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A new electrocardiographic pattern indicating inferior myocardial infarction

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ABSTRACT

Background: We identified a specific pattern that does not display contiguous ST-segment elevation (STE), indicating acute inferior myocardial infarction (MI) with concomitant critical stenoses on the other coronary arteries. We sought to define the frequency, underlying anatomic substrate, diagnostic power and prognostic implications of this pattern.

Methods: One thousand patients with a diagnosis of non-STEMI were enrolled as the study group. Within the same date range, all patients with inferior STEMI and 1000 patients, who had been excluded for MI (no-MI), were also enrolled. The coronary angiograms were reviewed by two interventional cardiologists, who were blinded to the ECGs. Echocardiographic wall motion bullseye displays and coronary angiography maps were constructed for each group. The dead or alive status was checked from the electronic national database.

Results: The final study population consisted 2362 patients. The prespecified ECG pattern was observed in 6.3% (61/966) of the non-STEMI cohort and 0.5% (5/1000) of no-MI patients. These patients had a larger infarct size as evidenced by 24-hour troponin levels, higher frequency of angiographic culprit lesion, and higher frequency of composite acute coronary occlusion endpoint compared to their non-STEMI counterparts. On the other hand, they had a similar in-hospital (5% vs. 4%, respectively; $P = 0.675$) and one-year mortality compared to the patients with inferior STEMI (11% vs. 8%, respectively; $P = 0.311$).

Conclusion: We here define a new ECG pattern indicating inferior MI in patients with concomitant critical lesion (s) in coronary arteries other than the infarct-related artery. Patients with this pattern have multivessel disease and higher mortality.

Introduction

For more than a century, the electrocardiogram (ECG) has been the most accessible clinical tool for the diagnosis of acute myocardial infarction (MI) and defining its location. According to the classical teaching, infarct location has been assumed to reside under the leads showing ST-segment elevation (STE). Correspondingly, international guidelines necessitate STE in two contiguous leads of an individual localization group (i.e., anterior, lateral, inferior) for the diagnosis and localization of STEMI [1]. However, this framework is intuitive rather than being well evidence-based and the recent studies have revealed that the location of the leads demonstrating STE does not reliably indicate the location of infarcting myocardium [2,3]. Assigning lead groups instinctively to certain infarct locations may fail to classify MI location correctly [4], and has to rely upon unsupported theoretical explanations, such as an infarct-related artery supplying two coronary territories [5], when the ECG shows an atypical pattern with STE in different lead groups. Furthermore, the requirement of STE in two contiguous leads results in reduced sensitivity for STEMI in some configurations [6,7]; worse, it may prevent the discovery of new ECG patterns indicating acute coronary occlusion necessitating acute reperfusion.

Inspired by several clinical observations, we hypothesized that a subgroup of inferior "STEMI" may show such a peculiar pattern and may be incorrectly labeled as non-STEMI. When the ST-vector of inferior MI shifts slightly more rightward than usual, it is directed at right angles to aVF, projects to the negative pole of lead II, but still points to the positive pole of lead III. In this situation, standard 12-lead ECG only shows STE in lead III which accompanies ST-segment depression (STD) in

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leads I and II, and nearly isoelectric ST-segment in aVF. STD in lateral chest leads complements these changes. In this study, we sought to define frequency, underlying anatomic substrate, diagnostic power and prognostic implications of this pattern.

Materials and methods

The study was undertaken at Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, Istanbul, which has a large regional referral network for primary PCI. Institutional review board approval was obtained; the study was judged to be exempt from formal evaluation because it involved only analysis of existing records.

Starting from May 2017, we retrospectively enrolled the first 1000 patients with a diagnosis of non-STEMI (non-STEMI cohort, Group I). We also devised two control groups within the same date range, one of which consisted patients with inferior STEMI (STEMI cohort, Group II) and the other was comprised of another 1000 patients, randomly chosen from a computer-generated date list, who had been excluded for MI with serial unchanging ECGs and negative serial troponins for at least 12-hours after beginning of symptoms (control cohort, Group III). Each patient was included only once. Baseline characteristics were obtained via chart review and GRACE risk score at admission was calculated retrospectively [8].

