Treatment of Right Ventricular Infarction: Thrombolytic Therapy, Coronary Angioplasty or Neither?*

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The beneficial effects of reperfusion therapy in patients with inferior Q-wave myocardial infarction (IMI) have been less impressive than in patients with anterior Q-wave MI because of the lesser amount of myocardium at risk and the lower mortality rate in patients with IMI (1,2). Thrombolytic therapy or primary coronary angioplasty should be more successful in reducing infarct size and decreasing mortality in high risk patients with IMI. Also relevant, is the lack of definitive mortality data supporting angioplasty as the primary reperfusion strategy in patients with IMI, unless there are contraindications to thrombolytic therapy or the patient is in cardiogenic shock (1).

High Risk Inferior MI Subgroups
Three high risk subsets of patients with IMI have been defined by simple electrocardiographic (ECG) criteria (1,3–11). These include patients with associated left precordial ST-segment depression, particularly when persistent for several hours; those with third-degree AV block; and those with right precordial ECG ST-segment elevation indicating the likelihood of right ventricular (RV) infarction.

The importance of coexisting precordial ST-segment depression in leads V1–V4 in patients with inferior ST-segment elevation from acute MI has been well documented (3–6). The available data suggests that thrombolytic therapy or direct angioplasty is more likely to reduce infarct size and mortality in patients with IMI when precordial ST-segment depression is present (12–15).

The mortality rate for patients with third degree AV block due to IMI has been shown to be higher than in those without AV block (5,7–9); this is likely due to a larger infarct size rather than to the conduction abnormality or to associated multivessel coronary artery disease per se (1,5,9). While third degree AV block is associated with an increased risk for mortality, currently there are no data that demonstrate a definite reduction in the relative risk of mortality with reperfusion therapy (1).

Diagnosis of Right Ventricular Infarction
Right ventricular infarction (RVI) occurs in more than 30% of patients with acute inferior-posterior left ventricular (LV) MI, the incidence varying depending on the criteria used for its detection (10,11,16,17). Hemodynamically important RVI is an uncommon sequela of IMI with the clinical triad of hypotension, clear lung fields, and elevated jugular venous pressure occurring in less than 10% of patients presenting with acute IMI (16,17). With volume loading, the hemodynamic signs of RVI can be induced in an additional 20% of patients. The incidence of RVI in different series will vary depending upon the noninvasive or invasive indicators used for diagnosis (18–22). In various studies ST-segment elevation in the right-sided precordial leads; the presence of depressed RV global and regional function by radionuclide or 2D-echocardiographic techniques; or the presence of RV uptake of technetium pyrophosphate during infarct avid imaging have been used as diagnostic criteria (10,11,22). During hemodynamic measurements, a mean right atrial pressure ≥10 mm Hg, a right atrial to left atrial mean pressure ratio of >0.86 and a systolic pressure ≤100 mm Hg have been used to indicate the presence of significant RVI (16,24).

Reduced Susceptibility of the RV to Infarction
Importantly, many right coronary artery occlusions do not result in significant RV necrosis and dysfunction (11). This variance with left coronary artery stenoses may be due in part to the lesser RV myocardial oxygen demands during an acute coronary artery occlusion to the lower pressure, thinner walled ventricle; the greater systolic-diastolic flow ratio in coronary vessels perfusing the RV; an increased ability of the RV to extract more oxygen during hemodynamic stress; and the presence of extensive collateral vessels that protect the RV from irreversible ischemic damage (11). In detailed postmortem studies three-quarters of all cardiac specimens with RV necrosis had a >75% stenosis of the left anterior descending coronary artery, suggesting that diminished left or right collateral blood flow may be involved in the pathogenesis of RVI (25). However, RVI often occurs in patients with only right coronary artery stenosis. A recent retrospective study by Shiraki et al. (26) of 113 patients with a first acute MI due to right coronary artery occlusion demonstrated a significant association between preinfarction angina and a lower incidence of RVI. Preinfarction angina was an independent predictor of the absence of RVI (ST-elevation in V4R) in patients with acute IMI. Also, patients with preinfarction angina tended to have a more developed collateral circulation than those without angina.

*Editorials published in Journal of the American College of Cardiology reflect the views of the authors and do not necessarily represent the views of JACC or the American College of Cardiology.

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Despite the potentially life-threatening acute hemodynamic effects, most patients with ischemic RV dysfunction during acute IMI have spontaneous early hemodynamic improvement and subsequent recovery of RV function regardless of the patency of the infarct-related vessel. Nevertheless, RVI is associated with an increased risk of early morbidity and mortality in most studies (10,11,27,28).

