Management of Acute Myocardial Infarction With Hypotension*

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Abbreviations: ICD = cardioverter-defibrillator; LV = left ventricular; MI = myocardial infarction; RCA = right coronary artery; RV = right ventricular; RVMI = RV myocardial infarction; SVR = systemic vascular resistance

CASE PRESENTATION

A 75-year-old man experienced the sudden onset of severe substernal chest pain, dyspnea, and near syncope beginning at approximately 10:30 AM. He was brought to the hospital emergency department, where he was found to be dyspneic, diaphoretic, hypotensive (BP, 90/65 mm Hg), and bradycardic (heart rate, 55 beats/min). A cardiac examination disclosed normal precordial activity and faint heart sounds, with no detectable murmurs or diastolic sounds. The lungs contained bibasilar moist rales. The remainder of the general examination was normal. ECG on admission (Fig 1) disclosed junctional rhythm, with findings consistent with acute posterolateral myocardial infarction (MI).

The patient had been recently hospitalized elsewhere for congestive heart failure and discharged about 1 week before this episode. An echocardiogram 6 months ago had disclosed left ventricular (LV) hypertrophy with reduced global systolic function (ejection fraction approximately 35%). There was a long-standing history of hypertension and renal insufficiency (most recent BUN, 57 mg/dL; creatinine, 4.0 mg/dL). He had been a heavy cigarette smoker for many years.

After a brief discussion with the patient about the options of treating his coronary event (cardiac catheterization with attempts to mechanically revascularize vs pharmacologic intervention with thrombolytic agents), the first alternative was selected, and cardiac catheterization was performed within approximately 2 h of symptom onset. Coronary cineangiography disclosed the total occlusion of both the proximal circumflex and right coronary arteries (RCAs). The left main and left anterior descending coronary arteries were widely patent, with the latter containing minor luminal irregularities. Both occluded segments were dilated, and stents were then implanted.

Within the next 48 h, his blood creatine phosphokinase level reached a peak of 4,000 U, with an MB fraction > 300. For the first 4 days of hospitalization, hemodynamic stabilization was achieved; subsequently, the patient developed recurrent episodes of sustained ventricular tachycardia in the presence of persistent second degree (Mobitz type 1) heart block.

QUESTIONS FOR CONSULTANT

1. Please give your interpretation of the initial ECG, including your thoughts about the meaning of ST depression in the anterior and lateral leads, and how the various changes help in determining which coronary arteries are occluded. How does one determine the presence of RV involvement in the infarction process?
2. With regard to thrombolytic agents vs direct mechanical intervention, where do each of these measures fit into the management of acute MI in the present era? Does the presence of coexistent hypotension or RV involvement modify your approach?

3. What are the preferred therapeutic options for cardiogenic shock, pharmacologic and otherwise?

4. Given the presence of recurrent ventricular tachycardia, what criteria do you employ in deciding when to perform electrophysiologic testing? Under what circumstances would an implantable pacemaker-defibrillator be preferred? How do you decide which pharmacologic agents to use for the ventricular dysrhythmia, and when to use them?

**Comments By Consultant**

R. Joe Noble, MD

The ECG is quite interesting. The rhythm is either low-voltage atrial fibrillation or atrial arrest with a slow junctional escape rhythm. The pattern is that of right bundle-branch block with marked ST-segment elevation in the inferior and lateral precordial leads, with equally impressive ST-segment depression in the right and mid precordial leads and aVL.

In this setting of acute inferior infarction, manifested by the marked ST-segment elevation in the inferior leads, one should attempt to identify the “culprit” coronary artery, and, if possible, the location of the occlusion within that artery. Specifically, is the right coronary or the left circumflex coronary artery involved? And, if the RCA is involved, is the occlusion proximal to or beyond the origin of an RV marginal branch?

When the RCA is occluded proximal to the origin of a large RV marginal branch, RV myocardial infarction (RVMI) is generally part of the clinical syndrome. Electrocardiographically, that is generally manifested by ST elevation or, at least, the absence of ST-segment depression in lead V₁. The present ECG does not show this pattern, and thus we do not have ECG evidence of RV involvement.

