Non–Q-Wave Versus Q-Wave Myocardial Infarction After Thrombolytic Therapy

Angiographic and Prognostic Insights From the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries–I Angiographic Substudy

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Background—Although the stratification of patients with myocardial infarction into ECG subsets based on the presence or absence of new Q waves has important clinical and prognostic utility, systematic evaluation of the impact of thrombolytic therapy on the subsequent development and prognosis of non–Q-wave infarction has been limited to date.

Methods and Results—We examined 12-lead ECG, coronary anatomy, left ventricular function, and mortality among 2046 patients with ST-segment elevation infarction from the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries angiographic subset to gain further insight into the pathophysiology and prognosis of Q- versus non–Q-wave infarction in the thrombolytic era. Non–Q-wave infarction developed in 409 patients (20%) after thrombolytic therapy. Compared with Q-wave patients, non–Q-wave patients were more likely to present with lesser ST-segment elevation in a nonanterior location. The infarct-related artery in non–Q-wave patients was more likely to be nonanterior (67% versus 58%, \( P = .012 \)) and distally located (33% versus 39%, \( P = .021 \)). Early (90-minute, 77% versus 65%, \( P = .001 \)) and complete (54% versus 44%, \( P < .001 \)) infarct-related artery patency was greater among the non–Q-wave group. Non–Q-wave patients had better global (ejection fraction, 66% versus 57%; \( P < .0001 \)) and regional left ventricular function (10 versus 24 abnormal chords, \( P = .0001 \)). In-hospital, 30-day, 1-year, and 2-year (6.3% versus 10.1%, \( P = .02 \)) mortality rates were lower among non–Q-wave patients.

Conclusions—The excellent prognosis among the subgroup of patients who develop non–Q-wave infarction after thrombolysis is related to early, complete, and sustained infarct-related artery patency with resultant limitation of left ventricular infarction and dysfunction. (Circulation. 1998;97:444-450.)

Key Words: infarction ■ electrocardiography ■ thrombolysis ■ catheterization ■ prognosis

The stratification of patients with acute MI into ECG subsets by abnormal new Q waves has important clinical and prognostic use. Studies in the prethrombolytic era have shown lower in-hospital mortality among patients with non–Q-wave MI. Despite a better initial prognosis, non–Q-wave MI patients had more frequent infarct extension and reinfarction, resulting in a similar or worse long-term prognosis compared with those with Q-wave MI.1

Autopsy and coronary angiographic studies in the prethrombolytic era suggest that non–Q-wave MI is associated with less frequent coronary occlusion and a higher incidence of collaterals to the infarct-related artery.2–8 However, pathophysiological insights gained from these angiographic studies are limited by small patient numbers, selection bias,4,8 and delayed timing of evaluation.4–7

Although the incidence of non–Q MI appears to be increasing,9,10 systematic evaluation of the impact of thrombolytic therapy on the subsequent development of non–Q-wave MI has been limited.11 Furthermore, the prognosis of non–Q-wave MI patients after thrombolysis remains uncertain, with the majority of studies not reporting on clinical outcome. Controversy exists among the few trials stratifying patients by...
predischARGE ECG subsets, with some suggesting similar outcome,
and others describing significantly lower early and late mortality among non-Q- versus Q-wave MI patients.

Accordingly, we examined coronary anatomy, left ventricular function, and mortality among the GUSTO-I angiographic subset to gain further insight into the pathophysiology and prognosis of these two ECG categories of MI.

Methods
Randomization and Treatment Strategies
The entry criteria for enrollment into GUSTO-I and the GUSTO-I angiographic substudy have been described in detail. Briefly, patients with chest pain for <6 hours and ST-segment elevation compatible with acute MI were randomized to one of four treatment strategies for reperfusion: (1) streptokinase with subcutaneous heparin, (2) streptokinase with intravenous heparin, (3) alteplase given in an accelerated manner with intravenous heparin, or (4) streptokinase and alteplase with intravenous heparin. All patients received aspirin and, for those without a contraindication, intravenous followed by oral atenolol therapy. All other medications and the use of coronary revascularization were left to the discretion of the investigator.

