Validation of Electrocardiographic Criteria for Predicting the Culprit Artery in Patients with Acute Myocardial Infarction

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Abstract

In this study we compare a variety of ECG criteria for predicting the culprit artery in cases with ST-segment elevation myocardial infarction (STEMI). We also assess the performance of these criteria for patients who did not meet the STEMI criteria although having an acute myocardial infarction (AMI).

The study population consisted of 623 patients of whom 64.6% fulfilled STEMI criteria. All patients underwent percutaneous coronary intervention of a single vessel for treatment of AMI.

The criteria proposed by Tierala have the highest performance for detecting the culprit artery in STEMI cases, although for some arteries specificity and sensitivity values were low. Performances are significantly lower in patients who do not meet the STEMI criteria. Improved criteria that better predict the culprit artery in the whole group of AMI patients are currently under investigation.

1. Introduction

Early identification of the culprit artery in patients with symptoms of acute myocardial infarction (AMI) could reduce the time to reperfusion in Percutaneous Coronary Intervention (PCI) and permit a better risk stratification. The possibly infarct-related arteries (IRA) under consideration are the left anterior descending artery (LAD), the right coronary artery (RCA), and the left circumflex artery (LCx). Many algorithms have been proposed to predict the IRA based on measurements in the electrocardiogram (ECG) [1-8]. However, the ECG at any moment is the resultant of the positive and negative contributions of an infinity of variously directed vectorial electric forces. Due to these "cancellation" effects the relation between ECG changes and the location and extent of myocardial injury is very complex, which is detrimental to the proper identification of the culprit artery by means of ECG parameters.

All of the proposed algorithms have been developed for use in ST-elevated myocardial infarction (STEMI) and especially inferior ST-elevated myocardial infarction (ISTEMI). Although AMI that meets the STEMI criteria is thought to be more acute and life-threatening than NSTEMI, Chua et al. showed that patients with NSTEMI may have smaller infarct sizes, but that they have similar short- and long-term outcomes and a similar rate of reinfarction [9]. Wang et al. in a recent study on 47,454 patients with NSTEMI [10] argue that the emphasis is too much on STEMI versus NSTEMI. They also stress the need for algorithms that may identify the culprit artery in both types of AMI.

As a first step in addressing this challenging problem we wanted to validate the performance of currently existing criteria to predict the IRA in STEMI, ISTEMI, and NSTEMI groups.

2. Methods

The study population consisted of patients who underwent a PCI for treatment of AMI in one of the four participating centres. The database contained 659 well-documented PCI procedures of patients suffering from a single-vessel disease with TIMI (Thrombolysis In Myocardial Infarction) flow grade zero. For each patient a digital ECG had been recorded within a timeframe of maximally 2 hours before the first balloon inflation. All ECGs were processed by the Modular ECG Analysis System (MEANS) [11]. To reduce noise, MEANS computes a representative averaged beat for each of the twelve leads. From these averaged complexes the ECG measurements are derived. Cases with right or left bundle branch block were excluded (n = 36). STEMI was defined as an elevation at the J-point of ≥ 0.2 mV in two or more contiguous leads in leads V1, V2 or V3, and of ≥ 0.1 mV in other contiguous leads. Contiguity in the frontal plane is defined in the lead sequence aVL, I, inverted aVR, II, aVF, III. ISTEMI was defined as ST elevation in at least 2 of the leads II, III, and aVF. Table 1 presents the distribution of the culprit arteries in each subset.

Table 1. Distribution of the culprit arteries in the NSTEMI, NSTEMI, and ISTEMI groups.

Culprit artery	NSTEMI	STEMI	ISTEMI
LAD	112	150	32
RCA	70	184	178
LCx	56	51	41
Total	238	385	251

We compared the following criteria:

1) The sequential algorithm of Fiol et al. [3] was developed for RCA and LCx in cases with AMI of the inferoposterior wall and is shown in Figure 1.

