Short and Intermediate Clinical Outcome After Late Coronary Stenting in Myocardial Infarction

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Abstract

Background : The open artery theory has been proposed that late reperfusion of an occluded coronary artery favorably affects clinical outcome. Myocardial reperfusion can be achieved in acute myocardial infarction (AMI) by coronary angioplasty. Coronary stenting improves initial success rate and reduces rate of restenosis. However, there are limited data regarding intermediate outcome of late angioplasty with stenting.

Method : Between June 1998 and August 1999, one hundred and twenty-three patients with AMI, and forty-four patients (37 males, 7 females) underwent late coronary stenting. Mean age was 57 ± 10 years. Echocardiography was performed before the procedure and at 6-months follow-up.

Results : There were 36 Q-MI and 8 non Q-MI. The infarct-related artery (IRA) was left anterior descending artery (LAD) 55 per cent, left circumflex artery (LCX) 15 per cent, and right coronary artery (RCA) 30 per cent. Coronary stenting was successfully performed in all patients. Pre- and post-procedural diameter stenosis were 90.5 ± 8.9 per cent and 2.2 ± 6.5 per cent. Stent indications were suboptimal results (68.2%), intimal dissection (20.4%), and acute closure (11.4%). Over all in-hospital mortality was 2.27 per cent from sudden cardiac death. Mean follow-up was 11.41 ± 4.79 months. There were 1 MI (2.3%), 2 CHF (4.65%), 1 unstable angina pectoris (2.3%), 1 transient ischemic attack (TIA) (2.3%), and no cardiac death. LVEF showed improvement at 6-months follow-up (47.75 ± 11.55% *vs* 54.89 ± 14.76%, p value < 0.001)

Conclusion : Late coronary stenting of the IRA of patients with AMI is feasible, with few complications. There was improved LVEF and intermediate clinical outcome.

Key word : Late Reperfusion, Open Artery Theory, Intracoronary Stenting, Echocardiography

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Several studies of thrombolytic therapy for the late treatment of patients with acute myocardial infarction (AMI), late reperfusion, have shown that such treatment confers survival benefits although late reperfusion does not reduce the infarction size or preserve left ventricular (LV) function[1]. Recently, the open-artery theory has been proposed, which suggests that even if patency of the infarct-related artery (IRA) is achieved late, after AMI is complete, the long-term outcome of such patients is still better than the patients in whom patency is not achieved[2-4]. Nevertheless, thrombolytic therapy beyond 12 hours after the onset of symptoms did not improve the clinical outcome, probably because it was ineffective in establishing coronary patency [5]. With the benefit of percutaneous transluminal coronary angioplasty (PTCA), rates of coronary reperfusion have exceeded those achieved with thrombolytic therapy[6-7]. Nevetheless, PTCA has limitations of the technique[8-10] including problems of residual stenosis, recoil, repeated vessel occlusion, and persistent thrombosis. On the other hand, successful procedures which gained the patency of IRA demonstrated beneficial effects of late mechanical reperfusion on LV remodeling[11-13].

After the first clinical application of intracoronary stents in 1986[14], they have revolutionized the contemporary practice of percutaneous revascularization. Several multicenter trials have shown that stenting is effective in the prevention of abrupt coronary occlusion after threatened or acute vessel closure, resulting in better procedural success, lower acute adverse events, lower angiographic restenosis, greater improvement in coronary flow reserve and myocardial perfusion, and lower intermediate- and long-term target vessel revascularization, compared to conventional balloon angioplasty[15-19]. Recent studies have demonstrated that stenting in AMI is a useful and safe adjunct to PTCA[8,20-35]. However, there are limited data on late coronary artery stenting in patients with AMI. This prospective study aimed to evaluate the safety of late mechanical reperfusion with coronary stenting of the IRA and to assess any long-term beneficial effect over an 18 month period.

