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Characteristics of Inferior Myocardial Infarction With a Special Electrocardiographic Pattern (Aslanger) in Metabolic Syndrome

Aim	To evaluate the features of ST-segment elevation myocardial infarction with the Aslanger pattern in comparison with traditional forms of inferior myocardial infarction in metabolic syndrome.
Material and methods	This study included 30 patients with inferior myocardial infarction in the presence of metabolic syndrome: 9 patients with the Aslanger electrocardiographic pattern (group 1, age 59.7 [58.4; 63.1] years) and the rest with one of the traditional forms (control group, 59.9 [57.2; 63.8] years, matched by all criteria of metabolic syndrome). All patients underwent primary percutaneous intervention with assessment of the angiographic picture. The magnitude of ST-segment elevation was measured in lead III at the J point and following 0.06 seconds, and the optimal threshold value of this indicator was determined for a new picture of myocardial infarction.
Results	The infarct-related artery in the Aslanger pattern was more often the circumflex artery (p=0.0099), and coronary thrombosis was characterized by a lower TIMI thrombus grade (p=0.014). SYNTAX values for the Aslanger pattern and for the traditional picture of inferior infarction with ST elevation in lead II≥III were higher than for a similar picture with ST elevation in lead III>II. The level of cTnI at admission (p=0.013) and after 24 hours (p=0.0017), the platelet count (p=0.0011) and mean volume (p=0.0047) in group 1 had smaller values than with traditional inferior infarction. The ST elevation at J point and at J+0.06 s point for lead III with the Aslanger pattern was significantly lower than values of such shift in lead III>II and lead II≥III with traditional inferior infarction (p<0.001). An elevation value ≤1.5 mm at J point +0.06 s was a predictor of infarction with the Aslanger pattern. Constructing the ROC curve made it possible to determine that with the Aslanger pattern, the best cutoff value for this index is 2 mm.
Conclusion	Myocardial infarction with the Aslanger pattern as compared with traditional lower infarction in metabolic syndrome is characterized by specific individual angiographic signs, lower ST segment elevation, cTnI level, and thrombotic disorders.

Keywords	Myocardial infarction with the Aslanger pattern; metabolic syndrome
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Introduction

In 2020, a specific form of inferior ST-segment elevation myocardial infarction (STEMI) was described in which the corresponding segment displacement is present only in lead III but not in leads aVF and II, which are usual for this form of infarction [1]. This form is characterized by significant necrosis size (24 hour troponin surrogate marker), high risk (according to the GRACE scale) and multivessel coronary artery disease compared to non-ST-segment elevation MI (STEMI). In turn, the form of STEMI studied is comparable to traditional inferior STEMI in terms of in-hospital and annual mortality, but it is much less common than the latter, which may lead to diagnostic and tactical errors. The difference between the Aslanger pattern in this form of STEMI and an electrocardiographic (ECG) artefact Aslanger's sign should be mentioned; the artifact was reported ten years earlier than the Aslanger pattern and is explained by the movement of the electrocardiographic electrode located near the high-amplitude pulsation of the radial or posterior tibial arteries in hyperkinetic hemodynamics (severe anemia, cirrhosis, hyperthyroidism, etc.) [2, 3].

Metabolic syndrome (MS) is the most common comorbidity of MI [4]. This paper presents the characteristic features of inferior STEMI with the Aslanger pattern in MS. This is due to the fact that all reported clinical cases of

