

**A comparative study intravenous streptokinase and immediate coronary angioplasty for myocardial infarction**

<sup>1</sup>Dr. Utsavkumar Patel, Assistant Professor, General Medicine, Dr. M.K. Shah Medical College, Ahmedabad

<sup>2</sup>Dr. Kamlesh Patel, Assistant Professor, Anesthesiology, Swaminarayan Institute of Medical Sciences & Research, Kalol.

**Corresponding Author:** Dr. Utsavkumar Patel, Assistant Professor, General Medicine, Dr. M.K. Shah Medical College, Ahmedabad.

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**Conflicts of Interest:** Nil

**Abstract**

**Introduction:** Randomized comparisons of the two reperfusion strategies are scarce, despite the fact that intravenous thrombolytic therapy and urgent percutaneous transluminal coronary angioplasty are commonly used to treat acute myocardial infarction. We present the findings of a prospective, randomised experiment that contrasted intravenous streptokinase treatment with immediate coronary angioplasty (without prior thrombolytic therapy).

**Methods:** One of the two therapies was randomly allocated to 108 patients who had suffered an acute myocardial infarction. Before being released from the hospital, radionuclide scanning was used to determine the left ventricular ejection fraction. A quantitative coronary angiography was used to evaluate the extent of remaining stenosis in the arteries connected to the infarct.

**Results:** Seventy patients were scheduled for urgent angioplasty and the remaining 65 patients were assigned to receive streptokinase. Of the 50 patients who had angioplasty, 58 experienced technical successes with the treatment. Nine patients who were given streptokinase experienced an infarction recurrence, while none of the patients who were given angioplasty did ( $P = 0.002$ ). After their infarction, four patients in the angioplasty group and fourteen in the streptokinase group experienced unstable angina ( $P = 0.01$ ). Prior to discharge, the streptokinase group had a mean ( $\pm$ SD) left ventricular ejection fraction of  $45 \pm 12$  percent, while the angioplasty group had a mean ( $\pm$ SD) of  $45 \pm 10$  percent ( $P = 0.002$ ). In 58 percent of patients receiving streptokinase and 78 percent of patients receiving angioplasty, the artery linked to the infarct was patent ( $P$  is equal to 0.001). Quantitative coronary angiography showed that the angioplasty group had stenosis of 26

$\pm 15$  percent of the luminal diameter, while the streptokinase group had  $68 \pm 13$  percent ( $P < 0.001$ ).

**Conclusions:** Compared to intravenous streptokinase, immediate angioplasty following acute myocardial infarction was linked to a higher rate of patency of the infarct-related artery, a less severe residual stenotic lesion, better left ventricular function, and a lower incidence of recurrent myocardial ischemia and infarction.

**Keywords:** Coronary angioplasty, streptokinase, myocardial infarction, artery, intravenous, patency, therapy.

### Introduction

According to De Wood et al.'s 1980 study, an intraluminal coronary thrombus superimposed on an atherosclerotic lesion is the usual reason why acute transmural myocardial infarction is associated with total coronary blockage. The last ten years have seen a significant amount of study on the efficacy of coronary angioplasty and thrombolytic therapy in restoring patency in the coronary artery connected to an infarct. [1, 2]

While it might be helpful in situations where arteries linked to infarcts do not reperfuse, routine angioplasty after thrombolytic therapy typically does not provide any additional benefit. [3, 4]

Recent published randomized trials have demonstrated that the gold standard treatment for patients experiencing an acute myocardial infarction is a combination of streptokinase, aspirin, and heparin. [5] In order to prevent the potential side effects of thrombolysis, such as myocardial and intraplaque hemorrhage, coronary angioplasty is carried out immediately without first receiving thrombolytic therapy. [6] Thus, the best course of action for treating an acute myocardial infarction is to

undergo urgent angioplasty, according to some authors. [7]

This approach also has the advantage of reducing the hemodynamic impact of the underlying atherosclerotic lesion. However, another investigation stated that issues frequently arise after timely angioplasty. [8] With the exception of a tiny randomized study contrasting intracoronary streptokinase therapy with rapid angioplasty, there aren't many parallels between the two techniques. As a result, we performed a prospective, randomized research wherein patients with an acute myocardial infarction were treated with either immediate angioplasty or intravenous streptokinase. [9]

