Original Article

Comparison of Atherosclerotic Risk Factors and Hematologic Parameters Between Coronary Artery Ectasia and Obstructive Coronary Artery Disease

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ABSTRACT

Objectives: This study aims to compare atherosclerosis risk factors between patients with coronary artery ectasia (CAE) and those with triple-vessel coronary artery disease (3VD). CAE is characterized by diffuse dilation in epicardial coronary arteries and is associated with risk factors for atherosclerosis.

Methods: This retrospective cross-sectional study examined patients who underwent coronary angiography in our center between 2012 and 2016 and were diagnosed with CAE. Patients with 3VD served as the control group. Eligible patients' medical records were reviewed, and relevant data, including demographic and clinical characteristics, atherosclerotic risk factors, and laboratory and inflammatory markers, were collected and documented.

Results: The study population comprised 157 patients with CAE and 156 patients with 3VD. There was a significantly higher proportion of male patients in the 3VD group (P<0.001). Smoking (P<0.001), a history of acute coronary syndromes (P<0.001), and dyslipidemia (P<0.001) were significantly more prevalent in the 3VD group. The mean neutrophil-to-lymphocyte ratio was significantly higher in the 3VD group than in the CAE group (3.6±3.0 vs 2.3±1.9; P<0.001).

Conclusions: The findings of this study suggest that atherosclerosis risk factors and inflammatory markers may play a role in the development of CAE. ***(Iranian Heart Journal 2024; 25(3): 16-20)***

KEYWORDS: Coronary ectasia, Atherosclerosis, Hematologic parameter

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Coronary artery ectasia (CAE) is typically defined as segmental or diffuse dilation in a coronary artery more than 1.5-fold the diameter of the normal adjacent segment.[1] The reported incidence of CAE ranges from 1.2% to 4.9%.[2]

Approximately 10% of CAE cases can be attributed to a congenital etiology.[3] For the remaining patients, CAE is an acquired condition, with atherosclerosis, connective tissue disorders, Kawasaki disease, and inflammatory diseases being the most common underlying causes.[4]

In the majority of cases (80%–85%), CAE coexists with atherosclerotic coronary artery disease (CAD).[5] Similar to atherosclerotic CAD, inflammation plays a part in CAE pathogenesis. Higher levels of high-sensitivity C-reactive protein have been found in patients with CAE than in those with obstructive CAD.[6] The neutrophil-to-lymphocyte ratio (NLR), another indicator of inflammation, is higher in patients with isolated CAE and is correlated with the severity of ectasia.[7]

CAE is more prevalent in men, at a ratio of 3:1.[2] Similar to atherosclerotic CAD, hypertension is a risk factor for CAE.[8] Age and DM may be inversely associated with the incidence of CAE.[9] Some studies have found that dyslipidemia in patients with CAE is as frequent as that in patients with significant stenosis.[10] Evidence also indicates high smoking rates in patients with CAE compared with those with coronary stenosis.[11]

Symptoms of CAE range from the absence of symptoms to acute coronary syndromes.[6] In symptomatic cases, the most frequent presentation is angina.[12]

In the current absence of a definite treatment option, the prevention of CAE by modifying risk factors seems necessary. We sought to investigate atherosclerosis risk factors between patients with CAE and those with triple-vessel coronary artery disease (3VD) in a sample of the Iranian population.

METHODS

The present investigation retrospectively assessed a sample of Iranian patients who underwent coronary angiography between March 2012 and March 2016 at Rajaie Cardiovascular Medical and Research Center, Tehran, Iran. The study population consisted of 313 patients, divided into a CAE group (n=157) and a group with significant coronary stenosis but without CAE (n=156). Patients with significant valvular heart disease (more than moderate), previous percutaneous coronary interventions or coronary artery bypass graft surgeries, malignancies, current or recent (3 months) infectious diseases or corticosteroid consumption, and extreme ages (younger than 20 and older than 75) were excluded. The Framingham risk score (FRS) was calculated for each patient. The FRS considers coronary risk factors, comprising sex, systolic blood pressure, total cholesterol, high-density lipoprotein cholesterol, and smoking.[13]

CAE was defined as dilation in a coronary artery as much as 1.5-fold or more compared with the adjacent normal coronary segment in the absence of significant coronary stenosis. Significant coronary stenosis was defined as 50% or more stenosis in the left main artery and/or 70% or more stenosis in the other major epicardial coronaries.

