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Failed thrombolysis following acute myocardial infarction

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Clinical outcome following thrombolytic therapy for acute myocardial infarction (AMI) is strongly associated with patency of the infarct-related artery.[1-4] Unfortunately, thrombolytic therapy fails to restore patency in 15-50% of patients.[1,5-7] Early percutaneous transluminal coronary angioplasty (PTCA) of persistently occluded infarct-related arteries ('rescue' PTCA) may potentially improve outcome in patients with failed thrombolysis following AMI.[8]

Relationship between patency of the infarct-related artery and mortality

Survival following thrombolytic therapy for AMI is closely related to the early restoration of coronary blood flow in the infarct-related artery. Numerous studies have demonstrated a consistent relationship between mortality rates and TIMI flow grade at 90 minutes. The in-hospital mortality rate after MI is 3.9% for TIMI 3, 6.7% for TIMI 2, and 9.9% for TIMI 0 or 1 flow.[1-4,9] Since thrombolysis achieves TIMI 3 flow in less than 60% of patients,[1-3] coronary angioplasty performed early after thrombolytic therapy may improve coronary patency rates and thereby improve clinical outcomes.

Routine angiography and PTCA following thrombolysis

One possible management strategy to attempt to improve flow in the infarct-related artery and mortality is routine use of angiography and PTCA in all patients receiving thrombolytic therapy. Several randomized trials performed in the 1980s evaluated this strategy. The Thrombolysis and Angioplasty in Myocardial Infarction (TAMI) I,[11] the European Cooperative Study Group for recombinant tPA,[12] and the Thrombolysis in Myocardial Infarction (TIMI) II-A[13] studies compared immediate angiography and PTCA following thrombolysis with various conservative and delayed PTCA strategies. Routine immediate angiography and PTCA did not improve clinical outcomes in these studies and was associated with increased bleeding and a trend towards increased mortality. However, the selective use of angiography and PTCA in the subset of patients with failed thrombolysis was not specifically addressed in these studies. Furthermore, these studies were done prior to the use of coronary stenting and glycoprotein (GP) IIb/IIIa inhibitors. Patients treated with thrombolytic therapy have increased levels of platelet activation and aggregation, and are therefore predisposed to thrombotic complications during percutaneous coronary intervention.[40] GP IIb-IIIa antagonists appear to counteract this prothrombotic state.[41]

Rescue PTCA after failed thrombolysis

Rescue PTCA is performed when there is clinical evidence of failed reperfusion after thrombolysis. Only two small randomized trials have specifically studied the use of rescue PTCA, but analyses from other trials, databases, and case series have also evaluated this strategy (Table 1). Belenkie et al randomized 28 patients with a persistently occluded infarct-related artery following thrombolytic therapy more than 3 hours after the onset of AMI to either to rescue PTCA (n=16) or conservative treatment (n=12).[14] There was a nonsignificant trend for lower in-hospital mortality in the rescue PTCA group (6.3% versus 33.3%; p=0.13). Although the number of randomized patients was small, the trend toward a lower mortality supports the hypothesis that rescue PTCA may be beneficial.

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Table 1: Reported success rates and mortality in studies of rescue PTCA

First Author {Trial} (Reference No.)	Year	Rescue PTCA (N)	Controls* (N)	Success Rate‡ N (%)	Mortality Among Rescue Pts (%)	Mortality Among Control Pts (%)	P Value
Califf et al. {TAMI 5} [17]	1991	52	17	43 (82.7)	NG	NG	NG
Belenkie et al.[14] †	1992	16	12	13 (81.3)	6.3	33.3	0.13
Ellis et al. {Case Series Meta-analysis}[16]	1992	560	—	451 (80.5)	10.6	—	—
Steg et al. {CORAMI study group}[21]	1994	72	—	65 (90.3)	4.2	—	—
Ellis et al. {RESCUE}[15] †	1994	78	73	72 (92.3)	5.1	9.6	0.18
McKendall et al {TIMI I/ II Database}[20]	1995	33	100	27 (81.8)	12.1	7.0	NS
Gibson et al. {TIMI 4}[18]	1997	58	37	52 (89.7)	12.1	10.8	NG
Ross et al. {GUSTO-1 Angiographic}[19]	1998	198	266	175 (88.4)	11.1	7.9	NG
Gruberg et al.[22]	1998	21	3	20 (95.2)	4.3	0.0	NG
Garot et al. [23] §	1998	81	—	77 (95.1)	5.0	—	—
Juliard et al. [24] §	1999	50	—	47 (94.0)	4.0	—	—

NG = Not given NS = Not significant † = Randomized Trial

* Control patients considered to be patients with TIMI 0 or 1 flow in infarct-related arteries managed conservatively
‡ Successful rescue PTCA defined in most series as restoration of TIMI 2 or 3 flow and less than 50% residual stenosis.
§ Some overlap exists between the patients included in these two papers (personal communication).

