Original Research Article

Prevalence and Predictors of Coronary Artery Ectasia among Delta Population

Abstract

Background: Localized or widespread non-obstructive lesions of the epicardial coronary arteries, with a luminal dilatation 1.5 times that of the neighbouring normal segments or vessel diameter, have been identified as CAE. This research was performed to determine prevalence and predictors of CAE among Delta population in Egypt.

Methods: This cross-sectional research was conducted on 2850 cases over the age of 18 who came for coronary angiography with positive non-invasive diagnostic tests, acute coronary syndrome and stable CAD. Cases were divided into two groups Group I: CAE cases (n =108) and Group II Non-ectasia cases (n =2742). All cases were subjected to history taking, clinical examination, laboratory investigations, standard 12-leads ECG, resting transthoracic echocardiography (TTE) and coronary angiography.

Results: CAE occurred in 108 (3.79%) of the studied cases. Total cholesterol level, serum creatinine, were insignificantly different between both groups. CRP, NLR, MPV, and PLR, were significantly higher in CAE cases versus non-Ectasia cases CRP, NLR, PLR, and MPV is a good predicator for CAE , LVESD and LVEDD, were significantly higher in CAE group

Conclusions: The RCA was the most often affected coronary artery. The existence of CAE can be predicted using easily accessible clinical laboratory values such as CRP, Neutrophil to lymphocyte ratio, mean platelet volume, and platelet to lymphocyte ratio.

Keywords: Coronary Artery Ectasia, Delta Population, coronary angiography, transthoracic echocardiography

Introduction:

The worldwide prevalence of coronary artery disease (CAD) is frightening. Rapid global expansion in the incidence of many coronary artery disease risk factors has also been documented. Therefore, it is highly suggested to place an emphasis on preventative screening methods, early diagnosis, and effective and prompt therapy (1).

Localized or widespread non-obstructive lesions of the epicardial coronary arteries, with a luminal dilatation 1.5 times that of the neighbouring normal segments or vessel diameter, have been identified as Coronary artery ectassia (CAE). CAE without severe coronary artery stenosis is considered isolated. Myocardial infarction and angina pectoris can result from vasospasm, dissection, or thrombus in coronary arteries that have abnormally dilated in the absence of coronary artery disease (2).

Due to advancements in diagnostics and imaging techniques, such as coronary angiography, more ectasia instances are being identified and treated. The true incidence of ectasia is unclear since not all individuals with the condition have symptoms and undergo a coronary angiography. At autopsy and during coronary angiography, the reported incidence is between 0.3% and 4.9%. (3).

However, it is unknown why some individuals experience arterial dilatation in response to the same risk factors whereas others experience arterial blockage (4). Histopathological examination of CAE cases after death typically reveals widespread inflammatory infiltration of the coronary vascular wall (2).

Generally speaking, the left major coronary artery is not affected. In most cases of CAE, just one blood artery is affected. Critical aortic stenosis is characterised by disruptions in blood flow filling and washout. It has been noted during imaging that the dilated coronary segment

has delayed antegrade filling, segmental reverse flow phenomena, and local deposition of dye (stasis) (5).

The purpose of this research was to identify risk factors for CAE in the Delta region of Egypt and to assess the frequency with which it occurs.

Patients and Methods:

This cross-sectional research was conducted on 2850 cases over the age of 18 who came for coronary angiography. Inclusion criteria were positive non invasive diagnostic tests, acute coronary syndrome and stable CAD. The research was approved by the Ethics Committee of Faculty of Medicine, Tanta University, Egypt. There were adequate provisions to maintain privacy of participants and confidentiality of the data.

Exclusion Criteria were cases less than 18 years, patient with autoimmune disease or on immunosuppressive drugs and cases with recent infection, and chronic inflammatory diseases.

Cases were divided into two groups Group I: CAE cases (n =108) and Group II Non-ectasia cases (n =2742).

Complete history taking was performed (age, sex and comorbidities), the risk factor for cad were assessed (hypertension, diabetes, dyslipidemia, smoking, body mass index (BMI). The personal family history was taken as well to identify the genetic predisposition.

