Primary angioplasty had lower long-term mortality than did streptokinase in acute myocardial infarction

Zijlstra F, Hoorntje JC, de Boer MJ, et al. **Long-term benefit of primary angioplasty as compared with thrombolytic therapy for acute myocardial infarction.** N Engl J Med. 1999 Nov 4;341:1413-9.

QUESTION

In patients with acute myocardial infarction (MI), is primary angioplasty more effective than streptokinase for reducing long-term mortality?

DESIGN

Randomized {allocation concealed*},† blinded (outcome assessors),* controlled trial with median follow-up of 5 years.

SETTING

A hospital in the Netherlands.

PATIENTS

395 patients (mean age 60 y, 81% men) with symptoms of acute MI for > 30 minutes who presented within 6 hours of symptom onset or within 6 to 24 hours with persistent ischemia. Other inclusion criteria were ST-segment elevation and no contraindications to thrombolytics. During the study, patients with marked hemodynamic instability or electrocardiographic signs of extensive MI were excluded. 1 patient was lost to follow-up.

INTERVENTION

Patients received aspirin and heparin and were allocated to streptokinase, 1.5 million units over 1 hour with angiography as needed (n = 194), or to angioplasty (n = 201). Additional revascularization could be done for symptomatic ischemia.

MAIN OUTCOME MEASURES

All-cause mortality and combined mortality and nonfatal MI at ≤ 30 days and at 5 years.

MAIN RESULTS

Patients in the angioplasty group had lower rates of all-cause mortality, cardiac mortality, and combined mortality and nonfatal MI at ≤ 30 days and 5 years ($P \leq 0.01$ for all comparisons) (Table) and lower rates of death from heart failure (3% vs 10%, $P < 0.01$) or sudden death (3% vs 9%, $P = 0.02$) at 5 years than patients in the streptokinase group. Patients in the angioplasty group also needed additional angioplasty less often (4% vs 35% at ≤ 30 d and 26% vs 52% at 5 y, $P < 0.001$ for both). The groups did not differ for noncardiac mortality.

CONCLUSION

Immediate angioplasty had lower short- and long-term all-cause mortality and cardiac mortality than did streptokinase for patients with acute myocardial infarction.

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*See Glossary.

†Information supplied by author.

Immediate angioplasty vs streptokinase for acute myocardial infarction (MI)‡

Outcomes	Angioplasty	Streptokinase	RRR (95% CI)	NNT (CI)
All-cause mortality by 30 d	1%	7%	85% (43 to 96)	17 (10 to 42)
All-cause mortality by 5 y	13%	24%	44% (14 to 64)	10 (6 to 35)
Cardiac mortality by 30 d	1%	6%	85% (43 to 96)	17 (10 to 42)
Cardiac mortality by 5 y	7%	20%	58% (19 to 78)	8 (5 to 15)
Death or nonfatal MI by 30 d	0.5%	9%	95% (69 to 99)	11 (7 to 19)
Death or nonfatal MI by 5 y	6%	22%	71% (49 to 85)	6 (4 to 11)

‡Abbreviations defined in Glossary; RRR, NNT, and CI calculated from data in article.

COMMENTARY

Shorter time to initiation of reperfusion therapy, higher patency of an infarcted artery, and higher flow rates are associated with decreased morbidity and mortality in acute MI. Several conclusions can be drawn from the controversial and sometimes contentious comparisons between primary angioplasty and fibrinolytic therapy. Angioplasty produces higher rates of patency of an infarcted artery and more normal flow rates than does fibrinolysis, but time to reperfusion can be delayed 1 to 2 hours because of logistic delays in organizing the procedure. This delay neutralizes the potential benefit. Mortality rates in randomized trials are artificially low because of selection bias. Less than 20% of U.S. hospitals do angioplasty, and outcomes are better in high-volume centers. Therefore, replicating the benefits of angioplasty that were seen in the study by Zijlstra and colleagues is currently impossible for most patients. No difference in left ventricular ejection fraction, a prime prognostic indicator, has been shown between angioplasty and fibrinolysis, and inadequate statistical power exists in 10 small, randomized trials to evaluate mortality benefit.

Nevertheless, recurrent ischemia, reinfarction, and stroke rates are lower with angioplasty. Furthermore, it is superior in patients with cardiogenic shock and may be superior for congestive heart failure. It is also safer in elderly patients who have a 4% risk for intracerebral hemorrhage with fibrinolysis. To complicate the comparison further, endoluminal stenting may improve angioplasty results, and glycoprotein IIb/IIIa platelet receptor antagonists may improve both angioplasty and fibrinolytic results.

So what is a clinician to do? The answer is to treat as quickly as possible with the best strategy available. Deciding which options to use depends on angioplasty availability, patient age, symptom duration, heart rate and blood pressure, electrocardiographic abnormalities, comorbid conditions, and financial issues. The ultimate strategy may be a combination of prehospital administration of half-dose fibrinolytic drug; emergency department administration of glycoprotein IIb/IIIa antagonists; and if ST-segment elevation persists, rapid transfer to an interventional cardiology laboratory for angioplasty and stent implantation. But this remains to be tested.

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