The prespecified ECG pattern was defined as (1) any STE in DIII but not in other inferior leads, (2) STD in any of leads 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 (Fig. 1). This pattern was prospectively screened in both Groups I and III by two ECG reviewers (E.A. and A.T.C.), who were blinded to the angiographic and clinical outcomes. Also, one of the ECG reviewers (E.A.) reviewed all ECGs twice, three months apart, for the assessment of intra-observer variability. For multiple ECGs on the same patient, the earliest ECG with maximum ST-segment deviation was used. After calculation of intra- and inter-observer variability, a final composite evaluation by two reviewers was undertaken. Any disagreement was resolved by discussion and, if necessary, with the opinion of a third cardiologist (B.Ş.).

Echocardiographic wall motion score index was calculated using a 17-segment model of the ventricle and a scoring system as follows: 1, normokinesia; 2, hypokinesia; 3, akinesia; 4, dyskinesia. To explore the difference in infarct distribution, mean values of the wall motion score for each segment were separately calculated and color-coded bullseye displays were constructed for the prespecified ECG pattern and inferior MIs (Group II) [3,9].

The coronary angiograms were reviewed by two interventional cardiologists (E.B. and Ö.Y.), who were blinded to the ECGs. The diagnosis of acute coronary occlusion (ACO) was made by angiographic properties (appearance, presence of collaterals and crossing of the lesion) and rising cardiac biomarker levels. Because the artery may spontaneously open by the time of the angiogram in many cases of ACO, the investigators used predefined surrogate endpoints: a highly elevated peak troponin, i.e., peak troponin $I > 5.0$ ng/mL, which has been shown to be highly correlated with ACO, or culprit lesion on the angiogram and/or critical stenosis with less than Thrombolysis in Myocardial Infarction (TIMI) 3 flow plus a rising troponin in first 24 h (>20% from baseline) [1,10]. Any disagreement was resolved by a third cardiologists' opinion (A.S.).

Troponin I Abbott c4100i (Abbott Diagnostics, Chicago, IL, USA) was used as the troponin assay. Admission troponin was defined as the first troponin obtained at the emergency department or catheterization laboratory; before, during or immediately after cardiac catheterization. In addition to peak troponin level, a 24-hour troponin level was also sought, as it was shown to be better correlated with infarct size [11]. All patients were checked for guideline-recommended contemporary therapy. Each individual's vital status was checked from the electronic national database for one-year mortality.

Baseline characteristics were summarized using standard descriptive statistics. Comparisons of relevant parameters were performed by chi-square, Fisher's exact-test, student's *t*-test, Mann-Whitney U,

