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ABSTRACT

Objective: To describe patients with a distinct electrocardiogram (ECG) pattern without ST-segment elevation in the presence of an acute occlusion of the proximal left anterior descending (LAD) artery.

Design: Single-centre observational study.

Patients: Patients with acute anterior wall myocardial infarction who were referred for primary percutaneous coronary intervention (PCI) between 1998 and 2008.

Results: We identified patients with a static, distinct ECG pattern without ST-segment elevation and an occlusion of the proximal LAD artery during urgent coronary angiography before PCI. Of 1890 patients who underwent primary PCI of the LAD artery, we could identify 35 patients (2%) with this distinct ECG pattern. The ECG showed ST-segment depression at the J-point of at least 1 mm in precordial leads with upsloping ST-segments continuing into tall, symmetrical T-waves. Patients with this distinct ECG pattern were younger, more often male and more often had hypercholesterolaemia compared to patients with anterior myocardial infarction and ST-segment elevation.

Conclusions: In patients presenting with chest pain, ST-segment depression at the J-point with upsloping ST-segments and tall, symmetrical T-waves in the precordial leads of the 12-lead ECG signifies proximal LAD artery occlusion. It is important for cardiologists and emergency care physicians to recognise this distinct ECG pattern, so they can triage such patients for immediate reperfusion therapy.

The standard 12-lead electrocardiogram (ECG) remains the most important clinical tool for the timely identification and management of patients with acute myocardial infarction.[1] Accurate recognition of characteristic changes in the pattern of the QRS-complex, ST-segments and T-waves associated with acute proximal occlusion of a large epicardial coronary artery is essential in predicting the amount of myocardium at risk and guides decisions regarding the need for immediate reperfusion therapy.[2–4] In the early days of reperfusion therapy, patients would only qualify for thrombolytic therapy when there was unequivocal evidence of acute transmural ischaemia with cumulative ST-segment deviation of 12–15 mm present on the ECG.[5]

Nowadays, primary percutaneous coronary intervention (PCI) within 2 hours of first medical contact for acute transmural myocardial infarction is the preferred treatment for reperfusion.[6] According to the current guidelines, primary PCI should be performed in patients with a clinical presentation of ST-segment elevation myocardial infarction (STEMI) within 12 hours of symptom onset and persistent ST-segment elevation at the J-point in at least two contiguous lead with cut-offs of ≥2 mm in lead V2-V3 and ≥1 mm in the remaining leads.[7–9]

In the region serviced by our centre, a 12-lead ECG is recorded in patients with chest pain either on admission in the referring hospital or in the ambulance. This ECG is transmitted by telephone to the interventional operator on call who decides to transport the patients directly to the catheterisation laboratory. We recently described a distinct pattern on the 12-lead ECG without ST-segment elevation, which was strongly associated with proximal left anterior descending (LAD) artery occlusion.[10] From our database that comprises pre-hospital ECG, pre-procedural ECG, and the coronary angiogram, we further explored possible explanations for the absence of ST-segment elevation in the presence of proximal LAD artery occlusion causing a large area of anterior transmural myocardial ischemia.

METHODS

Case finding

In our institution, patients qualify for primary PCI when they present with ischaemic chest pain lasting for less than 12 hours together with an ECG with at least 1 mm ST-segment elevation in two contiguous leads. The 12-lead ECG is recorded either on admission to our cardiac emergency department, on admission to one of our referring hospitals or in the ambulance, and is routinely sent by telephone to the interventional cardiologist on call who makes the decision to transport the patient directly to the catheterisation laboratory.

Thus, the ECG is evaluated by a cardiologist with more experience in the interpretation of electrocardiographic changes together with the angiographic characteristics in patients with large, transmural infarctions. When there is ischaemic chest pain and the 12-lead ECG shows abnormalities but no clear ST-segment elevation, the decision to transport the patient is at the discretion of the interventional operator. The first patient with an ECG pattern as described was recognised in January 1998. This patient underwent emergency coronary angiography on the basis of persistent clinical symptoms of acute myocardial infarction and showed a proximal occlusion of the LAD artery. From that time onwards, patients with similar electrocardiographic changes were routinely accepted by our interventional operators to be transported directly to our catheterisation laboratory.

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As in the clinical protocol, all patients received aspirin (500 mg) and unfractionated heparin (5000 IU) during transport. As from 2005, clopidogrel (300 mg) was given during transport. Glycoprotein IIb/IIIa inhibitor administration was at the discretion of the interventional operator. Coronary angiography and, when feasible, angioplasty were performed using standard techniques.