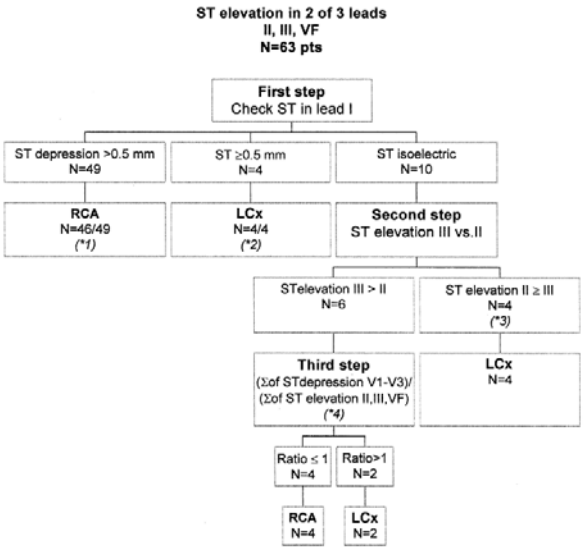

Figure 1. Algorithm of Fiol et al. [3] for predicting RCA or LCx as the culprit artery.

Fiol et al. also presented an algorithm for identifying the LAD as the IRA [12]. This was considered to be the case if ST elevation in the precordial leads exceeded the ST elevations in the inferior leads.

2) Tierala et al. predicted LAD to be the culprit artery if the maximal ST elevation occurred in the precordial leads V2 to V4 [4]. For ISTEMI cases Tierala used the algorithm as presented in Figure 2. In addition, the RCA was pointed out if ST elevation ≥ 0.1 mm in at least two of leads V1 through V3 with a maximum in lead V1 and without ST elevations in leads V4 through V6. Further, the LCx was held to be the culprit if the maximal ST elevation was in lead I or aVL with concomitant ST elevation in at least one of leads V4 through V6, but not in leads V1 through V3.

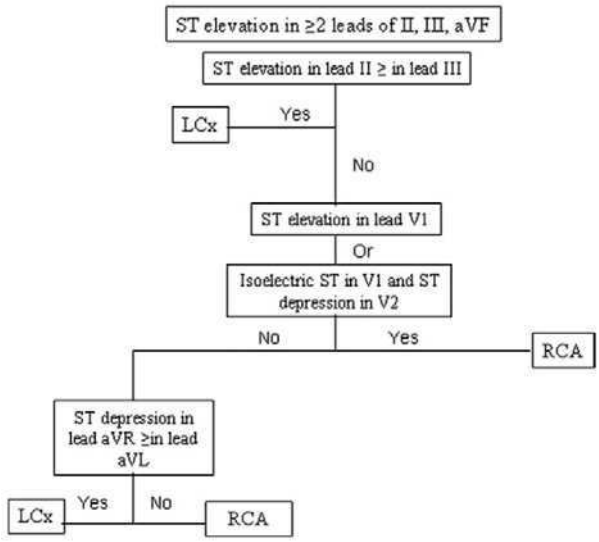

Figure 2. Algorithm of Tierala et al. [4] for predicting RCA or LCx as the culprit artery.

3) Wang et al. proposed an algorithm to not only predict the vessel containing the culprit lesion, but also to predict if the lesion was located proximally or distally in that vessel [5]. We refer to the original paper for details. In this study we combined the criteria for proximal and distal location for each IRA.

Additionally, we evaluated six simple criteria that were previously proposed for predicting RCA or LCx in ISTEMI patients. They are given in Table 2.

Unfortunately, some of the algorithms above do not quantitatively define ST elevation and depression. We define ST elevation in concordance with the STEMI criteria of the American Heart Association: amplitude at the J point ≥ 0.2 mV for leads V1, V2, V3 and ≥ 0.1 mV for the other leads [13]. For all leads, ST depression was defined as a negative J amplitude with absolute value ≥ 0.1 mV.

The performance for each of these algorithms and criteria is expressed by the Index of Merit, IM = sensitivity + specificity – 100 [14]. The index gives equal weight to sensitivity and specificity. For 100% perfect sensitivity and specificity, IM = 100. If cases are randomly called positive or negative, sensitivity = specificity = 50% and IM = 0.

Table 2. Sensitivity, specificity, and IM of the ECG criteria for RCA in the NSTEMI, STEMI and ISTEMI group.

