Henry Ford Hospital Medical Journal

Volume 39 | Number 3 Article 19

9-1991

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Borzak, Steven and Rosman, Howard S. (1991) "Non-Q Wave Myocardial Infarction," *Henry Ford Hospital Medical Journal* : Vol. 39 : No. 3 , 256-262.
Available at: https://scholarlycommons.henryford.com/hfhmedjournal/vol39/iss3/19

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Non-Q Wave Myocardial Infarction

Steven Borzak, MD,* and Howard S. Rosman, MD*

Non-Q wave myocardial infarction is a distinct and changing clinical entity characterized by lower initial mortality and a higher rate of reinfarction compared to Q wave infarction. Clinical and pathologic data suggest that the syndrome results from transient or incomplete coronary occlusion resulting in an infarct which is smaller than when Q waves are present. High-risk patients can be identified during hospitalization, allowing for aggressive therapy aimed at revascularization. Relatively few clinical trials have examined initial therapy or secondary prevention in this group of patients. These studies are reviewed and management guidelines suggested. (Henry Ford Hosp Med J 1991;39:256-62)

In 1919, Herrick (1) published a report of three patients who survived acute coronary thrombosis. He displayed the ECG of one patient where T-wave abnormalities, but no Q waves, were present. This was the first published illustration of what is now referred to as a non-Q wave myocardial infarction (MI). One year later, Pardee (2) described a patient with MI whose ECG initially demonstrated pathological ST segment elevation with subsequent development of a "deep S" wave. For decades this deep S wave (later called a Q wave) was the finding clinicians considered pathognomonic of infarction.

In 1942, Langendorf and Kovitz (3) reported a patient with clinical evidence of MI after hip surgery, where ECGs failed to show Q waves. At autopsy, the zone of infarction did not involve the endocardium, leading the investigators to postulate that ST and T-wave changes in the absence of Q waves were markers for subendocardial infarction. In 1954, Prinzmetal et al (4) suggested that the subendocardium was relatively "silent" electrocardiographically in dogs subjected to experimental ischemia. This report advanced the practice of referring to infarctions with Q waves as "transmural" and to infarctions with ST or T-wave changes but no Q waves as "subendocardial." Later findings by the same group invalidating the initial results went unnoticed (5). This terminology persisted despite clear evidence, most recently assembled by Spodick (6), that extent of infarction does not correlate with presence or absence of Q waves. Therefore, most investigators currently classify infarctions into electrocardiographic categories of "Q" versus "non-Q" rather than using the imprecise pathologic descriptors of "transmural" versus "nontransmural."

Pathophysiology

In 1980, DeWood and colleagues (7) settled a long-standing controversy by proving coronary thrombosis causative in the pathogenesis of infarction, rather than a secondary event. They performed early angiography in patients with infarction, which revealed a high incidence of thrombosis. In contrast, in 1986 the same group analyzed early angiograms of patients with non-Q wave MI and showed a much lower incidence of coronary occlusion and a higher incidence of collateralization of the infarct vessel (8). The finding of a patent infarct vessel has been reported by other groups when patients were studied within two weeks after non-Q wave MI (9,10). Pathologic studies conducted by Davies and Thomas (11) have revealed that plaque fissure is common in initiating the sequence of events triggering the coagulation cascade and leading to thrombosis at the site of a fixed coronary obstruction.

Reconciling the evidence of coronary thrombosis as the proximate cause of infarction with catheterization studies in patients showing open infarct-related arteries after non-Q wave MI has led to several hypotheses on the mechanism of non-Q wave MI (Table 1). The angioscopic visualization of dynamic, forming, and dissolving thrombus in the coronaries of patients with "intermediate" and unstable coronary syndromes (12) suggested that not all intracoronary thrombi are permanent and has led to the favored hypothesis that non-Q wave MI may be most often the result of transient coronary occlusion by thrombus.