Patient Population
Patients in the GUSTO-I angiographic substudy were randomly assigned to cardiac angiography at one of four times after the initiation of thrombolysis: 90 minutes, 180 minutes, 24 hours, or 5 to 7 days. The group undergoing angiography at 90 minutes underwent repeat study after 5 to 7 days, allowing for analysis of reocclusion and ventricular function at uniform times after therapy.

The GUSTO-I angiographic substudy consisted of 2431 patients from 75 North American, European, and Australian hospitals. Patients later excluded from the ECG analysis included those (1) without a baseline or follow-up ECG (n=135), (2) whose follow-up ECG was obtained <24 hours after thrombolytic administration (n=138), (3) who experienced reinfarction before a follow-up ECG was obtained (n=75), and (4) with confounding ECG factors that did not allow for an assessment of Q-wave MI (example, left bundle-branch block, paced rhythm, poor-quality ECG) (n=37). The remaining 2046 patients were included in this posthoc analysis (Fig 1).

Angiography
The protocol and core laboratory procedures of the angiographic substudy have been described in detail. Briefly, the infarct-related artery was identified through assessment of the initial ECG, ventriculographic location of contractile abnormality, and presence of stenosis or thrombus in the corresponding artery. Flow in the infarct-related artery was determined during the initial injection of contrast agent and graded as described in the TIMI trial. Coronary collateral score was grade 0, no angiographic filling of the infarct vessel distal to occlusion; grade 1, faint opacification or small fragments of the distal vessel visualized; grade 2, visualization of more than half of the estimated length of the distal vessel, although less opacified than a normal vessel of equal caliber; or grade 3, entire distal vessel wall visualized and densely opacified. Ventricular volumes and ejection fraction were calculated by the area-length method. Global left ventricular function was assessed by left ventricular ejection fraction and end-systolic volume index. Regional function was measured according to the method of Sheehan and Dodge and expressed as the number of abnormal chords in the infarct zone (defined by >2 SDs below the norm) and by the mean magnitude of depressed infarct-zone chords. Preserved wall motion was defined by all infarct-zone chords being normal. Patients who underwent angioplasty before the follow-up study at 5 to 7 days were excluded from the analysis of reocclusion.

Multivessel disease was defined as >75% stenosis in at least two vessels. Infarct related artery patency was defined as (1) open vessels (TIMI flow grades 2 and 3 combined) and (2) complete reperfusion (TIMI grade 3 flow). Infarct-related artery reocclusion was defined as TIMI grade 0 or 1 flow at follow-up in patients who had grade 2 or 3 flow at 90 minutes.

ECG Classification
A Q-wave MI was based on a follow-up ECG performed ≥24 hours after the initiation of thrombolyis. The determination of Q- and non–Q-wave ECG patterns was made by experienced readers without knowledge of the angiographic findings at the ECG Core Laboratory (Duke University); they used the Selvester QRS screening criteria for Q-wave (or Q-wave–equivalent) MI: (1) Q wave of ≥30 ms in aVF (inferior); (2) Q wave of ≥40 ms in I and aVL (lateral); (3) Q wave of ≥40 ms in ≥two of V4 through V6 (apical); (4) R wave of ≥40 ms in V1 (posterior); (5) any Q wave in V2 (anterior); and (5) R wave ≤0.1 mV and 10 ms in lead V2 (anterior).

Patients with evidence of prior MI on the baseline ECG were included because ST-segment elevation and careful comparison of the

### Selected Abbreviations and Acronyms

- GUSTO = Global Utilization of Streptokinase and Tissue plasminogen activator for Occluded coronary arteries (trial)
- MI = Myocardial infarction
- TIMI = Thrombolysis in Myocardial Infarction (trial)

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**Figure 1.** Disposition of patients; overall TIMI grade 3 infarct-related artery flow rates; and 30-day, 1-year, and 2-year mortality rates.
new MI location on the follow-up ECG allowed localization of the index infarct.

Clinical Follow-up
In-hospital recurrent ischemia, congestive heart failure, nonfatal stroke, cardiogenic shock, nonfatal reinfarction, revascularization, and mortality were documented in all patients. In addition, 30-day, 1-year, and 2-year mortality rates were recorded, with complete follow-up available in 95.4% of the patients.