Data collection

Between January 1998 and March 2008, a total of 1890 patients who underwent primary PCI for anterior myocardial infarction at our institution were recorded in the database. Of these patients, a set of "first medical contact" ECG (either ambulance ECG or referring hospital ECG) and pre-procedural ECG was recorded as part of standard care. In a retrospective manner, we were able to collect both ECGs in a dedicated database in 625 patients, together with prospectively collected data on baseline characteristics, risk factors, haemodynamic status on admission, transfer time intervals and total ischaemic times.

ECG analyses

After qualitative selection, ECGs of included patients were scanned into a computer environment and digitally analysed with freely available Image J software (version 1.41, 2008, http://rsb.info.nih.gov/ij/index.html). After digital calibration and magnification, the isoelectric baseline, J-point and landmarks for conduction analyses were identified by one investigator (NV) blinded to the patient and procedural characteristics. From this, J-point deviations and conduction durations were calculated by the computer software. These distances were noted on a form and manually entered into a digital database for further analyses. The TP-segment was considered the preferred isoelectric baseline.

Statistical analyses

Normally distributed, continuous variables are expressed as means (SD) and were compared using a Student t test. Other continuous data are presented as median with interquartile range (IQR) and compared using the Mann-Whitney U test as a non-parametric equivalent. All categorical variables are depicted using absolute and relative frequency distributions and the χ^2 test was used to make a comparison. For all tests, differences were considered significant if the two-sided p value was less than 0.05. All analyses were performed using SPSS software package.

RESULTS

Among the 1890 patients with an anterior myocardial infarction, a familiar ECG pattern with precordial ST-segment elevation was present in 1855 patients (98%), while 35 patients (2%) showed the new ECG pattern. In these 35 patients with the new pattern, the ECG showed absence of ST-segment elevation in leads V1 to V6. We observed overt ST-segment depression at the J-point (>1 mm) with upsloping ST-segments continuing into tall, symmetrical T-waves in the precordial leads. The QRS complexes were usually not widened or only slightly widened and in some but not all there was a loss of R-wave progression from V1 to V6. The intrinsicoid deflection was preserved in most patients. If present, reciprocal ST-segment depression in the inferior leads was modest. Remarkably, included patients showed a mean J-point elevation of approximately 0.5 mm in lead aVR (fig 1). Examples of this
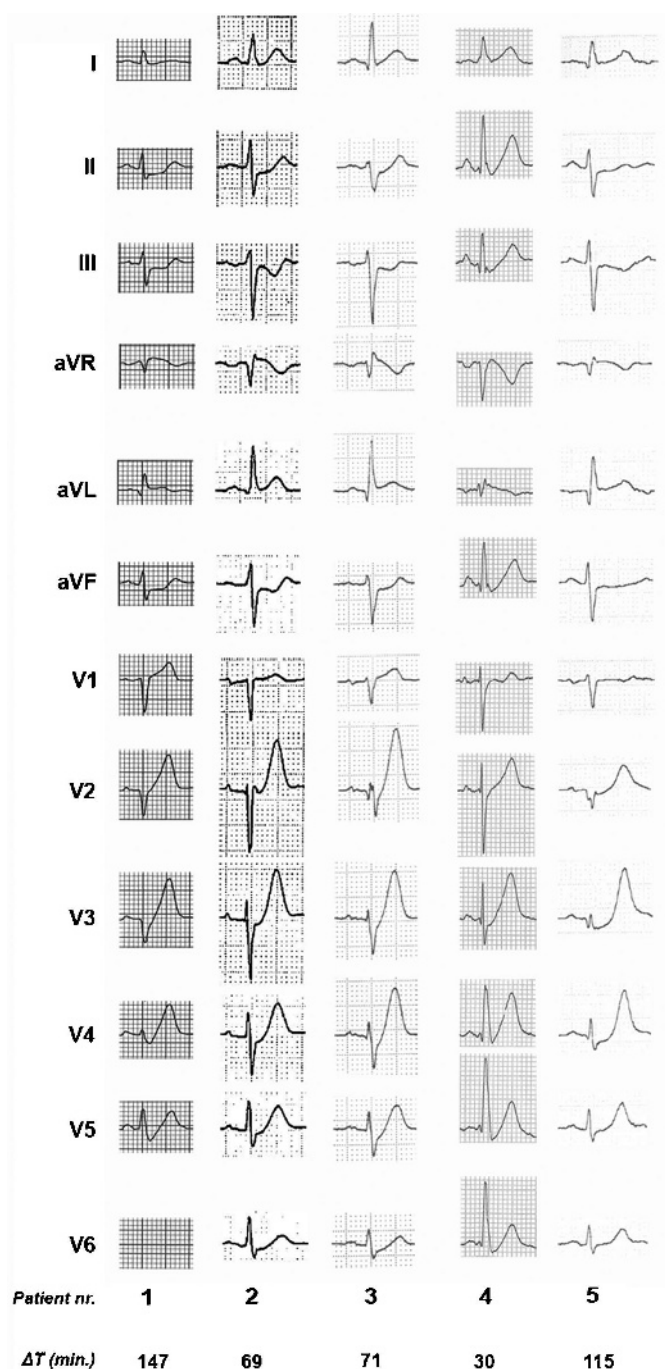

