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Role of Coronary Angioplasty in Acute Myocardial Infarction

Susheel K. Sharma, MD,* Kenneth Retter, MD,† and Fareed Khaja, MD‡

Aggressive approach in the form of thrombolysis and percutaneous transluminal coronary angioplasty (PTCA) has revolutionized the management of acute myocardial infarction (MI). Thrombolysis has emerged as the single most effective strategy to have favorably modified the natural history of acute MI. There now exists overwhelming evidence highlighting the beneficial effects of intravenous thrombolysis both in terms of limiting myocardial damage and improving survival (1-4).

PTCA has evolved simultaneously and been used in the management of acute MI (5,6). Whereas the guidelines for thrombolysis in acute MI are well established (7), considerable controversy exists as to the appropriate place of PTCA in the management of acute MI. We review the status of PTCA in acute MI, in light of available information from several recent randomized controlled trials, and attempt to establish pragmatic guidelines for its use in acute MI (8-10).

Pathophysiology of Acute MI and Rationale for Intervention Strategies

Ample evidence has confirmed atherosclerotic plaque fissuring followed by thrombus formation as the underlying pathologic process causing coronary artery occlusion resulting in acute MI. DeWood and colleagues (11) demonstrated occlusive thrombus in 86% of patients undergoing angiography within 4 hours of the onset of acute MI. Coronary occlusion results in myocardial ischemia, leading to ventricular dysfunction and myocardial necrosis. Restoration of antegrade flow during this early period of acute MI can arrest the progression of myocardial cell death and salvage function in jeopardized myocardium. This can be achieved chemically with thrombolysis and mechanically with PTCA.

PTCA: Definition of Terms

PTCA strategies can be divided into two broad groups:

1. Primary PTCA (PTCA without thrombolysis): This approach involves direct PTCA for acute MI as first-line therapy without initial thrombolysis.

2. Combination therapy: Under this strategy all patients at the time of presentation with an acute MI are first treated with thrombolysis. PTCA is integrated in this approach as prophylactic PTCA, salvage PTCA, or elective PTCA:
   A. Prophylactic PTCA (immediate PTCA following thrombolysis): This aggressive approach involves thrombolysis combined with immediate PTCA at the time of presentation for all patients with an acute MI who demonstrate significant residual stenosis following thrombolysis, regardless of symptoms. The rationale for this approach is that despite successful thrombolysis, the majority of patients are still left with an open but significantly stenosed coronary artery. PTCA is performed to open up the residual stenosis.
   B. Salvage PTCA (for failed thrombolysis): This approach involves PTCA only if the patient demonstrates failure of thrombolysis clinically either by having persistent ischemia in the form of ongoing chest pain or by hemodynamic deterioration such as worsening left ventricular failure or evolving cardiogenic shock (12,13).
   C. Elective PTCA (for postinfarction or provokable angina): This conservative approach involves thrombolysis as the initial therapy at the time of presentation with acute MI. PTCA is reserved only for patients who manifest ischemia later on during the hospital course, either in the form of postinfarction angina or provokable ischemia by exercise stress testing.

Various situations where PTCA can be considered in acute MI are schematically depicted in Fig 1.

Primary PTCA

Introduced by Hartzler and associates (6) in 1983, this approach involves direct transfer of the patient with an evolving acute MI to the cardiac catheterization laboratory without initial thrombolysis. Cardiac catheterization is performed to identify the infarct-related coronary artery, and mechanical reperfusion is achieved by the use of a guide wire and balloon.

Advantages of primary PTCA are:
1. It can be used for patients who have a contraindication to receiving thrombolytic therapy.
2. It has a higher recanalization rate. The recanalization rate with primary PTCA is 99% in patients with single-vessel disease (14) and 90% in patients with multivessel disease (15), compared to a 70% recanalization ceiling rate achieved with most available intravenous thrombolytic agents.

