Early and late Q wave regression in the setting of acute myocardial infarction

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Abstract

Abnormal Q waves after a myocardial infarction are not always an indicator of myocardial necrosis. In some cases these Q waves may disappear partially or completely in the evolution of the myocardial infarction. Five cases are described in whom complete Q wave regression and reappearance of R waves in the ECG leads corresponding to the affected area were observed. Q wave regression occurred early (hours) as well as late (months) after the myocardial infarction.

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The presence of Q waves does not always imply irreversible transmural necrosis.^{1–3} Q waves may be transient and represent intense but still reversible myocardial damage, reflecting a state of stunned or even hibernating myocardium.^{4–8} Although the time course of Q wave regression in the setting of myocardial ischaemia has been reported to last from several hours to several years,^{9–11} the precise significance of Q wave regression in the course of acute myocardial infarction is still unclear. We report a series of patients with variable temporal Q wave regression in the setting of acute myocardial infarction.

Patients

Among 312 patients with myocardial infarction admitted to our hospital over a 12 month period, we retrospectively identified five patients (2%) with transient abnormal Q waves and analysed the clinical presentation and time course of Q wave regression. Clinical, electrocardiographic, and angiographic features are presented in table 1. Patients 1–4 were thrombolysed with intravenous recombinant tissue plasminogen activator (r-tPA) within 2–4 hours of the onset of symptoms. All four patients had deep Q waves on presentation at the emergency department, with ST segment elevation. Pa-

tient 1 had Q waves in the inferior region and patients 2–4 had Q waves in the antero-septal region. While peak creatine phosphokinase MB (CPK-MB) cardiac enzymes (mean value 490 U/l) were observed at a mean of eight hours in patients 1–3, in patient 4 the peak value was reached at 24 hours after thrombolysis and was approximately four times higher.

In patients 1–3 symptoms rapidly disappeared and R waves reappeared in the corresponding regions at a time interval between 8–12 hours after thrombolytic treatment was initiated. All three patients had coronary angiograms (table 1) after 5–7 days, with an uneventful stay in hospital.

Four days after thrombolysis, patient 4 still had deep Q waves on precordial leads V1 to V3 with symptoms of congestive heart failure, caused by extensive anterior akinesia related to a 95% stenotic lesion of the left anterior descending coronary artery, which could be successfully dilated. Approximately six months later the patient developed recurrent chest pain with exertion. His resting ECG revealed the reappearance of R waves from V2 to V3, and a thallium-201 scintigraphy showed reversible ischaemia in the anterior region. A second cardiac catheterisation revealed normal left ventricular contractility with a normal left ventricular ejection fraction; there was, however, restenosis of the left anterior descending coronary artery at the site of the first percutaneous transluminal coronary angioplasty (PTCA). A second PTCA was successfully performed.

Patient 5 was admitted to hospital approximately three weeks after onset of his first episode of chest pain. At admission his ECG showed deep Q waves in precordial leads V1 to V3 (fig 1A, top), corresponding to akinesia of the left ventricular antero-apico-septal segments (Fig 1A, bottom) and to a severe stenosis of the proximal portion of the left anterior descending coronary artery (fig 1B, bottom), with collateral flow from the right coronary artery towards the left anterior descending

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Table 1 Clinical, electrocardiographic, and angiographic data in five male patients

Patient	Age (years)	Initial ECG Q waves	Thrombolytic treatment	Cardiac enzymes CPK-MB	Follow up ECG Q wave regression	Coronary angiogram
1	48	Q waves and ST elevation in leads II, III aVF	iv r-tPA 2.25 h after onset of symptoms	316 U/l MB 14% at 6 h	After 8–10 h	Normal LVEF, 95% stenosis RCD, successful PTCA
2	35	Q waves in leads V2, V3, and ST elevation in leads V2 to V6	iv r-tPA 4 h after onset of symptoms	538 U/l MB 21% at 8 h	After 8–10 h	LVEF 54%, AS hypokinesia, 95% stenosis LAD, successful PTCA
3	51	Q waves and ST elevation in leads V1 to V5	iv r-tPA 2.5 h after onset of symptoms	616 U/l MB 11% at 10 h	After 10–12 h	Normal LVEF, 90% stenosis LAD, successful PTCA
4	64	ST elevation in leads V2 to V6	iv r-tPA 2 h after onset of symptoms	1954 U/l MB 15% at 24 h	After several weeks	LVEF 47%, AS akinesia, 95% stenosis LAD, successful PTCA
5	57	Q waves in leads V1 to V3			After several weeks	LVEF 52%, AS hypokinesia, 95% stenosis LAD, successful PTCA

LVEF, left ventricular ejection fraction; AS, antero-septal; RCD, right coronary artery; LAD, left anterior descending coronary artery; PTCA, percutaneous transluminal coronary angioplasty.

