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Prognosis After Myocardial Infarction

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A substantial number of patients die in the first year after myocardial infarction. The major determinants of risk during this period appear to be the extent of either damaged or potentially ischemic myocardium and the degree of electrical instability. Anterior infarction, early left ventricular failure, late significant arrhythmias, and markedly reduced radionuclide left ventricular ejection fraction are the major clinical markers of risk.

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Each year in the United States approximately 500,000 patients are hospitalized with acute myocardial infarction (MI) as the single or primary diagnosis. Over 400,000 patients survive the hospital phase, with an average posthospital mortality rate of 10% in the first year, 5% in the second year, and 3 to 4% per year thereafter. The major mortality risk is in the first 6 months after hospitalization, with an almost equal distribution of deaths in the sudden (within 1 hour) and nonsudden categories.[1]

Numerous post-MI risk stratification studies have identified meaningful associations between selected or screened clinical factors and cardiac death.[2–4] Recent investigations have emphasized the importance of classifying post-MI risk by its functional or physiologic components. From the wide array of risk variables that have been reported, 4 major categories of functional risk emerge: extent of myocardial damage as reflected by global cardiac pump dysfunction; extent of viable myocardium at ischemic risk from residual atherosclerotic coronary artery disease; the degree of electrical instability from a complex interplay of the effects of ischemia, necrosis and fibrosis; and the work load on the heart as determined by the peripheral vascular resistance, the sympathetic nervous system and the cardiac output demands.

The hypothesis underlying this functional categorization of risk is that outcome can be improved by directing therapy to correct the disordered physiologic risks. The functional risks probably act as independent factors most of the time, but complex interactions may develop to greatly amplify the risk. For example, the coexistence of ischemic and electrical risks may be more than additive in the setting of transiently augmented sympathetic activity. Further understanding of the independence, interrelationships and interactions

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among the functional risk factors is required to optimize clinical management of individual patients and to develop rational approaches for intervention trials.

Functional Risk Factors

The physician obtains a vast amount of information from the history, physical examination and laboratory studies, and experience indicates that most of the important data can be extracted from just a few variables. Rather than presenting a complete list of risk variables, therefore, I will highlight only a few of the factors that seem to carry most of the physiologic weight in risk stratification and prognosis.

Clinical history: The presence of a prior MI before the index coronary event and functional limitation (New York Heart Association class II or IV) ≥1 month before hospitalization are meaningful indicators of an unfavorable outcome. Each of these variables may provide good qualitative measures of the extent of preexisting myocardial damage on which the acute MI is engrafted.

Physical examination: Auscultatory pulmonary rales in the upper lung fields (that is, more than bibasilar) and an unequivocal S_3 gallop are important findings during the patient's acute hospital course, and they reflect the extent of acute myocardial damage. The 1-year posthospital cardiac mortality is 3- to 4-fold greater in patients with than without rales. In our recent Multicenter Postinfarction Risk Stratification Study, pulmonary rales in the coronary care unit was the single most significant predictor of subsequent mortality from a large array of meaningful risk factors.[6]

Routine laboratory studies: Two variables that provide useful information about outcome are the location of the MI by ECG and the extent of the pulmonary congestion on chest roentgenogram. Anterior MI is associated with more extensive compromise of left ventricular (LV) function and a greater incidence of intraventricular conduction disturbances than MI at other locations. The mortality risk is considerably enhanced in patients with Q waves that involve leads V_1 and V_2. The 1-year cardiac mortality rate is proportional to the degree of pulmonary congestion on a chest roentgenogram taken in the coronary care unit. In our multicenter study, the 1-year posthospital cardiac mortality ranged from 3% for those with no congestion to 38% for those with transient pulmonary edema, with intermediate mortality rates for patients with bibasilar and interstitial congestion. The peak level of acute serum myocardial enzymes is of value in prognosis only if frequent serial enzyme determinations are recorded to obtain the full profile of the enzyme curve.[5]

Special laboratory tests: Three tests provide valuable information about the functional state of the circulatory system after MI. The radionuclide ejection fraction (EF), either by the multiple-gated or first-pass methods, quantitates the global contractile efficiency of the heart as a pump. The 1-year cardiac mortality risk increases exponentially for progressively lower EF values <0.40, with almost a 50% mortality for patients with an EF <0.20. In contrast, the cardiac mortality rate 1 year after hospital discharge is <4% for an EF >0.40.[6]

A low-level activity evaluation test (modified treadmill test) as proposed by Weld et al[7] provides dynamic information about overall cardiovascular performance. Although this test has been proposed as a means of obtaining prognostically useful information from dynamically induced myocardial ischemia,[8] data from our Multicenter Postinfarction Risk Stratification Study do not substantiate this point. Rather, the duration of symptom-limited activity emerges as the most powerful determinant of survival. Posthospital mortality is >14% at 1 year in patients performing <6 minutes of treadmill activity (about 2 METS), with mortality <3% in patients who complete the full 9-minute protocol (3 to 4 METS).