Figure 1 Examples of 12-lead electrocardiograms (ECGs) and left anterior descending (LAD) artery occlusion. 12-lead ECG recordings of five selected patients with precordial ST-segment depression at the J-point followed by peaked, positive T-waves. Lead aVR displays slight ST-segment elevation in the majority of cases. Emergency coronary angiography showed a proximal LAD artery occlusion in all five patients. ΔT denotes time between onset symptoms and depicted 12-lead ECG.

ECG pattern from other patients included in this cohort have been published previously.[10]

Invariably, this ECG pattern was associated with an occlusion of the proximal LAD artery on the emergency coronary angiogram. Although the ECG has features of hyperacute anterior transmural ischaemia (tall, symmetrical T-waves), which has been recognised as a transient feature dynamically changing into overt ST-segment elevation in the precordial

Figure 2 Serial electrocardiograms. Initial ECG (A) of a 52-year old man with clinical signs of myocardial infarction for approximately 90 minutes. (B) The pre-procedural ECG performed 71 minutes after the initial ECG (A) and roughly 2½ hours after onset of symptoms. The coronary angiogram performed shortly after this registration revealed a fresh occlusion of the proximal LAD artery.
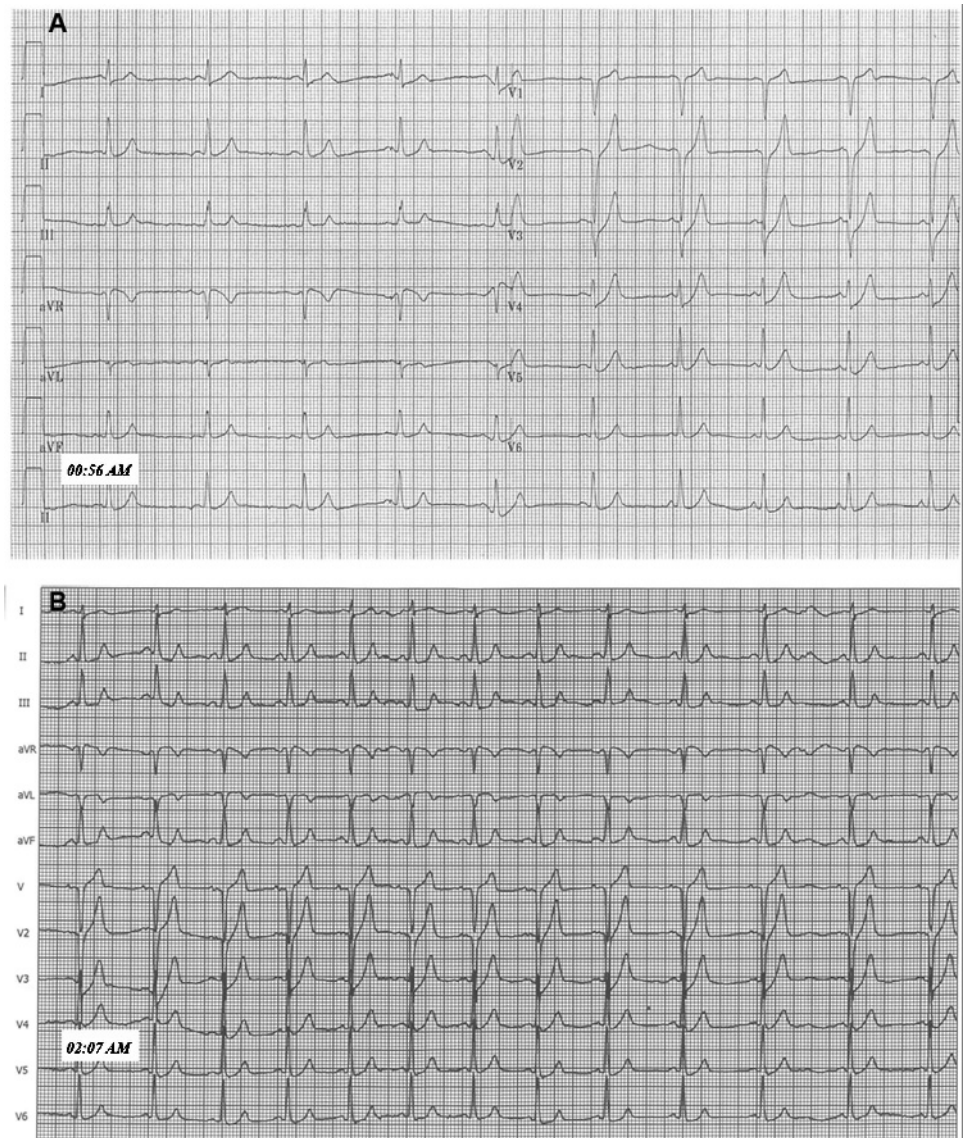
leads, we have observed this pattern as a static ECG pattern lasting from the time of first medical contact until the recording of the pre-procedural ECG and lasting until angiographic establishment of an occluded LAD artery (that is, approximately 60 minutes) (fig 2). T-wave inversion between first medical contact and arrival at the catheterisation laboratory was an occasional finding and occurred in lead aVF in four patients. We have never observed this ECG pattern in patients with transmural infarction and the right coronary artery or left circumflex coronary artery as the infarct-related vessel.

