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Research Article

The role of atherogenic index of plasma in coronary artery patients with high SYNTAXII score

Yüksek SYNTAXII skoru olan koroner arter hastalarında plazma aterojenik indeksinin rolü

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Abstract

Aim: We tried to evaluate the best diagnostic threshold value of the atherogenic index of plasma (AIP) with respect to coronary artery disease (CAD) and its relationship with SYNTAX II (SYNergy between percutaneous coronary intervention with TAXus and cardiac surgery) score (SSII).

Material and Methods: The research encompassed 280 consecutive patients with non-ST-segment elevation myocardial infarction (NSTEMI) undergoing coronary angiography, through which SSII was calculated based on initial coronary angiography by at least two independent cardiologists. The patients were separated into two groups low SSII (<23, n=116) and high SSII (≥23, n=164), while AIP was calculated by logarithmic conversion of triglyceride to high-density lipoprotein-to-cholesterol ratio.

Results: We determined some differences between the study groups in point of age, gender, smoking, hypertension, family history, diabetes mellitus, serum urea, C-reactive protein, hemoglobin level, left ventricular ejection fraction, and AIP (P <.05), which indicates a positive connection found between high SSII and AIP (r=0.343; p<0.001; statistically significant p<.05). As a result of multivariate logistic regression analysis, AIP was determined to be an independent risk factor for CAD and high SSII. In addition, we found that AIP values of 0.54 ng/ml and above could estimate the severity of coronary artery disease with 62.8% sensitivity and 60.5% specificity (area under the curve:0.676, %95 confidence interval, 0.613 – 0.739%; p<0.001).

Conclusion: AIP ratios were detected to be increased in patients with high SSII in comparison to those with low SSII. In addition, AIP was significantly independently connected with CAD and high SSII in the group with high SSII. In light of these findings, AIP, as a biomarker, may help prevent CAD.

Keywords: Atherogenic index of plasma, SYNTAX score II, acute coronary syndrome.

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Öz

Amaç: Koroner arter hastalığına (KAH) göre aterojenik plazma indeksinin (AIP) en iyi tanısal eşik değerini ve bunun SYNTAX II (SYNergy between percutaneous coronary intervention with TAXus and cardiac surgery) skoru (SSII) ile ilişkisini değerlendirmeye çalıştık.

Gereç ve Yöntemler: Araştırma, koroner anjiyografi uygulanan, ST segment yükselmesiz miyokard enfarktüsü (NSTEMI) olan ve en az iki bağımsız kardiyolog tarafından ilk koroner anjiyografiye dayalı olarak SSII'nin hesaplandığı ardışık 280 hastayı kapsamıştır. Hastalar düşük SSII (<23, n=116) ve yüksek SSII (≥23, n=164) olarak iki gruba ayrıldı. AIP, trigliseritin yüksek yoğunluklu lipoprotein-kolesterol oranına logaritmik dönüştürülmesiyle hesaplandı.

Bulgular: Çalışma grupları arasında yaş, cinsiyet, sigara kullanımı, hipertansiyon, aile öyküsü, diyabet, serum üre, C-reaktif protein, hemoglobin düzeyi, sol ventriküler ejeksiyon fraksiyonu ve AIP açısından bazı farklılıklar belirledik (P<.05), bu da yüksek SSII ile AIP arasında pozitif bir bağlantı bulunduğunu göstermektedir (r=0,343; p<0,001; istatistiksel olarak anlamlı p<0,05). Çok değişkenli lojistik regresyon analizi sonucunda AIP'nin KAH ve yüksek SSII için bağımsız bir risk faktörü olduğu belirlendi. Ayrıca 0,54 ng/ml ve üzerindeki AIP değerlerinin koroner arter hastalığının ciddiyetini %62,8 duyarlılık ve %60,5 özgüllükle tahmin edebildiğini bulduk (eğri altında kalan alan:0,676, %95 güven aralığı, %0,613 – 0,739; p <0,001).

Sonuç: Yüksek SSII'li hastalarda AIP oranlarının düşük SSII'li hastalara göre arttığı tespit edildi. Ayrıca yüksek SSII'li grupta AIP'nin KAH ve yüksek SSII ile anlamlı derecede bağımsız olarak ilişkili olduğu görüldü. Bu bulgular ışığında, bir biyobelirteç olarak AIP, KAH'ın önlenmesine yardımcı olabilir.