Figure 1 Top. Initial ECG of patient 5 showing pathologic Q waves in leads V1 to V3 (A) and ECG recorded seven months later with normal R waves in leads V1 to V3 (B). Bottom. Initial left ventricular angiogram of the same patient revealing dyskinesia of the antero-apico-septal segments (A) with severe stenosis of the proximal portion of the left anterior descending coronary artery (LAD) (B), and after successful PTCA (C); left ventricular angiogram seven months later showing normal regional function (D), but restenosis of the LAD artery (E); repeat PTCA was successful (F).

coronary artery. PTCA was successfully performed (Fig 1C). Approximately seven months later the patient again developed chest pain with exertion. Normal R waves of 4 mm in V2 and 7 mm in V3 were then visible on his standard ECG (fig 1B, top). The stress test revealed anteroapical ischaemia. At repeat catheterisation, the anterior akinesia was no longer present (fig 1D); however there was a tight restenosis of the left anterior descending coronary artery (fig 1E). Again, repeat PTCA was successfully performed (fig 1F).

Discussion

Our observations show that systemic thrombolysis resulted in near complete myocardial salvage after onset of symptoms in patients 1–3, directly reflected in the early electrocardiographic reappearance of R waves (table 1). In patient 4, Q waves were still present four days after thrombolysis. PTCA restored coronary flow, and was associated with subsequent Q wave regression and normalisation of left ventricular function. In patient 5, a situation of hypoperfusion over several weeks with subsequent recovery after coronary flow

restoration and late Q wave regression was observed.

While two extreme situations are illustrated with patients 1–3 and 5, patient 4's situation may be considered to be intermediate. Thus, while the first three patients showed Q wave regression during the first hours of an acute myocardial infarction, and the fifth patient represented a situation suggestive of hibernating myocardium with Q waves reflecting "electrical hibernation",^{4,6,8} wall motion abnormalities and Q waves were still present in patient 4 after systemic thrombolysis, compatible with mechanical and electrical stunning.^{3,5,7} However, as coronary blood flow was not fully restored, a state of myocardial hibernation was potentially developing in this patient, which could be reversed by PTCA. Thus, patient 4 provides an example of a situation in which the concepts of stunned and hibernating myocardium appear to have coexisted.^{6,7}

In previous reports the percentage of patients experiencing Q wave regression in the setting of myocardial ischaemia has ranged from 6.7–42%, corresponding to a time interval of one month to several years.^{9–11} Several case reports published in recent years^{2,3,12,13} have generally shown Q wave disappearance in the course of the early phase of an acute myocardial infarction or during unstable angina. The relation between the time interval of Q wave regression, the size of the infarct, and the prognostic implication of this Q wave regression remains unclear. While an association with smaller infarct sizes^{9,10,14} and a lack of correlation with the patency of the infarct related coronary artery or with improvement of left ventricular function have been reported,¹⁵ others have suggested that the presence of transient Q waves may identify patients with a potentially greater degree of myocardium at risk.^{16,17} Also, although some studies^{10,11,16} have pointed out that Q wave regression may not influence survival or rate of reinfarction, there is still disagreement about the prognostic implication of Q wave disappearance.

Although our series was small, our observations showed: (1) Q wave regression could occur both early (patients 1–3) and late (patients 4 and 5); (2) Q wave regression was mainly related to myocardial reperfusion, correlating to patency of the infarct related artery and changes in left ventricular function; (3) Q wave disappearance was not exclusively associated with a smaller infarct size, as illustrated in patient 4; and (4) Q wave regression was associated with a good prognosis.

This study of five cases draws attention to the need for prudent evaluation of patients with Q waves in the setting of myocardial ischaemia, because adequate treatment may still lead to notable recuperation of left ventricular function.

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IMAGES IN CARDIOLOGY

Metastatic renal cell cancer detected by transoesophageal echocardiography

A 63 year old man was referred to the outpatient cardiology laboratory for dobutamine echocardiography because of atypical chest pain. The transthoracic echocardiography had poor imaging quality but showed turbulent colour flow above the right atrium in the four chamber view. To define this further transoesophageal echocardiography (TEE) was performed (top). Where the inferior vena cava is normally seen, a large echodense mass (T) was seen protruding into the right atrium (RA). Colour flow of the same frame (bottom) shows turbulent flow (TL) of the inferior vena cava caused by a tumour obstructing the inferior vena cava–right atrium junction. The interatrial septum (IAS) is seen bulging towards the left atrium owing to high pressure in the right atrium cause by the obstructing mass. (LA, left atrium; SVC, superior vena cava.)

Further investigations with computed tomography and magnetic resonance imaging revealed a large renal mass invading the inferior vena cava and extending in to the right atrium. Biopsy results were consistent with clear cell carcinoma of the kidney. The patient opted for palliative therapy.

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