This novel ECG pattern, as described above, resolved after reperfusion in all included patients. After PCI of the LAD artery, we observed common ECG patterns associated with reperfusion, such as accelerated idioventricular rhythms, loss of R-waves in the precordial leads, modest residual ST-segment elevation or depression and T-wave inversion.

Baseline characteristics of the 35 patients with the above-described typical ECG pattern were compared with other patients with anterior myocardial infarction collected during the same time period, which showed that the selected patients were younger, more often male and more frequently had hypercholesterolaemia (table 1).

To further understand the mechanism of the absence of ST-segment elevation, we analysed electrocardiographic and angiographic characteristics. The ECGs with characteristic pattern were on average recorded approximately 1½ hours after symptom onset. There was normal sinus rhythm in nearly all cases (97%), a slightly accelerated heart rate (median 75 beats/min), an intermediate electrical axis in 30 cases (86%) and no marked conduction abnormalities (table 2). On coronary angiography, the culprit lesion invariably was the proximal LAD artery (either proximal to the first septal branch or distal to the first septal and proximal to the first diagonal branch). In three patients there was Thrombolysis In Myocardial Infarction 3 (TIMI-3) flow in the presence of a significant stenosis at the time of diagnostic angiography. Collateral filling of the LAD artery ranged from Rentrop class 0–3 and a "wraparound" LAD artery was present in approximately 50% of patients. There was no angiographic evidence of (transient) involvement of the left main coronary artery in any of the patients. The majority (67%) had isolated LAD artery disease without any significant coronary lesions elsewhere (table 2).

Despite adequate antegrade flow after the procedure in all cases, selected patients with the characteristic ECG pattern had

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Table 1 Baseline characteristics of primary PCI patients treated for anterior myocardial infarction

Characteristic	Anterior MI patients with described ECG pattern (n = 35)	Anterior STEMI patients (n = 1855)	p Value
Age (years)*	52 (8)	61 (13)	<0.001
Male gender	33 (94)	1331 (72)	0.002
BMI (kg.m^{-2})*	26 (3)	27 (6)	0.58
Risk factors			
Family history of CAD	15 (43)	708 (38)	0.57
Current smoking	19 (54)	779 (42)	0.15
Hypertension	7 (20)	557 (30)	0.20
Diabetes mellitus	3 (9)	219 (12)	0.79
Hypercholesterolaemia	15 (43)	372 (20)	0.001
Previous MI	2 (6)	237 (13)	0.31
Previous PCI	0	150 (8)	0.11
Previous CABG	0	5 (0)	1.00
Total ischaemic time (minutes) (median (IQR))	167 (126–235)	173 (126–248)	0.30
Timing ECG recordings (minutes) (median (IQR))†			
Symptom onset, ECG at first medical contact	92 (54–133)	96 (57–157)	0.36
ECG at first medical contact, preprocedural ECG	60 (42–101)	67 (52–95)	0.52