A number of lines of evidence have been marshalled by Gibson (13) favoring coronary occlusion followed by reperfusion as the most common mechanism of non-Q wave MI. First, pathologic studies by Freifeld et al (14) showed that patients dying of non-Q wave MI more often had patent infarct vessels and were

Submitted for publication: September 3, 1991.
Accepted for publication: October 24, 1991.
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Table 1
Proposed Pathophysiological Mechanisms of Non-Q Wave Myocardial Infarction

Occlusion-reperfusion of infarct vessel: • Spontaneous lysis of obstructive coronary thrombus • Relief of spasm which had caused coronary occlusion • Both
Incomplete occlusion of infarct vessel by plaque/thrombus
Increased demand in setting of fixed coronary obstruction
Occlusion of infarct vessel with low-grade collateral flow

twice as likely to manifest contraction band necrosis, a histologic feature of reperfusion, when compared with patients dying of similar-sized Q wave infarcts. Second, non-Q wave MIs tend to be smaller, with an earlier peak of serum creatine kinase MB (CK-MB), a serologic pattern suggesting "washout" of the enzyme in a reperfused zone similar to that observed after successful treatment with thrombolytic agents (13). One trial reported that of 53 consecutive patients given streptokinase (SK) for acute chest pain and ST elevation on the ECG, eight ultimately developed non-Q wave MI; six of the eight were treated within 1.5 hours of the onset of symptoms (15). Finally, Huey and colleagues (9) showed that up to one-half of patients who ultimately develop non-Q wave MI without thrombolytic treatment present with ST elevation on the initial ECG, a pattern previously thought to lead inevitably to Q wave development. These findings have subsequently been confirmed by others (10,16, 17). While it could be argued that ST elevation may not signify complete coronary occlusion, this finding together with the above arguments and other insights gained since the advent of the "thrombolytic era" suggest that non-Q wave MI differs from Q wave MI not as much in the initiating sequence of intracoronary thrombosis but more in the subsequent fate of the occluding thrombus. These studies also suggest that early thrombolytic therapy may "convert" a potential Q wave infarction into a non-Q wave MI. Thus, patients with non-Q wave MI in the 1990s may be a different group than those studied in earlier years, before the general use of the more sensitive and specific CK-MB enzyme assay to detect infarction (early 1980s) (18,19) and prior to the increased application of thrombolytic therapy (mid 1980s). This may explain why the incidence of non-Q wave MI is increasing (18,20); non-Q wave MI patients now account for more than 40% of all patients with MI at some institutions (21). Further research may help define anatomic, hematologic, and demographic characteristics that produce the differing rates of acute coronary occlusion in the Q wave and non-Q wave MI syndromes.

Natural History and Clinical Syndrome of Non-Q Wave MI

ECG criteria for non-Q wave MI

Q wave infarctions are characterized by the development of 30-msec or longer Q waves in at least two consecutive leads of the anterior (V1-V4), inferior (II, III, aVF), or lateral (I, aVL, V5, V6) distributions. Posterior infarctions characterized by prominent R waves and R/S ratio > 1 in leads V1-V2 are usually categorized as Q wave equivalent, as are anterior infarctions where "embryonic" R waves of less than 0.25 mm develop in V1-V4 (22). Presenting ECGs of patients with non-Q wave infarction may show initial ST elevation (9,10,16,17), in which case the diagnosis of non-Q wave MI must await subsequent evolution of the ECG over 24 to 48 hours. Patients with prolonged chest pain and ST depression or T-wave inversion on the initial ECG may initially be suspected of having non-Q wave MI, but distinction from unstable anginal syndromes must await serologic confirmation of infarction by CK-MB determination and clinical course.

Table 2
Clinical Profile of Q Wave Versus Non-Q Wave Myocardial Infarction Patients*

	Q Wave MI	Non-Q Wave MI	Number of Studies
Short-term mortality (5 to 30 days)	20%	10%	24
Long-term mortality (12 to 96 months)	26%	31%	21
Reinfarction (hospital stay-44 months)	6%	16%	14

*Pooled data from Gibson (13).
MI = myocardial infarction.

