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CARDIOLOGY Rounds™

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The role of angioplasty when thrombolysis fails

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The restoration of normal coronary artery blood flow early after the onset of acute myocardial infarction (MI) is associated with an improvement of both survival and left ventricular function.[1–4] Patients found to have normal (TIMI 3) flow at 90 minutes after thrombolysis have a 30-day mortality of 2.7%, in contrast to those with impaired (TIMI 2) flow or an occluded artery (TIMI 0–1 flow) who have mortality of 6.6% or 7.1%, respectively.[4] (TIMI flow grades are described in table 1.)

Thrombolysis has limited efficacy: r-tPA (recombinant tissue plasminogen activator) produces TIMI grade-3 flow in 54% of patients at 90 minutes; streptokinase produces grade-3 flow in only 30–33% of those receiving it.[2] Although new thrombolytics and anti-thrombotic agents are currently under investigation, it is likely that a core of thrombolytic failures will remain.

Failure of thrombolysis also includes early reocclusion of the infarct-related artery. After successful thrombolysis, early reocclusion occurs in approximately 15% of patients and is associated with an almost three-fold increase in mortality in comparison with those whose arteries remain patent.[5]

Rescue angioplasty is often attempted when thrombolysis fails, but is there evidence to support this course of management, and can we recognize patients who might benefit from the procedure? These rounds will focus on the recognition and management of patients with failed thrombolysis—patients in whom there is failure to restore normal blood flow within a short time from treatment.

Recognition of thrombolytic failure

As a way of identifying patients whose blood vessels have not reperfused, routine coronary angiography immediately following thrombolysis is not only impractical, but is also associated with high morbidity. How can we identify patients in whom reperfusion fails?

Relief of chest pain

Complete resolution of chest pain is associated with TIMI-2 or -3 flow within 90 minutes in more than 80% of patients.[6] However, change in chest-pain intensity is neither a sensitive nor a specific marker of arterial patency: a patent artery can be associated with persistent pain due to no reflow in the infarcted territory, and the termination or improvement of pain does not necessarily imply a patent artery.[6]

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Table 1: TIMI flow grades		
Grade	**Description**	**Angiographic findings**
0	No perfusion	No flow through the obstruction
1	Minimal perfusion	Contrast material passes beyond the area of obstruction but fails to make the entire coronary bed distal to the obstruction opaque.
2	Partial perfusion	Contrast material crosses the obstruction and makes the coronary bed distal to the obstruction opaque, but the rate of entry of contrast material into the vessel distal to the obstruction or its rate of clearance is reduced relative to the nonobstructed vessel.
3	Complete perfusion	Flow and clearance as in a normal vessel

Adapted from Rosenschein U, Roth A, Rassin T, et al. Analysis of coronary ultrasound thrombolysis endpoints in acute myocardial infarction (ACUTE Trial): Results of the feasibility phase. *Circulation* 1997;95:1411–1416.

ST-segment changes

ST-segment monitoring is useful to identify patients with failure to reperfuse after thrombolysis and to identify those at risk of reinfarction. A rapid, >50% decrease of ST-segment elevation is associated with a smaller MI and better global left ventricular function.[7] A further elevation of the ST-segment during the first hour after thrombolysis, followed by rapid decline, has been associated with early reperfusion.[8] In a TAMI study, 63% of patients with no ST-segment reduction or symptom improvement had patent infarct-related arteries at 90-minute angiography.[6] These patients probably had severe microcirculatory damage with impaired tissue perfusion despite a patent epicardial coronary artery.

In Wellens' study, improvement in ST-segment elevation by more than 25% within 90 minutes was associated with a likelihood ratio of 16.0 that the infarct-related artery had TIMI-2 or -3 flow restored.[9] Terminal T-wave inversion was found to have a likelihood ratio of 10.6 in the same study.[9] Fernandez et al showed that a >50% resolution of maximal ST-segment elevation at 60 minutes following administration of thrombolytics predicts a patent artery, with 96% sensitivity and 94% specificity.[10]

Biochemical markers

The early appearance of biochemical markers of myocardial injury has been recognized as an indication of early successful reperfusion by thrombolytic therapy. A recent study showed that, 90 minutes after thrombolysis with streptokinase, a less than ten-fold increase of either CK MB mass, myoglobin, or troponin T had 88–95% sensitivity and 49–65% specificity in predicting inability to attain TIMI grade-3 flow in the infarct-related artery.[11] The combination of clinical variables and biochemical markers further enhances the non-invasive prediction of reperfusion after MI.[12,13]

Myocardial imaging

Although myocardial perfusion imaging can be valuable in demonstrating the outcome of thrombolytic therapy, it is generally not available in emergency departments, nor is it likely to provide information rapidly enough to allow decisions to be made within 60–90 minutes after administration of the thrombolytic agent. Myocardial contrast echocardiographic imaging is a promising technique that is capable of revealing tissue perfusion after an intravenous injection of contrast medium.[14] Intracoronary contrast injection has been used to indicate restoration of tissue perfusion,[15] yet no prospective study has been completed that examines the value of intravenously administered echocardiographic contrast as a technique to predict coronary patency.