*Means (SD). Other data are expressed as n (%) unless otherwise indicated.
†Data available for 590 patients in reference population.
BMI, body mass index; CABG, coronary artery bypass grafting; CAD, coronary artery disease; ECG, electrocardiogram; IQR, interquartile range; MI, myocardial infarction; PCI, percutaneous coronary intervention; STEMI, ST-segment elevation myocardial infarction.

considerable loss of myocardium with a median creatine kinase isoenzyme MB (CK-MB) peak of 290 μg/l (table 2). Left ventricular function (LVF) was assessed by echocardiography during the days following primary PCI in 24 of the 35 patients and showed good LVF in 10 patients, impaired LVF in 10 patients, and poor LVF in four patients. During follow-up, one patient (3%) died within 30 days of the primary PCI procedure.

DISCUSSION
In this study we describe a novel, typical ECG pattern in patients with ischaemic chest pain and a large acute transmural anterior myocardial infarction, caused by an acute occlusion of the proximal LAD artery but without anterior ST-segment elevation. In approximately 2% of all patients presenting with anterior infarction, we observed this ECG pattern consisting of persistent ST-segment depression at the J-point with upsloping ST-segments continuing into tall, symmetrical T-waves in leads V1 to V6, accompanied by slight ST-segment elevation in aVR. This ECG pattern was invariably associated with a culprit lesion located in the proximal LAD artery, which was occluded in the majority of cases. Therefore, these patients qualify for immediate reperfusion therapy, preferably with primary PCI. In all patients included in this study, the characteristic ECG pattern immediately resolved after PCI of the LAD artery. The recognition of this ECG pattern in our centre and its correlation with the coronary angiogram occurred because all ECGs recorded "at first medical contact" were evaluated by the interventional operator on call and serial 12-lead ECGs were recorded before and during primary PCI in all patients. It is of great importance that all physicians and paramedics involved in triage of patients with chest pain do recognise this ECG pattern, and immediately refer these patients for primary PCI.

It goes without saying that the current computer algorithms designed to recognise ST-segment elevation will miss the above described ECG pattern warranting immediate primary PCI. In our opinion, guidelines for management of acute myocardial infarction should mention the likelihood of acute LAD artery occlusion in patients with chest pain with this typical ECG pattern. The subtle but characteristic electrocardiographic changes such as we describe in this study may be missed or misdiagnosed as reversible ischaemia,[8] which might substantially delay the transportation to a PCI centre or the start of reperfusion therapy. With the present day communication technologies, evaluation of the ECG by an experienced clinician is possible in all settings and in all regions and may decrease the considerable proportion of acute myocardial infarctions that remain unrecognised.[11]

The electrophysiological explanation of the observed ECG pattern remains elusive. We could not establish patient characteristics or angiographic characteristics that were unequivocally associated with the above described ECG pattern. We found that patients with this ECG pattern more often were young, of male gender and had hypercholesterolaemia, but other risk factors for coronary artery disease could not be related to this ECG pattern.[12] There was collateral protection as evident from Rentrop class-3 filling in only a few patients. A "wrap-around" LAD artery was present in approximately half of the patients, which is usually associated with ST-segment elevation in both precordial and standard inferior leads but not with absence of ST-segment elevation. There was no evidence of left main disease which could induce diffuse ischaemia of the whole left ventricle and the majority of patients had no angiographic evidence of significant coronary lesions besides an occluded LAD artery.

The classic description of the occurrence of ST-segment elevation, or the "pattern of injury" comes from experiments in dogs by Bayley et al.[13][14] Later studies showed that loss of action potential amplitude, action potential shortening and delayed activation are associated with ST-segment elevation and follow a characteristic time course.[15][16] The reason why our patients did not show ST-segment elevation in the presence of an occluded proximal LAD artery remains speculative. A possible explanation could be that the area of transmural ischaemia was very large, such that no injury currents were generated towards the