Clinical course and prognosis of non-Q wave MI

Course and prognosis differ between patients who do and do not manifest Q waves after infarction. The many studies comparing clinical characteristics of Q wave and non-Q wave MI patients published between 1970 and 1983 have been extensively reviewed by Gibson (13). Appreciable differences have not been found regarding patients' demographic characteristics. The key question posed by nearly all of the studies is: Does the prognosis of Q wave and non-Q wave MI patients differ in terms of death or reinfarction? The synthesis of Gibson's review of these studies is shown in Table 2. Event rates for mortality and reinfarction were obtained by pooling the results in Q wave and non-Q wave MI groups. Not all studies showed a statistically significant difference between groups for the variables shown, but aggregate differences are highlighted. Non-Q wave MI patients had lower short-term mortality (10% versus 20%) but similar or slightly higher long-term mortality (32% versus 26%). This discrepancy may be explained by the fact that nearly every study reviewed showed that non-Q wave MI patients have a smaller-sized infarct. More frequent reinfarction in non-Q wave MI (16% versus 6% in Q wave MI), sometimes fatal, would tend to equalize the long-term mortality rates.

In-hospital Evaluation of Non-Q Wave MI Patients: Risk Assessment

Patients with non-Q wave MI are a heterogeneous group regarding demographic characteristics, prior cardiac symptoms,

underlying coronary anatomy, ventricular function impairment, and, in particular, risk for future events (21). Some clinicians recommend that all non-Q wave MI patients undergo coronary angiography with consideration toward revascularization (23), while others suggest that a noninvasive assessment can identify patients at lower risk for death and reinfarction who would then be managed expectantly without routine angiography (13,24). We recommend the latter approach, based on 1) the lack of clinical trials showing benefit to patients of one approach over the other, and 2) the results of several trials of thrombolytic therapy in patients presenting with ST segment elevation. The Thrombolysis in Myocardial Infarction, phase IIB (TIMI-IIB) study showed that in patients who had been treated with thrombolysis, a group which differs somewhat from patients with non-Q wave MI, no benefit was achieved by requisite angiography and percutaneous transluminal coronary angioplasty (PTCA) in the absence of recurrent ischemia (25).

Recurrent or provokable ischemia

Numerous studies have shown that recurrent spontaneous or provoked ischemia dramatically increases the risk of early reinfarction and death in patients after a Q wave or an unspecified type of infarction (26). Similarly, in patients with non-Q wave MI, spontaneous postinfarction angina has been identified as a powerful predictor of subsequent events. Analysis of clinical features of the 576 patients enrolled in the multicenter Diltiazem Reinfarction Study (DRS) (22) revealed that the 43% of patients who had recurrent angina during the 14-day in-hospital follow-up period had a nearly fourfold higher risk of reinfarction (12.2% versus 3.6%) and death (6.1% versus 1.5%) when compared with patients not experiencing angina (27). Furthermore, patients whose angina was associated with ST or T-wave changes were 3.8 times more likely to have reinfarction and 7.5 times more likely to die compared to patients with angina not manifesting transient ECG changes (27). Reinfarction itself was also a powerful predictor of in-hospital death: 16.7% of patients with reinfarction died as compared to 2.4% of patients without reinfarction (22). When comparing all non-Q wave MI patients, Maisel et al (28) also found that patients with reinfarction had a higher in-hospital mortality (43% versus 8%) and one-year mortality (65% versus 16%) than those without reinfarction.

Evidence of ischemia provoked by predischarge submaximal exercise testing is also a predictor of increased risk of future cardiac events. In a prospective study of 87 patients with non-Q wave MI, Gibson et al (10) found that 36% of patients had ST depression, 30% had angina, and 60% had a redistribution thallium defect on submaximal predischarge treadmill testing. Subsequently, over a median duration of 30 months of follow-up, 14 of the 16 reinfarctions involved the infarct zone and all occurred in patients whose predischarge study had a redistribution thallium defect (10).

Anterior versus inferior-lateral location

Several studies have shown that patients with anterior non-Q wave MI are more likely to experience future adverse events. Kao et al (21) analyzed consecutive cardiac care unit admissions and found that in 135 patients, anterior non-Q wave MI location had a 3.8-fold relative risk of death and a 4.1-fold risk of reinfarction compared with inferior-lateral location. In a retrospective analysis of patients in the Multicenter Investigation of the Limitation of Infarct Size study, Stone et al (29) confirmed that anterior location conferred a worse prognosis than inferior location, even when data were adjusted to compare infarcts of similar size as determined by magnitude of peak CK release. Kao and colleagues (21) hypothesized that anterior infarcts in the left anterior descending (LAD) artery distribution may be more likely to have larger areas of residual ischemic myocardium than infarcts in the inferior location with the smaller distribution zones of right or left circumflex arteries. In addition, congestive heart failure, another risk factor for mortality, is more likely to occur at presentation or develop in the setting of an anterior infarct (29).