Statistical Analysis
Descriptive statistics (percentages for discrete variables; medians with 25th and 75th percentiles for continuous variables) were generated for baseline characteristics and for ECG, angiographic, and clinical outcomes. Comparison of baseline characteristics and clinical outcomes between patient groups was carried out using likelihood-ratio \( \chi^2 \) or Fisher’s exact tests for differences in proportions of categorical variables, and Wilcoxon rank sum tests was used for differences in median values of continuous variables. Kaplan-Meier estimates were used to obtain survival rates at 1 and 2 years, and curve comparisons were made using the log-rank test. A modified, backward elimination logistic regression model was used for prediction of 2-year mortality. The presence of non–Q-wave MI was added to this model, which contains the variables found to be predictive of 30-day mortality in the overall GUSTO-I trial: age, height, weight, systolic blood pressure, overall GUSTO-I trial: age, height, weight, systolic blood pressure, and atrial fibrillation (Table 1). Non-Q-wave MI patients were more likely to be female and to have a history of smoking. Presence of non–Q-wave MI was added to this model, which contains the variables found to be predictive of 30-day mortality in the overall GUSTO-I trial: age, height, weight, systolic blood pressure, heart rate, infarct location, previous MI, time to treatment, diabetes, smoking status (current and former), thrombolytic strategy, previous bypass surgery, hypertension, and previous cerebrovascular disease. Final data analysis was performed at Duke University; 2-year survival analysis was performed at the George Washington University Medical Center.

Results
Characteristics of Post-thrombolytic Non–Q-Versus Q-Wave MI Groups
Of the 2046 patients in this analysis, 409 (20%) developed a non–Q-wave MI. Follow-up ECGs were acquired 6.9 (4.2, 10.0) and 7.4 (4.3, 10.8) days after thrombolysis in the non–Q- and Q-wave groups, respectively (\( P=.12 \)). Comparison of the baseline characteristics of patients in the non–Q- and Q-wave groups revealed similar age, sex, and coronary artery disease risk factor profiles (Table 1). Non-Q-wave MI patients were more likely to be female and to have a history of smoking. Presentation characteristics revealed similar pretreatment heart rate, blood pressure, Killip class, and time from chest pain onset to thrombolytic administration. Maximal CK and CK-MB values were significantly lower among the non–Q- versus the Q-wave group: median of 742 versus 1853 IU (\( P=.0001 \)) and 79 versus 159 IU (\( P=.0001 \)), respectively. The 20% rate of non–Q-wave MI development did not differ by thrombolytic regimen received.

Qualifying ECG Findings
ECG indicators of infarct severity were fewer and of less magnitude in non–Q- compared with Q-wave MI patients: the median number of leads with ST-segment elevation of \( \geq 0.1 \text{ mV} \) was 3 (3, 5) versus 4 (3, 6) leads (\( P=.0001 \)); and the median of the maximal ST-segment elevation in any lead was 0.2 (0.1, 0.3) versus 0.3 (0.2, 0.4) mV (\( P=.0001 \)). Non–Q-wave MI patients were more likely to present with ST-segment elevation in an inferior (63% versus 56%) or other (4% versus 2%) location compared with more frequent anterior ST-elevation in the Q-wave group (34% versus 42%; overall \( P=.012 \)).

