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ORIGINAL PAPER

A. A. Khattab
M. Abdel-Wahab
C. Röther
B. Liska
R. Toelg
G. Kassner
V. Geist
G. Richardt

Multi-vessel stenting during primary percutaneous coronary intervention for acute myocardial infarction

A single-center experience

Abstract *Background* Recanalization of the culprit lesion is the main goal of primary angioplasty for acute ST-segment elevation myocardial infarction (STEMI). Patients presenting with acute myocardial infarction and multivessel disease are, therefore, usually subjected to staged procedures, with the primary percutaneous coronary intervention (PCI) confined to recanalization of the infarct-related artery (IRA). Theoretically at least, early relief of stenoses of non-infarct-related arteries could promote collateral circulation, which could help to limit the infarct size. However, the safety and feasibility of such an approach has not been adequately established. *Methods* In this single-center prospective study we examined 73 consecutive patients who had an acute STEMI and at least one or more lesions ≥70% in a major epicardial vessel other than the infarct-related artery. In the first 28 patients, forming the multi-vessel (MV) PCI group, all lesions were treated during the primary procedure. In the following 45 patients, forming the culprit-only (CO) PCI group, only the culprit lesion was treated during the initial procedure, followed by either planned-staged or ischemia-driven revascularization of the non-culprit lesions. Fluoroscopy time and contrast dye amount were compared between both groups, and patients were followed up for one year for major adverse cardiac events (MACE) and other significant clinical events. *Results* The two groups were well balanced in terms of clinical characteristics, number of diseased vessels and angiographic characteristics of the culprit lesion. In the MV-PCI group, 2.51 lesions per patient were treated using 2.96 ± 1.34 stents (1.00 lesions and 1.76 ± 1.17 stents in the CO-PCI group, both $p < 0.001$). The fluoroscopy time increased from 10.3 (7.2–16.9) min in the CO-PCI group to 12.5 (8.5–19.3) min in the MV-PCI group ($p = 0.22$), and the amount of contrast used from 200 (180–250) ml to 250 (200–300) ml, respectively ($p = 0.16$). Peak CK and CK-MB were significantly lower in patients of the MV-PCI group (843 ± 845 and 135 ± 125 vs 1652 ± 1550 and 207 ± 155 U/l, $p < 0.001$ and 0.01, respectively). Similar rates of major adverse cardiac events at one year were observed in the two groups (24% and 28% in multi-vessel and culprit treatment groups, $p = 0.73$). The incidence of new revascularization in both infarct- and non-infarct-related arteries was also similar (24% and 28%, respectively, $p = 0.73$). *Conclusion* We may state from this limited experience that a multi-vessel stenting approach for patients with acute STEMI and multi-vessel disease is feasible and probably safe during routine clinical practice.

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Ahmed A. Khattab, MD (✉)
Mohamed Abdel-Wahab, MD
Caroline Röther, MD · Branislav Liska, MD
Ralph Toelg, MD · Guido Kassner
Volker Geist, MD · Gert Richardt, MD
Herz-Kreislauf-Zentrum
Segeberger Kliniken, GmbH
Am Kurpark 1
23795 Bad Segeberg, Germany
Tel.: +49-4551/802 98 94
Fax: +49-4551/802 48 05
E-Mail:
ahmed.khattab@segebergerkliniken.de

Our data suggest that this approach may help to limit the infarct size. However, larger studies, perhaps using drug-eluting stents, are still needed to further evaluate the safety and efficiency of this procedure, and whether it is associated with a lower need of subsequent revascularization and lower costs.

Key words stents – multi-vessel disease – acute infarction

Introduction

Infarct size is an important determinant of prognosis in patients with acute myocardial infarction [1]. Early reperfusion of the infarct-related artery (IRA) is undoubtedly the most important intervention to limit the infarct size [2]. Primary stent implantation of the IRA has proven to be the reperfusion modality of choice [3]. Additional factors that may contribute to limitation of infarct size in association with reperfusion include relief of coronary spasm, prevention of damage of microvasculature, improved systemic hemodynamics and development of collateral circulation.

The magnitude of coronary collateral flow is indeed one of the principal determinants of infarct size [4]. Some collaterals are seen in nearly 40% of patients with an acute total occlusion and more begin to appear soon after total occlusion occurs [5]. The presence of collaterals is usually associated with high-grade stenoses and multi-vessel coronary artery disease. However, and according to the recommendations of current guidelines, staged procedures are usually performed in the presence of multi-vessel disease, with the primary procedure being limited to recanalization of the IRA, except for patients presenting with cardiogenic shock [6, 7]. It seems reasonable to investigate an alternative strategy, based on rapid relief of all significant lesions in the non-IRA besides recanalizing the IRA when dealing with multi-vessel disease patients, as an effort to promote collateral circulation and further limit the infarct size. The aim of this study was to evaluate the safety and feasibility of such an approach in an everyday clinical setting.