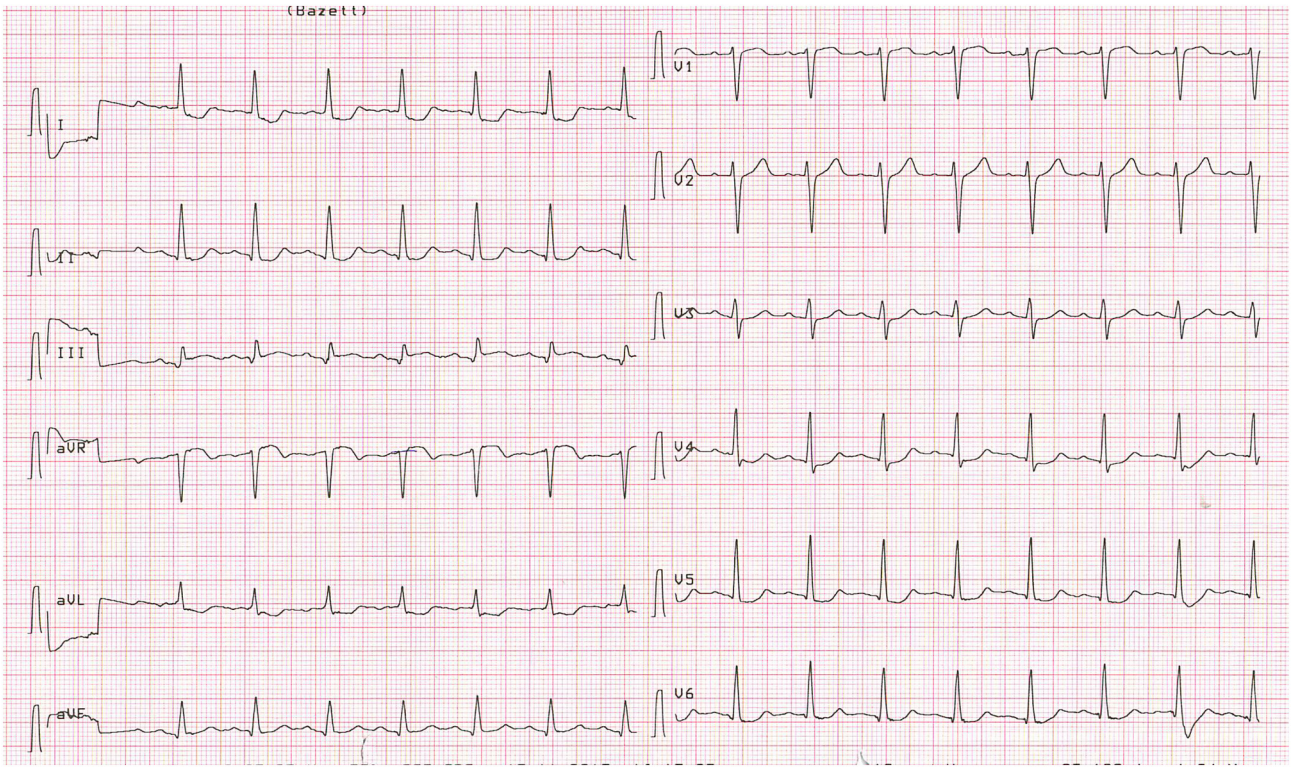

Fig. 1. The specific ECG pattern. The prespecified ECG pattern was defined as (1) STE in DIII but not in any other inferior lead, (2) ST depression in any of leads V4 to 6 (but not in V2) with a positive (at least terminally positive) T-wave, (3) ST in lead V1 higher than ST in V2.

Table 1
Baseline characteristics.*,†

Characteristic	Group I (N = 966)		Group II (N = 396)	Group III (N = 1000)
	Group IA (n = 61)	Group IB (n = 905)		
Age – years	66 ± 11	61 ± 13	59 ± 12	48 ± 16
		0.002	<0.001	<0.001
Male sex – no. (%)	34 (56)	586 (65)	314 (79)	646 (65)
		0.155	<0.001	<0.001
Medical history – no. (%)				
Hypertension	48 (79)	546 (60)	171 (43)	195 (20)
		0.004	<0.001	<0.001
Diabetes	36 (59)	335 (57)	104 (26)	83 (8)
		0.001	<0.001	<0.001
Dyslipidemia	16 (26)	153 (17)	104 (26)	207 (21)
		0.064	0.996	0.996
Smoking	15 (25)	376 (42)	230 (58)	483 (48)
		0.009	<0.001	<0.001
Prior MI	20 (33)	260 (29)	70 (18)	89 (9)
		0.499	0.006	0.006
Prior PCI	15 (25)	197 (22)	59 (15)	111 (11)
		0.606	0.056	0.056
Prior CABG	8 (13)	92 (10)	13 (3)	63 (6)
		0.465	0.001	0.001
Clinical parameters				
Systolic blood pressure – mmHg	154 ± 29	147 ± 28	132 ± 29	139 ± 24
		0.062	<0.001	<0.001
Heart rate – $min.^{-1}$	84 (27)	81 (28)	74 (22)	77 (19)
		0.105	<0.001	<0.001
ECG to PCI time – min.	3450 (4245)	2760 (4740)	35 (7195)	N/A
		0.205	<0.001	N/A
Killip Class		0.791	0.156	<0.001
1	56 (93)	838 (93)	379 (96)	1000 (100)
2	1 (1)	23 (2)	2 (1)	0 (0)
3	3 (5)	39 (4)	9 (2)	0 (0)
4	1 (1)	5 (1)	5 (1)	0 (0)
GRACE risk score	152 (36)	148 (43)	144 (36)	129 (44)
		0.013	0.016	<0.001
Laboratory investigations				
Creatinine – mg/dl	1.1 (0.6)	0.9 (0.4)	0.8 (0.2)	0.8 (0.2)
		0.019	<0.001	<0.001
Hemoglobin – g/dl	11.8 (2.7)	13.1 (2.9)	13.6 (2.1)	13.9 (2.5)
		0.004	<0.001	<0.001
Admission troponin I – ng/ml	0.495 (2.757)	0.383 (1.793)	3.112 (15.898)	0.002 (0.003)
		0.111	<0.001	<0.001

CABG, coronary artery by-pass grafting; ECG, electrocardiogram; GRACE, Global Registry of Acute Coronary Events; MI, myocardial infarction; PCI, percutaneous coronary intervention.
* The presented are mean ± standard deviation or median (interquartile range), and *P*-values.
† *P*-values on the second rows are for comparison with the patients in the Group IA (the patients with the prespecified ECG pattern).

