1.09)	0.59 (0.24-1.36)	0.603
Urea (mg/dL)	31 (18-87)	31 (16-100)	0.725
Creatinine(mg/dL)	0.74 (0.42-2.33)	0.87 (0.51-2.07)	0.004
GFR (mL/min/1.73m^2)	97.59 ± 19.58	76.43 ± 19.64	0.001
Glucose (mg/dL)	99 (70-399)	98 (76-284)	0.975
TC (mg/dL)	216.09 ± 47.26	197 ± 42.97	0.074
HDL-C (mg/dL)	44 (22-98)	41 (25-75)	0.224
TG (mg/dL)	138.5 (46-904)	156 (39-824)	0.634
LDL-C (mg/dL)	133 (70-400)	120 (54-210)	0.047
TyG index	8.88 (7.66-12.10)	8.97 (7.71-10.93)	0.714

HT: Hypertension, DM: Diabetes mellitus, Hgb: Hemoglobin, WBC: White blood cell, Hct: Hematocrit, Plt: Platelet, RDW: Red blood cell distribution width, GFR: Glomerular filtration rate, TC: Total cholesterol, HDL-C: High density lipoprotein cholesterol, TG: Triglyceride, LDL-C: Low density lipoprotein cholesterol, TyG: Triglyceride glucose index.

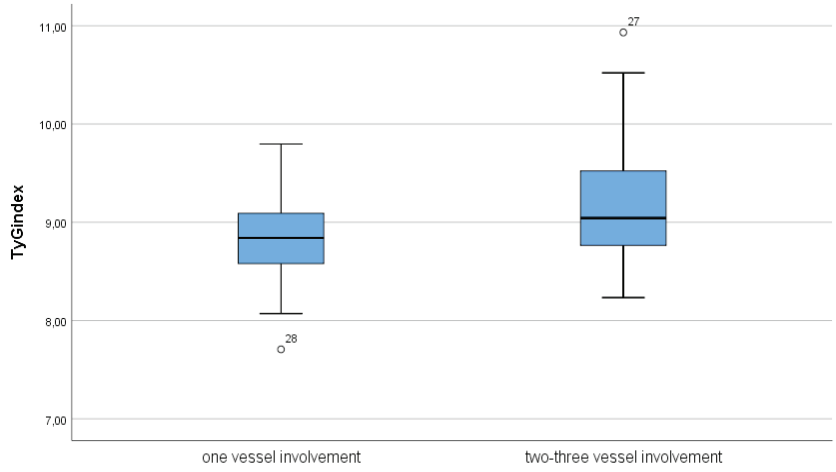

Figure 1. TyG index values of patients who had one vessel and two-three vessel CAE.

Twenty-five patients (41.0%) had one-vessel involvement, 24 (39.3%) patients had two-vessel involvement and 12 (19.7%) patients had three-vessel involvement. Of the patients who had one vessel involvement, RCA was the most commonly affected vessel (11, 44%), followed by LAD (8, 35%), left main coronary artery (LMCA) (4, 16%) and Cx (1, 4%). When we analyzed all CAE patients without considering the number of vessel involvement, the frequency of the affected vessel, in decreasing order, was LAD (39, 63.9%), RCA (33, 54.1%), Cx (28, 45.9%), and LMCA (9, 14.8%).

When patients who had one-vessel involvement and two/three-vessel involvement were compared, it was found that TyG index was statistically significantly higher in patients who had two/three vessel involvement (8.80 ± 0.48 vs 9.14 ± 0.62, p= 0.027) (Figure 1). Furthermore, TyG index of the patients who had two-three vessel CAE was significantly higher compared to the patients who had normal coronary arteries (9.27 ± 0.67 vs 9.00 ± 0.84, p= 0.034). Since the number of HT patients was found to be higher in the study group, we also analyzed the number of HT patients in one-vessel CEA involvement with two/three-vessel CAE involvement. We did not find any differences with respect to HT between one-vessel involvement and two/three-vessel involvement (n= 14, 56% vs n= 27, 75%, respectively, p= 0.121). According to correlation analysis, TyG index had statistically significant but a weak correlation with the number of ectatic vessels (r= 0.275, p= 0.032). ROC curve analysis showed that TyG index was useful for the prediction of two/three vessel involvement (AUC= 0.653, CI 95%= 0.515-0.792, p= 0.043) (Figure 2). TyG index value of 9.16 had 41.7% sensitivity and 84% specificity for the prediction of ectasia severity. According to logistic regression analysis, TyG index and the presence of DM were independent predictors of CAE severity (involvement of two/three vessels) (for DM, OR= 4.145, p= 0.044, 95% CI= 1.038-16.555 and for TyG index, OR= 3.167, p= 0.035, 95% CI= 1.082-9.267).