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3. There is no risk of cerebral hemorrhage. The most catastrophic complication of thrombolysis with profound long-term disability is intracerebral hemorrhage. The reported incidence of intracerebral hemorrhage is 0.1% to 0.2% with streptokinase (1,2), 0.5% with tissue plasminogen activator (16), and 0.4% with anisoylated plasminogen streptokinase activator complex (17). There have been no reports of intracerebral bleeding after primary PTCA therapy.

Disadvantages of primary PTCA are:

1. There are several logistical constraints. The most obvious problem is to get the patient in the cardiac catheterization laboratory within 4 to 6 hours of the onset of symptoms. This also requires the availability of facilities and skilled personnel able to perform expeditious PTCA. If applied on a large scale, the cost of the procedure may become prohibitive.

2. The high reocclusion rate (13%) following primary PTCA negates the initial gain and requires a repeat PTCA.

3. There are also some technical problems. Prompt and sudden mechanical reperfusion with primary PTCA has been associated with complicating arrhythmias and reperfusion injury especially in patients with inferior wall MI (18). Distal embolization of the thrombus can compromise collaterals. Although initial recanalization of the infarct-related artery is achieved in a high proportion of cases, due to an unstable evolving situation the artery may close again despite full anticoagulation and intracoronary nitroglycerin administration. This open-close-open artery cycle may sometimes be prolonged before final success is achieved.

Despite its disadvantages, primary PTCA is a useful approach to consider in the following specific situations:

1. When a patient presents with cardiogenic shock.

2. If associated conditions such as recent major surgery, gastrointestinal bleeding, recent stroke, or refractory hypertension contraindicate the use of thrombolysis.

3. When the diagnosis of acute MI is uncertain, such as in patients with a convincing history of acute MI but uninterpretable ECG due to left bundle branch block. The risks of thrombolysis may outweigh its benefits in this situation if the diagnosis turns out to be incorrect.

4. When patients with known coronary anatomy develop acute MI in-hospital at a time when rapid access to the catheterization laboratory supported by skilled operators experienced in performing expeditious PTCA is available. However, initial treatment with intravenous thrombolysis is still advised as preparations are made to transport the patient to the catheterization laboratory.

Wider application of primary PTCA beyond specific situations should wait until further information is available from large-scale trials proving its superiority over thrombolytic therapy.

**Lessons From Randomized Trials**

The impetus for combining thrombolysis therapy with PTCA stemmed from the hypothesis that the two-way approach of clot dissolution and plaque compression should achieve better revascularization with minimal residual stenosis.

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**Fig 1**—During a MI there usually is total occlusion of one of the three major coronary arteries. This can be opened either by primary PTCA or by intravenous thrombolysis. Successful thrombolysis frequently results in a high-grade residual stenosis that may cause ischemia. This can be treated by prophylactic or elective PTCA (see text). If thrombolysis is unsuccessful, the artery remains closed. This situation may require salvage PTCA.

The goal of three significant trials—the Thrombolysis and Angioplasty in Myocardial Infarction study, the European Cooperative Study, and the Thrombolysis in Acute Myocardial Infarction phase 2 trial—was to clarify the role of PTCA following thrombolysis. Each trial was different in design. The results of these three randomized trials are not encouraging for the routine immediate PTCA strategy, even though patients were entered early after symptom onset when the potential for myocardial salvage was the greatest (Table). Although the reinfarction and reocclusion rates were similar, the in-hospital mortality, hemorrhagic complications, and the need for emergency coronary bypass grafting were greater in patients randomized to the immediate PTCA strategy. Also, the residual stenosis noted within 24 hours of receiving thrombolytic therapy may improve over the next 24 hours and may not be severe enough to require PTCA. Thus, routine immediate PTCA following thrombolysis for acute MI has no additional benefit and may in fact be harmful.
Salvage PTCA for Failed Thrombolysis

Salvage PTCA is a fallback measure for failed thrombolysis, rather than a primary strategy. Unfortunately, there are currently no definite noninvasive means of identifying patients who fail thrombolysis. Certain clinical situations such as persistent chest pain, hemodynamic deterioration in the form of worsening heart failure, or cardiogenic shock suggest failed thrombolysis and necessitate salvage PTCA. Undertaken in the immediate period following acute MI, salvage PTCA carries greater risk in terms of higher acute complication rate.

