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Original article

A New Predictor of Obstructive Coronary Artery Ectasia in Patients with Non-ST-Elevation Acute Coronary Syndrome: The Atherogenic Index of Plasma

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ABSTRACT

Introduction: Coronary artery ectasia (CAE) is one of the uncommon cardiovascular disorders with a clinical spectrum ranging from asymptomatic cases to myocardial infarction. Atherosclerosis plays a pivotal role in the pathogenesis of CAE. Recently, it has been determined that the atherogenic index of plasma (AIP) is a strong predictive marker for atherosclerosis. The aim of this study was to investigate whether there is a relationship between obstructive CAE and AIP in patients with non-ST segment elevation acute coronary syndrome (NSTE-ACS).

Materials and methods: In this retrospective study, hospital electronic patient records were retrospectively examined. A total of 213 NSTE-ACS patients were included in the study. Patients were divided into two groups according to non-obstructive and obstructive CAE. The AIP value was compared between groups and regression analysis evaluated whether it is an indicator to predict the risk of obstructive CAE.

Results: The AIP value was found to be numerically and statistically significant in the obstructive CAE group compared to the non-obstructive CAE group. The multivariate logistic regression analysis identified AIP as a predictor of obstructive CAE in NSTE-ACS patients in the receiver operating curve analysis, AIP values above 0.33 had 90% sensitivity and 68% specificity to predict obstructive CAE in NSTE-ACS patients.

Conclusions: AIP values were increased in the presence of obstructive CAE in NSTE-ACS patients. Our findings suggest that AIP may be involved in the pathogenesis of obstructive CAE.

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Un nuevo predictor de ectasia obstructiva de la arteria coronaria en pacientes con síndrome coronario agudo sin elevación del segmento ST: el índice aterogénico del plasma

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RESUMEN

Introducción: La ectasia arterial coronaria (EAC) es uno de los trastornos cardiovasculares poco frecuentes con un espectro clínico que va desde casos asintomáticos hasta infarto de miocardio. La aterosclerosis juega un papel fundamental en la patogenia de la CAE. Recientemente, se ha determinado que el índice aterogénico del plasma (AIP) es un fuerte marcador predictivo de aterosclerosis. El objetivo de este estudio fue investigar si existe una relación entre el CAE obstructivo y la PAI en pacientes con síndrome coronario agudo sin elevación del segmento ST (SCASEST).

Materiales y métodos: En este estudio retrospectivo, se examinaron retrospectivamente las historias clínicas electrónicas de los pacientes. Se incluyeron en el estudio un total de 213 pacientes con SCASEST. Los pacientes se dividieron en dos grupos según el CAE obstructivo y no obstructivo. El valor de AIP se comparó entre grupos y el análisis de regresión evaluó si es un indicador para predecir el riesgo de EAC obstructivo.

Resultados: Se encontró que el valor AIP era numérica y estadísticamente significativo en el grupo CAE obstructivo en comparación con el grupo CAE no obstructivo. El análisis de regresión logística multivariable identificó a la AIP como predictor de EAC obstructiva en pacientes con SCASEST en el análisis de la curva operativa del receptor, valores de AIP superiores a 0,33 tenían una sensibilidad del 90 % y una especificidad del 68 % para predecir EAC obstructiva en pacientes con SCASEST.

Conclusiones: Los valores de AIP se incrementaron en presencia de EAC obstructivo en pacientes con SCASEST. Nuestros hallazgos sugieren que AIP puede estar involucrado en la patogenia de CAE obstructivo.

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1. INTRODUCTION

Coronary artery ectasia (CAE) is the dilatation of a coronary arterial segment to a diameter at least 1.5 times of the distal segment of the same coronary artery or adjacent coronary artery [1]. The incidence of CAE cases in angiographic examinations is 0.3% to 5.3%, and the presence of occlusive lesions has been rarely reported [2]. Damage to the coronary artery intima secondary to inflammation and cytokines released during the subsequent inflammation determine the clinical severity and prognosis of ectatic vessels [3]. Though the mechanism of CAE is unclear, it is known that inflammation, endothelial dysfunction, and vasculitides play a major role in the pathogenesis [4]. However, a complete consensus has not been reached on which patient is at risk of CAE and in which patient CAE may lead to an occlusive lesion.

