



Evaluation of Underlying Coronary Stenosis Severity in Patients with Acute MI after Thrombus Aspiration Using Export Catheter

Morteza Safi, MD*, Leila Nojoomizadeh, MD, Maryam Taherkhani, MD, Mohamadreza Motamedi, MD, Habibollah Saadat, MD, Mohammad Hasan Namazi, MD, Hossein Vakili, MD, Shahram Taghdisi, MD

Modarres Hospital, Cardiovascular Research Center, Shaheed Beheshti University of Medical Sciences, Tehran, Iran.

Received 26 April 2009; Accepted 11 August 2009

Abstract

Background: Early clinical and retrospective angiographic evaluations indicated that in patients with acute myocardial infarction (MI), vulnerable plaques most often represented a mild luminal stenosis. More recent studies drawing upon prospective angiograms, however, have demonstrated that in majority of patients with acute MI, the underlying stenosis is significant.

Methods: Twenty-eight patients with acute MI candidate for thrombectomy were enrolled in this study. Thrombectomy was performed using export thrombectomy catheters. After the injection of nitroglycerin, the severity of the lesions was determined by two independent operators.

Results: Between April 2007 and February 2008, 28 patients, comprised of 26 men and 2 women with acute MI, were evaluated. The underlying stenosis severity was $\geq 50\%$ in 22 (78.6%) patients and $< 50\%$ in the remaining 6 (20.4%) patients (P value < 0.01). The right coronary artery was the most common vessel involved in the lesions $< 50\%$.

Conclusion: Contrary to the general belief of many cardiologists, the majority cases of myocardial infarction occur in consequence of significant stenoses.

J Teh Univ Heart Ctr 4 (2009) 222-225

Keywords: Myocardial infarction • Thrombectomy • Coronary stenosis

Introduction

Myocardial infarction (MI) is most often caused by the rupture of atherosclerotic lesions in a coronary artery, leading to the formation of a thrombus that plugs the artery and thus prevents it from supplying blood to the heart.¹ Less frequently, however, MI is secondary to coronary spasm, coronary embolism, and thrombosis in non-atherosclerotic normal vessels.²

It should be noted that in many cases of MI, no history

of stable angina presages the acute event. Several clinical observations have thus far posited that many MI cases result not from high-grade stenoses but from lesions that do not limit the flow.³ In some studies, coronary angiography performed at some stages before ST-segment elevation myocardial infarction shows that an assessment of the angiographic severity of coronary stenosis may be inadequate to accurately predict the time or location of a subsequent coronary occlusion that will beget MI.⁴⁻⁷ In contrast to what is often believed, some recent studies have demonstrated that

*Corresponding Author: Morteza Safi, Associate Professor of Cardiology, Department of Cardiovascular Medicine, Modarres Hospital, Shaheed Beheshti University of Medical Sciences, Tehran, Iran. P.O.Box: 199873438. Tel: +98 21 22083106. Fax: +98 21 22083106. E-mail: mortezasaafi@yahoo.com.



the majority of MI cases occur due to significant stenoses.⁸

The foregoing discrepancies prompted us to investigate the underlying stenosis severity in our interventional center with more than 75 primary percutaneous coronary interventions (PCI) per year.

Methods

Between April 2007 and February 2008, 107 consecutive patients were admitted to Modarres Hospital for primary PCI due to acute MI. Among them, 28 patients (26 men and 2 women) who had indications for thrombectomy were recruited in the present study.

Thrombectomy with export catheters was performed in the patients, who had both large amounts of myocardium in jeopardy and large thrombus burden or TIMI (thrombolysis in myocardial infarction) of 0 or 1 after the wiring of the infarct-related artery (IRA).⁹ After thrombectomy, 200-400 µg nitroglycerin was injected intracoronarily. Culprit lesions were determined with respect to occlusion sites before wiring in the IRA with TIMI 0 or the site of thrombus in spontaneously recanalized IRA.

Patients without TIMI 2 or 3 or with a residual large thrombus at the site of culprit lesions were excluded.

Pre-PCI severity of stenosis was determined from two orthogonal standard views by two independent operators.

Results

Between April 2007 and February 2008, 28 patients, consisting of 26 (92.9%) men and 2 (7.1%) women aged 56.4 ± 11 (range: 33-87) years, were enrolled in this study. The culprit lesions were distributed, with 10 in the left anterior descending (LAD) artery, 4 in the left circumflex (LCX) artery, and 14 in the right coronary artery (RCA).

The underlying stenosis severity was $\geq 50\%$ in 22 (78.6%) patients and $< 50\%$ in the remaining 6 (21.4%) (P value $< 0/01$); consequently, the percentage of underlying stenosis in all the patients with acute MI who underwent thrombectomy was estimated to be at least 63 to 93 percent (CI=95).

There were no differences in sex between the lesions $\geq 50\%$ and lesions $< 50\%$ stenosis (P value < 0.7). In patients

over 60 years of age, there was no lesion $< 50\%$ stenosis (Table 1). In just one (16.7%) patient with lesion $< 50\%$ stenosis and in 3 (13/6%) patients with lesions $\geq 50\%$ stenosis, the LCX was involved. The RCA was the most common vessel amongst the patients with $< 50\%$ stenosis, but all the lesions in the LAD were more than 50% stenosis. Ignoring the LCX, in 52.6% of the cases, the LAD was responsible for lesions $> 50\%$ stenosis (Figure 1).