MATERIAL AND METHOD

Study Patients

The study population was selected from all patients who were admitted to the coronary care unit (CCU) of Phramongkutklao Hospital, with acute MI from June 1998 to August 1999 and on whom coronary angiography was performed. The diagnosis of AMI was based on typical chest pain lasting > 30 minutes that was accompanied by ST-segment elevation or depression ≥ 0.1 mv in 2 or more adjacent electrocardiographic leads and an increase in creatinine kinase (CK) concentration to three times the upper normal limit with a concomitant increase in the creatinine kinase-MB (CK-MB) fraction. The coronary angiograms were reviewed and stent implantation was offered to the patients when the culprit lesion on the IRA was suitable for stenting. Criteria for coronary artery stenting included (1) the residual diameter of the stenotic lesion was ≥ 50 per cent, (2) suboptimal results, acute or threatened vessel closure. Exclusion criteria for stenting included patients who had (1) a left main coronary stenosis, (2) diffuse, long, tortuous lesions of the IRA, (3) contraindications for the use of aspirin or ticlopidine, (4) refused the procedure and (5) severe peripheral vascular disease. Patients with stent implantation within the time ranging between 12 hours and 16 days after the onset of symptoms were eligible for this study.

Antithrombotic regimen

All patients received 250 mg of ticlopidine and aspirin (gr V) twice daily for at least 2 days before the procedure. After stenting, the patients received 250 mg of ticlopidine twice daily for another 28 days. Aspirin was continued indefinitely.

Catheterization and Angiography

Standard coronary angiogram (CAG) technique with multiple angulated projections to visualize the IRA was performed to evaluate severity and collateral of the lesion.

Angioplasty procedure and Stent implantation

The culprit lesion on the IRA suitable for angioplasty was set to undergo dilatation. Before PTCA, heparin was given to achieve an activated clotting time (ACT) of > 250-300 s. PTCA was performed according to the standard technique to restore patency and anterograde flow. The heparin bolus was repeated hourly during the procedure when needed. For patients who met the criteria for stenting of the IRA, stent implantation was obtained by using a monorail balloon catheter technique. The balloon size was carefully selected so that the dia-

meter at the nominal inflation pressure was equal to or slightly larger than the diameter of the reference vessel, which was estimated angiographically by digital comparison with the diameter of the guiding catheter. The balloon-mounted stent was positioned carefully to ensure coverage of the whole lesion and was deployed at 6-14 atm for 30-60 seconds. A short, noncompliant balloon catheter was then used for final, high-pressure stent dilatation (12-16 atm). Any persistent dissection was treated with additional stents. After the procedure, sheaths were removed when the ACT fell below 170 s. Standardard 12 leads electrocardiograms (ECG) and cardiac enzymes were measured immediately and 6 hours after the proedure.

Methods of assessment

Baseline clinical assessment included peak cardiac enzymes and standard 12 leads ECG after admission.

The echocardiogram was performed in a standardized fashion during admission and will be compared with values obtained 6 months after MI. Most studies were performed with a Hewlett-Packard Sonos 5500 machine. The rest were obtained using a Hewlett-Packard Sonos 1,000 machine.

Follow-up

Patients were clinically followed-up initially at 2-4 weeks in the out patient clinic, and then every 1-3 months. Long-term follow-up was conducted with regard to cardiac events as end points, including cardiac death, recurrent ischemia, and target vessel revascularization (TVR). Long-term follow-up was based on a systemic review of all of the hospital and out patient charts to assess rehospitalization during the follow-up period. Coronary angiography was repeated if recurrent myocardial ischemia was suspected. A follow-up echocardiogram was executed 6 months later.

Definitions

Clinical events were stratified into cardiac and noncardiac origin. Cardiac events included death of cardiac origin, MI, coronary artery bypass surgery, or repeated coronary angioplasty of the stented vessel. All deaths were considered cardiac unless a noncardiac cause was clearly identified. Diagnosis of a recurrent infarction was based on typical chest pain with new electrocardiographic changes and increased creatinine kinase. Bypass surgery referred to emergency or elective bypass operation of the stented coronary artery. Repeated coronary angioplasty was a repeat intervention to the stented lesion. Noncardiac events included death other than from cardiac cause, cerebrovascular accident, severe peripheral vascular event, or severe hemorrhagic event. Diagnosis of cerebrovascular accident was made when a neurological deficit occurred. A severe peripheral vascular event was the formation of a pseudoaneurysm or an arteriovenous fistula at the access site that required operation. A severe hemorrhagic event referred to a bleeding complication that required operation, blood transfusion, or both.

