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Abnormal Q Waves on the Admission Electrocardiogram of Patients with First Acute Myocardial Infarction: Prognostic Implications

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Summary

Background: Q waves developed in the subacute and persisting into the chronic phase of myocardial infarction (MI) usually signify myocardial necrosis. However, the mechanism and significance of Q waves that appear very early in the course of acute MI (<6 h from onset of symptoms), especially if accompanied by ST elevation, are probably different.

Hypothesis: This study assesses the prognostic implications of abnormal Q waves on admission in 2,370 patients with first acute MI treated with thrombolytic therapy <6 h of onset of symptoms.

Results: Patients with abnormal Q waves in ≥2 leads with ST-segment elevation (n = 923) were older than patients without early Q waves (n = 1,447) (60.6 ± 11.9 vs. 58.8 ± 11.9 years, respectively; p = 0.0003), and had a greater incidence of hypertension (34.3 vs. 30.5%; p = 0.05) and anterior MI (60.6 vs. 41.1%; p<0.0001). Time from onset of symptoms to therapy was longer in patients with Q waves upon admission (208 ± 196 vs. 183 ± 230 min; p = 0.01). Peak serum creatine kinase (2235 ± 1544 vs. 1622 ± 1536 IU; p<0.0001), prevalence of heart failure during hospitalization (13.8 vs. 7.0%, p<0.0002), hospital mortality (8.0 vs. 4.6%; p = 0.02), and cardiac mortality (6.6 vs. 4.5%, p = 0.11) were higher in patients with anterior MI and with abnormal Q waves than in those without abnormal Q waves upon admission. There was no difference in peak creatine kinase, prevalence of heart failure, in-hospital mortality, and cardiac mortality between patients with and without abnormal Q waves in inferior MI. Multivariate regression analysis confirmed that mortality is independently associated with presence of Q waves on admission (odds ratio 1.61; 95% CI 1.04–2.49; p = 0.04 for all patients; odds ratio 1.65; 95% CI 0.97–2.83; p = 0.09 for anterior wall MI.

Conclusion: Abnormal Q waves on the admission electrocardiogram (ECG) are associated with higher peak creatine kinase, higher prevalence of heart failure, and increased mortality in patients with anterior MI. Abnormal Q waves on the admission ECG of patients with inferior MI are not associated with adverse prognosis.

Key words: acute myocardial infarction, electrocardiogram, mortality, prognosis, Q waves, thrombolytic therapy

Introduction

Q waves developed in the subacute and persisting into the chronic phase of myocardial infarction (MI) usually signify myocardial necrosis. However, the mechanism and significance of Q waves that appear very early in the course of acute MI (≤6 h from onset of symptoms), especially if accompanied by ST elevation, are probably different.[1–7] Of the patients admitted within 1 h of onset of symptoms, 53% had abnormal Q waves on admission even before reperfusion therapy had been initiated.[1] Some have reported that early pathologic Q waves develop especially after reperfusion;[3–5,8] however, others have found these to be associated with a larger ischemic zone and ultimate necrotic area.[1,2,9] It has been reported that early Q waves (<6 h) do not signify irreversible damage, nor do they preclude myocardial salvage by thrombolytic therapy.[2,10] Moreover, transient Q waves have been observed in patients and produced experimentally in animals during ischemic episodes.[7,10–12] The prognostic importance of abnormal Q waves already present on admission in patients with acute MI undergoing thrombolytic therapy has not yet been reported. This study assesses the correlation between hospital mortality and presence of abnormal Q waves in the

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leads with ST elevation in the admission electrocardiogram (ECG) of patients with first acute MI treated with intravenous thrombolytic therapy.

Materials and Methods

Patients

The study included patients enrolled in 25 medical centers in Israel from September 1991 through February 1993. All patients were admitted within 6 h of onset of symptoms, with chest pain lasting at least 20 min and ≥0.1 mV of ST segment elevation in two or more limb leads or ≥0.2 mV in two or more consecutive precordial leads. Only patients without previous MI (determined by medical history and absence of abnormal Q waves in the leads without ST-segment elevation) were included. Patients with intraventricular conduction defect, ventricular rhythm, or ECG signs of left ventricular hypertrophy were excluded. Patients received either streptokinase, front loaded tissue plasminogen activator (TPA), or a combination of streptokinase and TPA according to the GUSTO-I protocol.[13] All patients received oral aspirin 160–325 mg/day and either intravenous or subcutaneous heparin for at least 48 h.[13]

Baseline Characteristics and Clinical End Points

Clinical data as to gender, age, history of diabetes mellitus, hypertension, previous angina pectoris, smoking status, Killip class on admission, type of thrombolytic therapy, time from onset of symptoms to thrombolysis, were recorded. End points were peak serum creatine kinase, severe heart failure (worst Killip score ≥ 2), and hospital mortality.