Atherosclerosis is the primary contributing factor in CAE cases [5]. Coronary artery diseases (CADs) are the leading causes of mortality among cardiovascular diseases (CVDs), and undesirable cardiovascular events secondary to atherosclerosis are involved in their pathogenesis [6]. Lipids play a critical role in the process of atherogenesis and are known as strong risk factors for CVD [7]. Triglyceride (TG) and high-density lipoprotein-cholesterol (HDL-C) are lipids that can be readily measured in routine clinical practice. In recent studies, the atherogenic index of plasma (AIP) obtained by logarithmic TG to HDL-C ratio has been shown to be associated with hypertension (HTN), diabetes mellitus (DM) and heart diseases. The AIP value is associated with the size of pre- and anti-atherogenic lipoprotein particles and represents the authentic relationship between protective and atherogenic lipoproteins [8-10]. In addition, AIP is a novel marker for atherosclerosis and an independent risk factor for subclinical CAD [11]. Although a relationship between atherosclerosis and AIP has been demonstrated, there is no up-to-date study examining the relationship between AIP and obstructive CAE.

In the light of this information, the aim of our study was to

investigate whether there is a relationship between AIP and obstructive CAE.

2. MATERIAL AND METHODS

2.1. STUDY POPULATION

In this single-center study, patients hospitalized and treated for non-ST segment elevation acute coronary syndrome (NSTE-ACS) and CAE between January 2013 and June 2021 were retrospectively examined, and 213 patients were included in the study.

The diagnosis of non-ST-elevation MI (NSTEMI) was made according to the following criteria [12]:

1. Typical chest pain lasting 30 minutes or longer.
2. Positive for cardiac troponin.
3. Absence of ST segment elevation in any lead on superficial electrocardiogram.

Patients with a history of CAD, chronic kidney disease [estimated glomerular filtration rate<30 (mL/min/1.73m2)], abnormal liver dysfunction, recent diagnosis of stroke, active infection, malignancy, pathological Q wave on electrocardiogram, heart failure (left ventricular ejection fraction of ≤40%), moderate–severe heart valve disease, diagnosed cardiomyopathies, statin use, and those <18 years of age were not included in the study.

The physical examination and demographic as well as clinical characteristics of the patients were obtained from the electronic medical records. Laboratory parameters of blood samples taken from antecubital peripheral veins at the time of admission (such as troponin value, liver and kidney function tests, whole blood count, and coagulation parameters) and on the morning after admission, following at least 8 hours of overnight fasting (such as TG and HDL-C) were obtained from medical electronic records. AIP was calculated using the obtained data and the following formula: AIP=log (TG/HDL-C).

The study approval was obtained from the ethics committee of our university (Decision number: 2011-KAEK-27/2021-2100169939). The study was performed in accordance with the Declaration of Helsinki.

2.2. CORONARY ANGIOGRAPHY

Coronary angiographies (GE Healthcare Innova 2100, New Jersey, USA) were performed by an experienced cardiologist using the standard Judkins technique with the femoral or radial approach. Angiographic images were evaluated by two experienced cardiologists.

Before the procedure, 600 mg clopidogrel or 180 mg ticagrelor was administered in addition to 300 mg aspirin for antiplatelet therapy. The definition of responsible lesions was decided by evaluating different images. Very high-risk NSTEMI patients underwent coronary angiography within 2 hours, whereas the procedure was performed within 24 hours in the remaining NSTEMI patients. During the procedure, the guiding catheter was set to achieve TIMI 3 flow into the responsible coronary artery, and an intravenous heparin (70 U/kg) bolus was followed by direct stenting of the appropriate lesions or stenting after balloon dilatation. In all patients without contraindications, β blockers, angiotensin converting enzyme inhibitors or angiotensin receptor blockers, and statin therapy were initiated.

CAE was defined as a dilatation with a diameter of 1.5 times the normal epicardial coronary artery compared to the normal coronary artery [13]. Stenosis of ≥70% was defined as obstructive CAD.

2.3. CAE CLASSIFICATION

Ectatic vessels were classified according to the Markis classification. Diffuse ectasia of 2 or 3 coronary arteries was classified as type 1, diffuse ectasia in a single vessel accompanied by localized ectasia in another vessel was classified as type 2, diffuse ectasia in a single vessel was classified as type 3, and segmental localized ectasia was classified as type 4 [14]. The patients were divided into two groups as group 1 with non-obstructive CAE and group 2 with obstructive CAE.