Kruskal-Wallis H test as appropriate. A Cohen's κ test was run to determine the intra- and inter-observer agreement for the pattern recognition. Kaplan-Meier analysis was performed to determine the cumulative long-term mortality rates among groups, which were then compared using the log-rank test. A Cox-regression model was used to repeat the survival analysis after correction for baseline GRACE risk score. All statistical analyses were performed with SPSS software (version 24.0; SPSS Inc., Chicago, IL).

Results

One thousand patients with non-STEMI (Group I), 404 patients with inferior STEMI (Group II) and 1000 patients with chest pain ruled-out for MI (Group III) were enrolled during the study period. Thirty-four patients in Group I and eight patients in group II were excluded because of technically inadequate admission ECGs. Final study population consisted 966 patients in group I, 396 patients in group II and 1000 patients in group III.

The prespecified ECG pattern was observed in 6.3% (61/966) of the patients in the Group I (non-STEMI cohort) and 0.5% (5/1000) of the patients in the Group III (control cohort). Intra-observer ($\kappa = 0.829$; 95% confidence interval [CI], 0.757 to 0.901; $P < 0.001$) agreement was excellent, and inter-observer agreement ($\kappa = 0.635$; 95% CI, 0.529 to 0.741; $P < 0.001$) was good.

The patients with the prespecified ECG pattern in the Group I were renamed as Group IA and the remaining patients with non-STEMI were renamed as Group IB. The patients in Group IA were higher risk patients, as evidenced by higher baseline GRACE risk scores, with older age and higher frequency of comorbidities compared to the Group IB and the Group II. The comparison of baseline characteristics of these patients and the others were summarized at Table 1. When Group IA was compared with Group IB in terms of clinical outcomes, the patients in Group IA had a higher troponin rise in the first 24-hours, higher infarct size as evidenced by 24-hour troponin levels, higher frequency of angiographic culprit lesion and higher frequency of composite ACO endpoint (Table 2). They also had a higher frequency of circumflex artery involvement as the infarct-related artery, higher frequency of multivessel disease, higher frequency of the presence of concurrent chronic total occlusion, and a higher in-hospital and one-year mortality compared to Group IB.

On the other hand, Group IA had a similar in-hospital and one-year mortality compared to the patients in Group II despite more limited infarct size and a lower frequency of ACO (Table 2). Although both groups have similar wall motion scores on echocardiogram (Group IA, 20.5 vs. Group II, 20.2; $P = 0.558$), the Group IA had higher scores for anterior

Table 2
Distribution of coronary involvement endpoints across groups.*,†

	Group I (N = 966)		Group II (N = 369)	Group III (N = 1000)
	Group IA (n = 61)	Group IB (n = 905)		
Troponin level – ng/ml				
Admission troponin	0.495 (2.757)	0.383 (1.793)	3.112 (15.898)	0.002 (0.003)
		0.111	<0.001	<0.001
24-hour troponin	1.993 (6.460)	0.731 (4.508)	34.117 (38.528)	0.002 (0.003)
		0.034	<0.001	<0.001
Peak troponin	2.370 (6.397)	1.331 (5.966)	34.883 (38.193)	0.002 (0.003)
		0.117	<0.001	<0.001
Angiographic involvement – no./total no. (%)				
LMCA	4/52 (8)	33/717 (5)	14/396 (3)	0/2 (0)
		0.306	0.152	N/A
LAD	33/52 (63)	406/717 (57)	147/396 (37)	1/2 (50)
		0.336	<0.001	N/A
Cx	42/52 (81)	345/717 (48)	209/396 (53)	1/2 (50)
		<0.001	<0.001	N/A
RCA	37/52 (71)	357/717 (50)	311/396 (78)	1/2 (50)
		0.003	0.230	N/A
IRA – no./total no. (%)		0.014	<0.001	N/A
LMCA	1/50 (2)	5/531 (1)	3/396 (1)	
LAD	5/50 (10)	175/531 (33)	0/396 (0)	
Cx	25/50 (50)	153/531 (29)	131/396 (33)	
RCA	16/50 (32)	130/531 (24)	262/396 (66)	
Culprit plaque	47/51 (92)	181/664 (27)	396/396 (100)	N/A
		<0.001	<0.001	N/A
Angiographic ACO	13/52 (25)	117/717 (16)	275/396 (69)	N/A
		0.099	<0.001	N/A
Chronic total occlusion	13/52 (25)	108/717 (15)	15/396 (4)	N/A
		0.059	<0.001	N/A
Echocardiography				
Ejection fraction – %	50 (18)	50 (20)	50 (15)	60 (5)
		0.532	0.933	<0.001
Composite ACO endpoint – no./total no. (%)	33/61 (54)	245/897 (27)	382/395 (97)	0/1000 (0)
		<0.001	<0.001	<0.001
Mortality – no. (%)				
In-hospital mortality	3 (5)	12 (1)	15 (4)	0/1000 (0)
		0.028	0.673	<0.001
Long-term mortality	7 (11)	31 (3)	30 (8)	1/1000 (0)
		0.002	0.311	<0.001