### Material & Method

The research protocol was reviewed and approved by our institutional review board. The enrollment of patients began on August 20, 2022, and ended on February 10, 2023. Inclusion criteria were as follows: symptoms of acute myocardial infarction that persisted for more than 30 minutes, accompanied by an elevation of more than 1 mm (0.1 mV) in the ST segment in two or more contiguous electrocardiographic leads; presentation within 6 hours after the onset of symptoms (or between 6 and 24 hours, if there was evidence of continuing ischemia); an age of less than 76 years; and no contraindication to thrombolytic therapy, including previous stroke or other known intracranial disease, recent trauma or surgery, refractory hypertension, active bleeding, or prolonged cardiopulmonary resuscitation. Previous coronary-artery bypass grafting, previous Q-wave or non-Q-wave infarction, and cardiogenic shock were not reasons for exclusion. Before randomization we recorded each patient's age, sex, Killip class on admission, [10] electrocardiographic site of infarction,

history of infarction, heart rate, arterial pressure, time of onset of symptoms, and time of hospital admission.

### **Randomization and Treatment Protocol**

Using a closed-envelope method, patients were randomly assigned to one of the two therapy groups following the acquisition of informed consent. All of the patients had intravenous aspirin injections totalling 300 mg. They also got oral aspirin dosages of 300 mg daily and intravenous nitro-glycerine at a dose intended to sustain a systolic blood pressure of 110 mm Hg. A dose of intravenous heparin was administered to ensure that the activated partial-thromboplastin time remained at least two days above the usual value, ranging from two to three times. Twice a day, this partial-thromboplastin time was recorded. Just 13 patients had values less than two times the normal value, despite the fact that values more than three times the normal value happened at least once in 57% of the patients. Medication including lidocaine,  $\beta$ -adrenergic blockers, and calcium-channel blockers were administered solely based on the attending physicians' judgment. Fourteen percent of the patients in the streptokinase group and 16 percent of those in the angioplasty group received intravenous lidocaine; 31 percent and 41 percent, respectively, received calcium-channel blockers; and 39 percent and 27 percent received  $\beta$ -adrenergic blockers. Both coronary arteries were visualized; left ventriculography was not performed. The time from admission to the initiation of therapy was calculated as the time to the start of the streptokinase infusion or the first balloon inflation.

### **Study end Points**

The left ventricular ejection percent, vascular patency, and the rate of recurrent ischemia prior to discharge were the variables we measured. Prior to hospital discharge,

recurrent ischemia was defined as follows: recurrent myocardial infarction, which was defined as chest pain, changes in the ST-T segment, and a second increase in the creatine kinase level to more than twice the upper limit of normal, or an increase of  $>200$  U per liter over the previous value if the level had not dropped below the upper limit of normal. Stable angina was defined as chest pain and a positive exercise test. Unstable angina was defined as chest pain and changes in the ST-T segment at rest. Two cardiologists who were blind to the prescribed treatment examined all ECGs and lab values for signs of recurrent ischemia. ST-segment depression or a new elevation of the ST segment of at least 1 mm in two or more contiguous leads during chest pain were required electrocardiographically for a diagnosis of ischemia; alternatively, unchanged or pseudonormal ST segments had to be present during chest pain, with the T waves turning inverted after the pain subsided.

These inverted T waves needed to appear three hours after the chest pain reappeared and had to be at least 2 mm deep. In a supine posture, the patient had a symptom-limited bicycle exercise test with 10 W per minute increments. An exercise test with a ST depression of more than 1 mm, recorded 60 msec after the J point, was considered to indicate ischemia. A depression of more than one millimeter in the ST segment was thought to be indicative of ischemia in patients with baseline abnormalities in the ST-T wave.

Prior to being released from the hospital, the left ventricular ejection fraction was assessed using a radiological approach [11,12]. There has been prior documentation of the method employed in our facility. In short, it involved identifying red cells with  $[^{99m}\text{Tc}]$  pertechnetate and then using the multiple-gated equilibrium approach. The gamma camera (General

Electric, Milwaukee) was equipped with a parallel-hole collimator that was low-energy and multipurpose. Using the PAGE program, a computer (Star View, General Electric) automatically computed the worldwide ejection fraction.