Management of failed thrombolysis

The management of the patient who has persistent chest pain and neither electrocardiographic nor biochemical evidence to suggest early reperfusion remains controversial.

Repeat thrombolysis or rescue angioplasty are two reperfusion strategies that have been attempted.

Repeat thrombolysis

The angiogram of a patient with "thrombolytic-resistant" thrombi was taken 90 minutes after the initial thrombolytic was administered, and it showed TIMI 0–1 flow. Intracoronary r-tPA (50 mg administered over 10 minutes) was able to reperfuse 48% of the coronary arteries.[16] Repeated thrombolysis has been shown to be effective when recurrent ischemia occurs early after the initial dose of thrombolytic.[17] Complete resolution of myocardial ischemia was observed in 85% of patients within one hour of the second infusion, yet almost 50% later required coronary revascularization. Furthermore, important bleeding occurred in 19% of patients, and transfusions were required in 4%.

Recurrent chest pain and ST-segment re-elevation might respond to a second infusion of r-tPA. In a study by the European Cooperative Study Group, the administration of a half-dose of r-tPA within 24 hours of the first thrombolytic, and of a full dose after 24 hours, led to resolution of the ST-segment re-elevation within 100 minutes (median 50 minutes).[18] Patency of the infarct-related artery at 60 minutes after the second dose of r-tPA appeared to depend upon whether the patient was receiving heparin: on heparin, 73% patency; no heparin, 40% patency.[18]

Rescue angioplasty

Emergency angioplasty is often requested for patients who have persistent chest pain and ST-segment elevation after thrombolysis. Although pain can be rapidly relieved by successful angioplasty in this setting, ST-segment elevation often persists, and normal flow is not always achieved despite a patent vessel. It is also unclear whether ventricular function or survival are improved by early revascularization several hours after the onset of symptoms.

There have been two randomized trials to examine the value of rescue angioplasty. In a Calgary-based study, Belenkie et al randomized 28 patients to conservative treatment or immediate percutaneous transluminal coronary angioplasty (PTCA) after angiography performed at 257±57 minutes following the onset of symptoms.[19] There was one death among the 16 patients in the rescue angioplasty arm, and there were 4 deaths in the 12 patients randomized to conservative treatment. However, the difference was not statistically significant.

The other study—RESCUE—enrolled patients with a first anterior MI who had received thrombolysis and were shown to have an occluded infarct-related artery within 8 hours of the onset of symptoms.[20] Patients with cardiogenic shock and those with left main coronary artery disease were excluded. The primary end-point of the RESCUE trial was left ventricular ejection fraction (LVEF) after 25–35 days, with a value of 20% imputed for nonsurvivors. At that time, there was no difference between LVEF in the two groups (angioplasty 40±11%, conservative 39±12%). However, the exercise LVEF was slightly higher in the angioplasty group (angioplasty 43±15%, conservative 38±15%, $P<0.04$). This difference could be the result of a decrease in EF due to myocardial ischemia in the conservatively-treated group; or it could be due to an increase in EF in the angioplasty group, recognizing the contribution of stunned myocardium.

Thirty-day mortality was 9.6% in the conservatively-treated patients and 5.1% in the angioplasty group, and the incidence of severe heart failure (NYHA classes III or IV) was 7.0% in the conservative group and 1.3% in the angioplasty group. Individually, the two endpoints did not achieve statistical significance, however, when the endpoints of mortality and severe CHF were combined, the treatment effect was significant: the conservative group 16.6% versus 6.4% in the angioplasty group, $P<0.05$.

Michels and Yusuf[21] combined the rescue angioplasty data in a meta-analysis and found an overall 5.4% mortality for the angioplasty-treated patients and 12.9% mortality for those conservatively managed. The odds ratio for death at 30 days was 0.38 with a 95% confidence interval of 0.13 to 1.06. The researchers emphasized the need for further randomized clinical trials to establish the value of rescue angioplasty.