The ST-segment depression anteriorly is likely a reciprocal change to ST-segment elevation posteriorly, indicating posterior infarction. The ST-segment elevation in V₂ and V₆ indicates lateral wall involvement as well. The lateral wall and posterior wall involvement could reflect the occlusion of a large RCA or a large left circumflex coronary artery. When the circumflex artery is responsible for the lateral infarction, there is generally ST elevation (or at least no ST depression) in lead aVL. The absence of ST elevation in aVL would favor the occlusion of the RCA, rather than the left circumflex artery, as the culprit.

Using those rules (the absence of ST elevation in V₁ [excluding RCA occlusion proximal to an RV marginal branch], and the absence of ST elevation in aVL [excluding the left circumflex]) we are left with occlusion of the RCA distal to the origin of the RV marginal branch. The only problem with that conclusion is that the infarction appears clinically larger than would be anticipated with a distal RCA occlusion; in addition, the impressive ST elevation in the lateral precordial leads favors circumflex occlusion.

This combination of findings is probably explained by the simultaneous occlusion of both arteries (the RCA and the left circumflex artery), as described in the case presentation.

Certain clinical features (the hypotension, bradycardia, and heart block) would all be compatible with...
RVMI. One is thus led to question the specificity of the ST-segment depression in V₁ in excluding this possibility. Of course, the ST-segment orientation in V₁ actually results from two possible opposing forces: ST elevation due to RV involvement vs ST depression as a reciprocal to true posterior wall involvement.

One could argue that with the simultaneous occlusion of both the right and the left circumflex vessels, that the right ventricle is, in fact, involved, implying that posterior ST elevation prevails, thus explaining the anterior ST-segment depression. However, one additional hemodynamic factor seems to exclude RVMI: the pulmonary hypertension. An infarcted right ventricle would probably be incapable of generating systolic pressures in the 55 to 60 mm Hg range. Indeed, the pulmonary hypertension and elevated pulmonary capillary wedge pressure are likely a reflection of the preexistent LV dysfunction coupled with the additional, substantial acute LV injury.

RVMI could also be excluded with the recording of isoelectric ST segments in right precordial leads V₃ to V₆, or by echocardiographic evidence of a normal size, normally contracting right ventricle.

Thrombolysis and direct mechanical intervention (ie, percutaneous transcutaneous coronary angioplasty) are nearly equally efficacious in most patients with acute MI. One’s preference is generally determined by the situation: If an expert interventionalist is available with an open, operating laboratory, I strongly prefer acute angioplasty. If any delay were necessitated by the availability of the operator or the laboratory, then thrombolytic therapy would be preferred. Trials comparing the two approaches tend to favor intervention.

On the other hand, the prognosis of cardiogenic shock due to acute MI is extremely poor, and most clinicians believe that the only hope for survival lies with acute mechanical intervention. There are no large randomized trials to confirm this clinical suspicion, but the SHOCK trial (not yet published) supports the anecdotal clinical experience.

The same may be said for the management of patients with acute RVMI, particularly with hemodynamic compromise. Recent randomized trials support mechanical intervention in this setting.

The pharmacologic management of systemic hypoperfusion is challenging. The goal is to improve the cardiac output while lowering the elevated pulmonary capillary wedge pressure. The means by which this goal is accomplished depends, in large part, on the systemic vascular resistance (SVR).

A patient this ill certainly requires hemodynamic monitoring with a pulmonary arterial catheter. If the BP and SVR were elevated (which they are not, in this patient with hypotension), one could begin with a pure vasodilator, such as nitroprusside or an angiotensin-converting enzyme inhibitor, and adding dobutamine or milrinone, a phosphodiesterase inhibitor, if necessary to improve the cardiac output.

