Interpretation of acute myocardial infarction with persistent 'hyperacute T waves' by cardiac magnetic resonance

Alessandro Zorzi, Martina Perazzolo Marra, Federico Migliore, Giuseppe Tarantini, Sabino Iliceto and Domenico Corrado

European Heart Journal: Acute Cardiovascular Care
1(4) 344–348

© The European Society of Cardiology 2012

Reprints and permission:

sagepub.co.uk/journalsPermissions.nav

DOI: 10.1177/2048872612466537

acc.sagepub.com

Abstract

Peaked and tall T waves represent a hyperacute electrocardiogram (ECG) manifestation of coronary artery occlusion which usually evolves into ST-segment elevation. We sought to investigate using cardiac magnetic resonance (CMR) the myocardial tissue changes underlying an atypical ECG pattern of presentation of left anterior descending artery (LAD) occlusion consisting of persistent hyperacute T waves and mild ST-segment depression. This ECG pattern is often associated with the presence of collateral circulation, which may modulate myocyte action potential changes in response to ischemia and prevent the appearance of ST-segment elevation. However, CMR findings resembled those of typical anterior myocardial infarction with nearly transmural necrosis in the large myocardial area supplied by LAD. Accordingly, persistent hyperacute T waves should be regarded as an equivalent to ST-segment elevation and immediate reperfusion therapy should be considered.

Keywords

Myocardial infarction, acute coronary syndrome, cardiac magnetic resonance, ST segment, hyperacute T wave

Date received: 9 June 2012; accepted: 11 October 2012

Introduction

According to the electrocardiogram (ECG) presentation, acute coronary syndromes are traditionally classified as STEMI (i.e. ST-segment elevation acute myocardial infarction) and NSTEMI (i.e. non-ST-segment elevation acute myocardial infarction). ST-segment elevation is a marker of transmural ischemia secondary to acute coronary artery occlusion; on the contrary, ST-segment depression is usually a sign of subendocardial (non-transmural) ischemia.¹ Whereas ST-segment elevation is an indication for immediate coronary reperfusion, in NSTEMI, a tailored invasive approach is recommended.² Transient tall and peaked T waves (also called 'hyperacute T wave') may occasionally precede ST-segment elevation. We report herein the case of a patient with acute occlusion of the left descending coronary (LAD) artery and atypical ECG manifestation of persistent hyperacute T waves in association with ST-segment depression in the anterior precordial leads. The pathophysiological mechanisms responsible for such unusual ECG pattern and its clinical implications were investigated using contrast-enhanced cardiac magnetic resonance (CE-CMR).

Case presentation

A 73-year-old male was admitted to the emergency department for chest pain, which had started approximately 1 hour previously and was associated with moderate dyspnea. He showed dyslipidemia as the only coronary risk factor, and an ECG recorded a few months before was normal. On admission, an ECG revealed the presence of tall and peaked T waves associated with up-sloping ST-segment depression in leads V2–V4 (Figure 1(a)). The first troponin sample was 0.56 μl (normal value <0.045 μl). This ECG pattern remained unchanged on the tracking recorded 4 hours later (Figure 1(b)). The patient was diagnosed with NSTEMI, which was considered at high risk

Division of Cardiology, Department of Cardiac, Thoracic and Vascular Sciences, University of Padova, Italy

Corresponding author:

Domenico Corrado, Inherited Arrhythmogenic Cardiomyopathy Unit, Department of Cardiac Thoracic and Vascular Sciences, University of Padua Medical School, Via N. Giustiniani 2 35121 Padua, Italy.
Email: domenico.corrado@unipd.it

Figure 1. Time-course of electrocardiographic abnormalities. The first 12-lead ECG, which was recorded 1 hour after symptom onset, showed peaked and tall positive T waves (so-called hyperacute T waves) in V2–V6, associated with mild ST-segment depression, and ‘R on T’ premature ventricular beats (a). Four hours later, a second ECG revealed persistence of hyperacute T waves (b). A third ECG, which was recorded 24 hours after admission at the time of cardiac catheterization, showed QS complexes in V1–V4 with disappearance of ST-T abnormalities (c).

according to a GRACE² acute coronary risk score of 161 and treated with aspirine, clopidogrel, fondaparinux and nitrates. He was haemodynamically stable but continued to complain of recurrent chest pain with no significant ST-segment changes on 12-lead ECG telemetry monitoring. Thus, cardiac catheterization was postponed, being performed 24 hours after admission.² Coronary angiography and left ventriculography showed occlusion of the proximal LAD artery with intercoronary Rentrop 2

collateral circulation and moderate (ejection fraction 38%) left ventricular (LV) systolic dysfunction (Figure 2). The patient was treated with coronary angioplasty and two drug-eluting stents. Troponine I peaked at 224 µg/l at day 2. Post-procedural ECG showed QS complex in V1–V4 and normalization of the ventricular repolarization (Figure 1(c)). Afterwards, subacute ECG changes occurred consisting of T wave inversion in the leads exploring the myocardial territory supplied by the proximal LAD artery.