The outcomes of non-randomized studies have been used both in support of and against the value of rescue angioplasty. In these non-randomized studies, mortality appears to be higher in the patients treated with angioplasty, and left ventricular function is not improved.[22–26] Mortality appears especially high in patients when angioplasty is not successful, and outcomes appear to be improved when non-fibrin-selective thrombolytic agents are used. Unfortunately, due to the nature of non-randomized observations, differences in the baseline status of the patient might influence outcome more than the intervention. In the observational studies, patients managed with rescue angioplasty were more likely to be diabetic, to have more severely impaired left ventricular function, and to have significant hypoten-

sion. In several studies, the patients who underwent rescue angioplasty that failed had been in a critical state, and the angioplasty was attempted as a last resort in a desperate situation.

Angioplasty failure during rescue PTCA

Failure of rescue angioplasty has been defined by several criteria:

- a failure to restore normal blood flow
- the presence of a significant residual stenosis with reduced flow
- a coronary dissection with a residual stenosis
- the failure to restore blood flow despite successfully re-opening the occluded artery (no reflow).[27]

The GUSTO-I study identified severe heart failure as a powerful predictor of failure.[22] Thrombus load and a relative resistance of thrombi to fibrinolysis might be a significant reason for rescue angioplasty to fail.[27]

The impact of the glycoprotein IIb/IIIa inhibitors in this situation is as yet unproven. However, early reports suggest that they might be beneficial.[28] Intravascular ultrasound shows that local dissection or plaque rupture can be angiographically indistinguishable from thrombus.[27] Intracoronary stenting is likely to improve outcome when a large spontaneous dissection is present, even in the presence of thrombus.[29] The long-term outcome of patients who survive to hospital discharge is similar whether patency is achieved with angioplasty or with thrombolysis.[26] This suggests that long-term outcomes can improve favorably if the incidence of failed angioplasties can be reduced.

Reocclusion of the infarct-related artery after an apparently successful rescue angioplasty ranges from 7% to 21%. After successful thrombolysis, reocclusion rates range from 5% to 11%. Both the GUSTO-I[22] and the TIMI 4[23] studies indicated that reocclusion rates were higher in the rescue angioplasty patients than in those receiving thrombolysis. The major risk for reocclusion is shock, and the consequences of reocclusion include increased major bleeding (due to need for further anticoagulation), severe CHF, and increased mortality.[26,29] This implies that significant amounts of myocardium are salvaged by the procedure.

The observational studies suggest that the complications of rescue angioplasty differ according to the vessel treated. Right coronary artery rescue angioplasty results in a greater incidence of ventricular fibrillation at reperfusion, complete AV block, severe bradycardias, and abrupt closure. The left coronary interventions are complicated by a higher incidence of cardiogenic shock, hypotension, and a greater need for intra-aortic balloon-pump support

Conclusion

Early coronary artery patency after MI is associated with improved long-term survival. Thrombolysis reduces mortality by 20–40%, and both the reduction of mortality and the subsequent improved left ventricular function are related to the presence of normal coronary flow in the infarct-related artery. The success of reperfusion can be gauged with ECG and biochemical criteria within 90 minutes of treatment.

Thrombolysis results in normal coronary flow in less than 60% of patients treated with the most aggressive thrombolytic protocol. It remains to be demonstrated whether patients failing to show criteria for successful reperfusion can benefit from immediate rescue angioplasty. Randomized studies of rescue angioplasty, in patients identified by routine early coronary angiography to have thrombolytic failure, have shown a trend for a survival benefit subsequent to the procedure; these studies, however, were underpowered and did not produce statistically significant results. Results from nonrandomized studies are difficult to interpret, as the patients managed by rescue angioplasty were sicker than those managed conservatively. A common observation in these studies was the higher mortality in patients who failed to be reperfused after angioplasty. However, these patients were much sicker before the procedure, and no conclusion can be reached to show that failed angioplasty is the cause of an adverse outcome.

There is need for a larger multicenter study to establish whether rescue angioplasty is beneficial in patients who lack noninvasive evidence of reperfusion. The use of intracoronary stenting and powerful antiplatelet agents is likely to increase the success of the procedure, and it might increase the difference between interventionally and conservatively managed patients.