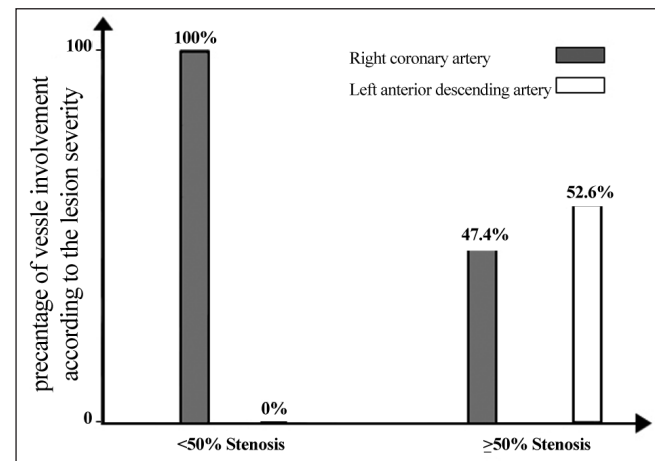


Figure 1. Relation between stenosis severity and vessel involvement (after ignoring left circumflex artery)

Discussion

Our study results highlight the significance of the underlying stenosis of the IRA in most patients with acute MI. In all of our patients, the door-to-balloon time was < 90 minutes, and two standard orthogonal views were obtained after the establishment of antegrade flow; the ischemic time, therefore, was not prolonged. Several studies have concluded that the majority of MI cases occur at locations of mild luminal stenoses.

Giroud et al. investigated 92 patients with coronary angiography before and after acute MI. The median time between the first coronary angiography and acute MI was 26 months (range: 1-144 months). Seventy-two (78%) segments responsible for a future acute MI were not significantly stenotic and it was concluded that acute MI was frequently related to a segment that was not the most stenotic one.⁵

Trabulo et al. evaluated 17 patients with pre- and post-MI angiography to determine the capability of coronary angiography to predict which arterial segment would be responsible for future acute MI. They concluded that coronary angiography, when performed in stable condition, had a low predictive value to determine the localization of arterial segment related to future acute MI.¹⁰ Little et al. investigated 29 infarction patients after a mean of 24 months (range: 1 to 11 months) following initial angiography and found that

Table 1. Relation between stenosis severity and patient's age and gender

	Stenosis severity		P value
	<50% stenosis	≥50% stenosis	
Sex			
Male	5 (83.37%)	21 (95.42%)	0.74
Female	1 (16.72%)	1 (4.64%)	
Age (y)			
<60	6 (100%)	13 (59.13%)	0.08
≥60	0	9 (40.94%)	

in 66% of them the artery subsequently occluded had <50% diameter stenosis.⁷ Hockett et al. assessed the severity of residual stenosis after successful thrombolysis in 60 patients and found that a residual stenosis <60% was present in 47% of the patients.¹¹ Libby et al. in a compilation of four studies with pre- and post-acute MI angiography reported that only approximately 15% of acute MI cases arose from lesions with a degree of stenosis >60% on an antecedent angiogram.¹²

In the present study and other similar articles reporting on the progression of insignificant lesions to total thrombotic occlusions, the mean interval between angiography and acute MI is 2.5 years with an interval as long as 12 or 18 years. These considerable time intervals could allow for the growth of small lesions before acute occlusion.¹³ On the other hand, these studies indirectly suggest that virtually all of the mature men and women of the industrialized world have a constant predictable risk of catastrophic coronary event. Be that as it may, several recent studies have concluded that the majority of MI cases occur in consequence of significant stenoses.

Ledru et al. evaluated 84 patients with acute MI and a coronary angiogram performed within the preceding 36 months. Coronary angiograms after MI showed that the culprit lesions were more symmetrical and more severe than those in the controls.¹⁴ Joseph et al. came to this point that there was a strong association between the coronary territory of transient defects on myocardial perfusion imaging and the site of subsequent myocardial infarction. These observations support the concept that the hemodynamic significance of a coronary lesion is an important factor in the pathophysiology of acute MI.¹⁵

Histopathological studies from patients with fatal coronary events have consistently shown that at the site of plaque rupture with superimposed occlusive thrombosis, the underlying lesion is severe.¹³ Alderman et al. found out that in 2,161 stenoses assessed by coronary arteriography, the occlusion rate at 5 years' angiographic follow-up was strongly related to the initial lesion severity.¹⁶ Nobuyoshi et al. in 239 prospective patients reported similar findings.⁴

In order to preclude an overestimation of underlying stenosis through superimposed thrombus in our study, thrombectomy with export catheters was performed. Furthermore, thrombectomy was followed by an injection of 200-400 µg intracoronary nitroglycerin to relieve spasm at the culprit site caused by vasoconstrictors such as serotonin thromboxane and endothelin 1.