Statistical Analysis

Data are expressed as mean ± SD and compared by the student *t* test for paired data. A value of $p < 0.05$ was taken as statistically significant.

RESULTS

Baseline clinical and angiographic features

Between June 1998 and August 1999, there were 123 patients with AMI. 44 patients were eligible for coronary stenting. The mean age was 57 ± 10 years (range 32 to 77), and there were 37 men and 7 women. The baseline demographic, admission and angiographic features of the patients appear in Tables 1 and 2.

Stent implantation

63 stents were implanted into the 47 lesions of the IRA 6.49 ± 4.87 days after AMI (Table 3). These included 26 (41.1%) Nir Primo stents (Boston Scientific SciMed Corporation, Medinol Ltd.), 16 (25.4%) AVE stents (Medtronic Inc.), 8 (12.7%) MultiLink Duet stents (Guidant Europe SA), 5 (8%) GR II stents (Cook Inc. (Bloomington, IN, USA)), 5 (8%) Crossflex stents (Cordis Corporation), 1 (1.6%) Minicrown stent (Cordis Corporation), 1 (1.6%) Jostent (Jomed International AB (Drottninggatan, Sweden)) and 1 (1.6%) Bestent (Medtronic Instent (Minneapolis, MN, USA)). The indications of stenting were suboptimal results 30 (68.2%), intimal dissection 9 (20.4%), and acute closure 5 (11.4%). 1.43 ± 0.97 stents (range 1 to 6) were implanted per patient. The mean stent size was 3.05 ± 0.44 x 18.13 ± 5.47 mm. TIMI grade 3 flow was achieved in 35 (79.5%) patients and TIMI grade 2 flow was present at the end of the procedure in 9 (20.5%) patients. The minimal lumen dia-

Table 1. Clinical profiles of the patients (N = 44).

		%
Age (year)	57 ± 10	
Male/female	37/7	
Diabetes mellitus	15	34
History of systemic hypertension	18	41
History of hypercholesterolemia	30	68
Current smoker	14	32
Prior MI	7	16
Prior PTCA	3	7
Prior CABG	0	
Prior stroke or TIA	1	2.3
Q-wave infarction	36	82
Non-Q-wave infarction	8	18
ECG infarction location		
Anterior	25	56.8
Inferior	18	40.9
Lateral	1	2.3
Admission Killip class/complications		
Class I	24	55
Class II	8	18
Class III	5	11
Class IV	4	9
Intravenous thrombolytic therapy	12	27
Maximum CK (U/L)	2246.7 ± 1655.6	

Data presented are mean value ± SD or number (%) of the patients

Table 2. Angiographic characteristics.

		%
No. of diseased epicardial vessels		
Single	25	57
Double	12	27
Triple	7	16
IRA distribution (n = 47)		
Left main (LM)	0	
LAD	26	55.3
LCX	7	14.9
RCA	14	29.8
Infarct-related lesion location		
Ostial	2	4.26
Proximal	21	44.68
Middle	20	42.55
Distal or branch	4	8.51
Lesion type		
A	6	12.8
B_1	20	42.6
B_2	16	34
C	4	8.5
Restenotic lesion of the previous angioplasty	1	2.1
TIMI flow grade		
Grade 0/I	19	43.2
Grade II	14	31.8
Grade III	11	25

Table 3. Procedural data before and immediately after stenting.

		%
Time between infarction and intervention (days)	6.49 ± 4.87	
Final TIMI flow grade		
Grade 0/I	0	
Grade II	9	20.45
Grade III	35	79.54
Stent indications		
Suboptimal result	30	68.2
Intimal dissection	9	20.4
Acute closure	5	11.4
Stent size	3.05 ± 0.44 x 18.13 ± 5.47	

meter (MLD) of the IRA increased from 0.34 ± 0.30 mm to 2.81 ± 0.62 mm, and residual stenosis was 2.2 ± 6.5 per cent after the procedure (Table 4). There were no complications related to stent implantation of the non-IRA. Adjunctive antiplatelet agent and/or intraaortic balloon pump (IABP) used in the catheterization laboratory were infrequent; intravenous abciximab was given to 1 patient (2.27%) due to residual intracoronary thrombus, and IABP was supported for a few days in 3 patients (6.8%) due to periprocedural hypotension. No vascular complication at the access site occurred and no adverse reactions to ticlopidine developed.