ACO, acute coronary occlusion; Cx, circumflex artery; ECG; electrocardiogram; N/A, not available; LAD, left anterior descending artery; LMCA, left main coronary artery; IRA, infarct-related artery; RCA, right coronary artery.

* The presented are number (percentage) or median (interquartile range), and *P*-values.

† *P*-values on the second rows are for comparison with the patients with the prespecified ECG pattern.

segments and lower scores for inferior segments compared to Group II (Fig. 1). On angiogram, the Group II had significantly more proximal right coronary artery disease (Group IA, 14% vs. Group II, 44.9%; $P < 0.001$), while the Group IA had more circumflex artery involvement (Group IA, 50.0% vs. Group II, 33.1%; $P = 0.018$) as the infarct-related artery (Fig. 2). Patients in Group IA showed a higher mortality trend compared to Group IB and even Group II (Fig. 3A). In cox-regression, however, statistical significance was lost for all comparisons after correction for baseline GRACE risk score (Fig. 3B). But it should be noted that this procedure may have caused an overcorrection and resulted in a diminished power.

Discussion

We here define a new ECG pattern consisting of three criteria: (1) any STE in DIII but not in other inferior leads, (2) STD in any of leads V4 to V6 but not in V2, (3) ST in lead V1 higher than ST in V2 (Fig. 1). Although the patients with this pattern are classified as non-STEMI, they have an acute atherothrombotic event frequently resulting in inferior MI (more often occlusion or near occlusion of the circumflex artery than the right coronary artery) with at least one accompanying stable but critical stenosis in one of the non-infarct-related arteries. They tend to have multiple vessel disease, multiple comorbidities and higher baseline risk, and show an increased short- and long-term mortality. This pattern is not uncommon among patients classified as non-STEMI (6.3%) but seems to be relatively rare in patients who were ruled-out for MI (0.5%). Our data indicate that 13.3% of inferior MIs may present with this pattern and may be deprived of emergent revascularization therapy because of being incorrectly labeled as non-STEMI.

Although infarct location has been assumed to reside under the leads showing STE according to the classical teaching, the main determinant of the number and location of the leads that would show STE is actually the spatial orientation of the injury vector [3]. Frequently, the direction of the ST-vector overlaps with lead groups labeled with the same localization, but this is not always the case [3–5]. For example, a distal left anterior descending artery (LAD) occlusion causes ST-vector to be directed anterolaterally, which results in STE in V1–2 through V6, whereas a proximal LAD occlusion frequently displays STE limited to V1-V4 accompanied by STD in V5-V6, due to an ST-vector directed to dominant basal segments [3,12–15]. Moreover, standardized lead placement does not cover all of the possible ST-vector orientations. It has been long known that standard ECG does not show STE in inferolateral (formerly posterior) [9] or right ventricular MI due to the exclusion of posterior (V7–9) and right-sided leads (V3R-V6R) from standard lead list, which would normally demonstrate STE. As in the pattern presented here, a frontal ST-vector oriented to the edges of the standard 12-lead placement coverage may display STE only in one of the inferior leads and may dangerously cause a STEMI diagnosis to be missed.