An expert in nuclear medicine collected the ejection fraction data while keeping the clinical data hidden from view. Using software whose outcomes were independent of the operator guarded against potential prejudice. The method's repeatability is outstanding, with a mean ( $\pm$ SD) variation of  $1.0 \pm 1.1$  percent between duplicate measurements. [13]

Coronary angiography was used to evaluate arterial patency, which is defined as Thrombolysis in Myocardial Infarction (TIMI) grade 2 or 3 flow in the infarct-related coronary artery. To determine the rate of restenosis, angiography was performed again in the angioplasty group, ideally three months later. [14]

During the angiography examination, the patient's name, date of birth, and electrocardiographic site of infarction were the only information available. A cardiovascular analysis system based on a personal computer was used to quantitatively examine all vessels associated to infarcts (Cardiovascular Measurement System, Medis Medical Imaging Systems, Nuenen, the Netherlands) The fundamental algorithms have already been explained. The technology projects a chosen cinema frame onto a digital camera via a zoom lens using a high-quality converter. After that, the video signal of the enlarged area of interest was converted to digital format. In order to calibrate, the boundaries of a no tapering part of the catheter were determined automatically over a length of approximately 2 cm. To determine the contours of the vessel the user had only to indicate the beginning and end of the coronary segment to be analyzed, after

which a path was computed connecting these two points. The contour procedure was then performed iteratively by resampling the image along scan lines perpendicular to the path computed in the first iteration. Next, a matrix of cost coefficients was computed that represented for each point in the resampled matrix the edge strength based on the weighted sum of the first and second derivative functions. The initial contours were found by the minimal-cost contour-detection technique applied to the cost-coefficient matrix. In the second iteration, the contours determined in the first iteration functioned as models for the subsequent determination. The edge strengths were corrected for the limited resolution of the entire imaging chain, a procedure that is particularly important for the accurate measurement of small vessels. From the final contours, a new center line was computed. A diameter was determined in absolute terms (in millimeters) by computing along the vessel center line the shortest distances between the left and right contours. The reference diameter was defined as previously described. [15]

### **Statistical Analysis**

The intention to treat principle was applied to the analysis of all end points. The mean values were compared using the Student's t-test. A standard chi-square test was used to compare the frequencies of recurrent ischemia, vascular patency, and complications; however, in cases where an expected cell value of less than five was present, Fisher's exact test was employed. Every computed P value has two tails. Whereas discrete variables are shown as absolute values and percentages, continuous baseline and outcome variables are reported as means  $\pm$ SD in our data presentation.

**Results**

Table 1 lists complications and necessary steps. Bleeding complications were defined as bleeding that required a blood transfusion or that resulted in intracerebral haemorrhage. Compared to patients given streptokinase, those given rapid angioplasty saw less problems overall. Specifically, the group that underwent angioplasty experienced lower rates of death, haemorrhage, and heart failure. Only 8% of patients in the angioplasty group experienced recurrent ischemia, compared to 32% of patients in the streptokinase group (P<0.001).

Quantitative angiographic analysis of the infarct-related vessels is shown in Table-2. Restenosis, defined as stenosis of more than 50 percent in the dilated vessel, was observed in 11 of 50 patients in the angioplasty group (16 percent). Although evidence has accumulated that the incidence of restenosis reaches a plateau at three months, the clinical implications of restenosis will become clear only after at least six months of follow-up. Excluding all angiography without evidence of restenosis performed within three months after angioplasty, we found the rate of restenosis to be 11 of 42, or 22 percent.

Table1: Complications and Procedures in the Study Patients

Variable	Streptokinase Group [N= 50]	Angioplasty Group [N=58]	P Value
NO IN %			
Death	3 [4]	0	0.12
Stroke	3 [4]	0	0.49
Bleeding	6 [5]	2 [1]	0.29
Mechanical ventilation	1[1]	1[1]	1.00
Femoral artery repair	0	1[1]	0.39
Heart failure	7 [8]	5 [7]	0.27
Ventricular tachycardia and ventricular fibrillation	6 [6]	5 [5]	0.59
Bypass surgery*	1[2]	3 [5]	0.42
Any of the above	15 [25]	9 [10]	0.04

\* Coronary-artery bypass grafting was required within 24 hours after admission to the hospital.

Table 2: Quantitative Angiographic Data

Variable	Angioplasty Group			P Value	Streptokinase Group At 21± 30 Days [N=58]
	Before [N=50]	After [N=58]	Follow up at 78±58 days [N=50]		
-				-	-

Projections analyzed [no.]	2.0±0.3	2.2±0.4	2.2±0.3	-	2.1±0.5
Stenosis [%]	89±12	22±10	32±18	<0.001	70±15
Minimal luminal diameter [mm]	0.10±0.39	2.25±0.58	2.05±0.85	<0.001	0.70±0.57
Reference diameter [mm]	-	3.18±0.65	3.16±0.65	0.245	3.03±0.67
Largest balloon [mm]	2.97±0.36	-	-	-	-

Plus- minus [±] values are means ±SD.