Acute and follow-up LV functions

Echocardiogram before intervention and at a six-month follow-up was available for 43 patients. Global LV ejection fraction (LVEF) improved from 47.75 ± 11.55 per cent to 54.89 ± 14.76 per cent ($p < 0.001$) as shown in Table 5. Factors related

Table 4. Quantitative angiographic parameters.

	Before stenting	After stening	*p* value
RVD (mm)	2.8 ± 0.4	2.9 ± 0.4	< 0.0001
MLD (mm)	0.34 ± 0.3	2.8 ± 0.6	< 0.0001
DS (%)	90.5 ± 8.9	2.2 ± 6.5	< 0.0001

RVD, reference vessel diameter; MLD, minimal lumen diameter; DS, diameter stenosis

Table 5. Acute and follow-up LV function.

	Acute	Follow-up	*p* value
LVEF (%)	47.75 ± 11.55	54.89 ± 14.76	< 0.001
LVEDV (ml)	110.0 ± 38.7	140.0 ± 48.7	< 0.001
LVESV (ml)	58.6 ± 31.9	68.1 ± 45.3	NS
LVEDD (mm)	48.54 ± 7.65	53.03 ± 7.73	< 0.001
LVESD (mm)	36.9 ± 9.1	37.97 ± 10	NS

to improvement of LVEF were (1) anterior wall MI ($p < 0.001$), (2) inferior wall MI ($p = 0.006$), (3) single vessel disease ($p < 0.001$), (4) non total coronary occlusion ($p = 0.003$). Regional wall motion abnormalities (RWMA) and diastolic function were also improved in the majority (63%).

Clinical outcomes

The mean hospital stay was 8.97 ± 6.48 days. Major in-hospital adverse outcome was found in 1 patient who died 2 days after the intervention date due to sudden death.

During the mean follow-up period of 11.41 ± 4.79 months, 4 patients had adverse events: 1 patient had MI (2.3%), 2 patients evolved CHF (4.65%), 1 patient (2.3%) developed angina pectoris in whom CAG revealed instent restenosis to which successful PTCA was undertaken, and there was no cardiac death. Forty patients (93%) remained free of major cardiac events, of these, 38 patients (88.4%) were free of angina. Non-cardiac events were found in 2 patients, 1 patient suffered minor upper gastrointestinal bleeding and drug-induced cholestatic jaundice which recovered uneventfully after discontinuation of captopril and one patient had transient ischemic attack (TIA) investigation of which showed lacunar infarction by computerized tomography scan of the brain. No one died during the follow-up period.

DISCUSSION

The time course of the recovery of myocardial perfusion and function after infarction is unclear. It is known that patients with a patent IRA as a result of spontaneous or pharmacological reperfusion have better LV function and lower cardiac mortality rates than those with an occluded IRA[3, 36,37], a theory better known as open-artery hypothesis. Early reperfusion is able to salvage the myocardium within the ischemic region, preserve the RWM, and decrease the infarction size. Late reperfusion of an occluded coronary artery beyond the golden time period of myocardial salvage also has a beneficial effect associated with a better clinical outcome[3,37,38]. This effect of late reperfusion is time independent as long as some tissue perfusion remains. The possible mechanisms responsible for potential benefits of an open IRA include (1) improved healing of the infarct tissue and prevention of ventricular remodeling, (2) prevention on infarct expansion and ventricular dilatation, (3) awakening of the hibernating myocardium, (4) electrophysiologic effects, including a reduction in ventricular arrhythmias, lower incidence of induced VT, a reduced frequency of late potentials on a signal averaged ECG[39], and reduction in QT dispersion [40], (5) providing collateral blood supply to distant ischemic myocardium. Reestablishment of perfusion in the IRA is a major prognostic determinant in patients with AMI[36,37].