Impaired ventricular function

Clinical signs of heart failure as well as objective evidence of diminished ejection fraction are correlated with adverse outcomes following non-Q wave MI (17,29,30). Congestive heart failure was a univariate predictor of reinfarction and mortality in the 576 patients enrolled in the DRS, and this variable remained an independent predictor of both outcomes after Cox multiple regression analysis (30). Subset analysis of the Multicenter Diltiazem Postinfarction Trial dichotomized patients based on the presence or absence of pulmonary congestion and revealed that this clinical finding was a risk factor for reinfarction and cardiac death following non-Q wave MI. Furthermore, diltiazem administration led to an increase in death and reinfarction compared with placebo in this subset of patients (31). These findings support the already established importance of left ventricular (LV) function as the best predictor of outcome in patients with Q wave or unspecified infarction (26).

Electrocardiographic findings

A number of studies have shown that non-Q wave MI patients whose initial ECGs manifest ST depression are at greater risk of death or reinfarction (16,30,32,33). One-year mortality was 29% versus 11% when ST depression versus ST elevation was found on the initial ECG (16). However, initial ST depression often coexists with LV functional impairment (32,33). To investigate whether ST depression was an independent predictor of adverse outcome, Schechtman and colleagues (30) using Cox multiple regression analysis identified baseline and discharge ST depression as univariate predictors of one-year reinfarction and mortality but not of in-hospital reinfarction or mortality in the DRS population (31). The finding of persistent ST depression was the single most powerful independent predictor of both endpoints among all 24 variables examined. ST elevation present at discharge also predicted late mortality but not reinfarction.

The electrocardiographic diagnosis of LV hypertrophy has been shown in population-based studies to be independently associated with an increased risk of sudden death and acute MI (34). This relationship was confirmed in the non-Q wave MI population, where multivariate analysis of the DRS study popu-

Table 3
Clinical Predictors of Death or Reinfartion

Death	Reinfarction	References
Angina	Angina	19,22
Anterior location	Anterior location	21,29
CHF	CHF	17,21,31
ST depression	ST depression	16,31,33,34
LVH	LVH	17,31
Reinfarction		22,28
	Positive predischarge ETT	10

CHF = congestive heart failure, LVH = left ventricular hypertrophy, ETT = exercise treadmill test.

lation showed a twofold higher incidence of death and reinfarction at one-year follow-up (31).

Diagnostic Approach to the Non-Q Wave MI Patient

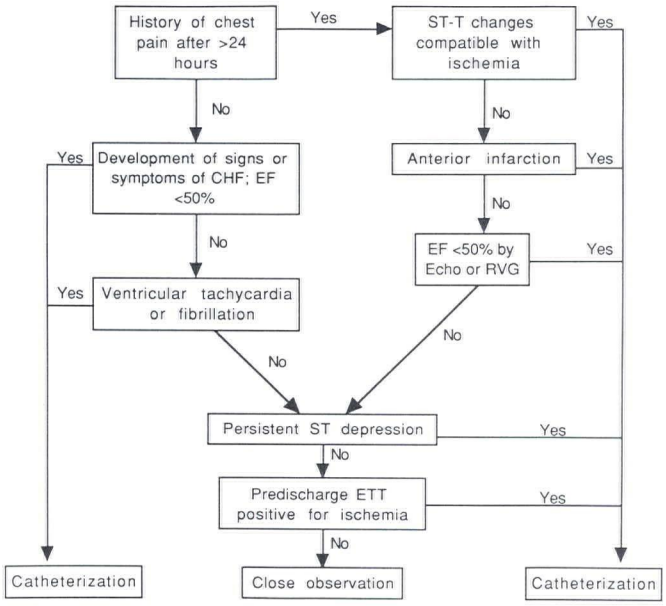
A recommended approach for diagnostic evaluation of the non-Q wave MI patient is based on the concept of risk stratification (Table 3). This scheme is illustrated in the Figure. This approach would allow for expectant management of an uncomplicated patient at lower risk, while providing an aggressive posture when death or reinfarction is relatively more likely. We do note that no prospective study has shown improved clinical outcomes after revascularization, whether by coronary artery bypass grafting (CABG) or PTCA in non-Q wave MI patients. Nevertheless, a consensus favors such mechanical intervention when severe or unstable symptoms or high-risk clinical situations are present (13,23,24).