Figure 2. Coronary angiography findings. Baseline antero-posterior view showing occlusion of the proximal left anterior descending coronary artery (a). The lesion was successfully treated by coronary angioplasty and stenting (b). Right anterior oblique revealing intercoronary collateral vessels from the right coronary artery to the left anterior descending coronary artery (arrows) (c).

Three days after admission, a CE-CMR scan (1.5-Tesla scanner, Magnetom Avanto, Siemens Medical Solutions, Erlangen, Germany) revealed the presence of full-thickness myocardial edema of the LV anterior wall, septum and apex on T2-weighted sequences, whereas T1-weighted inversion recovery post-contrast sequences showed late-gadolinium enhancement with the same regional distribution, involving the subendocardial and mediomural musculature (80–85% of wall thickness) but sparing the subepicardial layer (Figure 3). The estimated area at risk was 37% and infarct size was 32% with salvaged myocardium of 5%.³ The following hospital stay was uneventful and the patient was discharged at day 11. At 3-month follow-up, an echocardiogram showed persistence of LV systolic dysfunction with ejection fraction of 42% due to antero-septal akinesia.

Discussion

The electrophysiological bases and clinical significance of the ECG pattern of persistent ‘hyperacute T waves’ not evolving into ST-segment elevation in acute myocardial infarction (AMI) are not completely known.^{4,5} Previous reports indicated that persistent hyperacute T waves in anterior AMI are a favorable ECG sign, which suggests non-transmural myocardial ischemia. The hypothesized mechanisms include either flow preservation due to coronary subocclusion or coronary occlusion with subsidiary blood supply via collateral vessels. This interpretation has led to the current practice of initial conservative management of patients presenting with such an ECG pattern.^{6–9} This approach was recently called into question by the results of an angiographic study reporting a 2% prevalence of persistent hyperacute T waves in patients with acute occlusion of the LAD coronary artery, with only half presenting visible collateral vessels.^{10,11}

We reported a representative patient with evolving anterior AMI and the ECG pattern of persistent hyperacute T waves, who underwent comprehensive CE-CMR study. The CE-CMR findings provided insights into our understanding of the ischemia-induced myocardial changes underlying such atypical ECG pattern and its clinical implications.³ Whereas T2-weighted sequences demonstrated transmural myocardial edema in the large myocardial region supplied by the proximal LAD coronary artery, on T1-weighted inversion recovery post-contrast sequences the necrosis wave-front spared the epicardial layer. Except for the preservation of such a thin outer layer of LV musculature, the amount of myocardial necrosis evidenced by CE-CMR was comparable to that of anterior STEMI and accounted for the significant peak of enzymatic release, extent of subacute ECG repolarization changes and irreversible LV systolic dysfunction occurring despite delayed revascularization.¹² All these findings suggest that collateral circulation was unable to substantially limit the myocardial damage resulting from the LAD coronary artery occlusion. This is in agreement with previous studies showing that collateral circulation is less protective than expected in acute coronary occlusions and that patients with collateral vessels may have atypical clinical presentation potentially leading to delayed coronary interventions.^{13,14}

The electrogenetic mechanism of persistent hyperacute T waves is unclear. In patients with coronary artery occlusion, hyperacute T waves are usually transient and rapidly evolve to ST-segment elevation.¹ In the early phase of coronary artery occlusion the shortening of the subepicardial action potential (AP), resulting from activation of the ATP-dependent potassium current (I_{K-ATP}), is thought to increase the endo-epicardial voltage gradient of repolarization, which, in turn, generates tall and peaked T waves. As the ischemic insult prolongs, the epicardial AP dome may be completely lost leading to enhancement of transmural (i.e. endo-epicardial) and/or

Figure 3. Cardiac magnetic resonance findings. (Top) Increased signal intensity on T2-weighted sequences in the 4-chamber (a), 2-chamber (b) and mid short axis (c) views consistent with transmurular myocardial edema of the left ventricular anterior wall, ventricular septum, and apex (area at risk = 37%). (Bottom) T1-weighted inversion recovery post-contrast sequences in the same views (d–f) showing non-transmurular late-gadolinium enhancement suggestive of myocardial necrosis (infarct size = 32%) sparing the epicardial layers of the anterior wall and apical segments (arrowheads). Salvaged myocardium was 5%. An apical thrombosis is also evident (arrow).

regional (i.e. from the surrounding healthy area toward the ischemic area) dispersion of repolarization and giving rise to the ST-segment elevation.^{15,16} Despite LAD coronary artery occlusion, the presence of collateral circulation may have provided subsidiary blood supply which saved epicardial myocardium from dying and prevented the classic ST-segment elevation pattern from occurring.¹⁷ This pathophysiological hypothesis was supported by CE-CMR findings which demonstrated transmurular myocardial edema as a result of full-thickness myocardial ischemia but non-transmurular late-enhancement (i.e. not reaching the epicardium), suggesting that the outer LV myocardial layers of the infarcted areas were preserved from necrosis. From a clinical viewpoint, however, the ECG pattern of persistent hyperacute T wave heralded a large myocardial infarction which was comparable to typical anterior STEMI. Therefore, this ECG sign should be recognized as an atypical presentation of transmurular myocardial ischemia of the anterior wall so that immediate invasive therapy is warranted.