The reason for this atypical pattern with non-contiguous STE in inferior leads seems to be the average ST-vector not directed to the injury

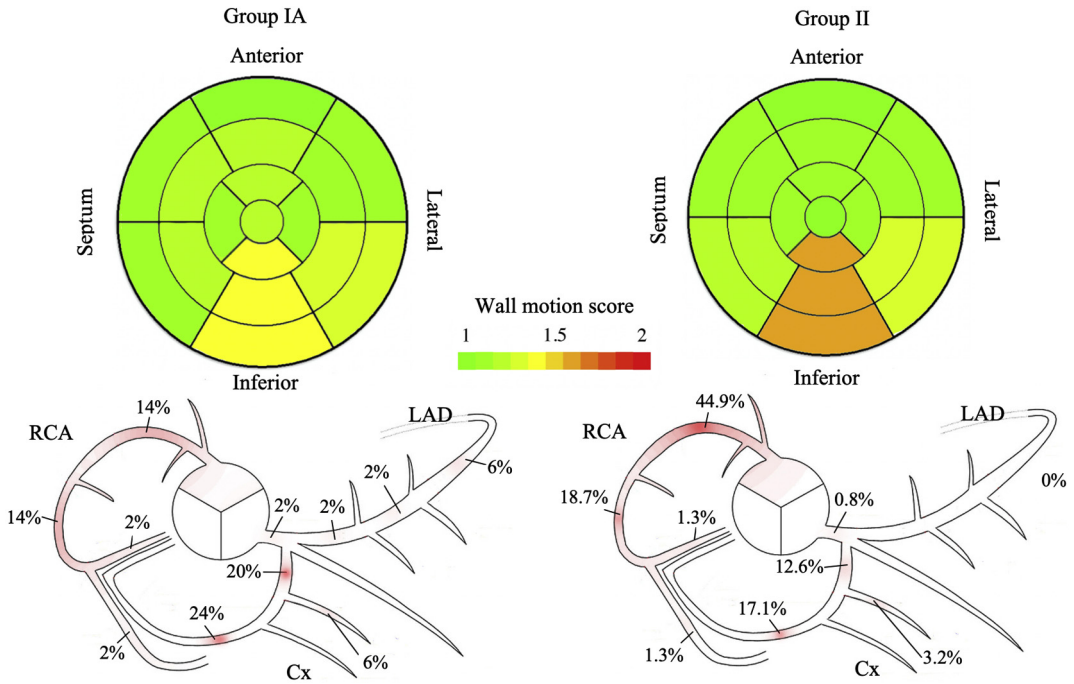

Fig. 2. The wall-motion score bullseye and culprit lesion localization in coronary tree maps for the patients with prespecified ECG pattern (Group IA) and the patients with inferior ST-segment elevation myocardial infarction (Group II). Cx, circumflex artery; LAD, left anterior descending artery, RCA, right coronary artery.

area located in the inferior wall. Theoretically, the average ST-vector that is more rightward than usual can be explained by the summation of the ST-vector of inferior MI and the ST-vector of subendocardial ischemia caused by the concurrent critical vessel disease. The ST-vector of inferior MI localizes the area of infarction and is directed inferiorly and frequently rightwards, whereas the ST-vector of subendocardial ischemia does not localize the area of ischemia and is directed to the lead aVR irrespective of involved coronary territory. The summation of these two vectors results in an average ST-vector directed rightwards at right angles to aVF, projects to the negative pole of lead II, but still the points to the positive pole of lead III. In this situation, standard 12-lead ECG only shows STE in lead III and aVR, which accompanies STD in lead I and II, and a nearly isoelectric ST-segment in aVF. Also, lateral chest leads show some STD due to the ST-vector pointing away from them. It seems inferior injury should be limited in degree and/or extent, otherwise the remaining inferior leads would show STE and the prespecified pattern turns into a straightforward inferior MI pattern. This also applies for inferolateral (formerly posterior) [9] involvement, as when posteriorly directed ST-vector is dominant, STD in V2 cannot be masked by benign anterior STE and becomes conspicuous. These can explain why the patients with the prespecified ECG pattern had more limited infarct size despite higher frequency of culprit lesion on their angiograms. The