Each participant underwent a thorough physical examination that included an assessment of their general health and vital signs including blood pressure and heart rate as well as an evaluation of their chest discomfort including its location, quality, timing, and referral patterns along with heart sounds and additional sounds as s3 or s4 and cardiac murmurs. laboratory investigations such as c-reactive protein (CRP), complete blood picture, neutrophil lymphocyte ratio, mean platelets volume, serum creatinine, serum cholesterol, platelets lymphocyte ratio and HbA1c.

Resting Transthoracic Echocardiography (TTE):

The used device were GE VIVID S5 and philips affiniti 70 new. The X 5-1 and X 7-1 phased array sector probes were employed for this investigation (frequency range 1.5-4.3 MHz). Once the images had been captured, they were saved on a hard drive and transmitted to a computer where they could be analysed later using analytic software (Q Lab 10). Parasternal images of the long axis of the left ventricle were captured in full volume mode for four heartbeats, as were parasternal images of the long axis and short axis of the aortic root in 3D zoomed mode.

Measurement of aortic root diameters by 2D echocardiography TTE:

Aortic annulus, sinuses of Valsalva, and sinotubular junction are standard measuring points for a heart's circumference. Using magnified photos taken at the end of diastole with the aortic valve leaflets inserted perpendicular to the aortic axis from leaflet leading edge to leaflet leading edge, the diameter of the aortic annulus was calculated using 2D TTE from the para sternal long-axis view. The aortic sinuses of Valsalva are the areas between the luminal surface of the aortic root's three bulges and the corresponding valve leaflets (7).

The aortic root's widest point is at the sinuses of Valsalva. The sinotubular junction is the point where the curved sinuses of Valsalva meet the tubular ascending aorta, and is denoted by an acute angle (7).

Coronary Angiography[(9)]

Invasive coronary angiography was done. Left and right guiding catheters introduced through the sheath in right femoral artery (trans-femoral approach). Assessment of lesions in 2 orthogonal views, and Syntax score calculation.

Syntax score is the total number of points given for each lesion in the coronary tree with a diameter narrowing of more than 50% in arteries larger than 1.5 mm in diameter.

Type I was defined as the presence of diffuse CAE in 2 or 3 coronary vessels; type II was defined as the presence of diffuse CAE in 1 coronary vessel and localised CAE in another vessel; type III was defined as the presence of diffuse CAE in 1 coronary vessel; and type IV was defined as localised or segmental CAE based on the angiographic anatomical distribution. **(Figure 1)**.

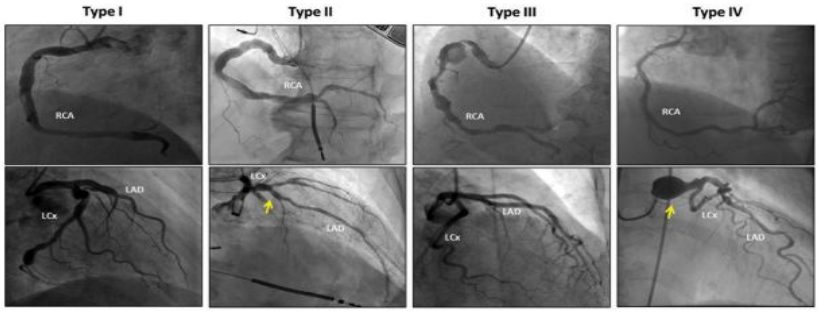

Figure 1: Angiographic characterization of CAE distribution according to the Markis classification. Type I: diffuse CAE in 2 or 3 coronary vessels. In these case, all 3 vessels present diffuse CAE. Type II: diffuse CAE in one coronary vessel (RCA) and localized CAE in another vessel (proximal LAD, arrow). Type III: diffuse CAE in only 1 coronary vessel (RCA, arrows). Type IV: localized or segmental CAE (in this case, massive dilatation of the LMCA, arrow). CAE = coronary artery ectasia; LAD = left anterior descending; LCx = left circumflex; LMCA = left main coronary artery; RCA = right coronary artery [(9)]

When there is a stenosis of more than 50% in two main coronary arteries, this is known as multivessel disease. The Thrombolysis in Myocardial Infarction (TIMI) frame count technique was utilised to assess blood flow within the coronary arteries and the severity of thrombosis, which ranged from 0 to 5. Thirteen had a thrombus load that was considered to be high if it had a TIMI-thrombus score of 4 or higher. Final TIMI 3 distal flows with less than 20% artery stenosis and no acute mechanical problems were considered successful angiograms. When there was no angiographic evidence of mechanical vascular blockage at the conclusion of the operation, a TIMI flow of 2 was considered indicative of a no-reflow occurrence. 14 Care-tracking protocols based on institutional guidelines were used to monitor cases [(10)].