With the initially low SVR and BP that this patient demonstrates, dobutamine may augment the cardiac output without further depressing the BP. The addition of milrinone may well be necessary as a positive inotrope and vasodilator, in order to decrease pulmonary capillary wedge pressure. A vasoconstricting dose of dopamine or norepinephrine will be required (if the systemic BP remains unacceptably low), or nitroprusside or an angiotensin-converting enzyme inhibitor (if the pulmonary capillary wedge pressure remains high).

Despite the logic of these approaches, the fact is that if serious hypotension persists, pharmacologic agents, alone, will not reverse this hemodynamic state; mechanical support, such as that provided by intraaortic balloon counterpulsation, will often be necessary.

Of more importance, however, is the immediate revascularization of the occluded vessel whenever possible. The rhythm is quite worrisome for several reasons. First, ventricular tachycardia not only appeared but increased in frequency following the second post-infarction day. Secondly, the patient has preexistent and significant LV dysfunction that increases the risk of sudden cardiac death associated with ventricular tachycardia. Third, the rhythm, itself, is malignant, in the sense that the ventricular tachycardia is rapid, symptomatic, and sustained. For all of these reasons, the patient’s future likelihood of sudden death is considerably heightened, and aggressive therapy is mandated.

There are two approaches to the prevention of sudden arrhythmic death: pharmacologic and interventional. One could perform electrophysiologic studies to learn the inducibility of the ventricular tachycardia and, having done so, learn its suppressibility with an antiarrhythmic agent, preferably amiodarone. Alternately, one could simply provide amiodarone in a loading dosage, and monitor the rhythm, to learn if it is effective in suppressing recurrences of the arrhythmia. Preferably a cardioverter-defibrillator (ICD) could be implanted, and this would best be accomplished with an electrophysiologic study to characterize the ventricular tachycardia, in order to employ the pacemaker antiarrhythmic device.

Precise guidelines for distinguishing between the amiodarone and the ICD route are not yet available. In the BASIS Trial, amiodarone improved survival in patients with complex arrhythmias post MI. In the Canadian (CAMIAT) and European (EMIAT) Tri-
als, amiodarone reduced arrhythmic deaths (but not all cause mortality) in post-MI patients.\textsuperscript{7,8} And, in a meta-analysis, amiodarone decreased the risk of death significantly in post-infarction patients.\textsuperscript{9} However, the risk was certainly not reduced to zero, and, as mentioned, all cause mortality was not substantially altered.

There is no question that arrhythmic mortality will be improved by the implantation of an ICD in a patient such as this (post MI with LV dysfunction).\textsuperscript{10,11} We will have to await the results of certain trials (MADIT-II and SCD-HEFT) for more definitive information. However, the initial reports from the MUSTT (Multicenter Unsustained Tachycardia Trial; not yet published) provide compelling support for a defibrillator, and that is what I would strongly favor. Amiodarone would be added in an effort to reduce the number of recurrences.

**Follow-Up Information**

In an effort to control the episodes of ventricular tachycardia, IV amiodarone was administered in a loading dose and followed by 400 mg/d po. An electrophysiologic study was subsequently performed, disclosing inducible sustained ventricular tachycardia and fibrillation despite this medication. In response to these findings, an automatic defibrillator with pacemaker capability was implanted on the following day. Amiodarone, 200 mg/d, was also continued. Prior to discharge from the hospital, a radionuclide ventriculogram demonstrated mild global LV hyokinesis with an ejection fraction of 42%. Approximately 2 weeks after release from the hospital, there was a spontaneous discharge of the implanted defibrillator, necessitating an increase of the amiodarone dosage to 300 mg/d. His clinical status has remained stable to the current date (6 months after release from hospital), and there have been no further discharges of his defibrillator. Subsequent electrocardiograms have demonstrated predominately sequential atrioventricular pacing as a result of the continued high-grade atrioventricular conduction abnormality.

**References**

5. O’Rourke RA. Treatment of right ventricular infarction: thrombolytic therapy; coronary angioplasty or neither? J Am Coll Cardiol 1998; 32:882–884