Correlation between the AHCPR (Agency For Health Care Policy and Research) risk stratification and angiographic morphology in non-ST-segment elevation acute coronary syndrome

ST yükselmesiz akut koroner sendromlu hastalarda Amerikan Sağlık Politikaları ve Araştırmaları Dairesi (AHCPR) risk sınıflaması ile anjiyografik morfoloji arasındaki ilişki

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ABSTRACT

Objectives: Risk stratification in acute coronary syndromes is an important diagnostic tool guiding future therapy. We evaluated the correlation between the AHCPR (Agency for Health Care Policy and Research) risk classification and angiographic morphology in patients with non-ST-segment elevation acute coronary syndrome (NSTE-ACS).

Study design: A total of 163 patients hospitalized with the diagnosis of NSTE-ACS were prospectively enrolled. All the patients underwent AHCPR risk analysis followed by coronary angiography. Based on the AHCPR system, the patients were classified as low (n=25, mean age 55±10 years), intermediate (n=55, mean age 58±10 years), and high (n=83, mean age 61±11 years) risk groups.

Results: The three groups were similar with regard to gender, age, and coronary heart disease risk factors (p>0.05). Comparison of the high-risk group with intermediate+low-risk group with regard to lesion morphology showed significantly higher rates of complex lesions (31.9% *vs.* 4.0%, p=0.001), total occlusion (23.2% *vs.* 0%, p=0.001), and intracoronary thrombosis (13% *vs.* 2%, p=0.02) in the high-risk group. In univariate analysis, high risk was significantly associated with the presence of complex lesion, total occlusion, intracoronary thrombosis, and TIMI flow <III. Of these, only the presence of complex lesion (p=0.005) and TIMI flow <III (p=0.02) were associated with high risk in multivariate analyses.

Conclusion: Our findings show that the incidence of high-risk coronary morphology is increased in NSTE-ACS patients having a high-risk profile according to the AHCPR classification.

ÖZET

Amaç: Akut koroner sendromlu (AKS) hastalarda risk sınıflaması tedavinin seçimi açısından önemlidir. Bu çalışmada ST yükselmesiz AKS'li hastalarda Amerikan Sağlık Politikaları ve Araştırmaları Dairesi (Agency for Health Care Policy and Research-AHCPR) risk sınıflaması ile anjiyografik morfoloji arasındaki ilişki araştırıldı.

Çalışma planı: Çalışmaya, ileriye dönük olarak, ST yükselmesiz AKS tanısı konan 163 hasta alındı. Tüm hastalara AHCPR risk değerlendirmesi yapıldı ve koroner anjiyografi yapıldı. AHCPR risk değerlendirme sistemine göre hastalar, düşük (n=25, ort. yaş 55±10), orta (n=55, ort. yaş 58±10) ve yüksek (n=83, ort. yaş 61±11) risk gruplarına ayrıldı.

Bulgular: Gruplar arasında cinsiyet, yaş ve koroner kalp hastalığı risk faktörleri açısından anlamlı fark yoktu (p>0.05). Lezyon morfolojisi açısından yüksek riskli grubun, düşük+orta riskli grupla karşılaştırılmasında, yüksek riskli grupta kompleks lezyon (%31.9 ve %4.0, p=0.001), tam tıkanıklık (%23.2 ve %0, p=0.001) ve intrakoroner trombüs (%13 ve %2, p=0.02) sıklıkları anlamlı derecede fazla bulundu. Tek değişkenli analizde, kompleks lezyon, tam tıkanıklık, intrakoroner trombüs ve TIMI <III akım olması yüksek riskle anlamlı ilişki gösterdi. Çokdeğişkenli analizde ise, kompleks lezyon (p=0.005) ve TIMI <III akım (p=0.02) varlığı yüksek risk ile anlamlı ilişki gösterdi.

Sonuç: Bulgularımız, AHCPR risk sınıflamasına göre yüksek risk grubunda değerlendirilen ST yükselmesiz AKS'li hastalarda yüksek riskli koroner morfoloji sıklığının da arttığını göstermiştir.

Received: August 14, 2009 *Accepted:* July 22, 2010

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A number of angiographic and angioscopic studies have demonstrated that acute coronary syndrome develops when the vulnerable or high-risk plaque undergoes disruption of the fibrous cap, disruption of the plaque being the stimulus for thrombogenesis.[1-12] After disruption of a vulnerable or high-risk plaque, reduction in the flow may be caused by a completely or subtotally occlusive thrombus.