Statistical Analysis

For the statistical analysis, we used SPSS 25. (IBM Inc., Chicago, IL, USA). Histograms and the Shapiro-Wilks normality test were employed to determine if parametric or nonparametric statistical testing was appropriate for the data. In order to compare the three groups, we used the F test, plus a post hoc (Tukey) test for pairwise comparisons. Parametric variables (such as age) were reported as means and standard deviations. Pairwise comparisons between two variables within the same group were analysed using the paired T test. The Kruskal-Wallis test was used to compare the means of non-parametric variables (such as VAS), and the Mann-Whitney (U) test was used to compare the two groups. Wilcoxon test was used to compare two factors amongst members of the same group. Chi-square analysis was used to determine statistical significance for categorical variables (such as sex) reported as frequencies and percentages. The cutoff for statistical significance was set at a two-tailed P value of less than 0.05.

Results:

CAE occurred in 108 (3.79%) of the studied cases. Age and BMI were insignificantly different between both groups. Sex was significantly different between both groups (P value= 0.001) (**Table 1)**

Table 1: Demographic data of the studied groups

		CAE cases (n =108)	**Non-ectasia cases (n =2742)**	**P value**
Age	**Mean ± SD**	56 ± 12.82	56.94 ± 11.68	0.540
	Range	35 – 75	33 – 75	
BMI	**Mean ± SD**	29.21 ± 7	28.54 ± 10.17	0.494
	Range	20 – 43	17 – 43	
Sex	**Male**	88 (81%)	1802 (66%)	**0.001***
	Female	20 (19%)	940 (34%)	

*: significant as P value ≤ 0.05.

Risk factors (HTN, dyslipidemia, and family history) were insignificantly different between both groups. But DM was significantly higher in non-ectasia cases (p =0.03) and smoking was significantly higher in CAE group (p =0.014). (**Table 2)**.

Table 2: Risk factors of the studied groups

	CAE cases (n =108)	**Non-ectasia cases (n =2742)**	**P value**
HTN	37 (34%)	1075 (39%)	0.351
DM	9 (8%)	457 (17%)	**0.03***
Dyslipidemia	70 (65%)	1661 (61%)	0.433
Smoking	68 (63%)	1382 (50%)	**0.014***
Family history	16 (15%)	577 (21%)	0.149

HTN: hypertension, DM: diabetes mellitus

Total cholesterol level, serum creatinine, were insignificantly different between both groups. CRP, NLR, MPV, and PLR, were significantly higher in CAE cases versus non-Ectasia cases (P value <0.05).HbA1c was significantly higher in non-ectasia cases versus CAE cases, Clinical examination (Systolic, diastolic blood pressure, heart rate, and chest pain) were insignificantly different between both groups (**Table 3)**.

Table 3: Laboratory investigations and Clinical examination of the studied groups

		CAE cases (n =108)	Non-ectasia cases (n =2742)	P value
CRP	**Mean ± SD**	3.21 ± 0.65	2.95 ± 0.61	**<0.001***
	Range	2.2 – 4.4	1.9 – 4.4	
NLR	**Mean ± SD**	2.82 ± 0.71	2.08 ± 0.54	**<0.001***
	Range	1.5 – 4.4	1.2 – 4.4	
MPV (fL)	**Mean ± SD**	8.62 ± 0.43	8.28 ± 0.45	**<0.001***
	Range	8.01 – 9.32	7.52 – 9.32	
PLR	**Mean ± SD**	123.65 ± 18.51	108.14 ± 19.8	**0.003***
	Range	94 - 159	75 - 159	
Total Cholesterol (mg/dL)	**Mean ± SD**	207 ± 55.4	212.27 ± 55.75	0.33
	Range	117 - 289	118 - 289	
Serum creatinine (mg/dL)	**Mean ± SD**	1.06 ± 0.12	1.05 ± 0.11	0.303
	Range	0.9 – 1.3	0.8 – 1.3	
HbA1c (%)	**Mean ± SD**	5.92 ± 1.25	6.18 ± 1.16	**0.04***
	Range	3.4 – 9.1	3.1 – 10.3	
Systolic blood pressure (mmHg)	**Mean ± SD**	126.96 ± 30.19	128.25 ± 32.85	0.371
	Range	72 - 181	65 - 181	
Diastolic blood pressure (mmHg)	**Mean ± SD**	72.58 ± 8.76	73.02 ± 09.9	0.797
	Range	62 - 89	58 - 89	
Herat rate (beats/min)	**Mean ± SD**	72.57±9.81	70.64 ± 11.04	0.08
	Range	54 - 89	53 - 89	
Chest pain	**Positive Typical**	61 (56%)	1779 (65%)	0.2
	Positive Atypical	16 (15%)	320 (12%)	
	Negative	31 (29%)	643 (23%)	