CORAMI = Cohort of Rescue Angioplasty in Myocardial Infarction, GUSTO = Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Arteries trial, RESCUE = Randomized Evaluation of Salvage Angioplasty with Combined Utilization of Endpoints study, TAMI = Thrombolysis and Angioplasty in Myocardial Infarction trial, TIMI = Thrombolysis in Myocardial Infarction trial

Reproduced from Goldman LE and Eisenberg ME, *Ann Intern Med* 2000;132(7):556-565.

The Randomized Evaluation of Salvage Angioplasty with Combined Utilization of Endpoints (RESCUE) study is the larger of two trials to specifically address the issue of rescue PTCA.[15] In the RESCUE trial, 151 patients with first anterior MI, treated with thrombolytic therapy and demonstrated to have an occluded infarct-related artery (TIMI 0 or 1 flow) within 8 hours of pain onset, were randomized to either rescue PTCA (n=78) or conservative medical management (n=73). No difference in the primary endpoint of the trial – resting ejection fraction – was noted at 30 days, although there was an improvement in the exercise ejection fraction at 30 days in the rescue PTCA group (rescue PTCA 43% versus conservative 38%; p=0.04). There were also trends towards lower 30-day mortality (5.1% versus 9.6%; p=0.18) and less severe congestive heart failure (New York Heart Association functional class III or IV) in the rescue PTCA group (1.3% versus 7.0%; p=0.11). A statistically significant benefit was reported in the rescue PTCA group for the combined outcome of death or severe congestive heart failure at 30 days (6.4% versus 16.6%; p=0.05).

The TAMI 5 trial also studied the use of rescue PTCA. The 575 patients treated with various thrombolytic strategies for AMI were randomized to one of two catheterization strategies: 1) an aggressive strategy with angiography performed at 90 minutes and rescue PTCA only attempted for TIMI 0 or 1 flow in the infarct-related artery (n=287), or 2) a conservative strategy with predischarge angiography at 5 to 10 days (n=288).[17] In-hospital event rates were low, but freedom from a composite endpoint of adverse outcomes (death, stroke, reinfarction, reocclusion, heart failure, or recurrent ischemia) favored the aggressive strategy (67% versus 55%; p=0.004). The major effect of the aggressive strategy was in reducing the incidence of severe recurrent ischemia (25% versus 35%); mortality rates were similar.

Two recently published trials randomized patients to one of 3 arms: thrombolysis alone, thrombolysis and immediate transfer for angiography and rescue PTCA for TIMI 0-2 flow, or immediate transfer for primary PTCA. Vermeer et al reported no statistically significant difference between groups.[37] In the PRAGUE trial,[38] there was a significant reduction in the primary endpoint of death, reinfarction or stroke for the invasive groups (23% thrombolysis, 15% thrombolysis and rescue PTCA, 8% primary PTCA, p<0.02).

In addition to the above studies, there have been several case series and non-randomized studies examining rescue PTCA[16,19-24] (Table 1). The case series suggest that technical success rates are improving with rescue PTCA, with 80% success rates reported in earlier case series and 88-95% success rates in more recent studies.

Potential limitations and complications of rescue PTCA

The identification of patients with failed thrombolysis following AMI is problematic. Reduction in chest pain, decrease in ST segment elevation, and arrhythmias, although suggestive of reperfusion, are not diagnostic. Among patients from the TAMI database, for example, 56% of patients without ST segment or chest pain resolution were still found to have patent arteries at 90 minutes after the initiation of thrombolysis, demonstrating the difficulty of clinical determination of reperfusion.[10] One limitation of the Belenkie and RESCUE trials described above is that randomization of patients was performed only after an occluded infarct-related artery was demonstrated angiographically. The Vermeer and PRAGUE studies did randomize patients prior to angiography, but did not attempt to select which patients had failed to reperfuse. In clinical practice, however, the main dilemma is trying to determine noninvasively which patients have not reperfused and may benefit from transfer to the cardiac catheterization laboratory.