Non-ST-segment elevation acute coronary syndromes are heterogeneous disorders associated with an increased risk for myocardial infarction and cardiac death.[13] Based on the evaluation encompassing medical history, physical examination, electrocardiography, and biochemical markers, several classifications have been recommended for the identification of the risk and selection of the management strategy in patients with NSTE-ACS.[14-19] In 1994, the Agency for Health Care Policy and Research published a definitive guideline for the diagnosis and management of unstable angina.[20] In a stepwise approach, the guideline stratifies patients with unstable angina into low, intermediate, and high risk subgroups based on the likelihood of coronary artery disease and the short-term risk for MI or death. The AHCPR guidelines have been validated in a population-based registry with regard to short-term prognosis and angiographic extent of CAD.[21,22] However, it is unknown whether these guidelines are useful in predicting the lesion morphology of CAD as assessed by coronary angiography.

The purpose of the present study was to assess the correlation between the AHCPR risk classification and angiographic morphology in patients with NSTE-ACS.

PATIENTS AND METHODS

Patients

A total of 163 patients admitted to the emergency department of our institution with NSTE-ACS were prospectively enrolled into the study. All patients underwent emergency, early or elective coronary angiography.

A two-step evaluation was performed on admission to the emergency department. First, angina pectoris was investigated as a possible cause of ischemic heart disease.[13] Patients with a high or intermediate risk for ischemic heart disease were included in the study with an initial diagnosis of NSTE-ACS. In the second step, patients underwent AHCPR risk analysis for early in-hospital death or non-fatal MI (Table 1).[13] All patients gave written informed consent to participate in the study and the study protocol was approved by the ethical committee.

Abbreviations:

ACS	*Acute coronary syndrome*
AHCPR	*Agency for Health Care Policy and Research*
CAD	*Coronary artery disease*
ECG	*Electrocardiography*
MI	*Myocardial infarction*
NSTE	*Non-ST-segment elevation*
PCI	*Percutaneous coronary intervention*

Cardiac catheterization

Emergent or early invasive intervention was performed for patients with refractory or recurrent ischemia, arrhythmia, or hemodynamic impairment. Selective coronary angiography and left ventriculography were performed in all patients using the Judkins technique. During ventriculography, 30-degree right anterior oblique and 45-degree left anterior oblique images were obtained. Then, left and right coronary angiographies and graft angiographies (arterial or venous) were performed at various positions.

Coronary angiographies were evaluated by two investigators blinded to the clinical features of the patients, and the vascular disease and ischemia-related artery were identified. Stenoses of greater than 50% and 70% were regarded as significant and critical, respectively. Patients were grouped as follows: normal coronary arteries, left main coronary artery disease, single-vessel disease, two-vessel disease, and triple-vessel disease.

Identification of the ischemia-related artery

The ischemia-related artery was the diseased vessel when only one vessel was involved. In case of multivessel disease, it was identified based on the coronary anatomy and localization of ECG change during chest pain. Coronary lesions with at least >70% visual diameter stenosis were regarded as the potential culprit lesion. When the patient had normal coronary arteries, or when the diseased vessel had less than 70% stenosis or in the presence of multiple coronary arteries responsible for ischemia (i.e. when the index artery could not be distinguished by ECG changes), the patient was excluded from subsequent analyses on the grounds that a single culprit lesion was not identified. In case of more than one obstructive lesion, the culprit lesion was identified on the basis of stenosis severity and/or presence of complex morphology (intracoronary thrombosis or total occlusion).[23]

Table 1. Short-term risk for death or nonfatal myocardial infarction in patients with unstable angina/non-ST-elevation myocardial infarction