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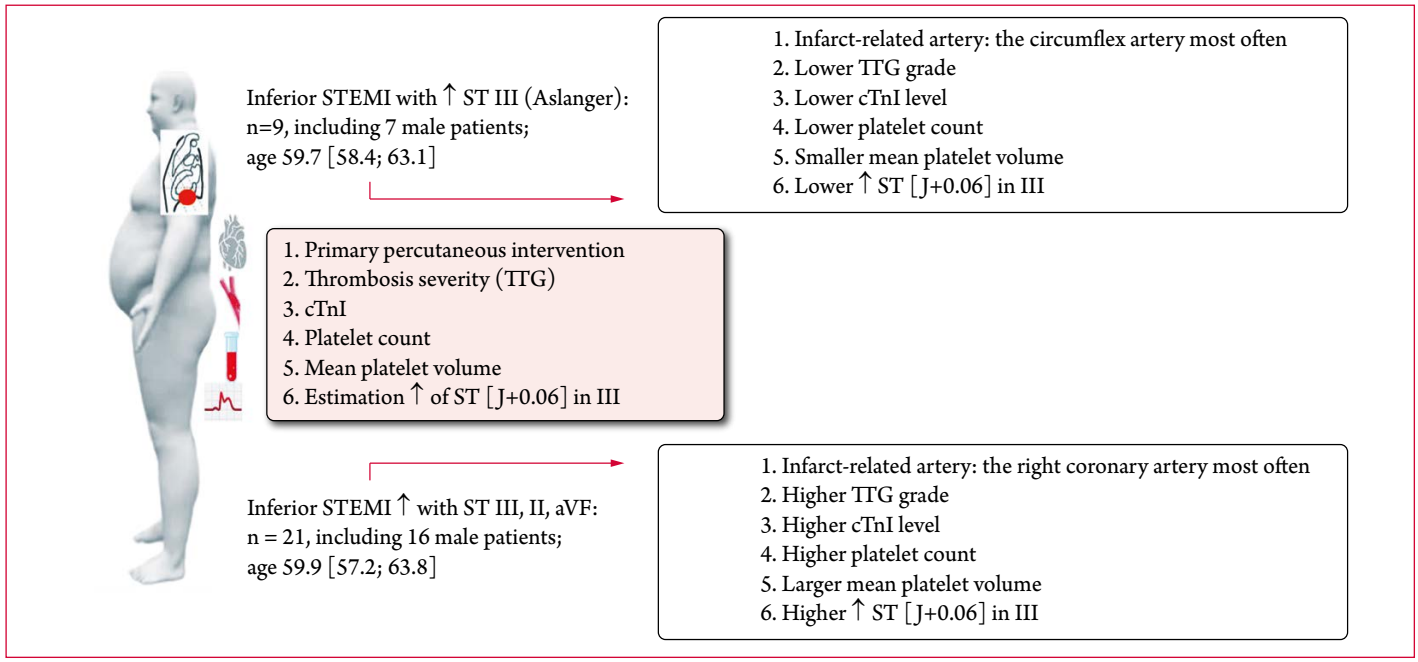
Central illustration. Characteristics of Inferior Myocardial Infarction With a Special Electrocardiographic Pattern (Aslanger) in Metabolic Syndrome



STEMI, ST-segment elevation myocardial infarction; NSTEMI, non-ST-segment elevation myocardial infarction.

Aslanger form MI were found to have the corresponding combination in this study.

Material and Methods

Thirty patients with inferior STEMI associated with MS who underwent primary percutaneous intervention (PCI), including 9 cases (group 1–7 males and 2 females, age 59.7 [58.4; 63.1] years) with the Aslanger pattern qualified by the three main features of the baseline ECG: 1) any ST-segment elevation in lead III but not in other inferior leads; 2) ST-segment depression in any of the leads from V4 to V6 (but not in V2) with a positive or terminally positive T-wave; 3) ST in lead V1 higher than ST in V2. The remaining 21 subjects (16 males, 5 females, age 59.9 [57.2; 63.8] years) were controls (Group 2); they were selected not only to balance the demographic characteristics of Group 1, but also to match it as closely as possible in terms of MS criteria: body mass index (BMI), waist circumference, hip circumference and waist-to-hip ratio, severity of arterial hypertension (AH), baseline (daily) blood pressure (BP) before the onset of MI, degree of dyslipidemia and hyperglycemia. The SYNTAX scale and a modified AHA/ACC classification proposed by the Society for Cardiac Angiography and Interventions (SCAI) with 4 grades of arterial defect severity were used to describe vascular lesions in predicting success and complications of coronary angioplasty: I. non-C lesion/patent vessel; II. C lesion/patents vessel; III. non-C lesion/occluded vessel; IV. C lesion/occluded vessel [5]. Type C included stenosis that met any of the following criteria: length >2 mm, pronounced proximal tortuosity, branching at >90 degrees, complete occlusion for >3 months. A vessel with acute thrombotic occlusion (total or subtotal) was considered an infarct-related artery (IRA). Angiographic evaluation of coronary thrombosis was performed using the TIMI thrombus grade (TTG) scale: TTG 0 – no angiographic signs of thrombosis, TTG 1 – likely presence of a thrombus, impaired wall contrast-enhancement, uneven vascular contours, TTG 2 – a thrombus of not more than ½ IRA diameter, TTG 3 – longitudinal size of a thrombus >½ but <2 IRA diameters, TTG 4 – longitudinal size of a thrombus >2 IRA diameters and TTG 5 – massive IRA thrombosis [6]. During ECG evaluation, ST-segment elevation was also calculated not only at the J point (III↑ST [J]) but also 0.06 seconds later in lead III (III↑ST [J+0.06]). All patients underwent echocardiography (GE Logiq F6, USA). Cardiac-specific troponin I (cTnI ELISA, USA) was measured at admission (cTnI-1) and 24 hours later (cTnI-24). The examination program included assessment of total cholesterol and high-density lipoprotein cholesterol (HDL–C), triglycerides (TG), creatinine with calculation of the Cockcroft-Gault glomerular filtration rate (GFR), glucose, platelet count and mean platelet volume (MPV) were determined in the complete blood count.