Anahtar kelimeler: Plazmanın aterojenik indeksi, SYNTAX II skoru, akut koroner sendrom.

Introduction

Acute myocardial infarction is a significant agent of mortality and morbidity, as well as one of the common public health problems seen worldwide. Atherosclerosis plays an important role in the emergence of most cardiovascular (CV) disorders [1,2], causing a progressive accumulation of fibrous tissue and cholesterol in the type of plaque, both resulting in contracting of the arterial lumen and being the primary factor of non-ST-segment elevation myocardial infarction (NSTEMI). Taking into consideration the fact that inflammation emerges as a constituent of the atherosclerotic process [3,4], it is now widely accepted that all developmental stages of atherosclerosis, including increased plaque instability, are mediated by inflammatory factors, resulting in clinical events such as unstable angina, myocardial infarction (MI), sudden death, and stroke [5,6].

Many types of research have revealed that dyslipidemia and inflammation are closely linked by the pathogenesis of atherosclerosis [7-9]. Low-density lipoprotein cholesterol (LDL-C) is considered an independent element and can be evaluated as a primary intervention target for coronary artery disease (CAD) [10-12], though the LDL-C target value has been an issue of discussion for a long [13,14]. Therefore, it is recommended to reduce LDL-C levels by 50% in clinical practice so as to eliminate CV risks [15].

The size of small, dense low-density lipoprotein (sdLDL), a subfraction of the LDL-C, has been widely investigated through research dealing with lipid metabolism [16,17]. Since sdLDL is assessed to be more atherogenic than floating LDL-C [17], it is widely evaluated as a risk agent for atherosclerosis and an indicator of CV disease [17,18]. However, its assessment is inadequate in clinical practice due to its complexity and high test cost. The logarithm of the molar ratio of triglyceride (TG) to high-density lipoprotein cholesterol (HDL-C) levels (log [TG/HDL]) was determined as the atherogenic index of plasma (AIP). Because AIP is indirectly strongly connected and reversely proportional to the diameter of LDL-C fragments and reflects sdLDL levels, it is referred to and used as a marker to predict cases of CAD and plasma atherosclerosis [19-21].

The SYNTAX II (SYNergy between cardiac surgery and percutaneous coronary intervention with TAXus) score (SSII) includes clinical variables, in addition to anatomical factors, to assess the extent and chance of developing CAD, and is mostly referred with the purpose of both coronary risk classification and evaluation of CAD prognosis [22]. Within this context, we evaluated the level of SSII and AIP in patients with NSTEMI encompassed in this study.

Material and Methods

Study Population

Totally 280 consecutive patients who applied to our emergency department with a first episode of NSTEMI were involved in the cross-sectional, single-center study from January 2021 to June 2022. Exclusion criteria include previous percutaneous coronary intervention (PCI) or coronary artery bypass grafting history, statin use, no fasting lipid blood results, presence of decompensated heart failure, severe liver and kidney diseases, autoimmune diseases, malignancies, hematological disorders, severe valve disease, inflammatory or infectious diseases. NSTEMI was diagnosed according to current guidelines [23,24].

Each patient was required to sign a written informed consent form, followed by the confirmation of the study protocol by the Bozok University Ethics Committee, Yozgat, Turkiye (Date/No:22 September 2017/189_2022.09.22_05). Thereby, we began to conduct the study protocol in line with the ethical guidelines of the 1975 Declaration of Helsinki.

The patients' age, gender, CV risk factors, and history of CAD were added to their files. Hypertension (HT) was determined as systolic blood pressure >140 mmHg and/or diastolic blood pressure >90 mmHg through two separate measurements or by means of any antihypertensive medication. In addition, we determined diabetes mellitus as fasting plasma glucose > 126 mg/dl or > 200 mg/dl via any antidiabetic drug use or any measurement.

Laboratory Measurements

Age, gender, CV risk factors, and laboratory analyses of all patients were received retrospectively from hospital medical documents [including complete blood count and standard biochemical parameters]. Values of lipid parameters were detected by means of the Beckman Coulter AU 5800 autoanalyzer subsequent to at least 12 hours of fasting, while AIP was calculated by the logarithmic transformation of the value of TG and HDL-k concentrations: log10[(TGx0,0113)/(HLDL-Cx0,0259)].