2.4. STATISTICAL ANALYSIS

Kolmogorov–Smirnov test was used to evaluate the distribution of continuous variables. The data that did not conform to normal distribution are expressed as median and percentiles (25th and 75th percentiles). Categorical variables are expressed as percentages and numbers. Chi-square test was used when comparing the probability ratios of categorical variables. For the comparison of continuous variables between groups, Mann Whitney U test was used. Receiver operating characteristic (ROC) curve analysis was performed to calculate the optimum cut-off values, sensitivity and specificity for obstructive coronary ectasia of AIP. Finally, the explanatory power of AIP, demographic and clinical variables on obstructive CAE are evaluated by logistic regression. P values of <0.05 were considered statistically significant. Statistical data were obtained using SPSS 20.0 (SPSS Inc, Chicago, IL, USA).

3. RESULTS

Our study consisted of 213 newly diagnosed NSTE-ACS (152 males, 61 females) patients. Patients were divided into two groups according to non-obstructive (n=141) and obstructive CAE (n=72). The clinical data of the study patients are shown in Table 1. No differences were observed between clinical features including age, gender, DM, body mass index, and HTN. Considering the biochemical parameters, TG levels were statistically and numerically significant in patients with obstructive CAE compared to those without ($p < 0.001$).

obstructive CAE group compared to the non-obstructive CAE group (Table 1).

The incidence of obstructive CAE in patients with NSTE-ACS was equal in the left anterior descending coronary artery (20.8%) and right coronary artery (20.8%). According to the Markis classification, type 3 obstructive CAEs were most common in NSTE-ACS patients (Table 2).

Variables that were found to be significant in univariate regression analysis were included in the logistic regression analysis. AIP was considered as a predictor of obstructive CAE in newly diagnosed NSTE-ACS patients (Table 3).

In the receiver operating characteristic analysis, AIP values

Table 1: Demographic and laboratory findings of patients

Clinical characteristics	Non-Obstructive CAE (n=141)	Obstructive CAE (n=72)	p value
Age (years, mean)	65 (54-71.50)	65.50 (55.25-70)	0.999
Gender (female/male)	44/97	17/55	0.318
BMI (kg/m^2)	25 (24-27)	25 (23-26.75)	0.209
Heart rate (bpm)	79 (71-91)	79 (70.25-92)	0.706
DM (n, %)	4 (2.8)	6 (8.3)	0.091
HTN (n, %)	8 (5.7)	5 (6.9)	0.766
COPD (n, %)	5 (3.5)	2 (2.8)	1.000
Smoking (n, %)	3 (2.1)	5 (6.9)	0.123
ACE/ARB (n, %)	10 (7.1)	7 (9.7)	0.687
Statins (n, %)	6 (4.3)	3 (4.2)	1.000
Beta-blockers (n, %)	6 (4.3)	1 (1.4)	0.427
Biochemical variables	**Non-Obstructive CAE (n=141)**	**Obstructive CAE (n=72)**	**p value**
Glucose (mg/dL)	124 (106-138)	131 (112.5-141.75)	0.121
Creatinine (mg/d))	0.74 (0.65-0.81)	0.76 (0.66-0.80)	0.923
Hemoglobin (g/dL)	12.70 (11.8-14.1)	12.60 (11.3-13.7)	0.107
White blood cell count (x10^3 /mL)	7.50 (6-10.3)	7.75 (6.12-12.9)	0.265
Neutrophil count (x10^3 / mL)	3.50 (1.7-7.6)	3.50 (2.4-7)	0.722
Lymphocyte count (x10^3 / mL)	1.40 (0.80-1.80)	1.40 (0.82-2.1)	0.705
Triglyceride (mg/dL)	132 (105-142)	161 (132.25-174.75)	<0.001
HDL-C (mg/dL)	53 (41-57)	45 (39.25-54.75)	0.117
LDL-C (mg/dL)	143 (105.50-155)	152.50 (109-162.75)	0.095
Troponin (ng/m)	7.44 (5.80-9.41)	8.2 (6.16-9.52)	0.642
CRP (mg/L)	1.65 (1-6.33)	5 (1.26-6.47)	0.017
AIP	0.42 (0.33-0.51)	0.52 (0.39-0.61)	<0.001
Decision (n, %)	**Non-Obstructive CAE (n=141)**	**Obstructive CAE (n=72)**	**p value**
Medical follow-up	141		
PCI		61	
CABG		11	