Methods

Design and population

This is a single-center, prospective observational study to determine the safety and feasibility of multi-vessel stenting during primary percutaneous coronary intervention (PCI). In the period from February 2004 to November 2005, 73 consecutive patients presenting with an acute ST-segment elevation myocardial infarction (STEMI) were included. Inclusion criteria were the presentation within 12 h of the onset of symptoms and the presence of multi-vessel coronary artery disease on angiography suitable for percutaneous intervention. Multi-vessel disease was defined as being the presence of at least one lesion ≥70% in a major epicardial vessel or one of its branches other than the IRA. Patients were excluded when the non-IRA diameter was <2.5 mm or was totally occluded or showed extensive calcification. Patients with significant left main disease or previous myocardial infarction were also excluded. In the first 28 patients, forming the multi-vessel (MV) PCI group, all lesions were treated during the primary procedure. In the following 45 patients, forming the culprit-only (CO) PCI group, only the culprit lesion was treated during the initial procedure, followed by either planned-staged or ischemia-driven revascularization of the non-culprit lesions.

Procedures

Patients were treated according to the standard care of treatment for patients with acute STEMI. A qualifying coronary angiography including a left ventriculography in RAO 30° and LAO 50° was performed. After inclusion, the activated clotting time (ACT) was measured and an intra-arterial heparin bolus was given to maintain the ACT ≥300 s or ≥250 s in case of glycoprotein (GP) IIb/IIIa receptor antagonists' administration. ACT was repeated every 30 min until procedure end. The use of GP inhibitors was strongly recommended (to be in line with current guidelines), but was left to the operator's discretion. A loading clopidogrel dose of 300 mg was given as soon as possible after inclusion and continued as a daily dose of 75 mg for at least 4 weeks. A 500 mg IV aspirin dose was given before PCI and continued indefinitely at a daily oral dose of 100 mg.

Both groups were treated with bare metal stents. In the MV-PCI group, all lesions in the IRA and non-IRAs were treated using the cobalt chromium MULTI-LINK-VISION™ RX Coronary Stent System (Guidant). The IRA was always treated first, then the non-IRAs. Direct stenting was always attempted in

the non-IRAs. In the CO-PCI group, either the cobalt chromium MULTI-LINK-VISION™ RX Coronary Stent System (Guidant) or the cobalt chromium PRO-Kinetic Coronary Stent System (Biotronik) was used during both initial and staged procedures. Only the culprit lesion in the IRA was treated during the initial procedure. The decision for further staged intervention with or without objective evidence of ischemia was left to the treating physician. Angiographic success was defined as in-stent residual stenosis ≤20% with TIMI 3 flow for both the infarct-related as well as the non-infarct-related arteries. Fluoroscopy time and contrast amount were recorded for both groups.

Follow-up and endpoints

Patients received medications according to current guidelines, including aspirin and clopidogrel as previously described, a statin, a beta-blocker and an angiotensin converting enzyme inhibitor. Total CK, CK-MB and Troponin T were measured on admission, every 6 h in the first 24 h, then serially until normalization. Thirty-day major adverse cardiac events (MACE), defined as death, myocardial re-infarction and/or target vessel revascularization (TVR), were recorded. Re-infarction was defined as recurrent chest pain associated with new ischemic electrocardiographic changes or re-elevation of serum cardiac markers. TVR included repeat PCI or bypass surgery. Cerebrovascular accidents, defined as any neurologic event considered to represent a hemorrhagic or nonhemorrhagic stroke, bleeding requiring surgery and/or blood transfusion, and all other complications requiring a specific intervention or leading to prolonged hospitalization were also recorded. Patients were then followed up for one year for further occurrence of MACE as well as for the need for any revascularization (in both target and non-target vessels).

Statistical analysis

All data analyses were performed with the Statistical Package for Social Sciences (SPSS for Windows 13.0, SPSS Inc.) software. Discrete variables are presented as counts and percentages. Continuous variables are presented as mean values ± SD or medians (25th, 75th percentile) when indicated. Proportions were compared by the chi square test or the Fischer's exact test, and continuous variables between groups were compared by the student's *t* test or the Mann-Whitney rank-sum test, as appropriate. A probability value < 0.05 was considered significant.