Bedside assessment

An especially vigilant approach is warranted given the tendency for non-Q wave MI patients to experience recurrent angina, infarction, and death as described previously. In particular, patients should be questioned daily about the development of ischemic or congestive heart failure symptoms. The physical examination should focus on the development of tachycardia, hypotension, pulmonary rales, or new gallop rhythms. A subtle deterioration in ventricular function in a resting, asymptomatic patient may be the only sign of recurrent ischemia or infarction. The appearance of any of these findings justifies a repeat ECG to look for new ischemic changes.

Noninvasive assessment

Since significant impairment of LV function can be asymptomatic or compensated by medical therapy, we believe an objective assessment of LV function by echocardiography or radionuclide ventriculography is warranted prior to discharge. Patients with impaired LV function are more likely to have multivessel coronary disease and would be most likely to benefit from mechanical revascularization (35).



Figure—Algorithm for in-hospital evaluation of the non-Q wave MI patient. The treatment strategy is based on the presence or absence of high-risk clinical markers. See text for details.

While the appearance of complex ventricular ectopy was found to occur in 22% of non-Q wave MI patients in one series, this study did not differentiate sustained or symptomatic ventricular tachycardia or fibrillation from lower grade arrhythmias, nor did it find that ventricular ectopy predicted subsequent events (10). However, the small sample size of this subgroup makes a type II error possible. Because late (occurring after the first 24 hours) sustained or symptomatic ventricular tachycardia or fibrillation occurring in the absence of electrolyte disturbance or severe LV dysfunction may signify ischemia, we feel that this set of circumstances warrants coronary angiography. Continuous bedside and/or holter monitoring is recommended for the first 48 to 72 hours after symptom onset (35).

The submaximal exercise test has become the standard of care for MI patients prior to discharge who do not experience an in-hospital complication (26). If resting ST abnormalities such as LV hypertrophy or digitalis effect preclude interpretation of exercise-induced ECG changes, then thallium imaging is recommended (35). Patients who manifest ischemia by the development of angina, ECG changes, or diagnostic thallium alterations can then be referred for angiography.

Invasive assessment

Once a patient has been identified as high risk, catheterization with coronary angiography is recommended (Table 4). The goal of this procedure is to define coronary anatomy in consideration of revascularization by PTCA or CABG. Accordingly, a relative contraindication to catheterization would be a patient's unwillingness to consider either revascularization procedure.

Table 4
In-hospital Evaluation of Patients with Non-Q Wave Myocardial Infarction

Procedure	Inclusion	Purpose
Daily bedside examination	All patients	Assess for recurrent angina or CHF
Telemetry or Holter monitoring (at least 72 hours)	All patients	Detect "late" ventricular tachycardia or fibrillation
Echocardiography or radionuclide ventriculogram	All patients	Determine overall ventricular function and regional contractility
Predischarge ECG	Patients without prior indication for angiography	Detect persistent or new ST depression
Submaximal predischarge exercise test	Patients without prior indication for angiography	Detect exercise-induced ischemia
Coronary angiography	See Figure	Define coronary anatomy prior to possible revascularization