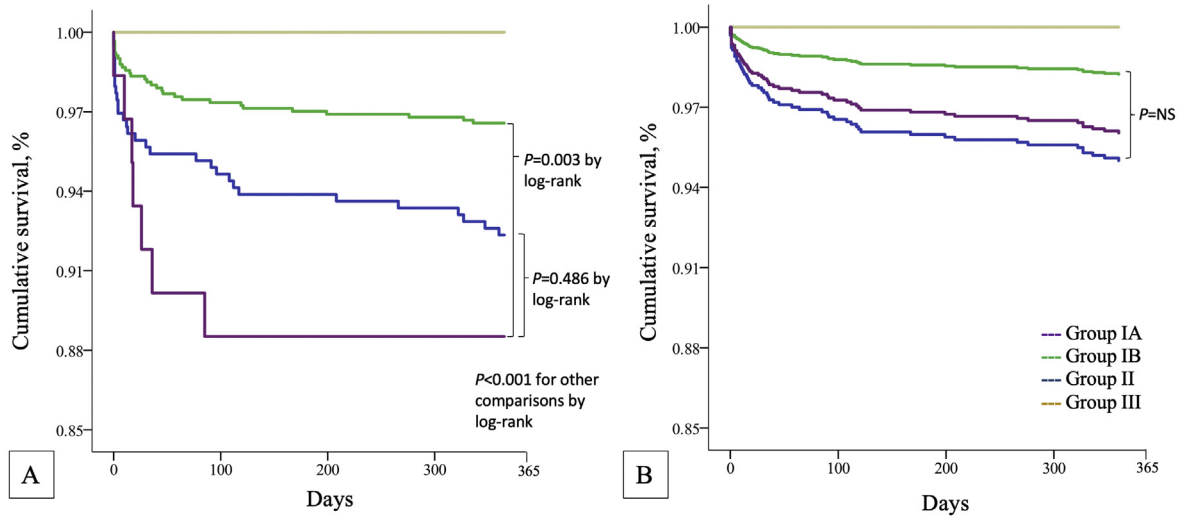

Fig. 3. Cumulative survival of according to the groups. A, Kaplan-Meier curves are presented for the patients with the prespecified ECG pattern (Group IA, purple line), with non-STEMI (Group IB, green line), with STEMI (Group II, blue line) and control patients (Group III, brown line). B, Cox-regression curves for survival estimates after correction for baseline GRACE risk scores. It should be noted, however, this may represent an overcorrection and have resulted in a diminished power. ECG, electrocardiogram; GRACE, Global Registry of Acute Coronary Events; STEMI, ST-segment elevation myocardial infarction.

requirement of a more limited inferior injury area may also explain why circumflex artery is more commonly involved, as it generally perfuses a more limited area compared to right coronary artery. On the other hand, since there is more than one lesion on coronary angiogram, this may cause some confusion about which lesion should be emergently opened. Our data indicates that the lesion on the artery that supplying inferior wall is generally the culprit one and should be considered first.

Our study has several limitations. Firstly, this is a retrospective study which might cause bias. Secondly, although we report a considerably higher frequency of ACO in the patients with the prespecified pattern, it is hard to formulate a universal ACO definition. Thirdly, the only explanation for rightward ST-vector resulting in the described pattern may not be the coincidence of an ACO supplying inferior wall with subendocardial ischemia caused by other critical stenoses. Theoretically, an isolated basal inferoseptal infarction or an acute inferior MI in the presence of previous infarctions that may change the orientation of lesion vector can also cause a similar picture. Lastly, this pattern may represent a chronic change from a previous ischemic insult as seen in a limited number of the patients in the control group.

In conclusion, we here define a new pattern frequently indicating an inferior MI with concomitant critical lesion(s) in coronary arteries other than the infarct-related artery. Recognition of this pattern is important since (1) it indicates an acute atherothrombosis event that frequently leads to inferior MI despite ECG not showing contiguous STE, and (2) the patients with this pattern have higher short- and long-term risk for mortality.

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Declaration of competing interest

The authors report no relationships that could be construed as a conflict of interest.

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References

[1] Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Executive Group on behalf of the Joint European Society of Cardiology (ESC)/American College of Cardiology (ACC)/American Heart Association (AHA)/World Heart Federation (WHF) Task Force for the Universal Definition of Myocardial Infarction. Fourth Universal Definition of Myocardial Infarction. J Am Coll Cardiol. 2018;72:2231–64.