	NSTEMI			STEMI			ISTEMI		
ECG criteria	Sens	Spec	IM	Sens	Spec	IM	Sens	Spec	IM
Fiol	20.0	97.0	17.0	90.2	82.1	72.3	94.4	38.4	32.8
Tierala	20.0	95.8	15.8	91.3	80.6	71.9	94.4	39.7	34.1
Wang	10.0	98.8	8.8	68.5	86.6	55.1	70.8	67.1	37.9
ST↓ I ≥ 0.1 mV [2]	10.0	93.5	3.5	48.4	91.5	39.9	50.0	78.1	28.1
ST↓ aVL ≥ 0.1 mV [1]	17.1	95.2	12.3	88.0	77.6	65.6	90.4	38.4	28.8
ST↑ III > ST↑ II, ST↓ I ≥ 0.1 mV [2]	10.0	94.0	4.0	48.4	91.5	39.9	50.0	78.1	28.1
ST↓ aVR ≥ 0.1 mV [8]	100.0	2.4	2.4	67.9	33.3	1.2	68.0	57.5	25.5
ST↓ V3 / ST↑ III > 1.2 [7]	71.4	36.9	8.3	96.2	4.0	0.2	97.2	5.5	2.7

Table 3. Sensitivity, specificity, and IM of the ECG criteria for LCx in the NSTEMI, STEMI and ISTEMI group.

	NSTEMI			STEMI			ISTEMI		
ECG criteria	Sens	Spec	IM	Sens	Spec	IM	Sens	Spec	IM
Fiol	3.6	100.0	3.6	17.6	98.5	16.1	41.5	94.3	35.7
Tierala	1.8	100.0	1.8	31.4	98.5	29.9	43.9	94.3	38.2
Wang	1.8	100.0	1.8	37.3	88.0	25.3	46.3	81.0	27.3
ST↓ I ≥ 0.1 mV	91.1	7.1	–1.8	86.3	29.6	15.9	82.9	46.7	29.6
ST↓ aVL ≥ 0.1 mV	92.9	8.8	1.7	54.9	55.1	10.0	41.5	86.7	28.2
ST↑ III > ST↑ II, ST↓ I ≥ 0.1 mV	91.1	6.6	–2.3	86.3	29.6	15.9	82.9	46.7	29.6
ST↓ aVR ≥ 0.1 mV	3.6	98.9	2.5	52.9	70.4	23.3	61.0	64.8	25.8
ST↓ V3 / ST↑ III > 1.2	66.1	75.3	41.4	13.7	97.6	11.3	9.8	97.6	7.4

3. Results

Tables 2 and 3 show the sensitivity, specificity, and IM of the ECG criteria for predicting RCA and LCx, respectively. As can be seen, reasonable performance (IM > 60) is only shown in the STEMI group by the algorithms of Fiol and Tierala and by the "simple" criterion ST↓ aVL ≥ 0.1 mV, and that only for RCA. Sensitivities of these algorithms are good, but specificities are moderate (around 80%). For LCx, the algorithms of Fiol and Tierala show excellent specificities, but sensitivities are low yielding poor overall performance. In fact, for all other algorithms and situations performance is disappointing, if not disastrous. In the subgroup of ISTEMI cases, none of the "simple" criteria had an IM > 30.

In Table 4 results of the algorithms by Fiol, Tierala, and Wang are presented for LAD. Tierala performs best in the STEMI group, with comparable sensitivity but higher specificity as the algorithm by Fiol. The sensitivity of the algorithm by Wang is low in our study population. In the NSTEMI group, all algorithms perform poorly.

Table 4. Sensitivity, specificity, and IM of the ECG criteria for LAD in the NSTEMI and STEMI groups.

	NSTEMI			STEMI		
Criteria	Sens	Spec	IM	Sens	Spec	IM
Fiol	83.0	60.3	43.4	88.7	87.7	76.3
Tierala	64.3	82.5	46.8	86.7	96.2	82.8
Wang	18.8	96.0	14.8	55.3	90.6	46.0

4. Discussion

We compared the performance of previously proposed criteria for predicting the culprit artery in patients with AMI. Overall, our results indicate moderate to poor performance for STEMI cases. The Tierala criteria have reasonable performance for LAD and RCA, but some of the sensitivity and specificity values are low. For LCx, performance of all algorithms is poor (IM < 40).

Our results are difficult to compare with those in previous reports because of differences in the composition of the study population, in location and severity of the lesions in the arteries, time between ECG recording and PCI, etc.

In NSTEMI cases, performances significantly decreased. This of course does not come as a surprise since the studied criteria were not designed for this group of patients, but considering recent reports that show similar outcomes for STEMI and NSTEMI patients, our findings underline the need for new criteria to detect occluded arteries in the NSTEMI group. Such new criteria are also of clinical relevance considering the large proportion of NSTEMI cases in our study population. Finally, our results also suggest that criteria for the STEMI group need to be improved to be useful in clinical practice.

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