Electrocardiographic Evaluation

All admission ECGs were analyzed by three investigators who were blinded to the clinical data and hospital course. The site of infarction was determined as follows: ST-segment elevation in V_1–V_4: anterior wall; ST-segment elevation in leads II, III, and aVF: inferior wall; ST-segment elevation in either leads I and aVL or leads V_5 and V_6 without ST elevation in either inferior or anterior leads: lateral wall; and ST-segment elevation in inferior and anterior leads: anterior and inferior. Patients were classified into two groups based on the presence or absence of abnormal Q waves in two or more leads with ST elevation (Fig. 1). Abnormal Q waves were defined by the criteria developed by Selvester *et al.*:[1,14] leads I, II, III, aVL, aVF, V_5, and V_6—≥30 ms; lead V_4—≥20 ms; and leads V_1–V_3—any Q wave. We cannot exclude that in some patients abnormal Q waves preexisted in the leads with ST-segment elevation on the admission ECG.

Statistical Analysis

Mean ± standard deviation (SD) were calculated for continuous variables (i.e., age, time from onset of symptoms, and serum creatine kinase levels), and absolute and relative frequencies were measured for discrete variables. In the case of continuous variables, differences between groups were analyzed for statistical significance by the Student's *t*-test. The chi-square test was applied to compare differences between discrete variables. A logistic regression model was fitted for all patients and for anterior and inferior MI separately. The dependent variable was hospital mortality, and the independent variables were gender, age, history of diabetes mellitus, hypertension, angina pectoris, smoking status, time from onset of symptoms to therapy, Killip score on admission, number of leads with ST-segment elevation, and presence of Q waves on admission. Odd ratios and 95% confidence intervals (CI) were calculated from the estimate parameters of the logistic regression model. All tests of significance were two-tailed. P values of < 0.05 were considered statistically significant.

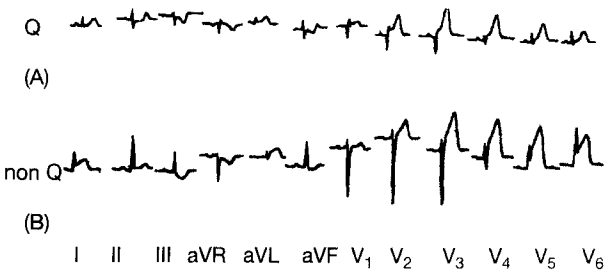

FIG. 1 Admission electrocardiograms of patients with first anterior wall acute myocardial infarction who were admitted within 6 h of onset of symptoms: (A) with pathologic Q waves in leads V_1–V_3; (B) without pathologic Q waves.

Results

Baseline Characteristics

The study population included 2,370 patients of whom 923 (39%) had abnormal Q waves on admission and 1,447 (61%) did not (Fig. 1) (Table I). Patients with abnormal Q waves on admission were older and had a greater incidence of hypertension than patients without Q waves. No difference was found in the male-to-female ratio and in the prevalence of angina, diabetes mellitus, and smoking between the groups. More patients with than without abnormal Q waves on admission had anterior wall MI. Prevalence of heart failure on admission (Killip score >1) was higher in patients with than without Q waves on admission. Time interval from onset of symptoms to therapy was shorter in patients without abnormal Q waves. There was no difference between the groups in the allocation to the various treatment regimens.

Among patients with first anterior wall infarction, there was no difference in mean age, male-to-female ratio, history of previous angina, hypertension, and diabetes mellitus between the groups (Table I). More patients in the group with abnormal Q waves were current smokers. Prevalence of heart