[2] Fiol M, Carrillo A, Cygankiewicz I, Velasco J, Riera M, Bayés-Genis A, et al. A new electrocardiographic algorithm to locate the occlusion in left anterior descending coronary artery. Clin Cardiol. 2009;32:E1–6.

[3] Bozbeyoğlu E, Aslanger E, Yıldırımtürk Ö, Şimşek B, Hünük B, Karabay CY, et al. The established electrocardiographic classification of anterior wall myocardial infarction misguides clinicians in terms of infarct location, extent and prognosis. Ann Noninvasive Electrocardiol. 2019;24:e12628.

[4] Bozbeyoğlu E, Aslanger E, Yıldırımtürk Ö, Şimşek B, Karabay CY, Türer A, et al. An algorithm for the differentiation of the infarct territory in difficult to discern electrocardiograms. J Electrocardiol. 2018;51:1055–60.

[5] Bozbeyoğlu E, Yıldırımtürk Ö, Aslanger E, Şimşek B, Karabay CY, Özveren O, et al. Is the inferior ST-segment elevation in anterior myocardial infarction reliable in prediction of wrap-around left anterior descending artery occlusion? Anatol J Cardiol. 2019;21:253–8.

[6] Durant E, Singh A. Acute first diagonal artery occlusion: a characteristic pattern of ST-elevation in noncontiguous leads. Am J Emerg Med. 2015;33:1326.e3-5.

[7] Sclarovsky S, Birnbaum Y, Solodky A, Zafrir N, Wurzel M, Rechavia E. Isolated mid-anterior myocardial infarction: a special electrocardiographic sub-type of acute myocardial infarction consisting of ST-elevation in non-consecutive leads and two different morphologic types of ST-depression. Int J Cardiol. 1994;46:37–47.

[8] Fox KA, Dabbous OH, Goldberg RJ, Pieper KS, Eagle KA, Van de Werf F, et al. Prediction of risk of death and myocardial infarction in the six months after presentation with acute coronary syndrome: prospective multinational observational study (GRACE). BMJ. 2006;333(7578):1091.

[9] Bayés de Luna A, Wagner G, Birnbaum Y, Nikus K, Fiol M, Gorgels A, et al. A new terminology for left ventricular walls and location of myocardial infarcts that present Q-wave based on the standard of cardiac magnetic resonance imaging: a statement for healthcare professionals from a committee appointed by the International Society for Holter and Noninvasive Electrocardiography. Circulation. 2006;114:1755–60.

[10] Dumas F, Manzo-Silberman S, Fichet J, Mami Z, Zuber B, Vivien B, et al. Can early cardiac troponin I measurement help to predict recent coronary occlusion in out-of-hospital cardiac arrest survivors? Crit Care Med. 2012;40:1777–84.

[11] Boden H, Ahmed TA, Velders MA, van der Hoeven BL, Hoogslag GE, Bootsma M, et al. Peak and fixed-time high-sensitive troponin for prediction of infarct size, impaired left ventricular function, and adverse outcomes in patients with first ST-segment elevation myocardial infarction receiving percutaneous coronary intervention. Am J Cardiol. 2013;111:1387–93.

[12] Arbane M, Goy JJ. Prediction of the site of total occlusion in the left anterior descending coronary artery using admission electrocardiogram in anterior wall acute myocardial infarction. Am J Cardiol. 2000;85:487–91.

[13] Engelen DJ, Gorgels AP, Cheriex EC, De Muinck ED, Ophuis AJ, Dassen WR, et al. Value of the electrocardiogram in localizing the occlusion site in the left anterior descending coronary artery in acute anterior myocardial infarction. J Am Coll Cardiol. 1999;34:389–95.

[14] Fiol M, Cygankiewicz I, Guindo J, Flotats A, Genis AB, Carreras F, et al. Evolving myocardial infarction with ST elevation: ups and downs of ST in different leads identifies the culprit artery and location of the occlusion. Ann Noninvasive Electrocardiol. 2004;9:180–6.

[15] Taglieri N, Saia F, Alessi L, Cinti L, Reggiani ML, Lorenzini M, et al. Diagnostic performance of standard electrocardiogram for prediction of infarct related artery and site of coronary occlusion in unselected STEMI patients undergoing primary percutaneous coronary intervention. Eur Heart J Acute Cardiovasc Care. 2014;3:326–39.