Meta-analysis performed by Weaver et al [41] revealed that primary PTCA appeared to be superior to thrombolytic therapy for the treatment of AMI in view of cardiac and non-cardiac events and good results have been noted with primary stenting after MI[21,24,28]. Several trials demonstrated that stenting of the culprit lesions compared to PTCA in AMI was safe, feasible, effective[32,42], associated with superior 6 month angiographic results[35,43], lower rate of restenosis and major adverse cardiac events[33], and significantly reduced the need for target vessel revascularization (TVR) at 8 months follow-up[42,44]. The earlier the patency of an occluded vessel is restored, the better the outcome, whether a beneficial effect can be achieved with a delayed restoration of coronary patency after infarction is less clear[3,5]. Improved LV function has been demonstrated with late elective revascularization of the occluded coronary arteries after AMI when viable myocardium is present[12]. The pilot study conducted by Pfisterer et al[45] supported the

hypothesis that myocardial viability persists for 2-3 weeks but not 3 months after MI and it may be worthwhile to restore blood flow to large myocardial territories in this time period. Our results indicated that a late, including very late (from 12 hours to 16 days after AMI), coronary stenting for an occluded IRA provided a high initial success rate and had a beneficial effect on cardiac events over a 6-month period, with an improvement of LV systolic and diastolic function, and a reduction in infarction size being a possible mechanism.

It is of interest that despite this recovery of regional function and improvement of LV function, global ventricular enlargement did occur[46,47]. Contradictory to other studies[13,48,49], that showed smaller LVEDV and LVESV after late mechanical opening of the IRA. These may be from a rather late [50] in time interval between infarction and intervention resulting in less myocardial salvage and less improvement of collateral flow to the infarct zone. Indeed, infarct expansion can be detected within 24 hours from the onset of infarction[51]. Although LVEF and LVESV are clearly related to each other and are the significant predictive factors of prognosis, i.e. patients who died had higher ESV for a given EF than those who survived, this tendency became apparent only when EF was reduced below 50 per cent or ESV was increased above 100 ml[52]. In our study, follow-up data of LVESV and LVEF was 68.1 ± 45.3 and 54.89 ± 14.76, respectively, implying that this relationship may be no longer significant.

Other mechanisms that may be the causes of these differences were the problems of "no reflow" at the myocardial level in those who rapidly achieve and maintain TIMI Grade III flow result in microvascular damage[53]. Although stenting itself reduced the relative risk of restenosis by 20 per cent to 30 per cent[16,17], silent instent restenosis may actually occur[54] even if the majority of patients were free of angina symptoms. Meta-analysis of several trials showed the incidence of about 4 per cent to 16 per cent to display angiographic evidence of restenosis without symptoms of angina at routine follow-up angiography[55]. In our study, direct clinical follow-up was obtained and the majority (90.7%) showed evidence of a good clinical outcome, angiographic restenosis may actually be higher than that of clinical restenosis (9.3%). This disproportion between clinical and anatomical restenosis may be one of the causes of silent ischemia responsible for the ventricular remodeling in our study i.e. increase in LVEDD.

Study Limitations

Potential limitations of this study need to be addressed. First, the patients in this study were not randomized. There was no control group of patients not receiving stents and therefore the clinical outcome can only be compared to similar patient groups treated with or without intracoronary stents in earlier reports. Second, the numbers in our study are relatively small, and therefore the ability of the study to differentiate outcomes between variables is limited. However, further randomized study should be performed to identify the appropriate time for late coronary stenting in patients with AMI.

SUMMARY

The present study suggests that late coronary stenting to the culprit lesion of the IRA of patients with AMI is safe, feasible, having few complications with a high success rate. It results in good clinical outcomes not only a high event-free survival rate but also an improved LV function over the short and intermediate term.