Table 2 Angiographic and procedural characteristics of selected cohort

Characteristic	Anterior MI patients with described ECG pattern (n = 35)
Electrocardiography	
Rhythm	
Sinus rhythm	34 (97)
Atrial fibrillation	1 (3)
Heart rate (min⁻¹) (median (IQR))	75 (64–87)
Electrical heart axis	
Intermediate	30 (86)
Left	5 (14)
Conduction (ms)	
PR interval*	165 (19)
QRS duration*	88 (12)
QTc interval*	394 (24)
Time from symptom onset to ECG recording (minutes) (median (IQR))	98 (57–145)
Coronary angiography	
Single-vessel disease	24 (67)
Dominant RCA	30 (86)
Location culprit lesion in LAD artery	
Proximal to 1st septal, proximal to 1st diagonal branch	23 (66)
Distal to 1st septal, proximal to 1st diagonal branch	12 (34)
Pre-PCI TIMI flow 0–1	30 (86)
Collateral circulation	
Rentrop class 0–1	25 (29)
Rentrop 2	9 (26)
Rentrop 3	1 (3)
Wrap around LAD artery	20 (57)
Procedural complexity	
GP IIb/IIIa receptor inhibitors used	7 (20)
IABP inserted	4 (11)
Post-PCI TIMI 3 flow	32 (91)
Laboratory	
Potassium on admission (mmol/l)*	3.9 (0.4)
Peak CK-MB value (μg/l) (median (IQR))†	290 (155–471)

*Means (SD). Other data are expressed as n (%) unless otherwise indicated.
†Peak CK-MB available in 32 cases.
CAD, coronary artery disease; CK-MB, creatine kinase isoenzyme MB; ECG, electrocardiogram; GP, glycoprotein; IABP, intra-aortic balloon pump; LAD, left anterior descending; LM, left main coronary artery; PCI, percutaneous coronary intervention; RCA, right coronary artery; TIMI, Thrombolysis In Myocardial Infarction.

precordial leads but only directed upwards to standard lead aVR. Some of our patients did show progressive loss of R-wave potential in the hours and days following primary PCI suggesting a large area at risk. Theoretically, an anatomical variant of the Purkinje fibres with endocardial conduction delay could result in the described ECG pattern. An alternative explanation might be that the absence of ST-segment elevation may be related to the lack of activation of sarcolemmal adenosine triphosphate (ATP)-sensitive potassium (K_{ATP}) channels by ischaemic ATP depletion as has been shown in (K_{ATP} knockout) animal models of acute ischaemia.[17]

Limitations

This study has several limitations. We did not routinely record leads V7-V12 and may have missed ST-segment elevation present in the postero-lateral leads. However, a 12-lead ECG was routinely recorded "at first medical contact" and there was normal R-wave amplitude transition in the precordial leads. Moreover, ST-segment elevation in the postero-lateral leads is usually associated with occlusion of the left circumflex artery and its branches, not with LAD artery involvement. Second, we

did not have the availability of continuous ST-segment monitoring and do not have data on electrocardiographic changes that may have occurred between "first medical contact" and arrival at the catheterisation laboratory. However, we have documented that the ECG pattern on arrival at our catheterisation laboratory was unchanged in many patients and that this pattern was present at the time the LAD artery occlusion was diagnosed, thus ruling out the possibility that this ECG pattern is a transient feature that was present either before or after overt ST-segment elevation in most of our patients. Third, we did not record endocardial or epicardial potentials during the primary PCI procedure in order to map action potential propagation and local action potential duration. Finally, owing to the retrospective collection of the ECG data, we had electrocardiographic data from a selection of patients with anterior myocardial infarction in the reference group. Therefore, any comparison should be considered hypothesis generating. In addition, we have no information from patients in whom the ECG was not sent to our institution. Thus, the 2% incidence of this ECG pattern is probably an underestimation of the true incidence in patients with acute ischaemic chest pain. The retrospective design of this study precludes a precise assessment of diagnostic accuracy of this new ECG pattern and therefore large, prospective registries should be undertaken to determine both a diagnostic and prognostic value of this ECG pattern. In our experience, the positive predictive value of this ECG pattern was 100%, but we can not rule out false positive cases, which were not presented by referring physicians.

Conclusions

Our study describes the occurrence of a distinct ECG pattern of persistent ST-segment depression at the J-point with upsloping ST-segments continuing into tall, symmetrical T-waves in the precordial leads in patients with acute, proximal LAD artery occlusion. The incidence of this ECG pattern may be approximately 2% of all anterior myocardial infarctions admitted to a dedicated pre-hospital triage network. Recognition of this ECG pattern is of vital importance to triage these patients for immediate reperfusion therapy.

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Notes