We did not use intravascular ultrasound (IVUS) or angiography, which could have afforded us a more optimal intraluminal evaluation, although it is deserving of note that the said modalities are time-consuming and not routinely recommended in primary PCI. We succeeded in performing direct stenting in all the cases; nonetheless, there were a few cases of wasting of the balloon during inflation, which assisted us in the estimation of lesion severity. In our study, there were 10

(36%) cases with LAD, 14 (50%) with RCA, and 4 (14%) with LCX occlusions; that is compatible with the infarct location reported in the Ferobert and Kim studies.^{8, 17} The assessment of severity was made via visual estimation as a routine.

The present study has some limitations, the most salient of which is the fact that the study population is a special group amongst acute MI patients because not all AMI patients need thrombectomy. The results of the present study, therefore, cannot be generalized to all acute MI patients. That there were only two women in our study population also renders the results unsatisfactory with respect to gender.

Conclusion

Our study showed that the majority of MI cases occurred in lesions with significant stenoses. This finding is in sharp contrast to the hitherto widespread belief amongst many cardiologists. Future studies with larger study populations are required to further substantiate our findings.

Acknowledgment

This study was approved and was supported by Modarres Hospital, Shaheed Beheshti University of Medical Sciences.

References

1. Virman R, Burke A, Willerson JT, Farb A, Narula J, Kolodgie FD. The pathology of vulnerable plaque. In: Virmani R, Narula J, Leon MB, Willerson JT, eds. *The Vulnerable Atherosclerotic Plaque: Strategies for Diagnosis and Management*. Massachusetts: Black Well; 2007. p. 21-37.
2. Burke AP, Virmani R. Pathophysiology of acute myocardial infarction. *Med Clin North Am* 2007;91:553-572.
3. Shah PK. Mechanisms of plaque vulnerability and rupture. *J Am Coll Cardiol* 2003;41:S15-22.
4. Nobuyoshi M, Tanaka M, Nosaka H, Kimura T, Yokoi H, Hamasaki N, Kim K, Shindo T, Kimura K. Progression of coronary atherosclerosis: is coronary spasm related to progression? *J Am Coll Cardiol* 1991;18:904-910.
5. Giroud D, Li JM, Urban P, Meier B, Rutishauer W. Relation of the site of acute myocardial infarction to the most severe coronary arterial stenosis at prior angiography. *Am J Cardiol* 1992;69:729-732.
6. Ambrose JA, Tannenbaum MA, Alexopoulos D, Hjem dahl-Monsen CE, Leary J, weiss M, Borrico S, Gorlin R, Fuster U. Angiographic progression of coronary artery disease and the development of myocardial infarction. *J Am Coll Cardiol* 1988;12:56-62.
7. Little WC, Constantinescu M, Applegate RJ, Kutcher MA, Burrows MT, Kahl FR, Santamore WP. Can coronary angiography predict the site of a subsequent myocardial infarction in patients with mild to moderate coronary artery disease? *Circulation* 1988;78:1157-1166.
8. Frøbert O, van't Veer M, Aarnoudse W, Simonsen U, Koolen JJ, Pijls NH. Acute myocardial infarction and underlying stenosis severity. *Catheter Cardiovasc Interv* 2007;70:958-965.
9. Antoniucci D, Valenti R, Migliorini A. Thrombectomy during



- PCI for acute myocardial infarction. *Catheter Cardiovasc Interv.* 2008;71:863-869.
10. Trabulo M, Silva JA, Mesquita A, Palos JL, Seabra-Gomes R. Predictive value of coronarography in the localization of arterial lesions responsible for future infarcts of the myocardium. *Rev Port Cardiol* 1996;15:11-16.
 11. Hackett D, Davies G, Maseri A. Pre-existing coronary stenoses in patients with first myocardial infarction are not necessarily severe. *Eur Heart J* 1988;9:1317-1323.
 12. Libby P, Theroux P. Pathophysiology of coronary artery disease. *Circulation* 2005;111:3481-3488.
 13. Fishbein MC, Siegel RJ. How big are coronary atherosclerotic plaques that rupture? *Circulation* 1996;94:2662-2666.
 14. Ledru F, Thérroux P, Lespérance J, Laurier J, Ducimetière P, Guermontprez JL, Diébold B, Blanchard D. Geometric features of coronary artery lesions favoring acute occlusion and myocardial infarction: a quantitative angiographic study. *J Am Coll Cardiol* 1999;33:1353-1361.
 15. Galvin JM, Brown KA. The site of acute myocardial infarction is related to the coronary territory of transient defects on prior myocardial perfusion imaging. *J Nucl Cardiol* 1996;3:382-388.
 16. Alderman EL, Corley SD, Fisher LD, Chaitman BR, Faxon DP. Graphic follow-up of factors associated with progression of coronary artery disease in the coronary artery surgery study (GASS). GASS participating investigators and staff. *J Am Coll Cardiol* 1993;22:1141-1154.
 17. Kim HW, Klem I, Shah DJ, Wu E, Meyers SN, Parker MA, Crowley AL, Bonow RO, Judd RM, Kim RJ. Prevalence and prognostic significance in patients with suspected coronary disease. *PLoS Med* 2009;6:e1000057.