Platelet-Lymphocyte Ratio *: significant as P value ≤ 0.05.

ECG changes were insignificantly different between both groups . LVESD and LVEDD, were significantly higher in CAE group (p =0.005, 0.033) but EF and LA were insignificantly different between the two groups. **[Table 4].**

Table 4: Standard 12-leads ECG and Resting Transthoracic Echocardiography (TTE) of the studied groups

		CAE cases (n =108)	Non-ectasia cases (n =2742)	P value
ECG	**Positive changes**	67 (62%)	1472 (54%)	0.107
	Negative changes	41 (38%)	1270 (46%)	
LVESD (cm)	**Mean ± SD**	3.13 ± 0.75	2.9 ± 0.82	**0.005***
	Range	1.8 – 4.1	1.5 – 4.1	
LVEDD (cm)	**Mean ± SD**	4.97 ± 0.73	4.83 ± 0.48	**0.033***
	Range	3.8 – 6.1	4 – 6.1	
EF (%)	**Mean ± SD**	62.12 ± 5.68	61.09 ± 8.89	0.009
	Range	53 - 72	49 - 72	
LA (mL/m^2)	**Mean ± SD**	41.12 ± 10.41	42.61 ± 8.51	0.197
	Range	20 - 55	29 - 55	

LVESD: Left Ventricular End-Systolic Diameter, LVEDD: Left Ventricular End-Diastolic Diameter, EF: Ejection fraction, LA: Left Atrial

CAE affected LAD vessel in 75 (69%) cases, LCX vessel in 58 (54%) cases and RCA vessel in 83 (77%) cases. CAE affected a single vessel in 29 (27%) cases but affected multi vessel in 79 (73%) cases. Regarding

Regarding Markis classification in CAE cases without lesions, 13 (43.33%) cases are classified as Type I, 3 (10%)as type II, 8 (26.67%) cases as Type III and 6 (10%) cases were classified as type IV [**Table 5**].

Table 5: Coronary angiography of CAE cases

		CAE cases (n =108)
Type of affected vessel	**LAD**	75 (69%)
	LCX	58 (54%)
	RCA	83 (77%)
Number of vessels	**Single vessel**	29 (27%)
	Multi-vessel	79 (73%)
Markis Classification of ectasia without lesion (n =30)	**I**	13 (43.33%)
	II	3 (10%)
	III	8 (26.67%)
	IV	6 (10%)

CAE: Coronary artery ectasia, LAD: left anterior descending artery, LCX: left circumflex artery, RCA: Right coronary artery

CRP is a good predicator for CAE (AUC =0.609, 95% CI =0.553- 0.664, p value <0.001). At cut off value >3, it has 60% sensitivity and 52% specificity.

NLR is a good predicator for CAE (AUC =0.787, 95% CI =0.749- 0.822, p value <0.001). At cut off value >2.8, it has 53.85% sensitivity and 91.14% specificity.

PLR is a good predicator for CAE (AUC =0.703, 95% CI =0.661 – 0.743, p value 0.001). At cut off value >109, it has 80.77% sensitivity and 53.8% specificity.

MPV is a good predicator for CAE (AUC =0.696, 95% CI =0.654 – 0.737, p value <0.001). At cut off value >8.3, it has 76.92% sensitivity and 50.21% specificity [Figure 2].