TABLE I Baseline patient characteristics

	All patients			Anterior myocardial infarction			Inferior myocardial infarction		
	Q− n = 1447 n (%)	Q+ n = 923 n (%)	p Value	Q− n = 561 n (%)	Q+ n = 550 n (%)	p Value	Q− n = 805 n (%)	Q+ n = 358 n (%)	p Value
Age (years)									
Mean ± SD	58.8 ± 11.9	60.6 ± 11.9	0.0003	60.0 ± 11.5	60.5 ± 12.3	NS	58.3 ± 12.0	60.6 ± 11.6	0.002
Male	1121 (77.5)	718 (77.8)	NS	452 (80.7)	424 (77.1)	NS	606 (75.3)	283 (79.1)	NS
Previous angina	403 (27.9)	287 (31.1)	NS	173 (30.8)	169 (30.7)	NS	213 (26.5)	116 (32.4)	0.04
Hypertension	442 (30.5)	317 (34.3)	0.05	178 (31.7)	174 (31.6)	NS	238 (29.6)	133 (37.2)	0.01
Diabetes mellitus	254 (17.6)	161 (17.4)	NS	98 (17.5)	96 (17.5)	NS	145 (18.0)	60 (16.8)	NS
Smoking	652 (45.1)	389 (42.1)	NS	221 (39.4)	248 (45.1)	0.05	395 (49.1)	136 (38.0)	0.0005
Killip on admission >1	202 (14.0)	176 (19.1)	0.0009	99 (17.6)	124 (22.5)	0.04	92 (11.4)	47 (13.1)	NS
Time to therapy (min)	183 ± 230	208 ± 196	0.01	185 ± 336	208 ± 226	NS	182 ± 124	207 ± 143	0.003

Abbreviations: n = number, SD = standard deviation, NS = not significant.

failure on admission (Killip score > 1) was higher in patients with than without Q waves on admission. Time interval from onset of symptoms to therapy tended to be shorter in patients without abnormal Q waves; this, however, had no statistical significance. Among patients with inferior wall MI, those without abnormal Q waves on admission were younger, had less history of previous angina and hypertension, and were more often current smokers. There was no difference between the group with and without pathologic Q waves in prevalence of heart failure (Killip > 1) on admission. Time from onset of symptoms to therapy was longer among patients with abnormal Q waves on admission.

Table II shows the outcome of all patients and identifies those with anterior and inferior wall MI. Among patients with anterior wall MI, peak serum creatine kinase levels were higher in the group with compared with the group without abnormal Q waves on admission. Severe heart failure (Killip score > 1) was more frequent and total hospital mortality was higher in patients with anterior wall MI and with abnormal Q waves on presentation. Cardiac mortality was also higher in these patients, but this did not reach statistical significance. There was no statistically significant difference in peak creatine kinase, prevalence of heart failure, total in-hospital mortality, and cardiac mortality between the group with and that without abnormal Q waves in inferior wall MI.

Pathologic Q waves on the predischarge ECG were found in almost all 890 patients (98.9%) with Q waves on admission compared with only 66.4% of the 1,412 patients without Q wave on admission, respectively ($p < 0.0001$). Predischarge ECGs were not available for 68 patients.

Parameters Affecting Total Mortality-Logistic Regression (Table III)

Multivariate analysis of all patients, taking into account gender, age, history of angina pectoris, hypertension, diabetes mellitus, smoking status, Killip class on admission, and time from onset of symptoms to therapy, demonstrated that hospital mortality was independently associated with female gender, diabetes mellitus, smoking status, age >60 years, Killip score on admission > 1, anterior wall infarction, and presence of abnormal Q waves on admission. The odds ratio for abnormal Q waves on admission was 1.61 (1.04–2.49; p = 0.04) (Table III). Multivariate regression analysis that included only

TABLE II Clinical outcome of the patients

	All patients			Anterior myocardial infarction			Inferior myocardial infarction		
	Q− n = 1447 n (%)	Q+ n = 923 n (%)	p Value	Q− n = 561 n (%)	Q+ n = 550 n (%)	p Value	Q− n = 805 n (%)	Q+ n = 358 n (%)	p Value
Peak creatine kinase (IU)									
Mean ± SD	1428 ± 1296	1920 ± 1573	<0.0001	1622 ± 1536	2235 ± 1544	<0.0001	1070 ± 1255	1181 ± 1571	NS
Worst Killip class >1	73 (5.0)	101 (11.0)	<0.0001	39 (7.0)	76 (13.8)	<0.0002	142 (17.7)	72 (20.1)	NS
Hospital mortality	46 (3.2)	60 (6.5)	0.0001	26 (4.6)	44 (8.0)	0.02	17 (2.1)	13 (3.6)	NS
Cardiac mortality	44 (3.0)	50 (5.5)	0.003	25 (4.5)	36 (6.6)	0.11	16 (2.0)	11 (3.1)	NS

Abbreviations as in Table I.