Differences between the two groups were calculated for continuous variables expressed as medians and 25th and 75th quartiles using the Mann-Whitney U test, and the Kruskal-Wallis H test was used to compare the three groups; conjugacy between individual continuous variables was assessed by calculating the Spearman rank correlation

coefficient; categorical variables were compared using the χ^2 test. Because PCI was performed by more than one operator, Cohen's kappa values were calculated for all angiographic indices. To identify new variables associated with the formation of Aslanger pattern MI, a logistic regression model was used (results were expressed as odds ratio [OR] with 95% confidence interval [95% CI]); when it was necessary to convert continuous variables into a categorical form, threshold values of the indicator were calculated using the logistic regression method. Then, a ROC curve was constructed taking into account the characteristics of an ECG pattern of inferior STEMI (categorical variable – Aslanger pattern or one of the traditional patterns) and III↑ST amplitude [J+0.06] (continuous variable). The optimal threshold in this model is determined by balancing sensitivity (Se) and specificity (Sp) – Se~Sp.

The study protocol conforms to the provisions of the Declaration of Helsinki and was approved by the local ethics committee of the Belgorod State National Research Institute (Minutes No. 16 dated 17.05.2022). The patients signed an informed consent form in accordance with the standard procedure prior to being included in the study.

Statistical analysis was performed using RStudio 4.3.1 and Statistica 12.0 for Windows.

Results

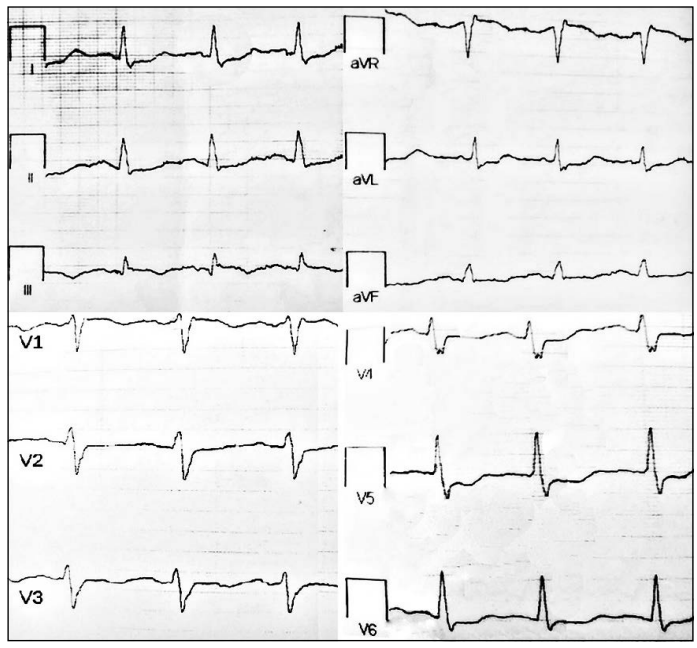
In both groups, angina syndrome was the main clinical manifestation of MI: 8 (88.9%) patients in Group 1 and 17 (85.7%) patients with conventional inferior infarction ECG pattern. At the same time, among those with the Aslanger pattern (Figure 1), pain developed for the first time in 5 patients; the rest sought medical attention for an increase in pain intensity (n=2) or duration (n=1).