Statistical Analysis

For ordinal and/or categorical variables with non-normalized distributions, nonparametric tests, such as the Wilcoxon signed-rank test and the Friedman test, were used. A *P* value <0.05 was considered statistically significant. All the statistical analyses were performed using the free version of SPSS 16.0.

RESULTS

The distribution of patients in the CAE group according to involved arteries is shown in Figure 1. Table 1 summarizes the

study population's demographic and clinical characteristics and coronary risk factors. The 2 groups were matched with respect to age. Male sex, smoking, and dyslipidemia were more common in the non-CAE group (*P* <0.001, *P*<0.001, and *P*<0.001, respectively). A history of previous acute coronary syndromes was significantly more frequent in the non-CAE group (*P*<0.001).

The median FRS was 12 (9–14) in the CAE group and 14 (12–15) in the non-CAE group, with the difference constituting statistical significance.

The NLR and the ratio between low and high-density lipoprotein levels were significantly higher in the non-CAE group (*P*<0.001 and *P*=0.012, respectively), whereas the mean high-density lipoprotein cholesterol level was significantly lower in the non-CAE group (*P*<0.001).

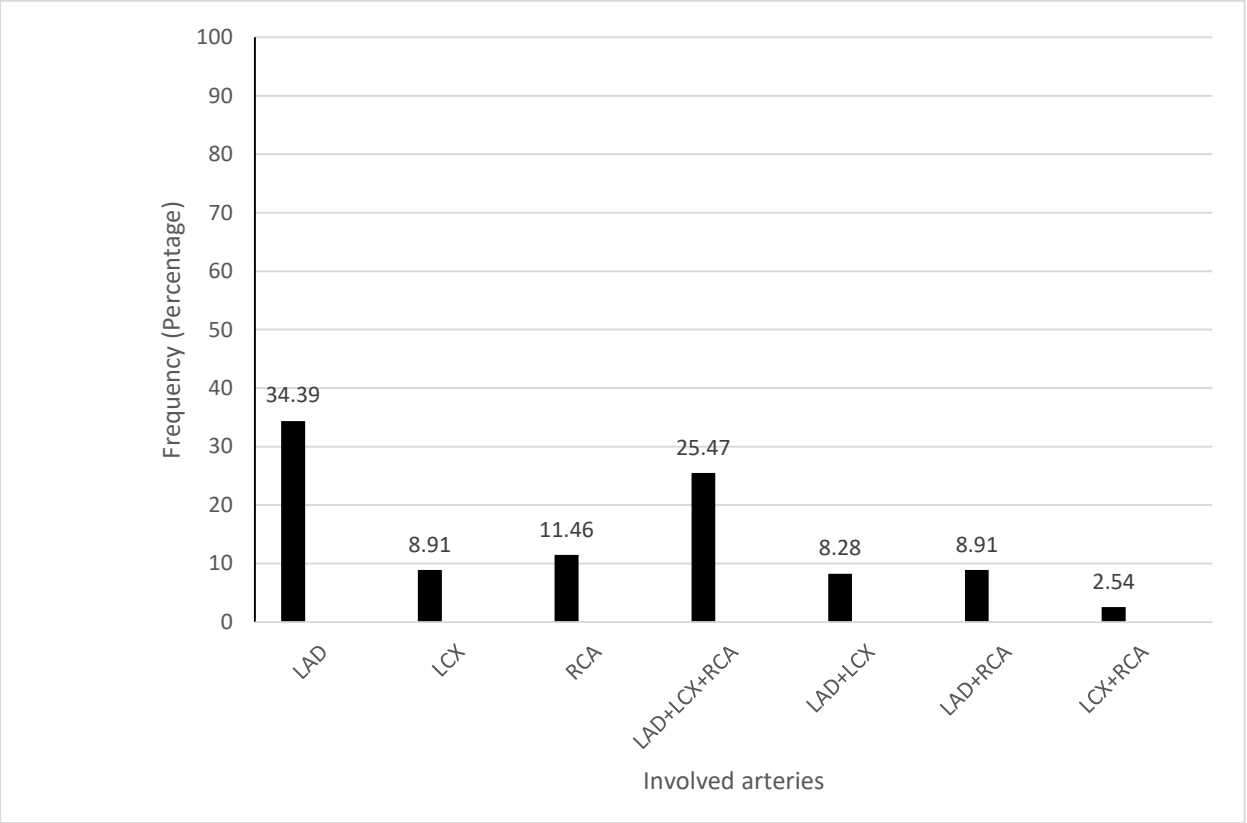

Figure 1: The figure showcases the distribution of the involvement of coronary arteries among the study population. LAD: left ascending coronary artery, LCX: left circumflex coronary artery, RCA: right coronary artery

Table 1: Baseline Characteristics and Coronary Risk Factors

Variable	CAE Group (n =157)	Non-CAE Group (n =156)	*P* value
Age, y	59.2±10.6	60.6±12.1	0.254
Male, n*%	(89) 56.7%	(120) 77.4%	<0.001
Diabetes mellitus	(50) 31.8%	(51) 32.7%	0.904
Hypertension	(88) 56.1%	(91) 58.3%	0.732
Dyslipidemia	(43) 27.4%	(77) 49.4%	<0.001
Smoking	(31) 19.7%	(62) 39.7%	<0.001

CAE: coronary artery ectasia

DISCUSSION

In the present study, the mean age was 59.2±10.6 years for the CAE group and 60.6±12.1 years for the non-CAE group, similar to the findings reported by Ozdemir et al[14] and Qin et al.[15]