Holter monitoring provides valuable insight into cardiac electrical activity over a 24-hour period. Although the primary value of this test is in characterizing ventricular arrhythmias, considerable information is also obtained about conduction disorders, ischemic ST-segment changes, heart rate trends during activity and sleep, and supraventricular arrhythmias. Several ventricular ectopic complex (VEC) grading schemes have been proposed,[9-11] and a rational and useful VEC categorization includes VEC frequency, the presence or absence of repetitive beats (>3 in a row), and the presence or absence of malignant characteristics of the repetitive pattern.[12] A 4-level, prognostically meaningful, postinfarction VEC grading system is as follows: Grade I (low risk)—average VEC frequency of <3/hour; if repetitive beats are present, the longest run must be <3 beats in a row; Grade II (intermediate risk)—average VEC frequency ≥3/hour or repetitive beats ≥3 in a row without malignant characteristics; Grade III (high risk)—average VEC frequency ≥3/hour and repetitive beats ≥3 in a row without malignant characteristics; and Grade IV (very high risk)—repetitive beats ≥3 in a row with malignant characteristics that relate to rate, duration or pattern of the tachycardia. The definition of "malignant" is still in flux, but coupling intervals <300 ms, duration >10 beats in a row at rates >150 beats/min, or torsades de pointes configurations are generally considered ominous.

Conclusion

Post-MI patients can be subgrouped according to mortality risk on the basis of physiologically meaningful variables. The presence or absence of various combinations of mechanical, electrical, ischemic and vascular parameters provide the necessary information for individualized risk stratification. This approach should permit the clinician to focus therapy on reversible dysfunction, thereby improving post-MI survival.

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Early Recognition of the Patient at Late High Risk: Incomplete Infarction and Vulnerable Myocardium

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The process of identifying patients with myocardial infarction (MI) at high risk after hospital discharge should begin at admission. By using basic clinical and laboratory information, enhanced by a wide variety of noninvasive tests, not only can individual patients at risk be recognized, but also the processes that determine risk can, at least in part, be appreciated. Outcome is affected by the extent of damaged tissue and, apparently, by the amount of potentially ischemic muscle. MI may change the coronary circulation such that a new and fragile balance between supply and demand results, both within and outside the infarct zone; that is, the infarct may be incomplete and the viable muscle within it may then be vulnerable to later ischemia. Muscle outside the infarct zone may be left in much the same precarious state. Also, coronary spasm may not be infrequent in the weeks after MI. These factors together may underlie recurrent post-MI myocardial ischemia.

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Because the complications of myocardial infarction (MI) cluster close to its onset, their treatment and prevention become the focus of coronary care. However, risk does not end after these first few days, but remains considerable for the first 6 to 12 months after hospital discharge. During this period, therefore, patients should be identified who will be at high risk of death or reinfarction during the first year after MI. Within the hospital period, the principal determinant of mortality is infarct size;[1] after that, survival has been clearly linked to left ventricular (LV) dysfunction and the extent of coronary artery disease.[2,3] Although considerable effort has been spent developing clinical and laboratory indexes to recognize the patient who will survive but remain at high risk, much less attention has been paid to the mechanisms of risk. This review defines the profile of the high-risk patient, emphasizing the information that may be derived from the initial period of coronary care, and then integrates this information in terms of pathogenesis.

MI is almost always the result of a sudden, severe and sustained reduction in blood flow. After this, there is often at least partial return of flow early enough to salvage tissue within the infarcted zone; that is, the infarct may be incomplete. Depending on the adequacy of its blood supply, this residual tissue may be vulnerable to myocardial ischemia; regions adjacent to the infarct may also be left in a precarious state. Adding to this instability is the possibility that coronary spasm may occur frequently in the weeks after MI. Thus, in some instances, the course of MI may be protracted, consisting of a series of discrete events, lasting not days but weeks. Only when this process is completed does the patient return to a relatively stable and low-risk state.

This view has been the basis for our management of post-MI patients. In the past 3.5 years, 313 patients with MI have been treated at the Cardiology Follow-Up Center of the Royal Victoria Hospital. All were ≤66 years old and none had other life-threatening diseases.

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