Feature	High risk (At least 1 of the following features must be present)	Intermediate risk (No high-risk feature, but must have 1 of the following)	Low risk (No high- or intermediate-risk feature, but may have any of the following)
History	Accelerating tempo of ischemic symptoms in the preceding 48 hours	Prior MI, peripheral or cerebrovascular disease, or CABG; prior aspirin use	
Character of pain	Prolonged ongoing (>20 min) rest pain	• Prolonged (>20 min) rest angina, now resolved, with moderate or high likelihood of CAD • Rest angina (>20 min) or relieved with rest or sublingual nitroglycerin • Nocturnal angina • New-onset or progressive CCS class III or IV angina in the past 2 weeks without prolonged (>20 min) rest pain, but with intermediate or high likelihood of CAD	• Increased angina frequency, severity, or duration • Angina provoked at a lower threshold • New-onset angina with onset 2 weeks to 2 months prior to presentation
Clinical findings	• Pulmonary edema, most likely due to ischemia • New or worsening mitral regurgitation murmur • S_3 or new/worsening rales • Hypotension, bradycardia, tachycardia • Age >75 years	Age >70 years	
Electrocardiogram	• Angina at rest with transient ST-segment changes >0.5 mm • Bundle-branch block, new or presumed new • Sustained ventricular tachycardia	• T-wave changes • Pathological Q waves or resting ST-depression <1 mm in multiple lead groups (anterior, inferior, lateral)	Normal or unchanged ECG
Cardiac markers	Elevated cardiac TnT, TnI, or CK-MB (i.e., TnT or TnI >0.1 ng/ml)	Slightly elevated cardiac TnT, TnI, or CK-MB (i.e., TnT >0.01, but <0.1 ng/ml)	Normal

CABG: Coronary artery bypass graft surgery; CAD: Coronary artery disease; CCS: Canadian Cardiovascular Society; CK-MB: Creatine kinase, MB fraction; ECG: Electrocardiogram; MI: Myocardial infarction; TnI: Troponin I; TnT: Troponin T.

Coronary morphology

The following features of the ischemia-related artery were evaluated:

1. Culprit lesion (eccentric, simple lesion, complex lesion, intracoronary thrombosis, total occlusion): The culprit lesion was categorized as either simple or complex.[23] A lesion was defined as complex when it was associated with intracoronary filling defect (thrombus appearance) and/or ulcerative, irregular borders, and overhanging edges.[8,24]

A proximal or distal filling defect of the culprit lesion, with at least three sides surrounded with contrast agent and displayed at multiple projections was defined as 'intracoronary thrombus'.[8] Eccentric lesion was defined as asymmetrical narrowing of a coronary artery.[24]

The TIMI (Thrombolysis In Myocardial Infarction) classification system was used for the description of intracoronary flow,[25] where absence of flow distal to the artery was regarded as total occlusion (TIMI 0).

2. Classification of the culprit lesion (type A, B, and C): This was a definition made to designate the suitability of the culprit lesion for intervention prior to revascularization by percutaneous coronary intervention, where complexity of the lesion increased from type A

Table 2. Demographic and basic clinical characteristics of NSTE-ACS patients by AHCPR risk groups

	Low risk group (Group A, n=25) (mean age 55±10 yr)		Intermediate risk group (Group B, n=55) (mean age 58±10 yr)		High risk group (Group C, n=83) (mean age 61±11 yr)		
	n	%	n	%	n	%	*p*
Sex							NS
Male	18	72.0	38	69.1	62	74.7	
Female	7	28.0	17	30.9	21	25.3	
Smoking	15	60.0	32	58.2	44	53.0	NS
Heredity	2	8.0	11	20.0	24	28.9	NS (0.06)
Hyperlipidemia	15	60.0	29	52.7	40	48.2	NS
Diabetes mellitus	9	36.0	11	20.0	26	31.3	NS
Hypertension	13	52.0	21	38.2	40	48.2	NS
Previous MI	0		16	29.1	40	48.2	**0.03** (A *vs.* C) **0.03** (B *vs.* C)
Previous revascularization	0		11	20.0	17	20.5	NS
Aspirin use	0		24/42	57.1	30/54	55.6	**0.02** (A *vs.* C) **0.02** (B *vs.* C)
Beta blocker use	8/16	50.0	10/42	23.8	13/54	24.1	NS (0.09)
Elevated cardiac TnT, TnI, or CK-MB (i.e., TnT or TnI >0.01 ng/ml)	0		35	63.6	60	72.3	**0.01** (A *vs.* C) **0.01** (B *vs.* C)

NSTE-ACS: Non-ST-segment elevation acute coronary syndrome; AHCPR: Agency for Health Care Policy and Research; MI: Myocardial infarction; Revascularization (PCI or CABG); TnI: Troponin I; TnT: Troponin T; CK-MB: Creatine kinase, MB fraction; NS: Not significant (p>0.05).

to C, the latter denoting more complex lesions including diffuse stenosis and extended total occlusion.[26]

Statistical analysis

All parameters were categorized as numeric or categorical, and analyzed using Epi Info 2000 and SPSS/PC+ 10.0 statistical software. For analyses, the chi-squre test, Fisher's exact chi-square test, Student's t-test, analysis of variance, and Tukey test were used, where appropriate. Total occlusion, intracoronary thrombus, complex lesion, and TIMI flow <III were analyzed by multiple logistic regression. A *p* value of less than 0.05 was considered significant.

