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Incomplete Versus Complete Myocardial Infarction

Mihai Gheorghiade, MD,* and Sidney Goldstein, MD*

Incomplete myocardial infarction (MI), when compared with a complete MI, is characterized by a small infarct size and a large mass of viable but jeopardized myocardium within the perfusion zone of the infarct-related vessel that is manifested clinically by early recurrent infarction. The pathophysiology involves early spontaneous or thrombolytic reperfusion. Clinical (i.e., residual ischemia), electrocardiographic, and echocardiographic findings and magnitude of serum cardiac enzyme elevations should be taken into account in diagnosing an incomplete MI. (Henry Ford Hosp Med J 1991;39:263-4)

The observation that the ischemic event associated with thrombotic occlusion of the coronary artery can be interrupted with thrombolytic therapy has led to the recognition of a new ischemic syndrome, the incomplete myocardial infarction (MI) (1). These observations have also called our attention to the fact that early spontaneous reperfusion may occur in patients who experience a non-Q wave MI (2). These two clinical presentations of incomplete infarction identify patients with initially small MIs who are at risk of early recurrence of myocardial ischemia involving a relatively large area of viable but jeopardized myocardium in the infarct-related artery (3).

Patients recovering from a large but completed MI have a well-defined increased mortality related to the extent of myocardium lost (4). In contrast, patients with an incomplete ischemic event, despite an initial modest insult, are prone to reinfarction shortly after their event, leading to a significant increase in mortality related to further tissue loss and recurrent ischemia (5). Patients who experienced early thrombolytic or spontaneous reperfusion comprise the patient population of incomplete infarction. Both groups of patients with incomplete infarction are bound together by the patent but diseased vessel, the high rate of early reinfarction, and a limited initial insult.

Given the heterogeneity of the non-Q wave infarction group (6) that includes patients with prior Q waves, different electrocardiographic locations, and even normal ECGs (7), and the fact that in patients receiving thrombolytic therapy the presence of Q waves does not always predict a completed event or transmurality (8-10), it is not surprising that the ECG alone will not properly identify patients with an incomplete infarction (11-13). The frequent presence of a Q wave in the incomplete thrombolytic infarct is partially related to the duration of symptoms and the protocol demand for significant ST and T-wave changes before administering thrombolytic therapy (14,15). In addition, the absence of Q waves is not always correlated with a small or subendocardial MI, particularly if the myocardial segments are supplied by the right or the circumflex coronary artery (16). In this era of thrombolytic therapy, the absence or presence of Q waves on the ECG may not properly identify patients with incomplete infarction.

When applied to the individual patient, it is therefore more useful to divide postinfarction patients, regardless of whether or not they have spontaneous reperfusion or receive thrombolytic therapy, into those who have a completed versus incomplete MI. In determining the incompleteness of MI, a number of factors come into play. The presence of Q waves, the location of the ST and T-wave changes (5), and the degree of ST depression (17,18) all are important. Peak serum creatine phosphokinase elevation may have significance in differentiation of the event. Small elevations of creatine phosphokinase suggest an incomplete event (16,19). The magnitude of wall motion abnormality in the area of the infarct-related artery, evaluated either by echocardiography or angiography, provides important information as to the completeness of the MI. However, early reperfusion of the occluded artery is not always accompanied by immediate recovery of the contractile function in the infarct-related artery segments (stunned myocardium). This condition in which the myocardium is viable but not contractile may persist much longer if the infarct-related artery is occluded or has a tight stenosis (hibernating myocardium) (20,21). Identification of areas of viable but not contractile myocardium (stunned or hibernating) may be possible by observing functional recovery by two-dimensional echocardiography during infusion of a small dose of dibutamine (22,23). In addition, residual ischemia, spontaneous or provoked in the area of the infarct-related artery, plays a pivotal role in identifying patients with an incomplete infarction (24).

In order to create a framework to describe the features of incompleteness of infarction, we propose the following:

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I. Suspect incomplete infarction when:
   1. Non-Q wave without thrombolytic therapy, or
   2. Small elevation usually less than 500 IU/L of the creatine phosphokinase.
   3. A. Angiographic or echocardiographic evidence of preserved function in the infarct-related area, or
      B. Angiographic or echocardiographic evidence of a large area of viable but noncontractile myocardium (stunned or hibernating myocardium) identified by low-dose dobutamine infusion.
   4. Residual ischemia present (postinfarction angiography or exercise-induced ischemia).

II. Complete infarction:
   1. Persistent Q waves.
   3. No evidence of residual ischemia.
   4. Large area of ventricular akinesis in infarct-related artery resulting from irreversibly damaged myocardium.

The criteria proposed are not rigid but require physician interpretation. A definition of the completeness of the infarction, taking these factors into consideration, is more complex than the simple presence or absence of a Q wave. For example, the patient with a Q wave MI with small elevation of the creatine kinase serum level and preserved wall motion in the infarct-related area by echocardiography will be assigned to the incomplete MI group. On the other hand, the patient with a non-Q wave infarct who has a high total creatine kinase serum level and by echocardiography akinetic wall in the infarct-related artery with no residual ischemia will be assigned as one with completed MI.

It is clear that reperfusion, either therapeutic or spontaneous, often leaves the artery with surprisingly little angiographic evidence of luminal narrowing (25). It is therefore important to realize that the therapy of the incomplete infarction lies in the realm of prevention of a recurrent thrombogenic and reocclusive process. This may have a mechanical answer in surgical or intracoronary prevention of a recurrent thrombogenic and reocclusive process.

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