Angiographic Analysis

We also performed coronary angiography via the Standard Judkins technique (Expo; Boston Scientific Corporation, Natick, Massachusetts, USA) and Siemens Axiom Sensis XP device (Munich, Germany), displaying each coronary artery on at least two perpendicular planes. As well as performing PCI according to clinical practice standards through iopromide (low osmolarity and nonionic contrast agent), we digitally documented all coronary angiographic images so as to make a quantitative analysis.

Digital angiograms were evaluated by at least two detached and qualified interventional cardiologists, along with calculating SSII scores, which indicated no difference between interventional cardiologists in terms of value. They also used the online SSII Calculator version 2.1 (www.syntaxscore.com) in order to score each lesion in epicardial arteries ≥1.5 mm in diameter and producing ≥50% stricture. According to this scoring system, the study groups were separated into two groups the low SSII group with values below SSII 23 and the high SSII group with an SSII value of 23 and above.

Statistical Analysis

We also conveyed statistical analyses by means of IBM SPSS Statistics for Windows, version 25.0 (IBM Corp., Armonk, N.Y., USA), while assessing the normality of the distribution of continuous variables via the Kolmogorov-Smirnov test. Based on the distribution pattern, Mann–Whitney U test or independent sample t-test was applied to compare continuous variants between both groups. Categorical variants were given as frequency (percentage), while their comparisons were made via the Chi-Square test. We performed univariate and multiple variable logistic regression analyses in order to detect the independent markers of the emergence of high SxS II. Two-tailed Pearson correlation analyses were performed to analyze correlations between Syntax score II and AIP. The results were expressed as correlation coefficient (r) and p values. We evaluated the value of AIP via receiver operating characteristic curve (ROC) analysis, along with diagnosing the values of high SxS II. Two-tailed p-value <0.05 was assessed to be remarkably significant.

Results

Demographic, basic clinical, and laboratory characteristics of the study groups, consisting of 280 patients in total, are displayed in Table 1. A statistical AIP significant difference was detected between the two groups with respect to age, gender, smoking, HT, family history, diabetes mellitus (DM), serum urea, hemoglobin, serum C-reactive protein (CRP), left ventricular ejection fraction (LVEF) and AIP. However, according to univariate logistic regression analysis, age, gender, smoking, HT, family history, DM, serum urea, hemoglobin level, LVEF, and AIP levels were detected to be independent markers for the high SSII group. On the other hand, age, gender, LVEF, and AIP were determined as independent determinant values in the high SSII group based on the multivariate logistic regression analysis (Odds Ratio respectively (OR): 1.325, %95 confidence interval (CI): 1.178-1.490; p<0,001; OR: 0.164, %95 GA: 0.033-0.807; p=0.026; OR: 0.747, %95 CI: 0.658-0.849; p<0.001; OR: 2.683, %95 CI: 1.552-4.369; p<0.001; Table 2). It was also detected that there was a positive correlation between AIP and high SSII (r=0.343; p<0.001; Figure 1). In addition, we found that AIP values of 0.54 ng/ml and above could estimate the severity of coronary artery disease with 62.8% sensitivity and 60.5% specificity (area under the curve:0.676, %95 confidence interval, 0.613 – 0.739%; p<0.001); figure 2).

Table 1. Baseline clinical and laboratory parameters of the study population.

	SxS II < 23 (n=116)	SxS II ≥ 23 (n=164)	P
Age, years	52 (46-58)	64.50 (56-69.75)	<0.001
Gender, male, n (%)	102 (87.9)	112 (68.3)	<0.001
Smoking, n (%)	101 (87.1)	105 (64)	<0.001
Hypertension, n (%)	29 (25)	69 (42.1)	0.003
Family history, n (%)	34 (29.3)	30 (18.3)	0.031
Diabetes, n (%)	16 (13.8)	55 (33.5)	<0.001
Urea, (mg/dl)	30 (26-36)	38 (28.50-46)	<0.001
Creatinine, (mg/dl)	86 (72-98.75)	0.85 (0.71-1.02)	0.568
Uric acid, (mg/dl)	5.30 (4.60-6.30)	5.60 (4.60-6.50)	0.464
Sodium, (mEq/L)	135 (134-137)	136 (133-138)	0.834
WBC count, x103	10.45 (8.60-13.40)	10.45 (8.10-12.37)	0.344
Neutrophil count, x103	7.10 (5.22-10.05)	7.70 (5.32-9.70)	0.898
Lymphocyte count, x103	1.80 (1.30-2.80)	1.70 (1.12-2.40)	0.240
Monocyte count, x103	600 (500-800)	600 (400-800)	0.277
Hemoglobin, g/dl	14.55 (13.52-15.20)	13.90 (12.90-15.10)	0.005
Platelet count, x103/mm3	235.50 (202-277)	228.50 (195-269.75)	0.400
Total cholesterol, (mg/dl)	187.50 (163.25-280.75)	190 (157-215)	0.837
Triglyceride, (mg/dl)	136.50 (77.25-182.50)	132.50 (84-194)	0.141
LDL, (mg/dl)	117.50 (103.75-141)	122.50 (100-152)	0.685
HDL, (mg/dl)	44.50 (40-52)	32 (25-37)	0.115
CRP, (mg/L)	0.42 (0.20-0.67)	0.48 (0.32-1)	0.017
EF, (%)	50 (48-55)	44 (35-50)	<0.001
AIP	0.44 ± 0.30	0.64 ± 0.32	0.003