BMI: Body mass index; DM: Diabetes mellitus; HTN: Hypertension; COPD: Chronic obstructive pulmonary disease; ACE: Angiotensin converting enzyme inhibitor; ARB: Angiotensin reseptor blocker; HDL-C: High-density lipoprotein cholesterol; LDL-C: Light-density lipoprotein cholesterol; CRP: C-reactive protein; AIP: Atherogenic Index of Plasma; PCI: Percutaneous coronary intervention; CABG: Coronary artery bypass grafting.

Though HDL-C and low-density lipoprotein-cholesterol (LDL-C) levels showed numerical differences between the groups, no statistical difference was observed. AIP values were numerically and statistically significant in the

above 0.33 showed 90% sensitivity and 68% specificity (area under the curve (AUC): 0.658, 95% CI: 0.581–0.734, $p < 0.001$) in terms of predicting obstructive CAE in NSTE-ACS patients (Figure 1). The distribution of the coronary

arteries based on the Markis classification and the presence of obstruction is shown in Figure 2.

Table 2: Angiographic characteristics of the study population			
Vessel (n, %)	**Non-Obstructive CAE (n=141)**	**Obstructive CAE (n=72)**	**Total (n=213)**
LAD	34 (24.19)	15 (20.8)	49 (23)
Cx	34 (24.1)	10 (13.9)	44 (20.7)
RCA	29 (20.6)	15 (20.8)	44 (20.7)
LAD-Cx	14 (9.9)	7 (9.7)	21 (9.9)
LAD-RCA	13 (9.2)	10 (13.9)	23 (10.8)
Cx-RCA	7 (5)	4 (5.6)	11 (5.2)
LAD-Cx-RCA	8 (5.7)	10 (13.9)	18 (8.5)
LMCA	2 (1.4)	1 (1.4)	3 (1.4)
Markis classification (n, %)	**Non-Obstructive CAE (n=141)**	**Obstructive CAE (n=72)**	**Total (n=213)**
Type 1	19 (13.5)	16 (22.2)	35 (16.4)
Type 2	21 (14.9)	15 (20.8)	36 (16.9)
Type 3	74 (52.5)	27 (37.5)	101 (47.4)
Type 4	27 (19.1)	14 (19.4)	41 (19.2)

LAD: Left anterior descending artery; Cx: Circumflex artery; RCA: Right coronary artery; LMCA: Left main coronary artery.

4. DISCUSSION

To the best of our knowledge, our study is the first of its kind to investigate the relationship between the presence of obstructive and non-obstructive ectasia and AIP in NSTE-ACS patients. This study concluded that the AIP value, which can be easily calculated, is associated with the presence of obstructive CAE and is a predictor of obstructive CAE risk.

Table 3: Analysis to identify the independent factors of obstructive coronary artery ectasia pattern			
Variables	**Odds Ratio**	**95% Confidence Interval**	**p-value**
Age	0.987	0.961-1.014	0.352
Gender	0.682	0.335-1.386	0.290
HTN	0.850	0.233-3.103	0.805
DM	0.273	0.069-1.083	0.065
COPD	1.241	0.213-7.245	0.810
CRP	0.932	0.865-1.005	0.068
Troponin	0.983	0.912-1.061	0.665
AIP	0.042	0.007-0.262	0.001

HTN: Hypertension; DM: Diabetes mellitus; COPD: Chronic obstructive pulmonary disease; CRP: C-reactive protein; AIP: Atherogenic Index of Plasma.

CAEs are uncommon coronary disorders and their pathophysiological mechanism is unclear [15]. As the pathophysiological mechanisms are not fully understood, there are no specific treatment methods and guideline recommendations to prevent the progression of the disease. CAEs may present with anginal attacks or ACS similar to CVDs [16, 17]. Anginal attacks are usually caused by slow and turbulent flow in dilated CAs, and turbulent blood flow may trigger the activation of atherogenic genes in the long term, leading to endothelial dysfunction and subsequent undesirable clinical outcomes, such as MI [18].