In the control group, these characteristics were similar: 10 patients had a primary pain syndrome, 4 and 2 patients reported an increase in pain frequency and duration, respectively, and one patient reported a decrease in the efficacy of conventional antianginal therapy. The reasons for calling an ambulance by the remaining patients were the onset of weakness (n=1 in Group 1 and n=3 in Group 2) and arrhythmia (n=1 in Group 2). Abdominal onset of inferior STEMI was not observed in these subjects. The mean time from symptom onset to the decision to call an ambulance and the total time from symptom onset to hospital admission in Group 1 were 60 [40; 120] minutes and 155 [100; 210] minutes, respectively, which were not significantly different from the control group (60 [40; 110] minutes and 150 [100; 210] minutes, respectively, p1=0.780; p^2=0.813). With the same amount of historical evidence of hypertension and the same level of daily BP prior to the development of MI, the median systolic BP (SBP) at admission was 8.8% higher in the Aslanger group than in the control group (p=0.032), whereas diastolic BP (DBP) did not reach a statistically significant difference between the groups (Table 1).

No differences were found between the two ECG forms of MI and in the severity of heart failure (HF) according to the Killip classification. On the contrary, the levels of cTnI-1 and cTnI-24 were 24.5% and 59.3% higher, respectively, in the traditional inferior form. Higher platelet count and MPV (by 5.1% and 20.5%, respectively) were observed in the same group.

For PCI performed by more than one operator, Coehn's kappa values ranged from 0.73 to 0.78. IRAs were more often the circumflex artery (LCx) in the Aslanger group and the right coronary artery (RCA) in the control group (p=0.047). In both groups, the lesion area was located in the proximal LCx or RCA (Table 2) and C-type plaques were prevalent in both complete (TIMI 0) and incomplete (TIMI I, II) occlusions. In the Aslanger pattern group 8 (88.9%) patients had TTG 1 coronary artery thrombosis, while 18 (85.7%) patients with the conventional form of inferior MI had more severe thrombosis TTG 2 and TTG 3 (p=0.162). Furthermore, the total SYNTAX score was lower for the ST III>II elevation pattern than for ST II≥III (p=0.005) and the Aslanger pattern (p=0.001). In addition, III↑ST (J) and III↑ST (J+0.06) in the latter case were significantly lower than the values of similar displacements in the III>II and II≥III forms of conventional inferior STEMI. According to the criterion of time in the intensive care unit (ICU), there was no significant difference between the groups, but among patients with the conventional ECG pattern of inferior infarction, there were 6 patients who stayed in the ICU for 48–72 hours: due to massive

Figure 1. Patient K., Aslanger pattern MI



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thrombosis requiring delayed stenting (n=4) and due to other complications (n=2).

Correlation analysis revealed a significant conjugacy of cTnI-24 and MPV only for conventional inferior MI (p=0.019), but not for the Aslanger pattern (Figure 2).

LCx as IRA, SYNTAX >12.3, cTnI-24 <6.88 ng/mL, III↑ST (J+0.06)≤1.5 mm were predictors of Aslanger pattern MI (Table 3).

The ROC curve was constructed for III↑ST (J+0.06) to verify its possible use as a diagnostic sign for this form of MI. The best cutoff for the Aslanger form was found to be 2 mm, above which ST elevation (J+0.06) in lead III would only

Table 1. Key clinical and laboratory characteristics of patients with Aslanger pattern and conventional ECG pattern of inferior STEMI

Parameters	Aslanger pattern, n=9	Conventional pattern, n=21	p
BMI, kg/m^2	27.4 [25.7; 31.2]	27.7 [25.3; 31.6]	0.861
WC, cm	95 [82; 97]	96 [81; 97]	0.95
History of hypertension, n	6	18	0.919
SBP, mm Hg	136 [128; 148]	124 [120; 136]	0.038
DBP, mm Hg	80 [76; 80]	76 [70; 80]	0.103
HR, bpm	78 [67; 82]	75 [65; 82]	0.563
DM, n	2	7	0.974
Glucose, mmol/L	5.1 [4.9; 5.4]	5.3 [4.6; 5.5]	0.813
HDL cholesterol, mmol/L	0.96 [0.93; 0.98]	0.97 [0.92; 0.99]	0.917
TG, mmol/L	1.80 [1.77; 1.82]	1.78 [1.76; 1.84]	0.796
GFR, mL/min	84.9 [77.2; 89.3]	85.3 [76.5; 90.1]	0.897
cTnI-1, ng/mL	1.63 [0.92; 2.41]	2.16 [1.03; 2.73]	0.013
cTnI-24 h, ng/mL	5.82 [4.31; 6.13]	14.50 [10.81; 19.03]	0.0017
Platelets, ×10^3/uL	262 [202; 273]	276 [269; 321]	0.0011
MPV, fL	8.9 [8.5; 9.5]	11.2 [10.3; 12.1]	0.0047
Killip I, n	7	16	0.789
Killip II, n	2	2	0.915
Killip III, n	–	3	0.665
Time in ICU, h	24 [24; 24]	24 [24; 48]	0.101