Results

Study population

Clinical and procedural characteristics of the study groups are shown in Tables 1 and 2. The two groups were similar with regard to age, sex, cardiovascular risk factors, left ventricular ejection fraction, number of diseased vessels, lesion type, use of GP antagonists and angiographic success. Significantly more

Table 1 Baseline clinical and angiographic characteristics of the study patients

Variable	MV-PCI (n = 28)	Culprit-only PCI (n = 45)	P value
Age in years	69 ± 12	65 ± 13	0.29
Male gender, n (%)	21 (75)	35 (78)	0.79
Diabetes mellitus, n (%)	2 (7)	7 (16)	0.47
Hypertension, n (%)	21 (75)	37 (82)	0.46
Dyslipidemia, n (%)	22 (79)	36 (80)	0.88
Current smoking, n (%)	10 (36)	18 (40)	0.71
Anterior infarction, n (%)	16 (57)	11 (24)	0.01
Inferior infarction, n (%)	12 (43)	34 (76)	
Cardiogenic shock, n (%)	1 (3.6)	2 (4.4)	0.86
Left ventricular ejection fraction (%)	47 ± 11	45 ± 11	0.45
2-vessel disease, n (%)	11 (39)	23 (51)	0.33
3-vessel disease, n (%)	17 (61)	22 (49)	0.36
Type of lesion in infarct artery			
A/B1, n (%)	17 (61)	19 (42)	0.12
B2/C, n (%)	11 (39)	26 (58)	0.12

MV-PCI Multi-vessel PCI

Table 2 Procedural characteristics of the study patients

Variable	MV-PCI (n = 28)	Culprit-only PCI (n = 45)	P value
Number of vessels treated, mean	2.17	1	< 0.001
Number of lesions treated, mean	2.51	1	< 0.001
Number of stents used per patient, mean ± SD	2.96 ± 1.34	1.76 ± 1.17	< 0.001
Use of glycoprotein IIb/IIIa inhibitors, n (%)	10 (36)	20 (44)	0.46
Angiographic success in infarct artery, n (%)	26 (93)	38 (84)	0.47
Angiographic success in non-infarct artery, n (%)	26 (93)	–	–
Fluoroscopy time in minutes, median (IQR)	12.5 (8.5–19.3)	10.3 (7.2–16.9)	0.22
Contrast dye amount in ml, median (IQR)	250 (200–300)	200 (180–250)	0.16

MV-PCI Multi-vessel PCI, *IQR* Interquartile range

patients with inferior wall infarction and fewer patients with anterior wall infarction were treated in the CO-PCI group. A similar low rate of cardiogenic shock was observed in both groups.

In the MV-PCI group, 2.51 lesions per patient were treated using 2.96 ± 1.34 stents (1.00 lesions and 1.76 ± 1.17 stents in the CO-PCI group, both $p < 0.001$). The median fluoroscopy time increased from 10.3 (7.2–16.9) min in the CO-PCI group to 12.5 (8.5–19.3) min in the MV-PCI group ($p = 0.22$), and the amount of contrast used from 200 (180–250) ml in the CO-PCI group to 250 (200–300) ml in the MV-PCI group ($p = 0.16$).

In-hospital and 30-day outcome

Peak CK and CK-MB levels were significantly lower in patients of the MV-PCI group (843 ± 845 and 135 ± 125 vs 1652 ± 1550 and 207 ± 155 U/l, $p < 0.001$ and 0.01, respectively). Follow-up data at 30 days are shown in Table 3. There were no significant differences between both study groups in the rates of death, re-infarction or TVR after 30 days. Combined MACE rates were similar (10.7% for the MV-PCI group and 9.1% for the CO-PCI group, $p = 0.82$). Two cases of subacute stent thrombosis were seen in the MV-PCI group; both occurred in the non-IRAs. Both led to a recurrent infarction and were treated by recurrent PCI. In the CO-PCI group, a single case of subacute stent thrombosis was recorded ($p = 0.56$).

One-year outcome

One-year follow-up was completed for 25 of the 28 patients of the MV-PCI group, and for 43 of the 45 patients of the CO-PCI group (follow-up rates of 89% and 96%, respectively). At one year, there were no significant differences between both groups in the cumulative rates of death, recurrent infarction and TVR (Table 4). The cumulative rates of MACE were 24% for the MV-PCI group and 28% for the CO-PCI group ($p = 0.73$). The incidence of any revascularization (in both target and non-target vessels) was also similar in both groups (24% and 28%, respectively, $p = 0.73$).