Dyslipidemia is a significant risk factor for atherosclerosis [19]. Plasma HDL-C levels have an inverse relationship with the risk of atherosclerosis [20]. Other studies have demonstrated the relationship between increased TG and LDL-C values and atherosclerotic processes [21, 22]. Given this information, it would be reasonable to hypothesize that CAEs, the pathogenesis of which is affected by atherosclerotic processes, are associated with dyslipidemia. HTN, advanced age, obesity, smoking, and hypercholesterolemia are among the major risk factors for CVDs [23, 24]. The formulas derived from the plasma lipid profile have been considered as predictors for CVD risk in recent years. In study conducted on CVD risk prediction with TG/HDL-C or LDL-C/HDL-C ratios, it has been shown that combined formulas are superior to a single lipid marker [25]. The main goal of various clinical study was to obtain a well-established predictor of the CVD risk instead of using the classical ratio [26]. In addition, it has been demonstrated that AIP is superior to HDL-C, LDL-C, and TG values in predicting the risk of CAD [27]. Therefore, AIP values may be related to obstructive CAE. Indeed, in

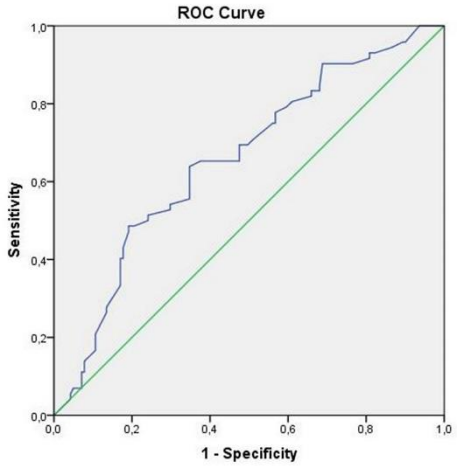

Figure 1: *Receiver operator characteristic curve of the AIP to predict obstructive CAE in patients with NSTE-ACS (90% sensitivity and 68% specificity AUC: 0.658, p<0.001).*

our study, AIP values were found to be significantly higher in the obstructive CAE group compared to those in the non-obstructive CAE group, whereas no differences were observed between the groups in HDL-C and LDL-C values. Moreover, we found that TG, an important parameter used in the calculation of AIP, was statistically and numerically significant in patients with obstructive CAE compared to those without. Hydrolysis of TGs by lipoprotein lipase yields TG-rich lipoprotein residues and fatty acids. Both lipolysis of fatty acids and lipoprotein residues have 4 times higher cholesterol transport capabilities [28]. Thus, it is likely that increased TG levels cause rapid progression of the atherosclerotic process and play an important role in the etiology of obstructive CAE.

The development of atherosclerotic plaque is associated with an increase in the small dense LDL-C (sdLDL-C) ratio. AIP is directly proportional to the increased number of sdLDL particles and inversely proportional to LDL-C particle size [29, 30]. In a previous study, the relationship between sdLDL and CAD has been demonstrated, and it has been shown that sdLDL plays a role in the atherosclerotic process [31]. Accordingly, increased AIP values in patients with obstructive CAE may be an indirect indicator of sdLDL values. In addition, the easy calculation of AIP may provide