Data are presented as medians and interquartile ranges Me [25%;75%]; BMI, body mass index; WC, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; DM, diabetes mellitus; HDL, high-density lipoprotein; TG, triglycerides; GFR, glomerular filtration rate; cTnI-1, baseline cardiac troponin level; cTnI-24, 24-hour cardiac troponin level; MPV, mean platelet volume; ICU, intensive care unit.

Table 2. Clinical examination findings in patients with Aslanger pattern and conventional ECG pattern of inferior STEMI

Parameters		Aslanger pattern, n=9	Conventional pattern, n=21		p / H#
			ST II≥III, n=4	ST III>II, n=17	
IRA	LCx, n	7	4	–	0.005
	RCA, n (%)	2	–	17	0.043
	Proximal, n	6	3	13	0.98
	Medial, n	3	1	3	0.781
	Distal, n	–	–	1	0.689
TTG 1, n		8	1	2	0.044
TTG 2, n		1	3	11	0.204
TTG 3, n		–	–	4	0.246
Non-C-type plaque, n		3	2	7	0.92
C-type plaque, n		6	2	10	0.987
SYNTAX, score		13.5 [12.5; 16.5]	15.5 [13.0; 17.5]	8.5 [7.0; 10.0]*	16.06#
III↑ST(J), mm		1.0 [1.0; 1.0]	2.5 [2.0; 3.0]	5.0 [5.0; 6.0]*	24.22#
III↑ST(J+0.06), mm		1.5 [1.5; 1.5]	3.5 [3.0; 4.0]	7.0 [6.0; 7.0]*	24.09#
aVR ↑ST(J) and/or (J+0.06)≥1 mm, n		7	–	–	0.003
V1 ↑ST(J) and/or (J+0.06)≥1 mm, n		1	–	1	0.77
EF, %		55 [50; 55]	55 [50; 57]	53 [50; 55]	0.234#

IRA, infarct-related artery; LCx, left circumflex artery; RCA, right coronary artery; ↑ST (J), ST-segment elevation at time point J; ↑ST (J+0.06), ST-segment elevation 0.06 seconds after the J time point; EF, ejection fraction. # Kruskal–Wallis H-test; * p < 0.001.

Table 3. Predictors of Aslanger pattern MI

Parameters	OR	95 % CI	p
LCx as IRA	14.88	2.02–109.74	0.01
SYNTAX>12.3	0.45	0.002–0.26	0.0039
cTnI-24 < 6,88 ng/mL	76	5.35–1079.5	0.002
III↑ST(J+0.06) ≤ 1.5 mm	48	3.86–596.96	0.0039

IRA, infarct-related artery; LCx, left coronary circumflex artery; OR, odds ratio; CI, confidence interval; cTnI-24, 24-hour cardiac troponin; MPV, mean platelet volume; ↑ST (J+0.06), ST-segment elevation 0.06 seconds after the J time point.

indicate one of the conventional forms of inferior STEMI (Figure 3).

When the model required the Se~Sp rule, the Se of this cutoff was 100% (95% CI: 62.0–100%), Sp – 81.0% (95% CI: 61.9–95.0%), positive predictive value, negative predictive value and its precision in diagnosing Aslanger pattern MI are 69.0% (95% CI: 53.0–90.0%), 100% (95% CI: 100–100%) and 87.0% (95% CI: 73.0–97.0%), respectively, with area under ROC curve 93.0 (95% CI: 85.0–99.8, p<0.001). The small number of observations of the new ECG form of MI, as shown in Table 3, while maintaining the rule of obtaining a statistically significant level for the characteristics mentioned, allows sufficiently wide CIs for some of them.