Elective PTCA for Postinfarction Angina or Provocable Angina

Elective PTCA for postinfarction angina or a positive exercise stress test offers several advantages over immediate PTCA. The procedure is not only safer but bleeding complications are minimized since PTCA is performed at a time distant from thrombolysis. A substantial proportion of patients may remain asymptomatic or may recanalize and therefore PTCA can be appropriately avoided. In terms of technical feasibility and success rates, both strategies are comparable. One potential disadvantage of this approach is denying the benefit of salvage PTCA for failed thrombolysis to a subgroup of patients in whom failed thrombolysis is not manifested clinically. Some evidence suggests that an open infarct-related artery favorably affects ventricular remodeling, functions as a conduit to collateral channels, enhances healing, and prevents postinfarction arrhythmias. However, it is not yet established whether salvage PTCA for this subgroup improves survival.

PTCA for Acute MI: The Henry Ford Hospital Experience

The Henry Ford Hospital experience of PTCA in acute MI (patients presenting within 24 hours of the onset of symptoms) consists of 96 consecutive patients seen between June 1984 and June 1991. PTCA was done in the infarct-related artery only in patients with persistent chest pain despite full medical treatment, those in whom thrombolysis failed, or in those with a contraindication to the use of thrombolytic therapy. Mean time to PTCA from the onset of chest pain was 9.9 hours (range 1 to 24 hours). The overall success rate of PTCA was 90% (86 of 96 patients). Thrombolytic therapy was given intravenously prior to PTCA in 19 (20%) patients and intracoronary during PTCA in 34 (35%) patients. There were 40 (42%) anterior, 47 (49%) inferior, and 9 (9%) lateral wall infarctions.

In terms of complications there was only one PTCA-related death and no emergency surgery. PTCA was unsuccessful in 10 (10%) patients. Long-term follow-up through July 1991 (mean 42 ± 23 months) is available in 79 (82%) patients. Of these 79 patients, eight have died, four of a definite cardiac cause, and 71 (90%) are alive. Of these 71 patients, 56 (71%) are asymptomatic and eight (10%) have minimal symptoms. Nine (11%) patients have moderate angina. Of the patients who underwent repeat cardiac catheterization for symptoms, 13 (16%) had restenosis of the previously angioplastied artery. Of these 13 patients, eight had repeat PTCA, two had elective coronary artery bypass grafting, and the remaining three were treated medically. Our experience suggests that PTCA can be performed with a high degree of success, low complication rate, and good long-term results in properly selected patients with acute MI.

Recommendations

Based on the results of the trials discussed as well as our own experience, we propose a practical approach to PTCA in acute MI (Fig 2). All patients with suspected acute MI who present within 6 hours of the onset of chest pain and have no contraindication to thrombolysis should receive intravenous streptokinase. If the diagnosis of acute MI is uncertain or a contraindication to thrombolysis exists, primary PTCA should be considered. This recommendation is operative only when it is suspected that a large amount of myocardium is at risk and the timing is favorable for immediate mobilization of facilities and personnel.

If the patient becomes unstable at any stage following thrombolysis, i.e., develops ongoing ischemia or cardiogenic shock or medically uncontrollable heart failure, cardiac catheterization with a view to salvage PTCA should be considered. Stable patients should be observed closely. If a predischarge submaximal

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<td>Immediate Versus Delayed PTCA Following Thrombolysis</td>
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TIMI = Thrombolysis in Myocardial Infarction trial (8), ECSG = European Cooperative Study Group (9), TIMI-2A = Thrombolysis in Myocardial Infarction phase 2A trial (10), CAGB = coronary artery bypass grafting, LVEF = left ventricular ejection fraction.
exercise stress test is positive in asymptomatic patients, cardiac catheterization should be performed. All other patients may be treated medically and followed, preferably with a repeat exercise stress test at six weeks. A positive stress test is an indication for cardiac catheterization with a view to PTCA.