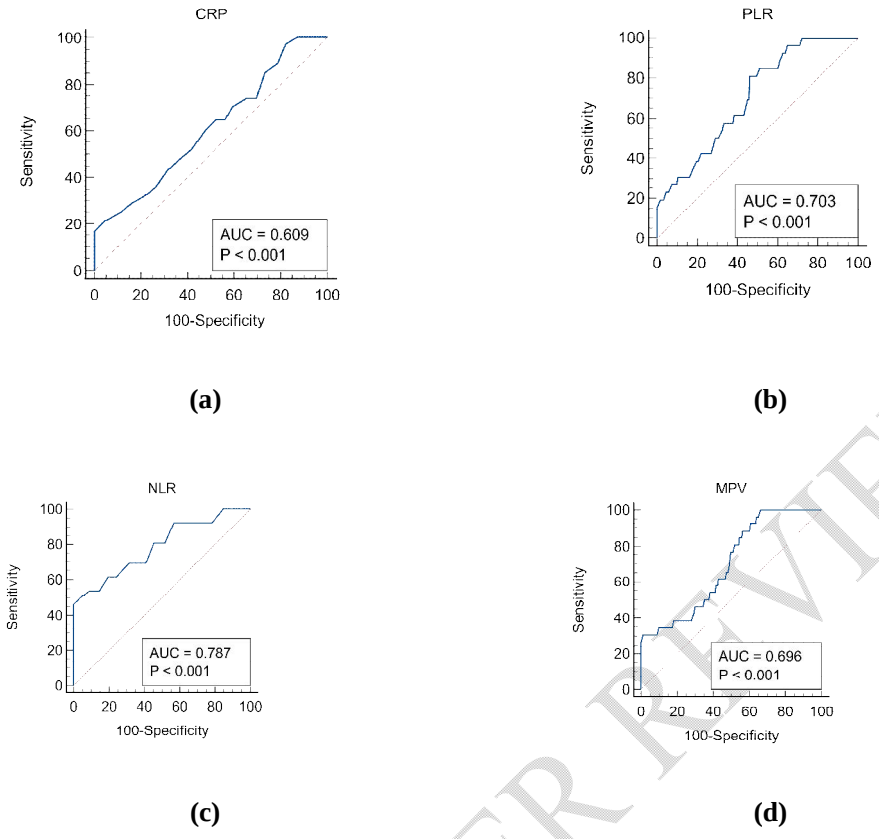

Figure 2: ROC curve of (a) CRP, (b) PLR, (c)NLR and (d)MPV for prediction of CAE

Discussion

In the present research, CAE occurred in 108 (3.79%) of the studied cases. A comparable research by **Willner et al., (2020)** [80] determined clinical outcome and CAE prevalence in a large cohort of Haifa, Israel, cases who had coronary angiography. Only 174 out of a total of 20455 investigations (0.85% of all angiograms, 161 people) were found to have CAE.

Additionally, **Doi et al., (2017)** [82] examined the incidence of major adverse cardiac events (MACE; defined as cardiac mortality and nonfatal myocardial infarction [MI]) in 1698 cases diagnosed with acute MI. Cases with and without CAE were compared for the incidence of MACE. Causing Adverse Events (3.0% of Research Participants) with a mean age of 63±13 and male concern 43 (84%) that was predominant.

Also, **Fan et al., (2020)**[83] examined the correlation between CAE and inflammatory markers such high-sensitivity C-reactive protein (hs-CRP) and interleukin-6 (IL-6). In their research, the prevalence of CAE among all angiography cases was calculated to be 302/6542 = 0.046, or 4.6%. Male percentage was 146 (67.3) in CAE cases.

Moreover, **Giannoglou et al., (2006)** [84]determined the prevalence of CAE in cases in Northern Greece Referred for Coronary angiography. In 10,524 consecutive cases CAE was found in 287 cases (2.7%). It was markedly more prevalent in men than in women (p <0.0001).

In agreement with our findings, **Moezi Bady et al., (2021)** [85] examined the prevalence of CAE in Eastern Iran and the variables contributing to it. The 2,795 cases who had scheduled coronary angiograms were the subjects of a cross-sectional research. We divided our cases into three groups based on their coronary artery disease status: CAE, Coronary artery stenosis and controls. Normal group (n = 744) CAS group (n = 1966) ectasia group (n = 85). The prevalence of CAE was 3.04%.