Some reports on the Aslanger pattern in MI mention two additional features recommended for consideration, but not related to the original author's description – ST-segment elevation in the aVR lead and ST-segment elevation in V1 (note: even the absence of ST depression in V2 is not mandatory) [7]. We used two criteria to evaluate the above recommendations: the frequency of the presence of ST elevation (J) and/or (J+0.06) ≥1 mm in aVR and similar displacement of ST (J) and/or (J+0.06)≥1 mm in V1. However, despite deliberately expanding the boundaries of the diagnostic feature to make it easier to detect, the first criterion was found in 77.8% of patients and the second criterion in only one patient (4.8%). Furthermore, no additional evidence of right ventricular (RV) infarction was observed in this patient. In the control group, one case with ST III>II and ST elevation in V1 was supplemented by short-term 1 mm elevation in V4R and V5R, and coronary angiography revealed occlusion of the RCA (proximal to the marginal branch), indicating additional damage to the RV.

Figure 2. Relationship between cTnI-24 and MPV in conventional inferior MI and Aslanger pattern MI

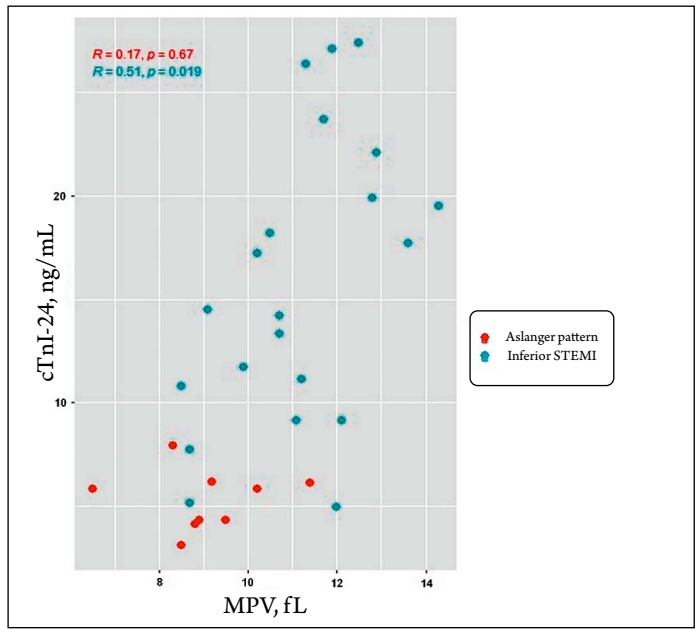
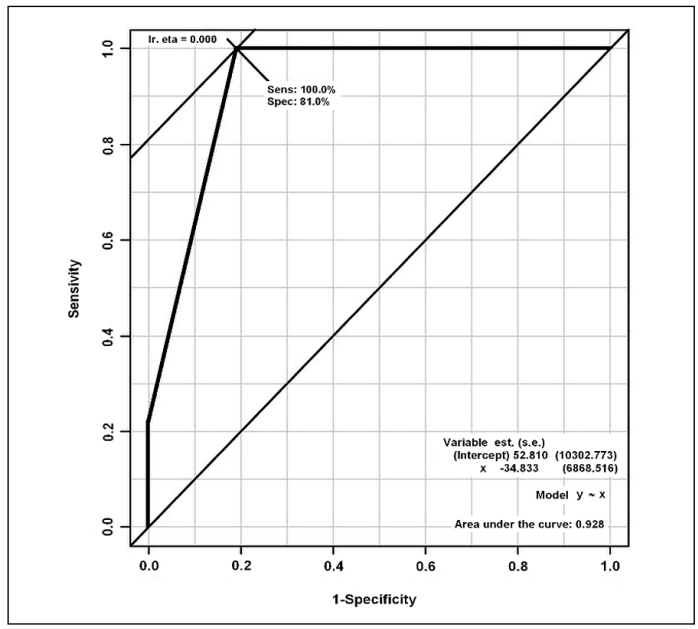


Figure 3. ROC curve of the best cutoffs of III↑ST[J+0.06] in the Aslanger pattern



Area under the curve – the area under the curve;
Intercept – intercept in the regression model;
x-intercept – the point where the line intersects the x axis.