### TABLE 1. Patient Characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Non–Q-Wave MI (n=409)</th>
<th>Q-Wave MI (n=1637)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>61 (53, 70)</td>
<td>61 (51, 69)</td>
</tr>
<tr>
<td>Sex, % male</td>
<td>73</td>
<td>80</td>
</tr>
<tr>
<td>History, %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>71</td>
<td>65</td>
</tr>
<tr>
<td>Hypertension</td>
<td>35</td>
<td>35</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>14</td>
<td>12</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>32</td>
<td>34</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>1.5</td>
<td>1.5</td>
</tr>
<tr>
<td>Angina</td>
<td>33</td>
<td>34</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>11</td>
<td>13</td>
</tr>
<tr>
<td>Coronary angioplasty</td>
<td>4.7</td>
<td>4.2</td>
</tr>
<tr>
<td>Coronary artery bypass graft surgery</td>
<td>3.2</td>
<td>4.8</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>72 (62, 85)</td>
<td>73 (62, 85)</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>126 (110, 142)</td>
<td>129 (112, 141)</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>78 (68, 90)</td>
<td>80 (70, 90)</td>
</tr>
<tr>
<td>Killip class, %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>88</td>
<td>87</td>
</tr>
<tr>
<td>II</td>
<td>10</td>
<td>11</td>
</tr>
<tr>
<td>III</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>IV</td>
<td>&lt;1</td>
<td>&lt;1</td>
</tr>
<tr>
<td>Time to treatment from chest pain onset, h</td>
<td>2.8 (2.0, 4.0)</td>
<td>2.8 (2.0, 3.8)</td>
</tr>
<tr>
<td>Maximal CK, IU</td>
<td>742 (315, 1374)</td>
<td>1853 (955, 3094)</td>
</tr>
<tr>
<td>Maximal CK-MB, IU</td>
<td>79 (42, 149)</td>
<td>159 (87, 286)</td>
</tr>
</tbody>
</table>

*Continuous variables are expressed as median value (25th and 75th percentiles).*

Cardiac Catheterization Findings
Patients with non–Q-wave MI were more likely to have an angiographic infarct-related lesion in the right coronary or left circumflex artery than were Q-wave MI patients (Table 2). Non–Q-wave MI patients were less likely to have a proximal site of occlusion (left main, proximal left anterior descending, circumflex, or right coronary artery) than were Q-wave MI

### TABLE 2. Coronary Angiographic Findings

<table>
<thead>
<tr>
<th>Coronary Angiographic Findings</th>
<th>Non–Q-Wave MI (n=409)</th>
<th>Q-Wave MI (n=1637)</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infarct-related artery, %</td>
<td>34</td>
<td>40</td>
<td>.029</td>
</tr>
<tr>
<td>Left anterior descending</td>
<td>49</td>
<td>45</td>
<td></td>
</tr>
<tr>
<td>Right</td>
<td>16</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Left circumflex</td>
<td>0</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>Left main</td>
<td>1</td>
<td>3.4</td>
<td></td>
</tr>
<tr>
<td>Bypass graft</td>
<td>33</td>
<td>39</td>
<td>.021</td>
</tr>
<tr>
<td>Proximal location within the infarct-related artery, %*</td>
<td>34</td>
<td>39</td>
<td>.103</td>
</tr>
<tr>
<td>Multivessel disease, %</td>
<td>12</td>
<td>14</td>
<td>.35</td>
</tr>
<tr>
<td>Collaterals, %</td>
<td>5.5</td>
<td>5.1</td>
<td>.74</td>
</tr>
<tr>
<td>Any</td>
<td>Grade 2 or 3</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Patients with a bypass graft as an infarct-related artery were not included in this analysis.*
TABLE 3. Coronary and Left Ventricular Angiographic Findings

<table>
<thead>
<tr>
<th>Angiographic Findings</th>
<th>Non–Q-wave MI (n=409)</th>
<th>Q-wave MI (n=1637)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patency, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TIMI grade 2</td>
<td>27</td>
<td>30</td>
<td>.29</td>
</tr>
<tr>
<td>TIMI grade 3</td>
<td>54</td>
<td>44</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Overall</td>
<td>81</td>
<td>74</td>
<td>.001</td>
</tr>
<tr>
<td>TIMI grades 2 and 3 flow combined</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>90 min</td>
<td>77</td>
<td>65</td>
<td>.001</td>
</tr>
<tr>
<td>180 min</td>
<td>82</td>
<td>78</td>
<td></td>
</tr>
<tr>
<td>24 h</td>
<td>89</td>
<td>85</td>
<td></td>
</tr>
<tr>
<td>5–7 d</td>
<td>87</td>
<td>83</td>
<td></td>
</tr>
<tr>
<td>Residual diameter stenosis, %*</td>
<td>75 (65, 87)</td>
<td>77 (67, 95)</td>
<td>.054</td>
</tr>
<tr>
<td>Reocclusion†</td>
<td>0.8</td>
<td>6.3</td>
<td>.015</td>
</tr>
<tr>
<td>Left ventricular function</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ejection fraction, %*</td>
<td>66 (57, 74)</td>
<td>57 (47, 67)</td>
<td>.0001</td>
</tr>
<tr>
<td>End-systolic volume index, mL/m²*</td>
<td>21 (14, 28)</td>
<td>27 (19, 37)</td>
<td>.0001</td>
</tr>
<tr>
<td>Wall motion, SDs/chord*</td>
<td>−1.76 (−2.83, −0.61)</td>
<td>−2.84 (−3.49, −2.04)</td>
<td>.0001</td>
</tr>
<tr>
<td>Abnormal chords, *</td>
<td>10 (0, 24)</td>
<td>24 (12, 36)</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Preserved regional wall motion, % of group</td>
<td>44</td>
<td>19</td>
<td>.0001</td>
</tr>
</tbody>
</table>