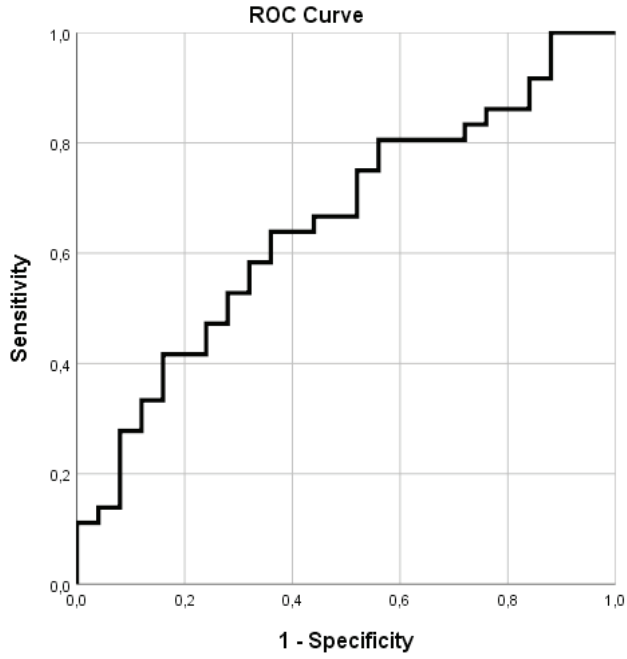

Figure 2. ROC curve analysis for prediction of CAE severity.

DISCUSSION

Our study showed that although TyG index was not different between patients who had normal and ectatic coronary arteries, patients with more diffuse involvement of ectasia had higher values of TyG index compared patients who had normal coronary arteries. TyG index could be useful in the prediction of the severity of coronary involvement in patients with CAE.

CAE is predominantly found in males with a male to female ratio of 3:1[23]. In the present study, 72.1% of the patients was male and LAD was the most commonly affected vessel. Although RCA has been reported to be the most frequently affected vessel in most of the studies, some studies have found different findings. Sultana et al. and Malviya et al. have found similar findings to our study, with the involvement of LAD in 63% and 59.6% of the cases, respectively[24,25]. In our study, 41% cases had single-vessel CAE, which is also in line with the previous studies[25].

The exact cause and pathophysiology of CAE is not fully understood; however, a variety of disorders including atherosclerosis, inflammatory diseases, connective tissue diseases, iatrogenic mechanisms and congenital causes have been reported to be associated with it. Several biochemical parameters have been investigated in order to determine risk factors and etiologic mechanisms underlying the disease process. Among laboratory parameters, we found that RDW and creatinine were significantly higher, whereas GFR was significantly lower in CAE patients. RDW has strong association with cardiovascular mortality and has been shown to be increased in CAE cases[26]. Yet, precise connection between CAE and increased RDW values has not been identified, factors that influence red blood cell homeostasis such as inflammation and oxidative stress might be the underlying link between two conditions[27,28]. Similarly, abnormalities in renal function are associated with CAE. Turkmen et al. have demonstrated that urinary excretion of albumin is increased in patients with CAE representing generalized condition that affects multiple tissues[29].

Insulin resistance can be considered as a syndrome which increases the risk of cardiovascular disease[30]. It appears to be related with dyslipidemia, hypertension, hypercoagulability, low-grade inflammation and other abnormalities. Zhang et al. have demonstrated that patients with CAE have higher insulin levels suggesting a common pathway in pathogenesis of CAE. In their study, magnitude of the lesions differed significantly among patients, patients with higher insulin levels had more severe disease[31]. This may be attributed to endothelial dysfunction, fat deposition in arterial wall, accelerated atherosclerosis and development of CAE[32]. Cao et al. have compared fasting insulin levels and HOMA-IR indices of patients with CAE, arteriosclerosis and normal coronary arteries. In their study, fasting insulin and HOMA-IR levels were significantly higher compared to that of patients with arteriosclerosis and normal coronary arteries. They have also found positive correlations between fasting glucose and HOMA-IR levels and the severity of CAE[33]. In this context, CAE might be considered as exaggerated vascular remodeling in response to atherosclerotic lesions[34].