Conclusions

In conclusion, we reported an unusual ECG presentation of AMI with persistent ‘hyperacute’ T waves and ST-segment

depression which did not evolve to ST-segment elevation, although coronary angiography revealed acute occlusion of the LAD artery. Cardiac CE-CMR allowed pathophysiological interpretation of such ECG findings by showing that the myocardial necrosis wave-front spared the subepicardial layer of LV musculature, which, in turn, may have preserved epicardial electrical activity and prevented the occurrence of ST-segment elevation. However, the present case indicated that the amount of myocardium saved by collateral circulation does not justify a conservative management of patients acutely presenting with chest pain, peaked and tall positive T waves, and ST-segment depression in anterior leads; instead, prompt coronary angiography and removal of LAD occlusion is warranted, because the total amount of myocardial necrosis and ensuing ventricular dysfunction may be severe and comparable to those observed in typical anterior STEMI.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflict of interest

None declared.

References

1. Sclarovsky S. Chapter 1, Angina at rest and acute myocardial ischemic syndromes. *Electrocardiography of acute myocardial ischemic syndromes*. London: Martin Dunitz Ltd., 1999.
2. Hamm CW, Bassand JP, Agewall S, et al. ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: The Task Force for the management of acute coronary syndromes (ACS) in patients presenting without persistent ST-segment elevation of the European Society of Cardiology (ESC). *Eur Heart J* 2011; 32: 2999–3054.
3. Francone M, Bucciarelli-Ducci C, Carbone I, et al. Impact of primary coronary angioplasty delay on myocardial salvage, infarct size and microvascular damage in patients with ST-segment elevation myocardial infarction. Insights from cardiovascular magnetic resonance. *J Am Coll Cardiol* 2009; 54: 2145–2153.
4. Eskola M, Nikus K and Sclarovsky S. Persistent precordial ‘hyperacute’ T waves signify proximal left anterior descending artery occlusion. *Heart* 2009; 95: 1951–1952.
5. Verouden NJ and De Winter RJ. Persistent precordial ‘hyperacute’ T waves signify proximal left anterior descending artery occlusion. The authors’ reply. *Heart* 2009; 95: 1952–1953.
6. Dressler W and Roesler H. High T waves in the earliest stage of myocardial infarction. *Am Heart J* 1947; 34: 627–645.
7. Sagie A, Sclarovsky S, Strasberg B, et al. Acute anterior wall myocardial infarction presenting with positive T waves and without ST segment shift. Electrocardiographic features and angiographic correlation. *Chest* 1989; 95: 1211–1215.
8. Stankovic I, Ilic I, Panic M, et al. The absence of the ST-segment elevation in acute coronary artery thrombosis: what does not fit, the patient or the explanation? *J Electrocardiol* 2011; 44: 7–10.
9. Zhong-qun Z, Nikus KC and Sclarovsky S. Prominent precordial T waves as a sign of acute anterior myocardial infarction: Electrocardiographic and angiographic correlations. *J Electrocardiol* 2011; 44: 533–537.
10. de Winter RJ, Verouden NJ, Wellens HJ and Wilde AA. A new ECG sign of proximal LAD occlusion. *N Engl J Med* 2008; 359: 2071–2073.
11. Verouden NJ, Koch KT, Peters RJ, et al. Persistent precordial ‘hyperacute’ T-waves signify proximal left anterior descending artery occlusion. *Heart* 2009; 95: 1701–1706.
12. Perazzolo Marra M, Lima J and Iliceto S. MRI in acute myocardial infarction. *Eur Heart J* 2010; 23: 284–293.
13. Desch S, de Waha S, Eitel I, et al. Effect of coronary collaterals on long-term prognosis in patients undergoing primary angioplasty for acute ST-elevation myocardial infarction. *Am J Cardiol* 2010; 106: 605–611.
14. Lonborg J, Kelbaek H, Vejlsstrup N, et al. Influence of pre-infarction angina, collateral flow, and pre-procedural TIMI flow on myocardial salvage index by cardiac magnetic resonance in patients with ST-segment elevation myocardial infarction. *Eur Heart J Cardiovasc Imaging* 2012; 13: 433–443.
15. Lukas A and Antzelevitch C. Differences in the electrophysiological response of canine ventricular epicardium and endocardium to ischemia. Role of the transient outward current. *Circulation* 1993; 88: 2903–2915.
16. Sclarovsky S and Nikus K. The electrocardiographic paradox of tako-tsubo cardiomyopathy-comparison with acute ischemic syndromes and consideration of molecular biology and electrophysiology to understand the electrical-mechanical mismatching. *J Electrocardiol* 2010; 43: 173–176.
17. Gorgels AP. Explanation for the electrocardiogram in subendocardial ischemia of the anterior wall of the left ventricle. *J Electrocardiol* 2009; 42: 248–249.