RESULTS

Based on the AHCPR risk classification system, the patients were classified as low (group A, n=25), intermediate (group B, n=55), and high (Group C, n=83) risk groups.

Demographic characteristics

Demographic characteristics of the patients are summarized in Table 2. The mean ages were 55±10, 58±10, and 61±11 years in groups A, B, and C, respectively. Although increased age is presumed to be associated with increased risk, there was no statistically significant difference between the three groups with regard to age (p=0.053). The groups were also similar in terms of gender (p>0.05).

The groups did not also differ in terms of coronary heart disease risk factors including smoking, hyperlipidemia, family history of coronary heart disease (heredity), hypertension, and diabetes mellitus. They also had similar heart rate and blood pressure values on admission.

Clinical characteristics

During hospitalization, the rates of recurrent ischemia were 20% (n=5), 38.2% (n=21), and 49.4% (n=41) in groups A, B, and C, respectively, with significantly higher rates in groups B and C compared to group A (p=0.04 and p= 0.02, respectively). Cardiac markers were positive in 58.3% (n=95) of the study patients, all of whom were in groups B (63.6%) and C (72.3%) (Table 2). Unstable angina pectoris and non-ST elevation MI were seen in 41.7% (n=68) and 58.3% (n=95) of the patients, respectively.

Table 3. Angiographic findings of NSTE-ACS patients by AHCPR risk groups

	Low risk group (Group A, n=25)		Intermediate risk group (Group B, n=55)		High risk group (Group C, n=83)		
	n	%	n	%	n	%	*p*
Normal coronary arteries	11	44.0	12	21.8	4	4.8	**0.01** (A *vs.* B) **0.002** (A *vs.* C)
Unidentified ischemia-related artery	3	12.0	4	7.3	10	12.1	NS
Lesion type							
Type A	7/11	63.6	14/39	35.9	17/69	24.6	**0.01** (A *vs.* B) **0.002** (A *vs.* C)
Type B+C	4/11	36.4	25/39	64.1	52/69	75.4	**0.002** (A *vs.* C)
LAD lesion	8/11	72.7	25/39	64.1	35/69	50.7	NS
Proximal lesion	8/11	72.7	21/39	53.9	35/69	50.7	NS
Multi-vessel disease	8	32.0	29	52.7	61	73.5	**0.01** (A *vs.* B) **0.002** (A *vs.* C)
Number of vessels (Mean±SD)	1.1±1.0		1.4±0.9		2.1±0.8		**0.01** (A *vs.* C)
TIMI flow <III	2/11	18.2	9/39	23.1	33/69	47.8	**0.001** (A *vs.* C) **0.01** (B *vs.* C)
Presence of collaterals	1/11	9.1	4/39	10.3	17/69	24.6	**0.02** (A *vs.* C)

NSTE-ACS: Non-ST-segment elevation acute coronary syndrome; AHCPR: Agency for Health Care Policy and Research; TIMI Flow: Thrombolysis In Myocardial Infarction; LAD: Left anterior descending artery NS: Not significant (p>0.05).

Coronary angiography

All patients underwent coronary angiography within 20 days of admission (mean 5 days). Angiographic findings of the patients are summarized in Table 3. A total of 27 patients (16.6%) had normal coronary arteries, with more patients in group A (44%) compared to group B (21.8%, p=0.01) and C (4.8%, p=0.002). Patients with multivessel disease were less frequent in group A (32%) compared to group B (52.7%, p=0.01) and group C (73.5%, p=0.002).

Ischemia-related artery could not be identified in 12%, 7.3%, and 12.1% of the patients in group A, B, and C, respectively (p>0.05). When the lesion type of ischemia-related artery is considered, type A lesion was more frequent in group A (63.6%) compared to group B (35.9%, p=0.01) and group C (24.6%, p=0.002). Type B+C lesion was seen in 36.4%, 64.1%, and 75.4% of group A, B, and C patients, respectively, with a significantly higher frequency in group C than group A (p=0.002).