TyG index is a simple calculation obtained from fasting glucose and TG levels. It is proposed as a surrogate marker for insulin resistance[35]. Since insulin resistance might play a role in the development of CAE, in our study, we wanted to measure TyG index values of CAE patients. We did not find any differences in TyG index values between the patient and control group. However, when patients who had two/three vessel involvement were compared with control group and patients with one vessel involvement, we found significant differences. These findings suggested that mild disease had only marginally elevated TyG index levels that did not reach statistical significance. According to our results, TyG index could be used to assess CAE severity. It was positively correlated with the number of CAE vessels. Moreover, patients with two/three-vessel involvement had higher TyG index values compared to controls. Our results also supported the hypothesis that insulin resistance and atherosclerosis were the part of the mechanism underlying the pathogenesis of CAE.

CONCLUSION

In conclusion, TyG index might be used in the assessment of severity of CAE. Its calculation is very easy and could be readily used in daily clinical practice. Large scale studies are needed to evaluate the value of TyG index in CAE patients.

Limitations

It was a single-center study with a relatively small sample size. Plasma insulin and HOMA-IR levels of the patients were not measured. TyG index was only measured at baseline, more frequent measurement could give more information. Last, long term follow-up of the patients was not conducted.

Ethics Committee Approval: This study was approved by Bakırköy Dr. Sadi Konuk Research and Training Hospital Clinical Research Ethics Committee (Decision no: 2021-19-01, Date: 04.10.2021).

Informed Consent: This is retrospective study, we could not obtain written informed consent from the participants.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept/Design - EO, İU, CY, AŞ; Analysis/Interpretation - CY, İBA, BM; Data Collection - EO, İU, AS; Writing - CY; Critical Revision - All of authors; Statistical Analysis - CY; Overall Responsibility - All of authors.

Conflict of Interest: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors declare that this study has received no financial support.