Results are expressed as mean ± SD or median (IQR) or frequency (%), SS, SYNTAX score, WBC: white blood cells, LDL: low-density lipoprotein cholesterol, HDL: high-density lipoprotein cholesterol, CRP: C-reactive protein, EF: Ejection fraction, AIP: atherogenic index of plasma (log(TG/HDL-C).

Table 2. Univariate and multiple variate logistic regression analysis shows the independent predictors of the presence of Syntax Score II ≥23.

	Univariate				Multiple variate			
		95% CI				95% CI		
	OR	Lower	Upper	P	OR	Lower	Upper	P
Age	1.145	1.107	1.185	<0.001	1.325	1.178	1.490	<0.001
Gender (male)	0.314	0.163	0.602	<0.001	0.164	0.033	0.807	0.026
Smoking	0.264	0.141	0.496	<0.001	0.761	0.137	4.233	0.755
Hypertension	2.179	1.293	3.673	0.003	0.360	0.090	1.442	0.149
Family history	0.540	0.308	0.948	0.032	0.779	0.183	3.318	0.736
Diabetes	3.154	1.698	5.859	<0.001	0.774	0.185	3.240	0.725
Urea	1.072	1.043	1.102	<0.001	0.983	0.905	1.068	0.683
Hemoglobin	0.801	0.684	0.938	0.006	0.903	0.588	1.386	0.641
CRP	1.479	0.928	2.359	0.100	1.284	0.546	3.019	0.567
EF	0.871	0.837	0.905	<0.001	0.747	0.658	0.849	<0.001
AIP	4.132	1.963	8.438	<0.001	2.683	1.552	4.369	<0.001

CRP: C-reactive protein, EF: Ejection fraction, AIP: atherogenic index of plasma (log(TG/HDL-C).

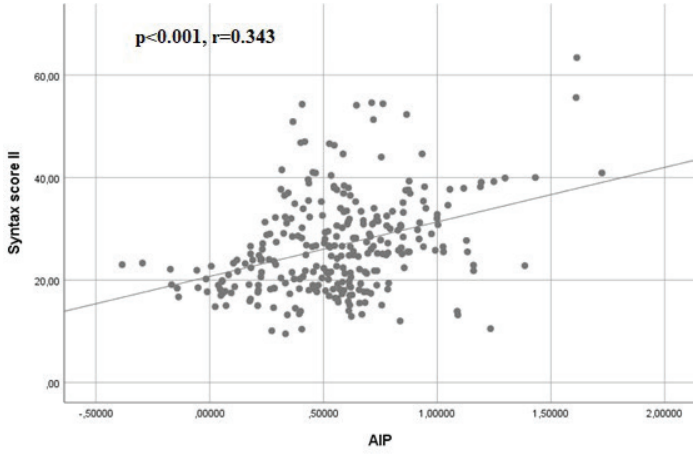

Figure 1. Relationship between Syntax score II and atherogenic index of plasma (AIP)