Discussion

This paper summarizes the cases of Aslanger pattern MI associated with MS. The low prevalence of this form of MI in clinical practice (according to Aslanger, there are only 6.3% of such patients) determined the small size of the experimental group [1], and the control group was formed to balance the first group in terms of MS criteria; in this regard, the proportion of Aslanger pattern examples relative to the control group was higher than in the original paper only because of this study design. The unusual electrocardiographic pattern of STEMI, which distinguishes it from the conventional pattern of inferior infarction, is that such an elevation is present in lead III but not in leads II and aVF. This makes it difficult to come to the right conclusion. In addition, ST segment depression in the V4-V6 thoracic leads may contribute to the misdiagnosis of STEMI. Finally, this ECG pattern is extremely rare in the absence of infarction, occurring in only about 0.5% of cases [1]. In general, the Aslanger pattern is characteristic of patients with MI associated with significant coronary artery disease. In our examples, this is evidenced by the prevalence of C-type plaque radiographic morphology, its proximal localization, and a significantly higher SYNTAX score than in the ST III>II form of conventional inferior STEMI. The multivessel nature of the disease and the approximately equal number of cases of LCx and RCA as IRA were

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previously reported [1]. In this study, IRA was mainly LCx, and the formation of this ECG pattern was associated with low detection of high-grade thrombosis (TTG≥3, univariate OR 48.0; 95% CI: 3.85–596.96; p=0.0038). Baseline (cTnI-1) and cTnI-24 levels appeared to be significantly lower in the Aslanger pattern, suggesting a smaller zone of necrosis than in conventional inferior infarction. Conversely, an increase in platelet count and mean platelet volume was observed for the latter. And a reliable relationship between MPV and cTnI-24 was observed only in this form of MI, but not in Aslanger pattern MI. In addition, very high-grade thrombosis (TTG ≥4) requiring delayed stenting to avoid no-reflow was observed only in conventional inferior MI. These peculiarities may result from the influence of MS factors and/or indicate the existence of different pathogenetic mechanisms for conventional and Aslanger pattern MI. And the same frequency of Killip classes between the groups, as well as the above-mentioned significant severity of coronary atherosclerosis, do not allow us to classify Aslanger pattern MI as a less severe form of MI. Taking into account the fact that the degree of ST segment elevation in lead III is much lower than in conventional inferior STEMI, it is reasonable to use the value of J+0.06 s point displacement for diagnosis: when constructing the ROC curve, the optimal cutoff is 2 mm subject to its compliance with the balance between sensitivity and specificity. The small number of clinical observations of the Aslanger pattern in our study do not allow, at the current stage, a reasoned discussion of the possibility of including additional ECG signs in the original criteria of this pattern, as well as a discussion of the role of individual MS factors (including DM) in its formation.

This study is limited by the small number of clinical observations of inferior infarction with conventional and Aslanger electrocardiographic patterns, as well as the use of only radiologic criteria of coronary artery disease.

Conclusion

Myocardial infarction with the Aslanger pattern, compared with conventional inferior myocardial infarction associated with the metabolic syndrome, is characterized by the specific nature of some angiographic features, lower ST-segment elevation in lead III, level of cTnI and thrombotic abnormalities, comparable X-ray morphology and localization of atherosclerotic plaque, frequency and severity of heart failure, and duration of stay in the intensive care unit after percutaneous coronary intervention.

No conflict of interest is reported.

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УВАЖАЕМЫЕ КОЛЛЕГИ!

Приглашаем Вас принять участие в работе Ежегодной Всероссийской научно-практической конференции «КАРДИОЛОГИЯ НА МАРШЕ 2024» и 64-й сессии ФГБУ «НМИЦК им. ак. Е. И. Чазова» Минздрава России. Конференция состоится 4–6 июня 2024 г. в ФГБУ «НМИЦК им. ак. Е. И. Чазова» Минздрава России (г. Москва, ул. Академика Чазова, 15А).

На Конференции будут представлены фундаментальные аспекты кардиологии, самые последние научные достижения и клинические подходы в области профилактики, диагностики, лечения и реабилитации сердечно-сосудистых и коморбидных заболеваний. Участниками Конференции станут ведущие ученые, клиницисты и организаторы здравоохранения из России и зарубежных стран.

Конференция будет проводиться при поддержке Министерства здравоохранения Российской Федерации, Департамента здравоохранения города Москвы, Национального медицинского общества профилактической кардиологии, Российского кардиологического общества, Российского научного медицинского общества терапевтов.

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