Acute Intervention During Myocardial Infarction in Patients with Prior Coronary Bypass Surgery

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Thrombolytic intervention with or without coronary angioplasty is becoming widely accepted as standard therapy for patients who present early with acute myocardial infarction. Thrombolytic therapy has been shown to improve left ventricular function and reduce mortality. Nearly all clinical trials of myocardial reperfusion have excluded patients with a history of coronary artery bypass grafting. Furthermore, the only available data for the use of thrombolytic agents in this patient group consist of case reports of intracoronary thrombolytic agents and a small series of intravenous thrombolytic agents. There are no published data for the use of angioplasty or combined thrombolysis and angioplasty in patients with acute infarction after bypass surgery. Considering the increasing number of patients undergoing coronary artery bypass grafting and their propensity for future cardiac events, important questions regarding the optimal treatment strategy for prior coronary artery bypass patients come to bear. Accordingly, we reviewed our experience in the treatment of patients after coronary artery bypass surgery in the setting of evolving myocardial infarction.

Records from the University of Michigan Cardiac Catheterization Laboratory were reviewed from January 1, 1984, to December 30, 1987, and a database of patients with a history of coronary artery bypass grafting and acute myocardial infarction was tabulated. Acute myocardial infarction was diagnosed when an episode of characteristic chest pain lasting ≥30 minutes was associated with a transient, temporally appropriate increase of the total serum creatine phosphokinase above the upper limit of normal with myocardial isoenzyme fraction greater than twice normal. Data were compiled regarding the presence and type of standard electrocardiogram tracings at the time of infarction. Details of acute interventions, when performed, were accumulated, including those concerning the infarct-related vessel and whether or not the patient was treated with thrombolytic agents, direct angioplasty or a combination of the 2. Status of the infarct-related vessel after intervention was also noted. Follow-up data regarding hospital discharge, repeat catheterization, exercise stress testing and the need for further intervention, including repeat bypass surgery, were also obtained.

Data are expressed as mean ± 1 standard deviation. A comparison of the efficacy of acute intervention as defined by infarct-related vessel patency after intervention was estimated by examining a cohort of patients who presented with acute myocardial infarction and had no previous history of bypass surgery treated with thrombolysis and/or angioplasty in the Thrombolysis and Angioplasty in Myocardial Infarction-I trial. Estimations of statistical significance were made using a chi-square test.

As listed in Table I, 40 patients met the criteria delineated previously during the time frame stated. The time since coronary artery bypass was 6 ± 3.5 years (range 2 months to 16 years). Age of the patients was 57 ± 10 years (range 33 to 77); 31 of 40 were male (72%). Although all patients presented with symptoms consistent with acute myocardial infarction and subsequently ruled in by enzymatic criteria, only 21 of 40 (53%) had typical ST-segment elevation or new Q waves. The remainder presented with ST depression or nonspecific electrocardiogram changes, thus not allowing localization of the site of myocardial infarction by these means. Thirty-five of the patients (88%) underwent diagnostic cardiac catheterization within close temporal relation to the onset of their symptoms. Of these patients, the infarct-related vessel could be reliably identified in 26 (74%); 12 were native vessel occlusions (46%) and 14 were saphenous vein grafts (54%). Left internal thoracic artery grafts were not present in any of our patients. Acute intervention was attempted in 20 of these patients in whom the infarct-related vessel could be ascertained. The time from onset of symptoms to intervention was 4 ± 1 hours (range 2 to 6).

Intravenous thrombolysis was only used in 4 (10%) of the patients (100 mg of recombinant tissue plasminogen activator in 2 cases, 1.5 million U of intravenous streptokinase in 1 case and 500,000 U of urokinase in 1 case) and was successful in establishing vessel patency in 3 (75%). The infarct-related artery of the patient with unsuccessful reperfusion was a native right coronary artery. Direct coronary angioplasty of the infarct-related vessel was performed in 9 patients (23%) and vessel patency was achieved in 7 (78%). The 2 unsuccessful coronary angioplasties were in saphenous vein grafts, 1 to the left anterior descending artery and 1 to an obtuse marginal branch. Combined angioplasty and thrombolytic therapy (intracoronary urokinase or streptokinase) was performed in 7 patients and resulted in reperfusion in 6 (86%). The unsuccessful combined therapy was in a patient with a native right coronary as the infarct-related vessel. Angiographically demonstrable embolic complications were not noted. The total success rate of all acute interventions in establishing infarct-related vessel patency was 16 of 20 (80%). A similarly treated cohort of patients obtained from the Thrombolysis and Angioplasty in Myocardial Infarction trials revealed a reperfusion rate of 92% in 835 patients with native coronary artery thrombosis receiving thrombolysis and/or coronary angioplasty (p = 0.06). Of the patients not undergoing acute percutaneous intervention in this study, 2 had
emergency repeat coronary artery bypass grafting and the remaining 18 were treated medically.

Four patients died in the hospital (10%). Two of these patients had been treated medically; 1 died during emergency repeat coronary artery bypass surgery and the other during acute coronary angioplasty (without combined thrombolysis). The intermediate-term postdischarge outcome was available in 38 of 40 patients. There were 4 deaths within the first 3 months, 3 in patients who had been treated medically and 1 by acute intervention went on to have successful revascularization procedures after their acute infarct; I was successfully dilated. One other patient from the combined treatment group had a delayed angioplasty of a noninfarct-related artery. Two patients in the medically treated group had delayed angioplasties: 1 was successful. Thus, 8 of 20 patients (40%) initially treated with acute intervention went on to have successful recanalization procedures after their acute infarct; I was lost to follow-up and 1 died. The remainder had patent infarct-related arteries at recatheterization or were asymptomatic clinically.