Table 3 Follow-up data at 30 days

Variable	MV-PCI (n = 28)	Culprit-only PCI (n = 45)	P value
Death, n (%)	1 (3.6)	2 (4.5)	0.84
Recurrent infarction, n (%)	2 (7.1)	2 (4.5)	0.64
Target vessel revascularization, n (%)	2 (7.1)	2 (4.5)	0.64
Stent thrombosis, n (%)	2 (7.1)	1 (2.3)	0.56
Cerebrovascular accidents, n (%)	1 (3.6)	1 (2.3)	0.74
Bleeding requiring transfusion and/or surgery, n (%)	1 (3.6)	2 (4.5)	0.84
Combined MACE[a], n (%)	3 (10.7)	4 (9.1)	0.82

MV-PCI Multi-vessel PCI, *MACE* Major adverse cardiac events
[a] MACE was defined as death, recurrent infarction or target vessel revascularization

Table 4 Cumulative follow-up data at one year

Variable	MV-PCI (n = 25)	Culprit-only PCI (n = 45)	P value
Death, n (%)	2 (8)	3 (7)	0.88
Recurrent infarction, n (%)	2 (8)	5 (12)	0.64
Target vessel revascularization, n (%)	5 (20)	9 (21)	0.93
Combined MACE[a], n (%)	6 (24)	12 (28)	0.73
Non-TVR, n (%)	2 (8)	6 (14)	0.7
Total revascularizations, n (%)	6 (24)	12 (28)	0.73

MV-PCI Multi-vessel PCI, *MACE* Major adverse cardiac events, *TVR* Target vessel revascularization
[a] MACE was defined as death, recurrent infarction or target vessel revascularization

Discussion

The indication for PCI in Europe has shifted towards acute coronary syndromes, as demonstrated by rising rates of interventions for acute myocardial infarction over the last decade [8]. Almost half of the patients presenting with acute STEMI have multivessel coronary artery disease on angiography and 60–90% of patients with cardiogenic shock have multi-vessel disease or left main disease [9]. Current practice usually confines intervention during the primary procedure to the IRA with a deferred approach to the other non-infarct vessels if needed. Beside reperfusion of the IRA, enhancement of collateral flow could help limit the infarct size, a major prognostic factor in patients with acute myocardial infarction. Immediate relief of flow-obstructing stenoses in non-IRAs during the primary procedure could, therefore, be of prognostic value.

Both short- and long-term outcome following multi-vessel intervention in the setting of acute myocardial infarction remain controversial, with only a very limited number of studies analyzing this strategy [10–12]. In a large retrospective study, Corpus et al. showed that multi-vessel PCI during the primary procedure was an independent predictor of MACE at long term mainly due to its high rate of TVR and re-infarction [10]. Furthermore, an increased risk of stent thrombosis was feared in pa-

tients with acute myocardial infarction subjected to multi-vessel stenting during the primary procedure. However, on the other hand, in a small randomized controlled trial, using modern, less thrombogenic stents, in conjunction with more effective antiplatelet drugs, complete revascularization with multi-vessel treatment during primary PCI appeared to be safer, without a significantly higher risk for in-hospital events [11]. Moreover, the high TVR rates associated with multi-vessel stent treatment have been substantially reduced with the introduction of the sirolimus-eluting stent when treating stable coronary artery disease patients [13].

We therefore believed it was reasonable to reinvestigate this approach of complete revascularization in patients with acute STEMI and significant multivessel disease during the primary phase for a positive effect on limiting the infarct size by using modern drug-eluting stents. However, such an approach would involve some difficulties; for this reason, we thought that the first essential step was to evaluate its feasibility and safety using a bare metal stent. We used a third generation stent system having good mechanical properties [14] to facilitate the procedure and followed up the patients for one year. We stented only lesions ≥70% after intracoronary administration of nitroglycerin in the non-IRAs in order to avoid stenting of functionally non-significant lesions.

As expected, both radiation time and contrast amount were higher in the group treated with multivessel stenting. However, the difference between the groups did not reach statistical significance, probably because of the small sample size. Nevertheless, the multi-vessel stenting approach seems feasible from a logistic point of view.