The timing of rescue angioplasty may critically affect the clinical benefit derived from the procedure. Excessive delays in performing rescue angioplasty may compromise the ability to salvage viable myocardium. The extent of myocardial salvage during rescue angioplasty may also be limited by impaired flow at the microvascular and myocardial level. Even after successful reperfusion with restoration of TIMI-3 flow in the infarct-related artery, 25-50% of patients have evidence of impaired myocardial perfusion, presumably related to distal embolization of platelet microthrombi, vasospasm, inflammation, and reperfusion injury.[42] The TIMI-14 trial has provided evidence that the GP IIb/IIIa inhibitor abciximab may not only improve the patency of the epicardial coronary artery, but may also enhance myocardial perfusion.[43,44]

A disturbing trend that has been found in almost all series is a mortality rate greater than 30% among patients with failed attempts at rescue PTCA.[14,18-20,26,27] These rates are substantially higher than the 7-11% mortality rates seen in patients with occluded infarct-related arteries who were treated conservatively.[2,15,18] However, it has also been noted that patients who die following unsuccessful rescue PTCA often have poor prognoses prior to the procedure.[19,26] Thus, a failed rescue PTCA by itself may not be directly responsible for the high mortality rates observed. Nevertheless, procedural failure is a potential limitation of rescue angioplasty. As noted above, recent studies indicate that success rates are improving over time. The use of stents and GP IIb/IIIa inhibitors result in higher procedural success rates and may therefore improve outcomes in this setting.

Rescue angioplasty was associated with increased bleeding and the need for emergency bypass surgery in earlier studies,[13,25] but complication rates appear to be dropping over time, perhaps partially related to less intensive anticoagulation. The GUSTO-1 angiographic substudy[19] showed no excessive bleeding and a low 1% rate of emergency CABG among the patients undergoing rescue PTCA.

Rescue PTCA combined with other treatment modalities

Stenting

Several case series have documented the successful use of stenting in patients undergoing rescue PTCA.[28,29,33] In a nonrandomized analysis of 108 consecutive rescue PTCA patients, Dauerman et al found improved angiographic results and less target vessel revascularization among the 45 patients who underwent stenting.[34] Thus, the limited data available support the feasibility, safety, and possible superiority of stenting in rescue PTCA.