*Continuous variables are expressed as median value (25th and 75th percentiles).

patients (33% versus 39%, P=.021). Patients with non–Q-wave compared with Q-wave MI were less likely to have multivessel disease (34% versus 39%, P=.103), particularly among those in whom the circumflex was the infarct-related artery (26% versus 42%). There was no difference in the frequency of identifiable collaterals between the non–Q- and Q-wave groups.

Complete infarct-related artery patency (TIMI grade 3 flow) was significantly greater among the non–Q-wave group (54% versus 44%, P<.001). Early (90-minute) infarct-related artery patency (TIMI grade 2 or 3 flow) was significantly greater among the non–Q-wave group (77% of 195 versus 65% of 790 patients, respectively; P=.001; Table 3). Angiograms obtained >180 minutes after the start of therapy showed consistently greater overall patency among the non–Q-wave group; however, these differences were not statistically significant. The non–Q wave group also showed a trend toward a lower median residual percent diameter stenosis of the infarct-related artery.

Five hundred eighteen patients had a patent infarct-related artery 90 minutes after treatment and underwent follow-up angiography at 5 to 7 days. Patients who subsequently developed a non–Q-wave MI had less reocclusion: overall reocclusion was 0.8% in the 119 patients who evolved a non–Q-wave MI compared with 6.3% of 399 patients who later developed a Q-wave MI (P=.015; Table 3).

Left Ventricular Function
Non–Q-wave MI patients had significantly better global left ventricular function, as indicated by greater median left ventricular ejection fraction, than did Q-wave MI patients (66% versus 57%, P<.0001, Table 3). In addition, end-systolic volume index was significantly lower among the non–Q-wave group (median 21 versus 27 mL/m², P=.0001).

Regional function in the infarct zone was also significantly better among non–Q-wave MI patients (median, −1.78 versus −2.84 SDs/chord; P<.0001). In addition, non–Q-wave MI patients had fewer abnormal chords (median, 10 versus 24 chords; P=.0001), and more of these patients showed preserved wall motion than did the Q-wave MI patients (44% versus 19%, P<.0001).

Consistent with the patency data, the left ventricular ejection fraction, end-systolic volume index, regional function in the infarct zone, number of normal chords, and number of patients with preserved wall motion was greater at all four angiographic time frames among non–Q-wave MI patients. Furthermore, in patients who underwent both 90-minute and 5– to 7-day follow-up study, better global (eg, median ejection fraction at 90 minutes: 66% versus 58% and 5 to 7 days: 68% versus 57%) and regional (eg, median infarct zone motion at 90 minutes: −1.84 versus −2.95 SDs/chord; 5 to 7 days: −1.27 versus −2.56 SDs/chord) left ventricular function was seen among the non–Q-wave group.

Clinical Outcomes
Compared with the Q-wave group, there was a trend toward lower in-hospital mortality among the non–Q-wave group (1.5% versus 3.0%, P=.067); this difference was maintained at 30 days (1.7% versus 3.2%, P=.082) and achieved statistical significance at 1 year (4.0% versus 7.0%, P=.021). Two-year mortality was also significantly lower among the non–Q-wave group (6.4% versus 10.1%, P=.02; Fig 2).