The RV ejection fraction increases significantly in the recovery period in survivors after acute RVI and the response of the RV ejection fraction during exercise is well preserved in patients 1–3 years after RVI (29). This remarkable capability of the low pressure, thinner RV to regain systolic function after MI explains the excellent short and long-term prognosis in many patients with RVI.

Reperfusion Therapy for RVI

In this issue of the Journal, Zeymer and associates (30) describe the prognostic impact of RVI in streptokinase-treated patients with acute IMI stratified to small or large “infarct size” using the sum of the ST-segment elevation from the 12 lead ECG. Of 522 patients with acute inferior IMI, ST-segment elevation of ≥0.1 mV in lead V_{4R} was present in 169 patients (32%). The higher 30-day cardiac mortality rates in patients with RVI were related to the larger infarct size and not to the presence or absence of RV using multivariate analysis. In this large multicenter trial, the intensity of the “ischemia” as measured by the sum of the ST-elevation at baseline was greater in patients with RVI, both in all patients and in those with large ST scores. The peak level of creatine kinase (from multiple enrolling sites) was statistically higher with RVI in all groups. In 69 patients with RVI undergoing coronary arteriography 90 minutes after the initiation of thrombolytic therapy, the infarct artery was the right coronary artery in all but two. The authors conclude that reperfusion therapy in IMI patients presenting with small ST-elevations in the 12 lead ECG, and thus a “small extent of infarction” probably is not indicated whether or not RVI is present unless advanced heart block or hemodynamic instability indicates a larger infarct. However, there was no noninvasive assessment of LV or RV size or function in these patients, only those eligible for thrombolytic therapy were included, and all had thrombolytic therapy within 6 hours of symptom onset.

In a relevant recent study using primary coronary angioplasty, Bowers and associates (31) performed echocardiographic studies before and after angioplasty in 53 patients with acute RVI. RVI was defined by 2-D echocardiographic evidence of ischemic RV dysfunction utilizing the combined presence of RV free-wall dysfunction, RV dilatation and depressed global RV function in patients with ST-segment elevations in leads II, III and AVF. These 53 patients were selected from a total of 290 patients with IMI who were screened. The patients presented 1.3–12.4 hours after the onset of symptoms. Single-vessel disease was present in 29 patients; the culprit lesion was in the right coronary artery in all cases and proximal to the major RV branches in 94%. Reperfusion of coronary blood flow in the major RV branches was successful in 41 patients (77%) and 12 patients had no or incomplete reperfusion with persistently impaired RV flow. Before angioplasty, all patients had severe inferior LV dysfunction but overall LV ejection fraction averaged 50 ± 8%. Untoward in-hospital events occurred more commonly in patients without successful reperfusion. However, the 58% mortality rate in those with incomplete reperfusion is very high, even for those with medically treated RVI. All five patients with unsuccessful reperfusion who survived to discharge had no subsequent evidence of right heart failure. Although this study supports the use of primary coronary angioplasty in patients presenting early after IMI who have echocardiographic evidence of RVI, it compares a relatively small number of patients with incomplete reperfusion (recurrent intracoronary thrombus or flow-limiting dissections in several) to patients who had successful reperfusion of the right coronary artery and its right ventricular branches.

The various different methods used to determine the presence or absence of RVI; the apparent relative resistance of the RV to irreversible myocardial ischemia; and the failure to randomize patients with unequivocal RVI to thrombolytic or primary angioplasty versus nonreperfusion therapy makes it difficult to determine which patients with IMI and RVI are likely to do better with reperfusion therapy as compared to a more conservative management. There is little information concerning patients with inferior and RV infarction who present after the first 12 hours; likely, they will do well considering the often spontaneous resolution of RV dysfunction in such patients.

Based on the available information it would seem prudent that all patients with evidence of an acute IMI have right-sided precordial ECG leads recorded. Patients with evidence of RVI by ECG criteria or by other noninvasive measures who have clinical evidence of depressed RV function are candidates for coronary angioplasty (if available), thrombolytic therapy (if not contraindicated), or volume and dobutamine infusion (if necessary) when reperfusion therapy is unavailable or contraindicated. Recent studies indicate that thrombolysis or primary angioplasty in the first 6 hours after IMI may improve the short-term survival in many patients with associated RVI.

Randomized trials of thrombolysis versus primary coronary angioplasty in patients with RV infarction who are not in cardiogenic shock would further our knowledge concerning the treatment of RVI and a comparison with RVI patients treated more conservatively would be useful in determining the cost effectiveness of different treatment strategies for patients with RVI.

References