TIMI flow <III was found in 18.2% in group A, 23.1% in group B, and in 47.8% in group C, where group C showed a significantly higher rate compared to group A (p=0.001) and group B (p=0.01) (Table 3).

The lesion morphology in the ischemia-related artery was examined among patients with and without high risk (group C *vs.* group A+B) (Table 4). Patients in the high-risk group showed a higher frequency of complex lesions compared to intermediate+low-risk group (31.9% *vs.* 4.0%, p=0.001) and higher rates of total occlusion (23.2% *vs.* 0%, p=0.001) and intracoronary thrombosis (13% *vs.* 2%, p=0.02), whereas frequencies of eccentric lesions were similar (21.7% *vs.* 34%, p=0.1).

Table 5 presents univariate and multivariate analyses of angiographic findings in relation with high risk. In univariate analysis, high risk was significantly associated with the presence of complex lesion, total occlusion, intracoronary thrombosis, and TIMI flow <III. Of these, only the presence of complex lesion (p=0.005) and TIMI flow <III (p=0.02) were associated with high risk in multivariate analyses.

Treatment

Percutaneous or surgical revascularization was undertaken in 40% (n=10), 65.5% (n=36), and 84.3% (n=70) of patients in the low-, intermediate-, and high-risk groups, respectively. The rate of emergent revascular-

Table 4. Distribution of angiographic findings of the ischemia-related artery

	Low and Intermediate risk groups (Group A+B, n=80)		High risk group (Group C, n=83)		
	n	%	n	%	*p*
Eccentric lesion	17/50	34.0	15/69	21.7	NS
Total occlusion	0		16/69	23.2	**0.001**
Intracoronary thrombus	1/50	2.0	9/69	13.0	**0.02**
Complex Lesion*	2/50	4.0	22/69	31.9	**0.001**

*Complex lesion with intracoronary filling defect (thrombus appearance) and/or ulcerative, irregular borders and overhanging edges; NS: Not significant (p>0.05).

ization was 10.8% in the high-risk group and 1.3% in the low+intermediate-risk group (p=0.02).

DISCUSSION

Many studies have demonstrated that the pathogenesis of NSTE-ACS is associated with atherosclerotic plaque disruption and presence of complex lesions (total occlusion, intracoronary thrombosis) on angiography, similar to other acute coronary syndromes.[1-11,24] Angiographic complexity of the lesions gradually increases from stable angina to unstable angina pectoris, Q-wave MI, and non-Q-wave MI.[24,27]

Unstable angina pectoris represents a heterogeneous group of disorders and many studies have examined the relation between the Braunwald classification and angiographic morphology. According to the Braunwald classification based on clinical presentation (Table 6), class III (resting angina within the last 24 hours), class C (post-MI angina), and class 3 (angina despite maximum anti-ischemic treatment) patients are accepted as having severe unstable angina pectoris.[12] Dangas et al.[28] examined the relation between the severity of clinical presentation and presence of complex lesion or intracoronary thrombosis and found that class III disease was associated with complex lesions and reduced TIMI flow (<III), while class 3 angina was associated with intracoronary thrombosis. Many other studies have supported these findings.[23,29,30] Patients admitted with ongoing angina at rest are categorized as class III according to the Braunwald classification, whereas they are classified as having high risk based on the AHCPR classification. In our study, the high-risk group was associated with reduced TIMI flow, and the presence of intracoronary thrombus, total occlusion, and complex lesion. These findings are in line with previous studies on clinical classification of angina.

Besides the Braunwald classification, the associations between coronary artery morphology and ECG or biochemical findings were also examined in patients with NSTE-ACS. The presence of ST depression on admission has been associated with angiographically high-risk lesions (complex lesion, intracoronary thrombosis, and total occlusion).[23,30] In our study, the frequency of ST depression was 0%, 2.9% and 97.1% among low-, intermediate-, and high-risk patients, respectively, with higher rates of reduced TIMI flow, intracoronary thrombosis, and total occlusion among high-risk patients, most of them having ST depression.

Table 5. Univariate and multivariate analysis of angiographic findings of the ischemia-related artery in relation with high risk

	Univariate analysis	Multivariate analysis		
	p	OR	95% CI	*p*
Total occlusion	**0.001**	0.3	0.06 - 1.9	NS
Intracoronary thrombus	**0.02**	1.0	0.1 - 7.9	NS
Complex Lesion*	**0.001**	20.0	2.4 - 160	**0.005**
TIMI flow <III	**0.0001**	2.8	1.1 - 7.1	**0.02**

*Complex lesion with intracoronary filling defect (thrombus appearance) and/or ulcerative, irregular borders and overhanging edges; NS: Not significant (p>0.05).