There was a male predominance in both groups, with a significantly higher proportion in the non-CAE group. Men accounted for 56.7% (n =89) of our study population, which is lower than the percentage of male patients in the investigations by Ozdemir et al[14] (76%, n=158) and Qin et al[16] (70%, n=70).

The frequency of smoking in the non-CAE group was significantly higher than that in the CAE group (39.7% vs 19.7%; *P*<0.001), which is not compatible with previous studies. In a study by Gunes et al,[17] 41.8% of patients with CAE were smokers, as opposed to 29.6% of patients with obstructive CAE.

Discordant with many previous studies that have shown that the frequency of DM is lower in patients with CAE, we found a similar rate of DM in the CAE and non-CAE groups (31.8% vs 32.7%).

The rate of hypertension was similar in the CAE and non-CAE groups (56.1%). In a study by Qin et al,[15] the prevalence of hypertension was significantly higher in patients with CAE than in those with coronary stenosis (80% vs 63%). In our study, 27.4% of the CAE group had hyperlipidemia, significantly lower than that in the non-CAE group (49.4%). In many previous studies, the prevalence of hyperlipidemia is high by comparison with our findings.[18]

The NLR was significantly higher in the non-CAE group. This finding suggests that inflammation may play a more significant role in the development of obstructive CAD compared with CAE. Previous studies have shown that the NLR is a significant inflammatory marker that could also be considered a mortality predictor.[19]

CONCLUSIONS

We studied atherosclerosis risk factors in patients with CAE. Smoking and dyslipidemia were more prevalent in patients with 3VD, among whom the NLR was also significantly higher. The findings of this study and similar previous studies show that atherosclerosis risk factors and inflammatory factors could also be considered CAE risk factors.

Ethical Approval

The study was approved by the Ethics Committee of Iran University of Medical Sciences (Ethics ID: IR.IUMS.REC.1398.363).

REFERENCES

1. Demopoulos VP, Olympios CD, Fakiolas CN, Pissimissis EG, Economides NM, Adamopoulou E, et al. The natural history of aneurysmal coronary artery disease. Heart 1997; 78: 136-41.

2. Hartnell G, Parnell B, Pridie R. Coronary artery ectasia. Its prevalence and clinical significance in 4993 patients. Heart 1985; 54: 392-5.