MACE rates at 30 days were similar in both groups (10.7% in the MV-PCI group and 9.1% in the CO-PCI group). These rates are less than those reported by Roe et al. [10], but more than those reported by Di Mario et al. [11]. In the MV-PCI group, two cases of subacute stent thrombosis were seen and both occurred in the non-IRAs, which could be a matter of concern. On revising the acute angiographic results of both cases, a type A dissection at the distal landing zone was identified in one case, and the implanted stent appeared to be oversized causing a distal step-down in the other case. Both cases had a recurrent infarction, were subjected to repeat PCI, and completed their follow-up. Although one of the cases occurred in spite of receiving a GP IIb/IIIa antagonist during the primary procedure, the value of GP IIb/IIIa inhibitors in this complex interventional setting remains undoubted. The single death case that occurred in the MV-PCI group during initial hospitalization was in a 91-year-old male patient with anterolateral wall infarction who presented with cardiogenic shock. Recanalization of the infarct-related artery (LCX) was followed by revascularization of the LAD and RCA in the same setting. The patient died one week later after initial hemodynamic stabilization.

Interestingly, and despite the significantly higher incidence of anterior wall infarctions in the MV-PCI group, peak CK and CK-MB levels were significantly lower, which may reflect a smaller infarct size in the group where complete revascularization was attempted during the initial procedure. This finding has to be interpreted cautiously, since objective evaluation of the final infarct size using echocardiography, nuclear imaging or delayed enhancement cardiac magnetic resonance (CMR) imaging has not been performed. Nevertheless, a recent small study by Hedström et al. [15] demonstrated a strong correlation between peak values of CK-MB and infarct size as estimated by delayed enhancement CMR, suggesting that these peak values can be used to estimate infarct size after primary PCI.

Cumulative MACE rates at one year were also similar in both groups (24% in MV-PCI group and 28% in the CO-PCI group). These rates are also comparable to those recorded by Roe et al. in multivessel and culprit-only PCI patients [12] and to the MACE rates of the culprit-only group in the study reported by Corpus et al. [10]. Again, with the significantly higher incidence of anterior wall infarctions in the MV-PCI group, one might have expected a worse outcome in this group of patients. In the era of thrombolytic therapy, patients with anterior infarction had a substantially worse in-hospital and follow-up clinical course compared with those with inferior infarction [16]. However, with the improvement in and widespread use of primary PCI techniques, a recently published study by Assali et al. [17] reported that mortality at hospital discharge, 30 days, and 6 months was highest in patients with right ventricular (RV) infarction, intermediate in patients with anterior infarction, and lowest in patients without RV myocardial involvement. In the current era, the modern reperfusion modalities and the intensity of medical therapy have probably limited the effect of infarct location on the long-term outcome.

Regarding the use of drug-eluting stents in this complex interventional setting, a recently published meta-analysis of six trials comparing drug-eluting with bare metal stents in acute infarction demonstrated what drug-eluting stents are well known to do, that is, reduce the need for repeat revascularization procedures [18]. However, with the level of uncertainty currently surrounding these devices [19], long-term follow-up data for a larger number of patients are needed to confirm the safety of drug-eluting stents

in this context. Moreover, the differences in real-world patients with acute myocardial infarction who likely have higher medication non-compliance than those enrolled in randomized trials and the risk of stenting deep into the necrotic lipid core of the acute coronary syndrome lesion are valid concerns currently limiting the use of drug-eluting stents during primary PCI [20]. Ongoing trials, such as the HORIZONS trial, may clarify the ultimate risk of drug-eluting stent use in the setting of an acute myocardial infarction, including both long-term safety and efficacy.

Study limitations

This study has several limitations. It is an observational single-center study with a limited number of patients analyzed. Patients were not stratified according to the severity of pump failure, which is an important aspect in patients for whom multi-vessel stenting is being considered. We cannot draw a firm conclusion about the similar incidence of any revascularization procedure in both groups at one year, as patients for whom planned staged procedures for non-culprit lesions were performed were not studied as a separate group. Furthermore, the smaller infarct size in patients in the MV-PCI group as detected by the lower CK and CK-MB values has not been confirmed with an imaging modality, making this finding only suggestive and not conclusive. Finally, although the use of GP inhibitors was strongly recommended (to be in line with current guidelines), the final decision was left to the operator's discretion, which explains why almost two-thirds of our patients were not treated with GP antagonists, which seem necessary in this complex interventional setting.

Conclusion

We can conclude from this limited experience that a multi-vessel stenting approach for patients with STEMI and multi-vessel disease is feasible and probably safe during routine clinical practice. Our data suggest that this approach may help limit the infarct size. We think that the next logical step is to initiate a large randomized trial, perhaps using drug-eluting stents, to further evaluate the safety of this procedure and whether it is associated with a lower need of subsequent revascularization and lower costs.

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