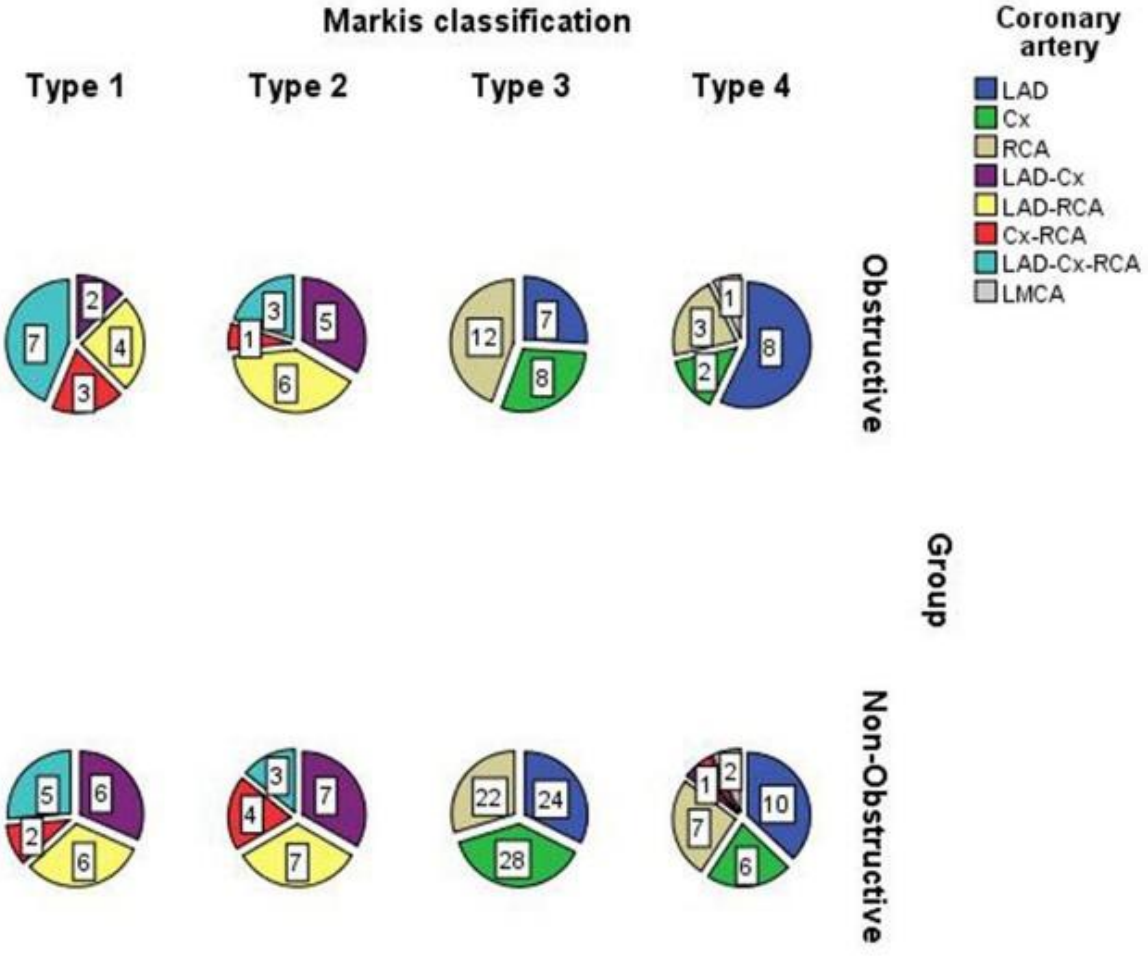

Figure 2: *The distribution of the coronary arteries based on the Markis classification and the presence of obstruction.*

clinicians with important cell-related knowledge, which may ultimately aid in treatment planning.

CAEs associated with atherosclerotic processes may also be be associated with inflammatory parameters. Despite the exclusion of infectious diseases in our study, C-reactive protein (CRP), a simple indicator of inflammation, was statistically higher in patients with obstructive CAE. However, CRP values were not significantly different on multivariate logistic regression analysis in NSTE-ACS patients with obstructive ectasia. Atherosclerotic processes are important in obstructive and non-obstructive CAE, and AIP values can indirectly offer the clinician remarkable insights into atherosclerotic processes.

Our study had some limitations. First of all, it was a single-center, retrospective study. Secondly, the study design precludes the understanding of the efficacy of primary preventive medical therapy in patients with non-obstructive ectasia. Our results were obtained based on the current clinical and laboratory results. Multicenter and prospective studies are needed to support the results of the present study and to eliminate its limitations. The relationship between sdLDL values and AIP may be a topic of investigation in prospective studies to improve our understanding of the atherosclerotic process in obstructive CAE.

5. CONCLUSIONS

Considering the findings in literature and of our study, we hypothesized that obstructive CAE lesions in NSTE-ACS patients may be associated with AIP. In particular, our study showed that obstructive CAE was independently and significantly associated with AIP values above 0.33 compared to non-obstructive CAE. AIP may be a useful parameter for atherosclerotic risk management in patients with obstructive and non-obstructive CAE.

6. CONFLICT OF INTERESTS

The authors have no conflict of interest to declare. The authors declared that this study has received no financial support.

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