TABLE III Baseline characteristics independently associated with hospital mortality- logistic regression model

	Odds ratio	95% Confidence interval	p Value
All patients			
Gender (females vs. males)	2.04	1.31–3.19	0.0027
Diabetes mellitus (yes vs. no)	1.64	0.99–2.68	0.06
Current smoking (no vs. yes)	1.92	1.03–3.58	0.05
Age (years)			
(60–69 vs. <60)	3.12	1.36–7.16	<0.0001
(≥70 vs. <60)	10.1	4.56–22.3	—
Killip on admission (>1 vs. 1)	2.48	1.58–3.88	0.0001
Anterior vs. inferior infarction	2.21	1.38–3.53	0.002
Abnormal Q waves on admission	1.61	1.04–2.49	0.04
Anterior myocardial infarction			
Gender (females vs. males)	2.28	1.33–3.89	0.005
Current smoking (no vs. yes)	2.69	1.20–6.01	0.03
Age (years)			
(60–69 vs. <60)	2.55	0.96–6.78	<0.0001
(≥70 vs. <60)	8.11	3.23–20.4	—
Killip on admission (>1 vs. 1)	2.66	1.56–4.55	0.0008
Abnormal Q waves on admission	1.65	0.97–2.83	0.09
Inferior myocardial infarction			
Age (years):			
(60–69 vs. <60)	6.23	1.31–29.7	<0.0001
(≥70 vs. <60)	22.0	5.04–96.3	—
Diabetes mellitus (yes vs. no)	2.36	1.05–5.30	0.04
Killip on admission (>1 vs. 1)	2.46	1.06–5.70	0.035

the 1,097 patients with first anterior wall MI showed a trend for an independent association between hospital mortality and presence of Q waves on the admission ECG. The odds ratio was 1.65 (95% CI 0.97–2.83; p = 0.09). However, presence of abnormal Q waves upon admission was not associated with adverse prognosis in patients with inferior wall MI.

Discussion

This retrospective analysis assessed the relationship between hospital outcome and presence of abnormal Q waves on admission in patients with acute MI treated with thrombolytic therapy. Abnormal Q waves on the admission ECG of patients without previous history of MI, admitted within 6 h of onset of symptoms, were relatively common (39%), as reported earlier,[1] and were more common in patients with anterior infarction (Table I). The average time from onset of symptoms to admission was longer in patients with abnormal Q waves upon admission, especially in those with inferior wall MI.

Abnormal Q waves on admission were associated with larger peak creatine kinase levels, higher prevalence of severe heart failure, and higher hospital mortality in patients with anterior but not inferior wall MI. Multivariate regression analysis confirmed the independent association of pathologic Q waves on admission with increased hospital mortality only in patients with anterior wall MI.

Previous studies showed that abnormal Q waves that appear within 6 h of onset of symptoms are associated with larger predicted and final infarct size.[1,2,9] However, in contrast to pathologic Q waves that appear later, early Q waves do not signify irreversible damage and do not preclude potential for myocardial salvage by thrombolytic therapy.[1,2,7,10–12] Such Q waves have been explained by a transient loss of electrophysiologic function due to intense ischemia ("myocardial concussion"[7,10,15]). In contrast, some investigators found that Q waves develop rapidly only after reperfusion,[3–5,8] that their presence may be masked by the injury current during ischemia,[5] and that they can be seen only after resolution of the injury current. However, these changes may reflect reperfusion injury, interstitial edema, or hemorrhage that may partially resolve later,[3] and may even predict enlargement of the final infarct size. Chuang and Spodick reported that in a third of 167 patients after suffering an inferior wall MI with Q waves in lead aVF, there were daily fluctuations in the presence of Q waves in the inferior leads during the chronic phase.[16] This was probably due to the breathing phase in which the ECG was recorded. Hence, respiratory variation may explain some of the fluctuations in Q waves, at least in the chronic phase of inferior wall MI. However, this does not explain the profound difference in prognosis between patients with and without abnormal Q waves on admission.

In the present study we included only patients without a previous history of MI. In the group with abnormal Q waves

we included only patients who had Q waves in the leads with ST-segment elevation but not those with abnormal Q waves in leads without ST-segment elevation. We cannot exclude the possibility that some of the patients had pre-existing Q waves in the territory of the acute MI. Previous silent MI in the same territory of the index infarction may explain the presence of Q waves in some of the patients. Furthermore, several medical conditions, such as dilated or hypertrophic cardiomyopathy, left ventricular hypertrophy, myocarditis, cardiac amyloidosis, chronic obstructive lung disease, various metabolic disturbances, and pancreatitis may also be associated with abnormal Q waves.[7, 9] All these conditions may be associated with increased mortality after acute MI.

Conclusion

Regardless of the underlying mechanism, early appearance of abnormal Q waves within 6 h of onset of symptoms of acute anterior but not inferior myocardial infarction is associated with larger infarct size and increased hospital mortality. It has yet to be determined whether other modalities of reperfusion such as primary angioplasty may improve outcome in patients with abnormal Q waves on admission compared with intravenous thrombolytic therapy. Presence of abnormal Q waves on admission should be considered in risk stratification of patients with acute anterior wall myocardial infarction.

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