In the current research, NLR, MPV and PLR were significantly higher in CAE cases versus non-Ectasia cases (P value <0.05).

Likewise, **Shereef and Kandeel (2019)** [13] CAE prediction accuracy was evaluated by measuring platelet-to-lymphocyte ratio, mean platelet volume, and neutrophil-to-lymphocyte ratio against the high-sensitivity C-reactive protein (Hs-CRP). A total of 60 individuals with chronic stable angina were divided into three categories. Twenty cases in Group 1 had CAE but no atherosclerotic coronary arteries; twenty cases in Group 2 had CAE plus atherosclerotic coronary arteries; and twenty cases in Group 3 had normal coronary angiography. They demonstrated that NLR, MPV and PLR were significantly higher in CAE cases versus normal coronary angiography cases (NLR 2.94±0.75 vs. 2.11±0.46; p= <0.001,

PLR 125.80±17.60 vs. 109.70±17.28; p=0.003, MPV (FL) 8.58±0.40 vs. 8.26±0.37; p=0.008).

Hyperdestructive thrombocytopenia, such as idiopathic thrombocytopenic purpura, can be distinguished from hypo-productive thrombocytopenia due to the presence of MPV, whose levels are greater in the former condition [(14)].

In the present research, CRP was significantly higher in CAE than cases without CAE ($p<0.05$).

In contrast to the present research results, **Fan et al., (2020)** [(15)]reported that there was non-significant difference in NLR and MPV (NLR: 8.18 ± 5.36 vs.8.41 ± 6.6; p=0.125). They also reported that hs-CRP (mg/L), 16.9 ± 3.82 vs. 25.6 ± 4.65; $P<0.05$) and these findings are comparable to ours.

In the present research, HbA1c was significantly higher in non-ectasia cases versus CAE cases. The current research results came in line with **Özkan et al., (2019)** [(16)]who said that there was insignificant difference regarding clinical presentations (SBP (mm Hg) 127.00±7.25 vs. 125.51±6.32; p=0.293), (DBP (mm Hg) 80.42±4.66 vs. 80.88±4.19; p=0.624) in CAE versus subjects with normal coronary arteries.

In the current research, LVESD and LVEDD, were significantly higher in CAE group (p =0.005, 0.033) but EF and LA were insignificantly different between the two groups. Comparable to the present research findings, **Fan et al., (2020)** [(15)]mentioned that there was an insignificant difference between CAE and non-CAE regarding %EF (P=0.343).

In the present research, CAE affected LAD vessel in 75 (69%) cases, LCX vessel in 58 (54%) cases and RCA vessel in 83 (77%) cases. CAE affected a single vessel in 29 (27%) cases but affected multi vessel in 79 (73%) cases. Regarding Markis classification in CAE cases without lesions, 13 (43.33%) cases are classified as Type I, 3 (10%) as type II, 8 (26.67%) cases as type III and 6 (10%) cases were classified as type IV.

In the current research, CRP is a good predicator for CAE (AUC =0.609, 95% CI =0.553-0.664, p value <0.001). At cut off value >3, it has 60% sensitivity and 52% specificity.

Our results are confirmed by **Shereef and Kandeel, (2019)** [13]who observed that Hs-CRP ≥2.35 mg/dl can predict CAE with sensitivity 95% and specificity 85% (P < 0.001).

In the present research, NLR is a good predicator for CAE (AUC =0.787, 95% CI =0.749-0.822, p value <0.001). At cut off value >2.8, it has 53.85% sensitivity and 91.14% specificity.

The findings of PLR are in the same line with **Shereef and Kandeel (2019)** [13] who stated that PLR with value ≥110.5 can detect CAE with sensitivity of 82.5% and specificity of 60% (95% CI, AUC = 0.756, P < 001).

The findings of this research about MPV are in harmony with **Shereef and Kandeel (2019)** [89]who noticed that MPV with value ≥8.25 can detect CAE with sensitivity of 72.5% and specificity of 55% (95% CI, AUC = 0.575, P = 0.007).

Limitations: It was a bi-center research, and the results may differ elsewhere. We have no healthy controls compared with CAE cases.