Treatment of Non-Q Wave MI Patients

Thrombolytic therapy

No study to date has conclusively reported the effect of thrombolytic therapy on death or reinfarction after non-Q wave MI. Such a study might be impossible to conduct because of the variability in presenting features seen in non-Q wave MI patients. Up to one-half of non-Q wave MI patients present with ST elevation on ECG, which is a general criterion for administration of thrombolytic therapy (25). Many patients who later develop a non-Q wave MI might initially be classified as having a transmural injury currently typical of an evolving Q wave infarct (9). Non-Q wave MI patients presenting with ST depression or T-wave inversion cannot be reliably distinguished from patients with unstable angina until CK-MB results and clinical course are apparent. The TIMI-IIIB study currently in progress is designed to compare low-dose tissue plasminogen activator with placebo in patients with chest pain and ECG changes other than new ST elevation or Q waves, but without enzymatic evidence of infarction; some of these patients are likely to develop non-Q wave MI. Thus non-Q wave MI patients are now more likely either to be given lytic therapy upon presentation with ST elevation or be managed without thrombolysis if presentation is with ST depression or T-wave inversion. Recommendations for lytic therapy in the latter group of patients must await the results of TIMI-IIIB and other trials.

Heparin and acetylsalicylic acid

No trial has examined the role of either intravenous (IV) heparin or acetylsalicylic acid (ASA) as primary treatment for patients with non-Q wave MI. Low-dose subcutaneous heparin has been recommended after infarction to prevent venous thrombosis and pulmonary embolism (35). Therapeutic IV heparin has been shown to be of benefit in unstable angina (22), and since a common pathophysiology has been proposed for unstable angina and non-Q wave MI (36), some investigators have advocated the use of this agent on presentation and for several days after non-Q wave MI. Heparin may be even more appealing with the knowledge that most infarct-related vessels are patent at angiography and that most reinfarctions occur in the same arterial distribution (10), but a firm recommendation cannot be made in the absence of a clinical trial. IV heparin followed by oral anticoagulation can be recommended when non-Q wave MI is accompanied by severe global hypokinesis, significant anterior akinesis, or directly visualized mural thrombus (35).

ASA has been shown to reduce mortality and reinfarction in unstable angina (37), but no trial has assessed prospectively its effect on modifying myocardial damage in patients with non-Q wave MI. Again, based on the postulated common pathophysiologic mechanisms of these two clinical entities, early ASA therapy is appealing. A much stronger argument can be made for ASA treatment in secondary prevention of future events.

Anti-ischemic therapy

Recurrent angina after non-Q wave MI should be promptly treated, followed by coronary angiography. There is little to suggest relative benefits of calcium channel blocking agents over β-adrenergic receptor blocking agents (beta-blockers) when ischemia recurs in the days after non-Q wave MI.

IV nitroglycerin has found growing support for its potential role in limiting infarct size and reducing mortality in patients with unspecified infarction (38,39). However, no study has addressed the non-Q wave MI population and current recommendations of the American College of Cardiology do not support IV nitroglycerin in all patients with infarction (35). Nitroglycerin is well established for relieving ischemic symptoms. Analgesia with morphine sulfate and sedation with short-acting benzodiazepines are appropriate for many patients experiencing angina.

Revascularization

No study has examined the effect of either PTCA or CABG on symptoms or survival after non-Q wave MI. When recurrent ischemia or infarction occurs, there is general agreement among cardiologists that catheterization followed by mechanical revascularization offers the best hope both for relief of symptoms and protection from further ischemia and infarction (13,24). Such an approach is particularly warranted when the jeopardized myocardium is in the anterior distribution of the LAD artery (16).

Secondary Prevention After Non-Q Wave MI

An important issue distinct from management of recurrent ischemia is routine prophylactic treatment of patients who are asymptomatic after non-Q wave MI (Table 5).

ASA and anticoagulation

ASA has been shown in a number of trials to reduce both death and reinfarction after unspecified infarction and should be given to patients after non-Q wave MI when no contraindications are present (37). Subgroup analysis of the Persantine-Aspirin Reinfarction Study suggested that the beneficial effects of antiplatelet therapy may be even more pronounced for non-Q wave MI than for Q wave MI patients (40).

Table 5
In-hospital Treatment of Non-Q Wave Myocardial Infarction Patients*

Indication	Treatment	Status of Recommendation
Primary modification of infarction	Thrombolytic therapy	Controversial
	Heparin	Controversial
	ASA	Recommended
	Nitrates	Acceptable
	Beta-blockers	Acceptable
	Calcium blockers	Controversial
Secondary prevention of death or reinfarction	ASA	Recommended
	Beta-blockers	Acceptable
	Calcium blockers	Acceptable
	Warfarin	Controversial

*For references, see text.