The number of patients undergoing coronary artery bypass grafting with saphenous vein grafts has been steadily increasing. Considering that the rate of late graft occlusion is approximately 4%/year and the yearly rate of myocardial infarction after coronary artery bypass surgery is approximately 3%, the number of patients at risk for these events continues to increase. Thus, this group of patients represents an increasing proportion of cases of acute myocardial infarction that may benefit from acute intervention. However, the experience on the
use of thrombolytic agents or angioplasty in these pa-
tients remains very limited.

This study reports our experience in patients after
coronary artery bypass grafting in the setting of acute
myocardial infarction. Although the number of patients
available for review is small when compared to large scale
clinical trials, it represents a large series of such patients
reported to date. Furthermore, we report all such patients
presenting to our institution regardless of whether or not
an intervention was performed.

We noted a limited ability of the standard electrocar-
diogram to corroborate acute myocardial infarction and
aid in ability to determine the infarct area. The prepon-
derence of non-Q-wave myocardial infarcts in our study
probably relates to either the finding that the infarct-
related vessel is less likely to be a main epicardial artery10
or to the presence of collaterals. Thus, such infarcts are
usually smaller in postcoronary artery bypass surgery
patients.11 This observation may impede the ability to
promptly intervene in these patients when they present
with acute myocardial infarction.

Furthermore, even with angiography the infarct-
related vessel could not be reliably identified in a fairly
large proportion of our patients. This phenomenon was
due to the presence of >1 vessel occlusion or multiple
discordant wall motion abnormalities. With our concern
over patient safety intervention with direct angioplasty or
intracoronary thrombolysis was not possible in this group.

However, in our patients in whom the infarct-related
vessel could be ascertained and acute intervention was
performed, the results as assessed by vessel patency com-
pared less favorably with a cohort of patients who never
had bypass surgery. Although the lesions encountered in
these patients may have been somewhat more "resistant"
to thrombolytic therapy or angioplasty, there was yet a
high degree of success. We did not encounter overt angi-
ographic evidence of embolic complications as reported by
others.12 Furthermore, more definitive revascularization
with redo surgery was then possible in many of these
patients at a later date. The limited number of patients in
our study preclude recommendations as to the general
experience of treatment such patients. However, based on this
experience acute intervention in this setting is a viable option,
although with a decreased reperfusion success rate, but with
a potentially positive impact on the prognosis of these
patients. There appears to be no legitimate reason to
exclude this important group of patients from prospective
reperfusion trials.

1. White HD, Norris RM, Brown MA, Takayama M, Maiselski A, Baas NM,
Ommiston JA, Whistock T. Effect of intravenous streptokinase on left ventricular
1987;317:850-855.

2. Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico
(GISSI). Effectiveness of intravenous thrombolytic treatment in acute myocardial


4. Kleiman NS, Berman DA, Gaston WR, Cashin WR, Roberts R. Early
angiographic evidence of embolic complications as reported by
others.12 Furthermore, more definitive revascularization

5. Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico
(GISSI). Effectiveness of intravenous thrombolytic treatment in acute myocardial

6. Topol EJ, Califf RM, George BS, Kerriakes DJ, Abbottsmith CW, Candela
RJ, Lee KL, Pitt B, Stack RS, O'Neill WW, and the Thrombolytics and Angio-
plasty in Myocardial Infarction Study Group. A randomized trial of immediate
versus delayed elective angioplasty after intravenous tissue plasminogen activator

7. Loop FD, Lytle BW, Gill CC, Golding LAR, Concorpe DM, Taylor PC.

8. Campeau L, Lespinciane J, Bouvier MO. Natural history of saphenous vein

9. Coronary Artery Surgery Study (CASS) Principal Investigators and Their
Associates. A randomized trial of coronary artery bypass surgery: Quality of life in

10. Crean PA, Waters DD, Bosch X, Peltier GB, Roy D, Theroux P. Angio-
pgraphic findings after myocardial infarction in patients with previous bypass
surgery: explanations for smaller infarcts in this group compared with control

11. Waters DD, Peltier GB, Hasche M, Theroux P, Campeau L. Myocardial
infarction in patients with previous coronary artery bypass surgery. JACC
1984;2:909-915.

12. De Feyter PJ, Serruya P, van den Brand M, Meester H, Beati K, Suryaprana-
uia H. Percutaneous transluminal angioplasty of a totally occluded venous bypass
graft: a challenge that should be resisted. Am J Cardiol 1989;64:88-90.

Effects of Verapamil on the Anaerobic Threshold and Peak Oxygen
Consumption in Effort Angina Pectoris

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The anaerobic threshold is the submaximal level of
exertion above which lactic acid progressively accumu-
lates in the blood and fatigue progressively increases
during incrementnal exercise.1,2 The anaerobic threshold is
a useful clinical measurement of submaximal exercise
performance in normal subjects1-3 and in patients with
congestive heart failure,4 angina pectoris5 and valvular
regurgitation,6 and has been used to assess drug interven-
tions.7 In patients with effort angina, ST-segment depres-
sion is reduced and exercise time increased after calcium
antagonists.8-9 If relief of myocardial ischemia by verapa-
mil allows an improvement in peak oxygen consumption (VO2peak),
there may also be an improvement in oxygen metabolism by exercising muscle and the anaerobic
threshold may increase, delaying the onset of fatigue.
This study determines whether verapamil alters the oxy-
genous consumption (VO2) at the anaerobic threshold, or the
perception of leg fatigue in patients with effort angina
pectoris.

Sixteen patients with stable exertional angina pectoris and positive exercise tests (Bruce protocol with at least 1-mm horizontal ST-segment depression) entered the study. No patient had chronic lung disease, valvular regurgitation, clinical heart failure or peripheral vascular
disease. All patients gave written informed consent