After adjustment for differences in baseline characteristics and other prognostic determinants of survival (eg, time to thrombolytic treatment, thrombolytic strategy) in a multivariable model, non–Q-wave MI was a significant independent predictor of lower 2-year mortality (odds ratio, 0.61; 95% confidence interval, 0.38 to 0.97; P=.044).
Non–Q-Wave vs Q-Wave MI After Thrombolysis

The use of in-hospital coronary revascularization procedures was similar among the non–Q- and Q-wave groups (42% versus 44%, P=.45); coronary angioplasty accounted for the majority of cases (33% versus 36%). Thirty-day and 1-year mortality rates were similar among the non–Q-wave MI patients, regardless of whether they underwent in-hospital coronary revascularization (1.7% versus 1.7% and 4.1% versus 4.7%, respectively).

Cardiogenic shock and congestive heart failure were less frequent in the non–Q versus the Q-wave group (1.5% versus 4.8%, P<.001; and, 11% versus 15%, P=.013, respectively). Rates of reinfarction (0.5% versus 0.2%, P=.35), recurrent ischemia (19% versus 16%, P=.29), and stroke (1.2% versus 1.3%, P=.85), were similar among the two groups.

Discussion

In our study, 20% of the 2046 GUSTO-I angiographic patients who were eligible for this secondary analysis and presented with ST-segment elevation were classified as evolving a non–Q-wave MI post-thrombolysis. This rate of non–Q-wave development is similar to the findings from the LATE substudy,16 the current angiographic substudy and overall GUSTO-I analyses15 represent the majority of cases (33% versus 36%). Thirty-day and 1-year mortality rates were similar among the non–Q-wave MI patients, regardless of whether they underwent in-hospital coronary revascularization (1.7% versus 1.7% and 4.1% versus 4.7%, respectively).

Cardiogenic shock and congestive heart failure were less frequent in the non–Q versus the Q-wave group (1.5% versus 4.8%, P<.001; and, 11% versus 15%, P=.013, respectively). Rates of reinfarction (0.5% versus 0.2%, P=.35), recurrent ischemia (19% versus 16%, P=.29), and stroke (1.2% versus 1.3%, P=.85), were similar among the two groups.

The differences observed between the studies of non–Q versus Q-wave survival likely relate to differences in patient selection (eg, age exclusion, prior MI exclusion), time from chest pain onset to initiation of thrombolytic therapy, and differences in post-thrombolytic treatment strategies (eg, use of adjunctive intravenous heparin, particularly among patients who received alteplase, and coronary revascularization).

In addition, multiple classifications and different time frames for infarct type stratification have been used; the ECG criteria for diagnosis of non–Q- and Q-wave MI have never been standardized. The simple screening criteria we used in this analysis have been validated with quantitative anatomic analysis and are easily applied by the clinician at the bedside. The optimal timing of ECG classification of non–Q- and Q-wave MI post-thrombolysis is also controversial25; perhaps, as
silent) left ventricular territory is at risk.

Given that a smaller (and possibly electrocardiographically silent) left ventricular territory is at risk.

right coronary artery and left circumflex culprit involvement, the infarct-related artery: only 33% of patients in the non–Q-wave group experienced MI from an occlusion of the left main, the infarct-related artery: only 33% of patients in the non–Q-wave group experienced MI from a Q- or non–Q-wave classification.

Another novel finding of this study is the more frequent finding of a more distal location of the culprit stenosis within the infarct-related artery: only 33% of patients in the non–Q-wave group experienced MI from an occlusion of the left main, proximal left anterior descending, left circumflex, or right coronary artery. In addition to the more common finding of right coronary artery and left circumflex culprit involvement, this more distal location within the infarct-related artery could account for the more frequent incidence of non–Q-wave MI given that a smaller (and possibly electrocardiographically silent) left ventricular territory is at risk.

Study Limitations

The current analysis was based on a follow-up ECG performed at ≥24 hours after the initiation of thrombolysis but before hospital discharge, with a median time of ≈1 week. Therefore, analysis of reinfarction was limited because most of these events (87 of 96) occurred before a follow-up ECG was obtained. In addition, patients who died before the acquisition of a follow-up ECG were not included in this analysis. Of the 385 patients (15.8%) enrolled in the GUSTO-I angiographic study excluded from the current analysis, 102 (26.5%) died within 30 days. However, 51% of these deaths occurred within 24 hours, and this time frame is too short to accurately assess ECG development or provide prognostic information from a Q- or non–Q-wave classification.