**Discussion**

Our study shows that in patients with acute myocardial infarction, direct coronary angioplasty results in a higher rate of patency of the infarct-related coronary arteries, less severe residual stenotic lesions, better left ventricular ejection fraction, and a lower incidence of recurrent myocardial ischemia than intravenous streptokinase. The patients in our angioplasty group had a rate of freedom from adverse events of 58 percent, significantly better than the rate in the streptokinase group (48 percent, P = 0.001).

**Conclusion**

The coronary anatomy was known soon after admission in the angioplasty group, clinical decision making was influenced by anatomical considerations. Finally, the implications of this study with respect to cost effectiveness require a formal analysis, which will be performed after one year of follow-up.

**References**

1. Prevalence of total coronary occlusion during the early hours of transmural myocardial infarction. N Engl J Med 1980; 303:897-902
2. Kennedy JW, Ritchie JL, Davis KB, Stadius ML, Maynard C, Fritz JK. The Western Washington randomized trial of intracoronary streptokinase in acute myocardial infarction: a 12-month follow-up report. N Engl J Med 1985; 312:1073-1078
3. The TIMI Study Group. Comparison of invasive and conservative strategies after treatment with intravenous tissue plasminogen activator in acute myocardial infarction: results of the Thrombolysis in Myocardial Infarction (TIMI) Phase II Trial. N Engl J Med 1989; 320:618-627
4. Califf RM, Topol EJ, Stack RS, et al. Evaluation of combination thrombolytic therapy and timing of cardiac catheterization in acute myocardial infarction: results of thrombolysis and angioplasty in

- myocardial infarction -- phase 5 randomized trial. *Circulation* 1991; 83:1543-1556
5. de Bono DP, Simoons ML, Tijssen J, et al. Effect of early intravenous heparin on coronary patency, infarct size, and bleeding complications after alteplase thrombolysis: results of a randomised double blind European Cooperative Study Group trial. *Br Heart J* 1992; 67:122-128
  6. Waller BF, Rothbaum DA, Pinkerton CA, et al. Status of the myocardium and infarct-related coronary artery in 19 necropsy patients with acute recanalization using pharmacologic (streptokinase, r-tissue plasminogen activator), mechanical (percutaneous transluminal coronary angioplasty) or combined types of reperfusion therapy. *J Am Coll Cardiol* 1987; 9:785-801
  7. Meier B. Balloon angioplasty for acute myocardial infarction: was it buried alive? *Circulation* 1990; 82:2243-2245
  8. Gacioch GM, Topol EJ. Sudden paradoxical clinical deterioration during angioplasty of the occluded right coronary artery in acute myocardial infarction. *J Am Coll Cardiol* 1989; 14:1202-1209
  9. O'Neill W, Timmis GC, Bourdillon PD, et al. A prospective randomized clinical trial of intracoronary streptokinase versus coronary angioplasty for acute myocardial infarction. *N Engl J Med* 1986; 314:812-818
  10. Killip T III, Kimball JT. Treatment of myocardial infarction in a coronary care unit: a two-year experience with 250 patients. *Am J Cardiol* 1967; 20:457-464
  11. Boudreau RJ, Loken MK. Functional imaging of the heart. *Semin Nucl Med* 1987; 17:28-38
  12. Jones RH. Use of radionuclide measurements of left ventricular function for prognosis in patients with coronary artery disease. *Semin Nucl Med* 1987; 17:95-103
  13. Remkes PAJ, Reiffers S, Thomas K, Zijlstra F. Effect van percutane transluminale coronaire angioplastiek bij eenvats coronair lijden op de veranderingen van de globale en segmentale linker ventrikel wandbeweging. *Ned Tijdschr Cardiol* 1991; 5:178-182
  14. Serruys PW, Luijten HE, Beatt KJ, et al. Incidence of restenosis after successful coronary angioplasty: a time-related phenomenon: a quantitative angiographic study in 342 consecutive patients at 1, 2, 3, and 4 months. *Circulation* 1988; 77:361-371
  15. Reiber JHC, van der Zwet PMJ, von Land CD, et al. Quantitative coronary arteriography: equipment and technical requirements. In: Reiber JHC, Serruys PW, eds. *Advances in quantitative coronary arteriography*. Dordrecht, the Netherlands: Kluwer Academic Publishers, 1992:75-111.