References


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Management of Cardiogenic Shock Complicating Acute Myocardial Infarction: The Henry Ford Hospital Experience and Review of the Literature

Ali R. Moosvi, MD,* Mihai Gheorghiade, MD,† Sidney Goldstein, MD,‡ and Fareed Khaja, MD++

Cardiogenic shock complicating acute myocardial infarction (MI) carries a high mortality which in some series prior to 1980 exceeded 80%. Neither the use of inotropic and vasopressor agents nor intraaortic balloon counterpulsation was found to improve survival in this group of patients. Intravenous thrombolytic agents improve survival in patients with acute MI, but their role in cardiogenic shock is unknown. Reports of the use of surgical and mechanical interventions in patients with severe left ventricular dysfunction were examined to determine if there was any benefit to be derived from restoring blood flow to ischemic areas of the myocardium. It was found that urgent placement of intraaortic balloon counterpulsation followed by coronary bypass surgery may improve survival rates and successful coronary angioplasty also appeared to benefit patients with cardiogenic shock. Similar improvement in survival has been reported after successful coronary reperfusion. In surgical series with predominantly nonmechanical causes of shock, survival has varied from 40% to 88%. Data from our five-year experience in the management of MI patients with cardiogenic shock suggest that coronary revascularization with coronary angioplasty or bypass surgery improves survival in patients with cardiogenic shock especially when performed within 24 hours of the onset of shock. (Henry Ford Hosp Med J 1991;39:240-4)

A acute myocardial infarction (MI) is complicated by shock in up to 15% of patients and carries a high mortality, exceeding 80% (1-6). After the introduction of coronary care units, even though overall mortality decreased in MI, only 19% of patients with shock survived (7).

Definition
There is no consistent definition of cardiogenic shock in the literature. Some earlier reports defined shock only on the basis of hypotension (systolic blood pressures below 90 mm Hg), while others insisted on the presence of peripheral circulatory collapse (1-22). Cardiac index and wedge pressures have not been included as part of the definition, even in more recent studies, because not all patients in these studies had Swan-Ganz catheterization performed (23-25). In our study a patient was considered to be in cardiogenic shock when: 1) arterial systolic blood pressure was less than 80 mm Hg without drugs or intraaortic balloon pump support or less than 90 mm Hg with such support; 2) there was no evidence of hypovolemia (pulmonary capillary wedge pressure greater than 12 mm Hg) and cardiac index was low (< 2.2 L/min/m²); and 3) the patient demonstrated clinical evidence of hypoperfusion (26).

Medical Therapy
Conventional medical therapy does not influence survival in patients with cardiogenic shock (8-22). Although inotropic agents, such as isoproterenol, produce initial improvement of hypotension in some patients with shock, they have not been shown to improve survival. Failure is presumably due to the positive inotropic effect being achieved at the expense of increased myocardial metabolism (12-15). Norepinephrine is considered to be superior to isoproterenol in the treatment of patients with shock because it increases arterial blood pressure by increasing systemic vascular resistance and, unlike isoproterenol, it improves myocardial lactate extraction and coronary perfusion (12,13). Binder et al (11) showed a small improvement in survival with norepinephrine. Similarly, digitalis does not produce any significant change in cardiac output, arterial pressure, or systemic vascular resistance. This lack of hemodynamic benefit may be due to the fact that cardiogenic shock is initiated by the loss of a large amount of myocardium (18). Vasoreactive, inotropic agents like dobutamine and dopamine have been utilized for the management of shock, but no benefit in survival has been demonstrated (8,18-20).
Intraaortic Balloon Counterpulsation

In a multicenter trial, Scheidt et al (27) studied the effects of intraaortic balloon counterpulsation in 87 patients with cardiogenic shock and demonstrated hemodynamic improvement. However, survival was not improved. A recent preliminary report by Waksman et al (28) suggested that survival was better in patients treated with balloon counterpulsation. However, in this report coronary revascularization was performed in 67% of patients. Thus, it appears that balloon support combined with revascularization, rather than balloon support alone, improves survival.