Conclusions:

The RCA was the most often affected coronary artery. The existence of CAE can be predicted using easily accessible clinical laboratory values such as CRP, Neutrophil to lymphocyte ratio, mean platelet volume, and platelet to lymphocyte ratio, which means that CAE is also severe inflammatory process.

References:

1. Jyothi L, Nuthalapati RK, Peddy SRJJoICoC. Prevalence of coronary artery disease and its relationship with various cardiovascular risk factors: Analysis of south Indian cases. J Cardiol. 2018;8:138-42.

2. Boles U, Rakhit R, Shiu MF, Patel K, Henein M. Coronary artery ectasia as a culprit for acute myocardial infarction: review of pathophysiology and management/Koroner arter ektazisi akut miyokart enfarktüsünde sorumlu: Patofizyoloji ve yönetim derlemesi. AKD. 2013;13:695-712.

3. Hsu P-C, Su H-M, Lee H-C, Juo S-H, Lin T-H, Voon W-C, et al. Coronary collateral circulation in cases of coronary ectasia with significant coronary artery disease. PloS one. 2014;9:8-17.

4. Kanbay M, Segal M, Afsar B, Kang D-H, Rodriguez-Iturbe B, Johnson RJ. The role of uric acid in the pathogenesis of human cardiovascular disease. Heart. 2013:12-30.

5. Zografos TA, Korovesis S, Giazitzoglou E, Kokladi M, Venetsanakos I, Paxinos G, et al. Clinical and angiographic characteristics of cases with coronary artery ectasia. Int J Cardiol. 2013;167:1536-41.

6. Goldstein SA, Evangelista A, Abbara S, Arai A, Asch FM, Badano LP, et al. Multimodality imaging of diseases of the thoracic aorta in adults: from the American Society of Echocardiography and the European Association of Cardiovascular Imaging: endorsed by the Society of Cardiovascular Computed Tomography and Society for Cardiovascular Magnetic Resonance. J Am Soc Echocardiogr. 2015;28:119-82.

7. Galea N, Piatti F, Sturla F, Weinsaft JW, Lau C, Chirichilli I, et al. Novel insights by 4D Flow imaging on aortic flow physiology after valve-sparing root replacement with or without neosinuses. Interact Cardiovasc Thorac Surg. 2018;26:957-64.

8. David TE. Aortic valve sparing operations. Semin Thorac Cardiovasc Surg. 2011;23:146-8.

9. Wang X, Montero-Cabezas JM, Mandurino-Mirizzi A, Hirasawa K, Ajmone Marsan N, Knuuti J, et al. Prevalence and Long-term Outcomes of Cases with Coronary Artery Ectasia Presenting with Acute Myocardial Infarction. Am J Cardiol. 2021;156:9-15.

10. Liem S-S, van der Hoeven BL, Oemrawsingh PV, Bax JJ, van der Bom JG, Bosch J, et al. MISSION!: optimization of acute and chronic care for cases with acute myocardial infarction. Am Heart J. 2007;153:14-20.

11. Willner NA, Ehrenberg S, Musallam A, Roguin A. Coronary artery ectasia: prevalence, angiographic characteristics and clinical outcome. Open heart. 2020;7:96-9.

12. Moezi Bady SA, Rezvanian E, AZDAKI N, KHOSRAVI BIZHAEM S, KAZEMI T. Prevalence of Coronary Artery Ectasia and Associated Risk Factors. Shiraz E Medical J. 2021;22:150-6.

13. Shereef A, Kandeel N. Predictors for coronary artery ectasia. J Indian Coll Cardiol. 2019;9:123-30.

14. Keser A, Özbek K, Ulucan Ş, Katlandur H, Bilgi M, Özdil H. Relationship between red cell distribution with levels and severity of coronary artery ectasia. Eur Rev Med Pharmacol Sci. 2016;20:1571-4.

15. Fan CH, Hao Y, Liu YH, Li XL, Huang ZH, Luo Y, et al. Anti-inflammatory effects of rosuvastatin treatment on coronary artery ectasia cases of different age groups. BMC Cardiovasc Disord. 2020;20:330-40.

16. Özkan B, Örsçelik Ö, Yıldırım Yaroğlu H, Balcı Ş, Özcan MK, Çelik A, et al. Association between serum adropin levels and isolated coronary artery ectasia in cases with stable angina pectoris. Anatol J Cardiol. 2019;22:250-5.