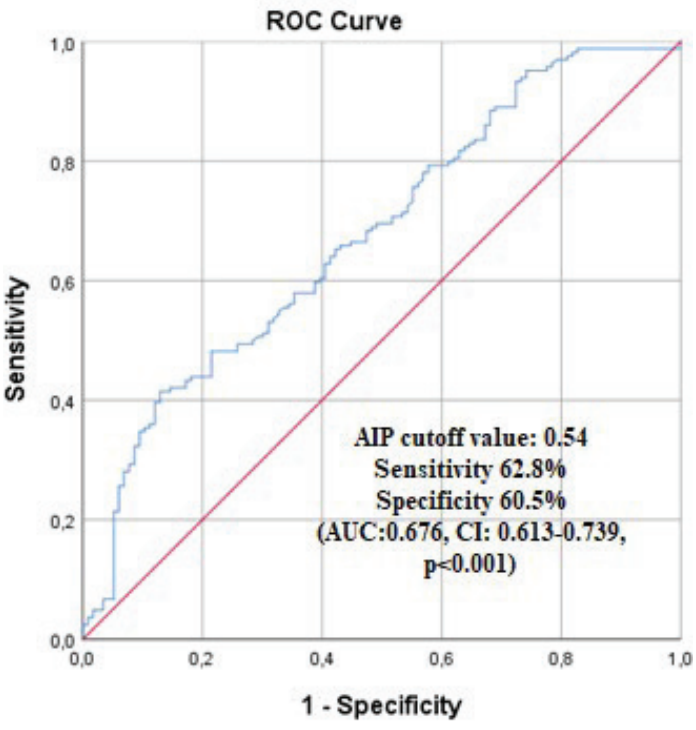

Figure 2. Receiver operating characteristic (ROC) curve for atherogenic index of plasma as a predictor of high syntax score II.

Discussion

AIP and high SSII were shown to be positively correlated according to the findings of the study. In addition, AIP was determined as the independent predictive value for High SSII. This study revealed that AIP as a comprehensive lipid index can be a strong marker for the risk of CAD. In light of these data, AIP may provide clinical utility in terms of examining the emergence of CAD in the following studies.

Since lipid metabolism disorder is considered a significant mechanism for the emergence and progress of CAD [10], most cases of dyslipidemia are triggered by low LDL-C and high TG. The fact that AIP is indirectly closely related to the diameter of LDL-C particles and indicates sdLDL levels makes it a more compatible index with the properties of blood lipids [19].

Studies show that smaller particle size promotes arterial wall penetration and deposition as an efficient cause of atherosclerosis. Therefore, the occurrence of plaque is considered a significant sensitive marker of CAD and atherosclerosis [17]. However, due to high costs and complicated techniques, measuring the ratios of sdLDL is rarely applied in today's medical treatments. AIP has been suggested to be adversely linked to the size of LDL particles. Thus, AIP can, in part, help as a proxy for sdLDL to evaluate potential plasma atherogenicity [19,25,26].

Moreover, numerous relevant studies have revealed that AIP is associated with obesity, HT, DM, insulin resistance, metabolic syndrome, the severity of CAD, and the incidence of acute coronary syndrome [27-29]. AIP may therefore provide a potential direction for CV risk research and the development of early detection, treatment, and intervention strategies for CAD in such patients.

It is confirmed through this study that AIP can be remarkably increased in high-risk CAD patients. Moreover, multivariate logistic regression analysis suggested that AIP can serve as an independent risk agent for CAD, as long as adjusted about age, gender, smoking, HT, family history, DM, serum urea, hemoglobin, serum CRP level, and LVEF (OR: 2.683, %95 CI: 1.552-4.369; P < 0.001). Çerik et al. In his study, CRP was found to be significantly higher in obstructive coronary ectasia than in isolated coronary ectasia [30]. CRP, an inflammatory marker, was also found to be statistically significant in our study (p=0.017).

The SSII is used for risk stratification of patients with CAD and to lead clinic staff into conducting an improved revascularization process. It also provides support for individualized treatment. Since AIP was detected to be remarkably increased in the high SSII group in comparison to the low SSII group, it may allow physicians to assess coronary artery severity early and non-invasively in CAD.

Study Limitations

Although AIP was determined to be positively correlated with high SSII in patients with acute coronary syndrome, this research has some limitations, such as the existence of a single center and therefore a small sample size, no follow-up data available due to its cross-sectional design, not being prospective and multi center, and not including other individual risk factors.

Conclusion

The atherogenic index of plasma can be suggested as an independent risk agent for CAD and high SSII. Therefore, it can be used as a diagnostic biomarker for early diagnosis of CAD and initiation of preventive treatment methods.

Declaration of Conflicting Interests

None.

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