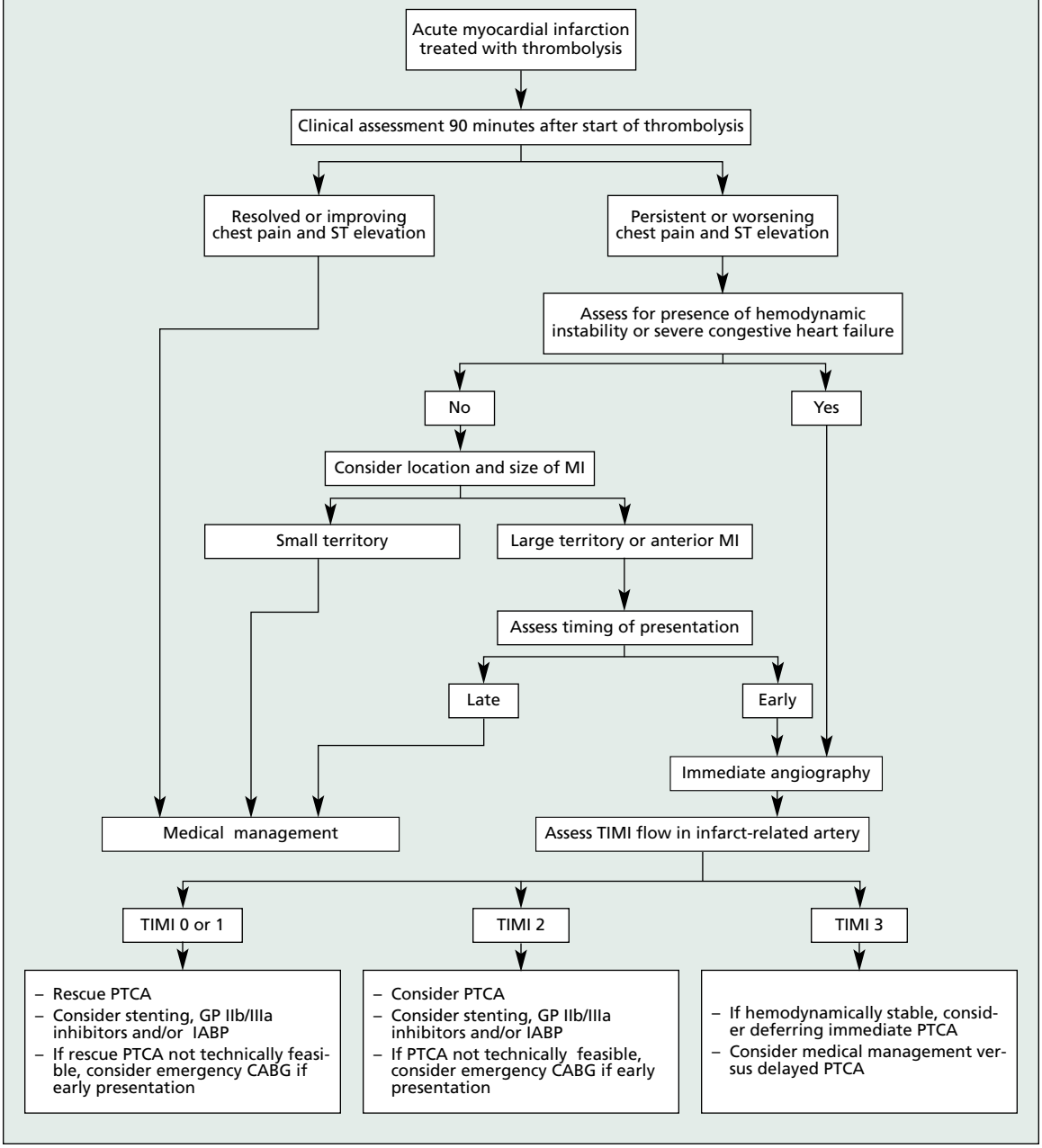
GP IIb/IIIa inhibition

The use of platelet GP IIb/IIIa inhibition has also been proposed as a means of improving outcomes among patients undergoing rescue PTCA, although at the risk of increasing bleeding complications.[39] Abciximab was associated with a significant reduction in the composite endpoint of death, MI and urgent reintervention among 22 patients who underwent rescue PTCA in the EPIC trial,[30] although major bleeding occurred in 9 of the15 abciximab-treated patients. In the GUSTO-III trial, 392 patients underwent rescue PTCA, with abciximab administered in a non-randomized manner to 83 of these patients. Treatment with abciximab was associated with lower 30-day mortality (3.6% versus 9.7%, P=0.042 after adjusting for baseline characteristics), but was also associated with more severe bleeding resulting in substantial hemodynamic compromise (3.6% versus 1.0%, p=0.08).[35] Cantor et al reported major bleeding in 23% of PTCA patients treated with abciximab within 24 hours of thrombolytic therapy.[36] Thus, the use of GP IIb/IIIa inhibitors during rescue PTCA likely improves clinical outcomes, but at the expense of increased bleeding complications.

Intraaortic balloon counterpulsation

The use of intraaortic balloon counterpulsation (IABP) has also been suggested as a means of improving outcome in rescue PTCA. The Randomized IABP study group randomized 182 patients sent for emergent cardiac catheterization within 24 hours of AMI, including 51 patients undergoing rescue PTCA, to the use of an IABP or standard care.[31] The investigators reported a significantly lower rate of a composite endpoint of death, stroke, reinfarction, emergency PTCA or CABG, or recurrent ischemia in the IABP group (13% versus 24%; p=0.04). Only one study specifically addressed the use of IABP in the setting of rescue PTCA. Ishihara et al assigned the first 20 consecutive patients with anterior MIs who underwent rescue PTCA to standard care and the next 40 similar

Figure 1: Approach to the patient with failed thrombolysis



patients to the use of an IABP.[32] The patients treated with an IABP had significantly decreased reocclusion rates (2.5% versus 25.0%; $p<0.05$), better evolution in predischarge mean ejection fraction (6.8% increase versus 2.0% decrease; $p<0.05$), and a non-significant trend towards decreased mortality (5.0% versus 20.0%; p=NS). These results suggest that an IABP may be helpful in patients undergoing rescue PTCA and that further studies are warranted.

Conclusion

To date, the benefits of rescue PTCA have not been clearly established, although there is a trend for improved outcomes in the two randomized trials of rescue PTCA. For this reason, if failed thrombolysis is suspected, immediate angiography and rescue PTCA should be considered, and a suggested clinical approach is summarized in Figure 1. The most benefit will likely be obtained in patients with hemodynamic instability, anterior, or large MIs within 8 to 12 hours from the onset of symptoms. The adjunctive use of GP IIb/IIIa inhibitors may improve clinical outcomes, but this benefit is offset by an increased risk of bleeding complications. Further studies are needed to determine the optimal management strategy for failed thrombolysis.

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