Table 6. Braunwald clinical classification for unstable angina pectoris

Responsible for the unstable clinical syndrome	
Class A	Secondary unstable angina; cause of noncoronary disease identified
Class B	Primary unstable angina
Class C	Post myocardial infarction angina pectoris; within 14 days of a myocardial infarction
Severity	
Class I	New-onset exertional chest pain and accelerated angina without rest pain
Class II	Rest angina without pain for 48 hours before angiography
Class III	Recent (<48 hours) rest angina
Intensity of medical therapy	
Class 1	No or minimal therapy (i.e., only aspirin and one class of antianginal agents)
Class 2	Remaining patients
Class 3	Patients with angina refractory to maximal medical therapy, including intravenous nitroglycerin and heparin

Unstable angina pectoris is frequently associated with intracoronary thrombosis and complex lesions, causing spontaneous or PCI-related complications. Elevated troponin I indicating myocardial injury is among the predictors of clinical prognosis. These indicators may be helpful in the evaluation of angiographic risk stratification and in guiding the supportive treatment. Benamer et al.[31] investigated the relation between increased troponin I levels and angiographic morphology of the culprit lesion in patients with unstable angina pectoris and found that increasing troponin I level within the first 24 hours was an independent predictor of a high-risk morphological pattern, particularly in terms of PCI complications. Several other angioscopy studies have also confirmed the role of troponin T as an independent predictor of coronary thrombus.[32,33]

Inflammation plays an important role in the development of atherosclerosis and pathogenesis of acute coronary syndromes, i.e. atheromatous plaque disruption. Although large studies in patients with NSTE-ACS have failed to demonstrate a relationship between C-reactive protein, the systemic inflammation marker, and a high risk morphology on angiography, such a relation could be established for clinical outcomes.[34-37] On the other hand, studies on serum neopterin levels demonstrated an association between serum neopterin concentrations and the presence of angiographically complex lesions in patients with unstable angina.[38,39] As increase in cardiac markers represents an indication of high risk in AHCPR classification, patients with high levels of troponin T or I were included in the high-risk group in the present study. These patients also had higher rates of reduced TIMI flow, intracoronary thrombosis, and total occlusion.

It has been demonstrated that clinical risk stratification according to the AHCPR guidelines correlates with the angiographic extent of CAD in patients who are referred for coronary angiography due to unstable angina. Specifically, low-risk patients are more likely to have normal coronary arteries or mild CAD, whereas intermediate- and high-risk patients are more likely to have significant CAD (1-, 2-, 3-vessel, or left main CAD).[21,22] Likewise, in the present study, the frequency of marked CAD or multivessel disease was significantly higher in the high-risk group. It is of note that the lesion types and morphological patterns defined in our study have not been addressed in other studies examining the AHCPR risk stratification and angiographic features of patients with NSTE-ACS. We found that type A lesion was more frequent in the low-risk group and type B+C lesion was more frequent in the high-risk group. Analysis of the relationship between these groups (group A+B and group C) and angiographic morphology showed significant associations for complex lesions and reduced TIMI flow (<III) in both univariate and multivariate analysis; however, association was not significant for total occlusion and intracoronary thrombosis in multivariate analysis.

In this study, coronary angiography was performed after a mean of five days of hospital admission, during which lesion morphology could have been changed. This might be a limitation to this study.

In conclusion, in view of the results of the present study, the AHCPR risk classification based on the above-mentioned and practically relevant clinical features seems to be effective in predicting high-risk morphology on angiography. Therefore, the possibility of a morphologically complex lesion should be kept in mind in cases in which the AHCPR classification suggests a high risk, even at the time of admission to the emergency department where the decision for timing and strategy of the treatment is made.

Conflict-of-interest issues regarding the authorship or article: None declared

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Key words: Angina, unstable/classification; coronary angiography; coronary artery disease; coronary stenosis; coronary thrombosis; myocardial infarction; risk assessment/methods; severity of illness index.

Anahtar sözcükler: Angina, kararsız/sınıflandırma; koroner anjiyografi; koroner arter hastalığı; koroner darlık; koroner tromboz; miyokart enfarktüsü; risk değerlendirmesi/yöntem; hastalık ciddiyet indeksi.