Thrombolytic Therapy

Intravenous thrombolytic agents have been shown conclusively to decrease mortality from MI except in patients with severe heart failure. In the latter, mortality was 70% in both streptokinase and placebo groups (29-32). In a study of 44 patients with cardiogenic shock, Kennedy et al (33) reported survival of 58% of patients with successful reperfusion compared to 16% when reperfusion with intracoronary streptokinase was unsuccessful.

Coronary Artery Bypass Surgery

In 1980, DeWood et al (34) demonstrated improved long-term survival in 19 patients treated with urgent coronary bypass surgery. Although this was a small, uncontrolled study, the results suggested that 1) early revascularization salvaged jeopardized myocardium and improved survival, and 2) surgery was feasible and indeed advantageous in a patient population considered to be “too sick for surgery.” In other reports which included patients with predominantly nonmechanical causes of shock, revascularization resulted in rates of survival from 40% to 88% (35-42).

There are several limitations in the surgical data on cardiogenic shock. First, surgical series are subject to considerable selection bias and not all patients with shock undergoing angiography were considered eligible for surgical revascularization. Second, it is difficult to compare various studies since the percentage of patients with surgically correctable mechanical causes, such as ventricular septal rupture or papillary muscle rupture, was based on different reports. Third, the definition of shock is not uniform in these studies. Fourth, like the medical series, surgical studies are small and nonrandomized.

Percutaneous Transluminal Coronary Angioplasty

Coronary angioplasty can be performed in acute MI with reperfusion rates from 70% to 90% (43,44). Lee et al (45) studied the effect of coronary angioplasty on survival in 24 patients with cardiogenic shock following acute MI. The 30-day survival was 77% in patients with successful angioplasty versus 18% in those whose angioplasty was unsuccessful. However, in this study none of the patients admitted between 1975 and 1982 were candidates for coronary angioplasty and were therefore treated conventionally. They served as historical controls for patients admitted between 1983 and 1985, all of whom were candidates for coronary angioplasty. Similarly, other studies have reported in-hospital survival of 59% to 71% when shock patients were treated with either primary angioplasty or the combination of successful thrombolysis and bypass surgery (23,24,46).

Henry Ford Hospital Study

The effects of revascularization by coronary angioplasty or bypass surgery on survival were evaluated in 81 patients with cardiogenic shock complicating acute MI admitted from January 1985 to May 1990 (26). Patients with MI in whom shock was secondary to mechanical causes, such as ventricular septal rupture or papillary muscle rupture, were excluded from the study. There were 1,346 patients admitted with confirmed acute MI during this period, of whom 81 (6%) patients with cardiogenic shock form the study population. Successful revascularization was achieved in 32 patients: by angioplasty in 22, bypass surgery in 2, and angioplasty followed by bypass surgery in 8 patients. The nonrevascularized group consisted of 49 patients; 41 had conventional medical therapy and 8 had failed angioplasty.

The clinical characteristics and hemodynamic data obtained at the onset of cardiogenic shock were similar in both groups. Ejection fraction was low in all patients but was a little higher in the revascularized patients (31% versus 25%, P = 0.04).