3. Chugh R, Perloff JK, Fishbein M, Child JS. Extramural coronary arteries in adults with cyanotic congenital heart disease. The American journal of cardiology 2004; 94: 1355-7.

4. Abou Sherif S, Ozden Tok O, Taşköylü Ö, Goktekin O, Kilic ID. Coronary artery aneurysms: a review of the epidemiology, pathophysiology, diagnosis, and treatment. Frontiers in cardiovascular medicine 2017; 4: 24.

5. Mesquita A, Silva J, Seabra-Gomes R. Coronary artery ectasia: clinical and angiographic characteristics and prognosis. Revista portuguesa de cardiologia: orgao oficial da Sociedade Portuguesa de

Cardiologia= Portuguese journal of cardiology: an official journal of the Portuguese Society of Cardiology 1993; 12: 305-10.

6. Rashid S, Gul U, Ali M, Sadiq T, Kiyani AM. Coronary artery ectasia: clinical and angiographic features. J Coll Physicians Surg Pak 2018; 28: 824-8.

7. Sarli B, Baktir AO, Saglam H, Arinc H, Kurtul S, Sivgin S, et al. Neutrophil-to-lymphocyte ratio is associated with severity of coronary artery ectasia. Angiology 2014; 65: 147-51.

8. Swaye PS, Fisher LD, Litwin P, Vignola PA, Judkins MP, Kemp HG, et al. Aneurysmal coronary artery disease. Circulation 1983; 67: 134-8.

9. Giannoglou GD, Antoniadis AP, Chatzizisis YS, Damvopoulou E, Parcharidis GE, Louridas GE. Prevalence of ectasia in human coronary arteries in patients in northern Greece referred for coronary angiography. The American journal of cardiology 2006; 98: 314-8.

10. Saglam M, Karakaya O, Barutcu I, Esen AM, Turkmen M, Kargin R, et al. Identifying cardiovascular risk factors in a patient population with coronary artery ectasia. Angiology 2007; 58: 698-703.

11. Bermúdez P, Martínez-Luengas L. Coronary ectasia: prevalence, and clinical and angiographic characteristics. Revista espanola de cardiologia 2003; 56: 473-9.

12. Yang J-J, Yang X, Chen Z-Y, Wang Q, He B, Du L-S, et al. Prevalence of coronary artery ectasia in older adults and the relationship with epicardial fat volume by cardiac computed tomography angiography. Journal of geriatric cardiology: JGC 2013; 10: 10.

13. Sohn C, Kim J, Bae W. The framingham risk score, diet, and inflammatory markers in Korean men with metabolic syndrome. Nutrition research and practice 2012; 6: 246-53.

14. Ozdemir E, Safak O, AltIn MP, Akgun DE, Emren SV, AvcI E, et al. Correlation Between the Severity of Coronary Artery Ectasia and Monocyte/Lymphocyte, Platelet/Lymphocyte, and HDL/LDL Ratios. Journal of the College of Physicians and Surgeons Pakistan 2020; 30: 235-9.

15. Qin Y, Tang C, Ma C, Yan G. Risk factors for coronary artery ectasia and the relationship between hyperlipidemia and coronary artery ectasia. Coronary artery disease 2019; 30: 211-5.

16. Shi S, Qin M, Shen B, Cai Y, Liu T, Yang F, et al. Association of cardiac injury with mortality in hospitalized patients with COVID-19 in Wuhan, China. JAMA cardiology 2020.

17. Gunes Y, Boztosun B, Yildiz A, Esen AM, Saglam M, Bulut M, et al. Clinical profile and outcome of coronary artery ectasia. Heart 2006; 92: 1159-60.

18. Almansori MA, Elsayed HA. Coronary artery ectasia–A sample from Saudi Arabia. Journal of the Saudi Heart Association 2015; 27: 160-3.

19. Kaya H, Ertaş F, İslamoğlu Y, Kaya Z, Atılgan ZA, Çil H, et al. Association between neutrophil to lymphocyte ratio and severity of coronary artery disease. Clin Appl Thromb Hemost 2014; 20: 50-4. doi: 10.1177/1076029612452116