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ผลการรักษาทางคลินิกระยะสั้นและระยะกลางในผู้ป่วยกล้ามเนื้อหัวใจตายเฉียบพลันโดยใช้บอลลูนและขดลวดขยายหลอดเลือดโคโรนารีที่ทำภายหลัง 12 ชั่วโมง

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การเปิดหลอดเลือดโคโรนารีที่อุดตันในผู้ป่วยกล้ามเนื้อหัวใจตายเฉียบพลันที่ทำภายหลัง 12 ชั่วโมง (late reperfusion) อาจยังมีประโยชน์ แต่ผลการขยายหลอดเลือดดังกล่าวด้วยบอลลูนมีข้อจำกัดจากอัตราการตีบซ้ำสูง การใส่ขดลวดถ่าง (stent) ช่วยเพิ่มความสำเร็จในการเปิดหลอดเลือดและลดอัตราการตีบซ้ำได้ แต่ยังขาดข้อมูลการติดตามถึงผลการรักษาด้วยวิธีนี้ จึงได้ทำการศึกษาผู้ป่วยกล้ามเนื้อหัวใจตายเฉียบพลันจำนวน 123 ราย ในระหว่างเดือนมิถุนายน พ.ศ. 2541 – สิงหาคม พ.ศ. 2542 มีผู้ป่วย 44 ราย เป็นผู้ป่วยชาย 37 ราย ผู้ป่วยหญิง 7 รายที่ได้รับการรักษาวิธีนี้ หลังจากมีอาการอย่างน้อย 1 วัน และรับการตรวจคลื่นเสียงสะท้อนหัวใจก่อนรักษาและ 6 เดือนถัดมา ผู้ป่วย 36 รายเป็นกล้ามเนื้อหัวใจตายเฉียบพลันชนิด Q wave (4 รายมีอาการช็อคจากการทำงานของหัวใจล้มเหลว) 8 รายเป็นชนิดไม่มี Q wave หลอดเลือดโคโรนารีที่เป็นสาเหตุของกล้ามเนื้อหัวใจตายเฉียบพลันคือ LAD 55% LCX 15% RCA 30% การเปิดหลอดเลือดได้ผลสำเร็จทุกราย โดยเส้นผ่านศูนย์กลางที่ตีบลดจาก 90.5 ± 8.9% เหลือ 2.2 ± 6.5% ขนาดขดลวดที่ใช้โดยเฉลี่ย 3.05 ± 0.44 x 18.13 ± 5.47 ม.ม. จำนวน 1.43 ± 0.97 อันต่อผู้ป่วย 1 ราย ข้อบ่งชี้ในการใส่ขดลวดได้แก่ ผลการขยายหลอดเลือดต่ำกว่ามาตรฐาน 68.2% ชั้น intima ฉีกขาด 20.4% และเกิดหลอดเลือดปิดเฉียบพลัน 11.4% อัตราตายโดยรวมในโรงพยาบาล 2.27% จากภาวะหัวใจตายเฉียบพลัน ระยะการติดตามผลการรักษาเฉลี่ย 11.41 ± 4.79 เดือน มีผู้ป่วยกล้ามเนื้อหัวใจตายเฉียบพลัน ภาวะเจ็บหน้าอกซ้ำ และภาวะสมองขาดเลือดชั่วคราวอย่างละ 1 ราย หัวใจวาย 2 ราย แต่ไม่มีรายใดเสียชีวิตจากโรคหัวใจ LVEF ที่ 6 เดือนดีขึ้นกว่าก่อนการเปิดหลอดเลือดอย่างมีนัยสำคัญ (47.75 ± 11.55% ก่อน และ 54.89 ± 14.76% หลัง ค่า P < 0.001) โดยสรุปการเปิดหลอดเลือดโคโรนารีที่อุดตันด้วยบอลลูนและขดลวดที่ทำภายหลัง 12 ชั่วโมงในผู้ป่วยกล้ามเนื้อหัวใจตายเฉียบพลันเป็นวิธีที่ได้ผลสำเร็จสูง มีภาวะแทรกซ้อนน้อย ให้ผลดีทั้งในระยะสั้นและระยะกลาง รวมทั้งทำให้การทำงานของหัวใจ (LVEF) ดีขึ้น

คำสำคัญ : การเปิดหลอดเลือดโคโรนารีที่อุดตันในผู้ป่วยกล้ามเนื้อหัวใจตายเฉียบพลันที่ทำภายหลัง 12 ชั่วโมง, ทฤษฎีการเปิดหลอดเลือด, การใส่ขดลวดถ่างหลอดเลือดโคโรนารี, คลื่นเสียงสะท้อนหัวใจ

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