While anticoagulation with warfarin was shown in a recent study to reduce death after unspecified infarction (41), routine administration to non-Q wave MI patients cannot be recommended in the absence of more traditional indications (visualized mural thrombus, akinetic anterior wall, atrial fibrillation).

Calcium channel blockers and the DRS

The DRS is the only published trial designed to examine secondary prevention specifically in the population of non-Q wave MI patients (22). A total of 576 patients were enrolled 24 to 72 hours after the index event and randomized to the double-blind administration of diltiazem or placebo. Patients were followed until discharge (a total of 14 days) and evaluated for the primary endpoint of reinfarction and secondary endpoints of recurrent angina and in-hospital mortality. Diltiazem was found to reduce early reinfarction from 9.3% in control patients to 5.2% in those receiving diltiazem ($P < 0.03$). The validity of this finding has been questioned, however, because a one-sided test of significance used in this analysis would not adequately account for the possibility of an adverse effect of treatment. Diltiazem had no significant effect on recurrent angina (41% versus 44% in placebo-treated patients) or on in-hospital mortality (3.8% versus 3.1% in the placebo group). Episodes of severe angina (requiring withdrawal from the trial for indicated calcium channel blockers or revascularization) were significantly reduced by 50%. No late follow-up on treatment effect was provided in the study. We note also that nearly two-thirds of patients in both treatment and control groups were receiving beta-blockers, which were not restricted by the study design.

A retrospective analysis of non-Q wave MI patients has been performed on results from the Multicenter Diltiazem Postinfarction Trial (31). This study was designed to examine the long-term effects of diltiazem from 3 to 15 days after unspecified infarction, with follow-up to an average of 25 months. Diltiazem had no effect on mortality or cardiac event rate overall (42), but subgroup post hoc analysis showed fewer cardiac events and a trend toward lower mortality in patients receiving diltiazem after a first non-Q wave MI. A detrimental effect was seen in all subgroups with pulmonary congestion as a manifestation of congestive heart failure (31). However, retrospective subgroup analysis has serious limitations in identifying treatment indications not originally specified by the study design (43).

Other calcium channel blocking agents (verapamil, nifedipine, and newer "second generation" agents) have not been examined specifically in non-Q wave MI patients. We conclude that data support the safe use of diltiazem for secondary prevention after non-Q wave MI in patients without congestive heart failure (35) but that unequivocal evidence of important benefit is lacking.

Beta-blocking agents

The use of beta-blocking agents after unspecified infarction has been found to reduce significantly both mortality and reinfarction; their administration has been recommended for at least two years after a MI (35). No trial has prospectively examined the non-Q wave MI population, but subset analysis of three studies—Metoprolol in Myocardial Infarction Study, the Beta-Blocker Heart Attack Trial, and the Timolol Myocardial Infarction Study—has yielded conflicting results, with only the latter study showing a statistically significant decrease in mortality but not reinfarction (44-46). The limitations of post hoc analysis have been extensively discussed in this context (43). Given the relative instability of the non-Q wave MI population compared to patients with Q wave infarcts, some investigators have argued that prophylactic beta-blockade might in fact be more beneficial in this subgroup. An answer to this intriguing issue must await a prospective trial.

Revascularization

No trial supports PTCA or CABG in asymptomatic patients following non-Q wave MI (13). We support the use of the predischarge treadmill test to direct patients who manifest exercise-induced ischemia toward catheterization and revascularization when appropriate.

Summary and Conclusions

Changes in medical practice in the 1990s—among them a better pathophysiologic understanding of the role of intracoronary thrombus and increased use of thrombolytic therapy in the early hours of MI—have changed the types and numbers of patients with the diagnosis of non-Q wave MI. Prior experience has shown that non-Q wave MI patients experience smaller infarcts, higher rates of reinfarction, and lower in-hospital mortality compared to Q wave MI patients. In-hospital management should focus on an aggressive search for spontaneous or provokable ischemia, depressed LV function, and other markers of increased risk of future events. Such patients should be considered for angiography and revascularization. Few clinical studies support specific secondary prevention strategies in this population other than those measures recommended for the postinfarction population at large. Firm recommendations for therapy must await future trials.

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