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Upcoming Scientific Meetings

28-30 Apr 1999
Clinical Cardiology Management and Diagnostic Dilemmas
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Abstracts of Interest

Decision analysis model of primary angioplasty versus thrombolysis in the treatment of myocardial infarction: A southwestern Ontario perspective

SRIDHAR D, GREGOR J, MASSEL D, TEEFY P, HUQ N. LONDON, ONTARIO

Primary angioplasty (PA) has been shown to improve short-term outcomes in patients with acute myocardial infarction (AMI). The upfront costs and resource utilization with PA are greater than with thrombolysis (THR). The purpose of this study was to assess the cost effectiveness of PA as a treatment strategy for AMI in a Canadian setting. The decision analysis model considered patients presenting within 6 hours of symptom onset with THR-eligible criteria for AMI. Clinical outcome data was abstracted from published large scale randomized trials comparing THR and moderate-size randomized trials of PA and thrombolysis. Economic data assessing direct costs was acquired from the London Health Sciences Centre hospital cost database and published literature. The primary endpoint was the incremental cost per life saved at one year. The baseline analysis assumed exclusive tissue plasminogen activator (TPA) use and GUSTO trial mortality and outcome results. The baseline analysis yielded a benefit of 6 lives per 1000 patients treated for one year. The average cost per patient over one year was US$7,114 for the PA arm compared to $6,882 per patient in the THR arm. The incremental cost benefit was $38.733 per life save at one year. The incremental cost was sensitive to changes in THR utilized, stent and abciximab use for PA along with appropriate outcome assumptions. Specifically, if present Canadian stent and abciximab utilization rates of 75% and 25% respectively along with 50% utilization of streptokinase and TPA are employed in the model, the incremental cost per life saved increases to $108,920. In conclusion, PA is moderately cost effective in terms of lives saved at one year. However, the incremental cost is sensitive to important changes in Canadian-based clinical practice.

A randomized comparison of primary angioplasty and thrombolytic therapy in elderly patients with acute myocardial infarction

DE BOER MJ, ZIILSTRA F, LIEM AL, ET AL. ZWOLLE THE NETHERLANDS.

The place of primary coronary angioplasty (PA) in elderly patients (pts) with acute myocardial infarction (MI) has yet to be determined. We performed a prospective randomized trial comparing PA with 1.500.000 IU intravenous streptokinase (SK), in pts with an acute MI, ≥76 yrs and no contraindications for thrombolytic therapy. Endpoints included death, recurrent MI, stroke or a combination. Sixty pts were randomized, 32 to PA and 28 to SK. Time from admission to start of SK infusion was 30 ± 15 min. Of pts randomized to PA, 29 actually underwent the procedure with a primary success rate of 29/29 (100%), and a time from admission to first balloon inflation of 74 ± 32 min. Follow-up was obtained after 30 days.

Conclusion: In this small series of elderly pts with MI, PA seems to have a significant survival benefit when compared with streptokinase therapy.

Baseline characteristics	SK	p	PA
Age (yr)	81±4	NS	81±4
Male	17 (61%)	NS	14 (45%)
Anterior MI	14 (50%	NS	11 (34%)
Previous infarction	5 (18%)	NS	2 (6%)
Results (3 months)			
Death	7 (25%)	0.02*	1 (3%)
Bleeding	2 (10%)	NS	2 (10%)
Stroke	1 (4%)	NS	0
Recurrent infarction	1 (4%)	NS	0
Combined endpoint	9 (32%)	<0.01	1 (3%)

*Odds ratio: 10 (95% confidence interval 1.2 - 90)

Acute infarct angioplasty: Differential mortality of trial eligible and ineligible patients.

DAUERMAN HL, PINTO DS, KALON KL, ET AL. BOSTON, MA

Studies comparing thrombolysis and primary angioplasty for acute myocardial infarction (AMI) have criteria that exclude many AMI patients. To evaluate the outcome in patients eligible vs. ineligible for a study like PAMI, we examined our 1997 database of 148 AMI angioplasty patients.

Methods: All patients had ST segment elevation/LBBB. Using the PAMI Stent trial criteria, there were 76 (51.3%) trial eligible versus 72 (48.7%) trial ineligible patients. The most common reasons for exclusion were shock (33.3%), thrombolytics (26.4%), late presentation (23.5%) and intubation (13.9%) with 21 patients (29.2%) having multiple exclusions. Analysis of baseline and procedural variables showed the ineligible group to have more high risk baseline markers and less use of a new device of Reopro during the procedure (see Table). Mortality in the ineligible group was over five fold higher. Multivariate predictors of mortality were shock, procedural failure, intubation, and LBBB.

Conclusion: 1) In a heterogeneous population with AMI, half of the patients are PAMI ineligible. 2) Ineligible patients have more adverse baseline and procedural variables and a five fold higher in-hospital mortality.

	Total (n=148)	Eligible (n=76)	Ineligible (n=72)	P value*
CHF (%)	53.8	42.6	64.1	0.03
VT/VF (%)	15.5	9.2	22.2	0.05
Reopro use (%)	37.8	50	25	0.003
Stent use (%)	71.6	61.6	61.1	0.01
Mortality (%)	12.2	3.9	20.8	0.003

*for comparison of eligible and ineligible group