REFERENCES

1. Hartnell GG, Parnell BM, Pridie RB. Coronary artery ectasia, its prevalence and clinical significance in 4993 patients. Br Heart J 1985;54:392-5. [Crossref]
2. Manginas A, Cokkinos DV. Coronary artery ectasias: Too large to miss? Eur Cardiovasc Dis 2006;2:1-6. [Crossref]
3. Sharma SN, Kaul U, Sharma S, Wasir HS, Manchanda SC, Bahl VK, et al. Coronary arteriographic profile in young and old Indian patients with ischemic heart disease: A comparative study. Indian Heart J 1990;42:365-9.
4. Chaithiraphan S, Goldberg E, O'Reilly M. Multiple aneurysms of coronary artery in sclerodermal heart disease. Angiology 1973;24:86-93. [Crossref]
5. Shiro I, Bannerman RM, Graf CJ, Brennan JC. Ehlers-Danlos syndrome with multiple arterial lesions. Am J Med 1969;47:967-77. [Crossref]
6. Türkmen M, Bitigen A, Esen AM. Coronary artery ectasia. J Med Sci 2006;26:68-72.
7. Markis JE, Joffe CD, Cohn PF, Feen DJ, Herman MV, Gorlin R. Clinical significance of coronary arterial ectasia. Am J Cardiol 1976;37:217-22. [Crossref]
8. Antoniadis AP, Chatzizisis YS, Giannoglou GD. Pathogenetic mechanisms of coronary ectasia. Int J Cardiol 2008;130:335-43. [Crossref]
9. Sorrell VL, Davis MJ, Bove AA. Current knowledge and significance of coronary artery ectasia: A chronologic review of the literature, recommendations for treatment, possible etiologies, and future considerations. Clin Cardiol 1998;21:157-60. [Crossref]
10. Androulakis AE, Andrikopoulos GK, Kartalis AN, Stougiannos PN, Katsaros AA, Syrogiannidis DN, et al. Relation of coronary artery ectasia to diabetes mellitus. Am J Cardiol 2004;93:1165-7. [Crossref]
11. Johanning JM, Franklin DP, Han DC, Carey DJ, Elmore JR. Inhibition of inducible nitric oxide synthase limits nitric oxide production and experimental aneurysm expansion. J Vasc Surg 2001;33:579-86. [Crossref]
12. Swaye PS, Fisher LD, Litwin P, Vignola PA, Judkins MP, Kemp HG, et al.Aneurysmal coronary artery disease. Circulation 1983;67:134-8. [Crossref]
13. Sadr-Ameli M, Sharifi M. The natural history of ectatic coronary artery disease. Iranian Heart J 2001;2:12-6.
14. Mrdović I, Jozić T, Asanin M, Perunicić J, Ostojić M. Myocardial reinfarction in a patient with coronary ectasia. Cardiology 2004;102:32-4. [Crossref]
15. Kühl M, Varma C. A case of acute coronary thrombosis in diffuse coronary artery ectasia. J Invasive Cardiol 2008;20:23-5.
16. DeFronzo RA. Insulin resistance, lipotoxicity, type 2 diabetes and atherosclerosis: The missing links. The Claude Bernard Lecture. Diabetologia 2010;53:1270-87. [Crossref]
17. Bornfeldt KE, Tabas I. Insulin resistance, hyperglycemia, and atherosclerosis. Cell Metabol Rev 2011;14:575-85. [Crossref]
18. Zhang Y, Ding X, Hua B, Liu Q, Gao H, Chen H, et al. Predictive effect of triglyceride glucose index on clinical events in patients with type 2 diabetes mellitus and acute myocardial infarction: Results from an observational cohort study in China. Cardiovasc Diabetol 2021;20:43. [Crossref]
19. Chiu TH, Tsai HJ, Chiou HYC, Wu PY, Huang JC, Chen SC. A high triglyceride-glucose index is associated with left ventricular dysfunction and atherosclerosis. Int J Med Sci 2021;18:1051-7. [Crossref]
20. Zheng R, Mao Y. Triglyceride and glucose (TyG) index as a predictor of incident hypertension: A 9-year longitudinal population-based study. Lipids Health Dis 2017;16:175. [Crossref]
21. Srinivasan S, Singh P, Kulothungan V, Sharma T, Raman R. Relationship between triglyceride glucose index, retinopathy and nephropathy in type 2 diabetes. Endocrinol Diabetes Metab 2020;4:e00151. [Crossref]
22. Guerrero-Romero F, Simental-Mendía LE, González-Ortiz M, Martínez-Abundis E, Ramos-Zavala MG, Hernández-González SO, et al. The product of triglycerides and glucose, a simple measure of insulin sensitivity. Comparison with the euglycemic-hyperinsulinemic clamp. J Clin Endocrinol Metab 2010;95:3347-51 [Crossref]
23. Lin CT, Chen CW, Lin TK, Lin CL. Coronary artery ectasia. Tzu Chi Med J 2008;20:270-4. [Crossref]
24. Sultana R, Sultana N, Ishaq M, Samad A. The prevalence and clinical profile of angiographic coronary ectasia. J Pak Med Assoc 2011;61:372-5.
25. Malviya A, Jha PK, Mishra A. Isolated coronary artery ectasia; clinical, angiographic, and follow up characteristics. Indian Heart J 2017;69:619-23. [Crossref]
26. Felker GM, Allen LA, Pocock SJ, Shaw LK, McMurray JJV, Pfeffer MA, et al. Red cell distribution width as a novel prognostic marker in heart failure: Data from the CHARM program and the Duke Databank. Am Coll Cardiol 2007;50:40-7. [Crossref]
27. Sezen Y, Bas M, Polat M, Yildiz A, Buyukhatipoglu H, Kucukdurmaz Z, et al. The relationship between oxidative stress and coronary artery ectasia. Cardiol J 2010;17:488-94.
28. Guo YL, Luo SH, Tang Y, Li JJ. Association of red cell distribution width with the presence of coronary artery ectasia. Clin Lab 2014;60:199-205. [Crossref]
29. Turkmen S, Yolcu M, Cagliyan CE, Sercelik A, Ipek E, Tekin K, et al. The relationship between microalbuminuria and isolated coronary artery ectasia. Eur Rev Med Pharmacol Sci 2014;18:1661-5.
30. Ginsberg HN. Insulin resistance and cardiovascular disease. J Clin Invest 2000;106:453-8. [Crossref]
31. Zhang X, Liu A, Guo M, Guan J, Cai S, Du Q, et al. Correlations among plasma insulin concentration, insulin sensitivity and coronary artery ectasia. Int J Clin Exp Med 2018;11:2325-30
32. Pino AD, DeFronzo RA. Insulin resistance and atherosclerosis: Implications for insulin-sensitizing agents. Endocr Rev 2019;40:1447-67. [Crossref]
33. Cao Y, Yin H, Yin H, Jiang Z, Wang T, Chen T. Associations of the insulin level and insulin resistance with the severity of coronary artery ectasia. Int J Clin Exp Med 2018;11:4937-42.
34. Devabhaktuni S, Mercedes A, Diep J, Ahsan C. Coronary artery ectasia-a review of current literature. Curr Cardiol Rev 2016;12:318-23. [Crossref]
35. 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. [Crossref]