Although this post hoc analysis does provide explanatory pathophysiological mechanisms for the development of non–Q-wave MI after thrombolysis, most patients who sustain a non–Q-wave MI do not present with ECG changes (eg, ST-segment elevation or left bundle-branch block) that would lead the physician at the bedside to administer thrombolytic therapy. There is evidence that patients without ST-segment elevation, particularly those with significant ST-segment depression (who often develop a non–Q-wave), have a much worse prognosis than patients with ST-segment elevation who receive thrombolysis. Finally, although patients with ST-segment elevation who evolve non–Q-wave MI are an important subgroup, they represent <50% of the overall non–Q-wave population.

Conclusions

The findings from this secondary GUSTO-I angiographic analysis indicate significant ECG, angiographic, and mortality differences between patients who evolve a non–Q- versus Q-wave MI after thrombolysis. The excellent prognosis among the subgroup of patients who develop non–Q-wave MI after thrombolytic therapy is related to early, complete, and sustained infarct-related artery patency with resultant limitation of left ventricular MI and dysfunction.

Acknowledgments

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References


Matetzky et al suggest, the prognostic value of this ECG stratification is best demonstrated at a later (pre-discharge) rather than an earlier (24 hours) time after thrombolysis.

Although the rate of in-hospital coronary revascularization was high (43%), this is comparable to that seen in the TIMI-II study, and one of the major findings in that study was a similar rate of reinfarction and death in the non–Q-wave group, regardless of the treatment strategy assigned. Finally, short- and long-term mortality rates were similar among non–Q-wave MI patients in the current analysis regardless of whether they underwent in-hospital revascularization.

Another novel finding of this study is the more frequent finding of a more distal location of the culprit stenosis within the infarct-related artery: only 33% of patients in the non–Q-wave group experienced MI from an occlusion of the left main, proximal left anterior descending, left circumflex, or right coronary artery. In addition to the more common finding of right coronary artery and left circumflex culprit involvement, this more distal location within the infarct-related artery could account for the more frequent incidence of non–Q-wave MI given that a smaller (and possibly electrocardiographically silent) left ventricular territory is at risk.

Study Limitations

The current analysis was based on a follow-up ECG performed at ≥24 hours after the initiation of thrombolysis but before hospital discharge, with a median time of ≈1 week. Therefore, analysis of reinfarction was limited because most of these events (87 of 96) occurred before a follow-up ECG was obtained. In addition, patients who died before the acquisition of a follow-up ECG were not included in this analysis. Of the 385 patients (15.8%) enrolled in the GUSTO-I angiographic study excluded from the current analysis, 102 (26.5%) died within 30 days. However, 51% of these deaths occurred within 24 hours, and this time frame is too short to accurately assess ECG development or provide prognostic information from a Q- or non–Q-wave classification.

Although this post hoc analysis does provide explanatory pathophysiological mechanisms for the development of non–Q-wave MI after thrombolysis, most patients who sustain a non–Q-wave MI do not present with ECG changes (eg, ST-segment elevation or left bundle-branch block) that would lead the physician at the bedside to administer thrombolytic therapy. There is evidence that patients without ST-segment elevation, particularly those with significant ST-segment depression (who often develop a non–Q-wave), have a much worse prognosis than patients with ST-segment elevation who receive thrombolysis. Finally, although patients with ST-segment elevation who evolve non–Q-wave MI are an important subgroup, they represent <50% of the overall non–Q-wave population.

Conclusions

The findings from this secondary GUSTO-I angiographic analysis indicate significant ECG, angiographic, and mortality differences between patients who evolve a non–Q- versus Q-wave MI after thrombolysis. The excellent prognosis among the subgroup of patients who develop non–Q-wave MI after thrombolytic therapy is related to early, complete, and sustained infarct-related artery patency with resultant limitation of left ventricular MI and dysfunction.

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Non–Q-Wave vs Q-Wave MI After Thrombolysis


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