The overall survival rate was 27% (22 of 81 patients). However, survival was significantly better in revascularized patients (18 [56%] of 32 patients) compared to the nonrevascularized group (4 [8%] of 49 patients) (P < 0.0001). This survival difference persisted at a mean follow-up of 21 ± 15 months, being 50% in the revascularized group versus 2% in the nonrevascularized group (P < 0.0001). The mean time from development of shock to revascularization in survivors was 12.4 ± 15 hours compared to 58.5 ± 93 hours in nonsurvivors (P = 0.004). The most important conclusion was that in-hospital survival was 77% (17 of 22 patients) when revascularization was performed within 24 hours compared to 10% (1 of 10 patients) when revascularization was performed after 24 hours (P = 0.0006).

Even though our institution’s experience is a nonrandomized, prospective observational study, we believe that the groups are comparable. Except for revascularization, no other variable appeared to contribute significantly to survival. These data suggest that coronary revascularization improves survival in cardiogenic shock, especially when it is performed early.

Time From Shock to Revascularization

Just as time from onset of symptoms to administration of a thrombolytic agent in acute MI is critical for survival, a time frame applies to the management of shock by performing revascularization procedures. DeWood et al (34) emphasized that survival was 75% when intraaortic balloon counterpulsation and surgery was performed within 16 hours from the onset of symptoms compared to 29% if this was achieved later. In a report from the Mayo Clinic on 45 patients with cardiogenic shock, the in-hospital survival was 71% with successful angioplasty compared to 29% when the procedure was unsuccessful. Ejection fraction is likely to improve when primary an-
Acute Myocardial Infarction and Clinical Shock

**Resuscitative Measures**
- Fluids
- Inotropes/Vasopressors
- Ventilatory Support

**BP, Organ Hypoperfusion**

**Hemodynamic Assessment**
- Swan Ganz Catheter
- Arterial Line

**Echocardiogram**

**Classify Shock**
- Cardiogenic
- Hypovolemic
- Other
  - Dehydration
  - Blood Loss
  - Plasma Loss
  - Tamponade
  - P. Embolism
  - Arrhythmias

**BP, Organ Hypoperfusion**

**Fig 1**—Suggested management of clinical shock in acute myocardial infarction.

**Non-Mechanical**
- Ventricular Dysfunction
- Severe Ventricular Septal Rupture
- Mitral Regurgitation

**Mechanical**
- IABP

**Emergency Cardiac Catheterization**

**Early Revascularization**
- PTCA/CABG

**Surgery**

**IABP**

**Fig 2**—Suggested management of cardiogenic shock in acute myocardial infarction.

gioplasty is performed within 4 hours of symptoms in patients with cardiogenic shock (23). Hence, early recognition of cardiogenic shock and prompt intervention are of prime importance.

An outline for the diagnosis and management of patients who develop hypotension during the course of acute MI is shown in Figs 1 and 2.

Hands et al (3) reviewed the Multicenter Investigation of the Limitation of Infarct Size data base and found that enzymatic evidence of infarct extension or reinfarction occurred in 23% of patients in whom cardiogenic shock developed, compared to a 7% incidence among those in whom shock did not develop. The mechanism by which revascularization improves survival remains to be defined, as shock usually occurs several hours to days following actual total occlusion of the infarct artery. Autopsy studies have demonstrated necrosis of greater than 40% of myocardium in patients who develop cardiogenic shock (47). Such patients also show progressive necrosis with infarct extension (48-50). Left ventricular dilatation may occur early and progress over time, leading to hemodynamic deterioration and sometimes rupture (51-60). There is experimental evidence that late reperfusion inhibits infarct expansion (60). It is possible that revascularization in human cardiogenic shock may be beneficial by preventing or limiting infarct extension and expansion and left ventricular dilatation.

Predictors of shock include history of previous infarction, diabetes, age greater than 65 years, admission radionuclide ejection fraction less than 35%, and a peak creatine kinase MB determination greater than 160 IU/L (3). We propose that in the clinical setting of acute MI, these predictors should be carefully evaluated and patients with these factors be closely monitored for hemodynamic deterioration. If deterioration develops, they should be considered for emergency revascularization by percutaneous transluminal coronary angioplasty and/or coronary artery bypass graft surgery.

**References**


