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Single vs multivessel treatment during primary angioplasty: results of the multicentre randomised HEpacoat™ for cuLPrit or multivessel stenting for Acute Myocardial Infarction (HELP AMI) Study

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DESIGN: Prospective randomized, multicentre study.
RATIONALE: Recanalisation of the culprit lesion is the main goal of primary angioplasty for acute myocardial infarction. With the exception of cardiogenic shock, staged procedures are performed in the presence of multivessel disease. The study hypothesis is that with modern non-thrombogenic stents (heparin coated) complete revascularization with multivessel treatment can be safely achieved during the primary angioplasty procedure with a lower need of subsequent revascularization procedures and at a lower cost.
ENDPOINTS: PRIMARY: 12-month incidence of repeat revascularization (any revascularization, infarct related artery as well as non-infarct-related artery). SECONDARY: (1) in hospital repeat revascularization, reinfarction and death; (2) total hospital cost (including a 12 months follow-up period).
METHODS: 69 patients with ST elevation Acute Myocardial Infarction (AMI), <12 hours after symptoms onset, undergoing primary angioplasty, with documented multivessel disease and both culprit lesion and 1 to 3 other lesions suitable for stent implantation. Unbalanced randomization between culprit lesion treatment only ($n = 17$) and complete multivessel treatment ($n = 52$, with 71 additional lesions treated).
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 complete multivessel treatment group 2.36 ± 0.64 lesions per patient were treated using 2.73 ± 0.78 heparin coated stents (1.00 lesions and 1.29 ± 0.61 stents in the culprit treatment group, both $p < 0.001$). The duration of the procedure increased from 53 ± 21 min (culprit treatment group) to 69 ± 32 min ($p = 0.032$) and the amount of contrast used from 242 ± 102 ml (culprit treatment group) to 341 ± 163 ml (multivessel complete treatment), $p = 0.025$. A similar low incidence of in-hospital major adverse cardiac events was observed in the 2 groups (0 and 3.8% in culprit and multivessel treatment groups, $p = 0.164$). The increase in the incidence of new revascularisation in the culprit treatment group at 12 month follow-up was not significant (35 vs 17%, $p = 0.247$) but was sufficient to compensate the initial higher in-hospital cost, with a similar 12 month hospital cost in the 2 groups (€22 330 ± €13 653 vs €20 382 ± €11 671, $p = 0.231$).
CONCLUSION: Multivessel treatment during primary PTCA was safe in this controlled trial. However, when only the culprit lesion was initially treated, the need for subsequent clinically driven revascularization remained low and no clinical or economical advantages were obtainable with a more aggressive initial approach. In clinical practice, a staged approach to multivessel treatment during primary angioplasty avoids to treat unnecessarily non clinically relevant lesions.
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Keywords: acute myocardial infarction – primary PTCA – heparin coating – stents

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Introduction

Prospective randomized studies comparing thrombolysis with primary angioplasty in patients with acute myocardial infarction (AMI) have demonstrated superior reperfusion rates and improved clinical outcome after angioplasty.[1] During primary angioplasty, most operators prefer to treat only the culprit vessel in the initial procedure, leaving other lesions to subsequent elective revascularization. The rationale of this choice is to avoid a severe impairment of left ventricular function in the setting of AMI, if occlusion or complications occur during treatment of a second/third lesion. This 'safe' approach exposes the patient to the risk of new ischemic events and may require a subsequent revascularization procedure. In the PAMI study[2] unscheduled catheterization was performed in 13.3% of patients assigned to PTCA and 6.2% of patients had a repeat in-hospital PTCA because of a recurrent ischemia or abnormal exercise test. Zijlstra[3] also reported a 9% of patients in the angioplasty group with recurrent ischemia and 25% of patients with in-hospital additional procedures (counterpulsation, re-PTCA and CABG). Furthermore, in the PAMI Stent Pilot Trial[4] Stone et al. reported that repeated predischarge catheterization was performed in 18% of patients in both studied groups (PTCA only vs stent) and staged PTCA of non infarct-related vessel was performed in 4.2 and 2.5% of patients, respectively.

The advances in stent design and implantation technique (direct stenting strategy, without predilatation) and the improvement in antiplatelet therapy (periprocedural utilisation of IIb-IIIa inhibitors, association of aspirin and clopidogrel) make treatment of all the suitable lesions feasible in the majority of patients with AMI in the first angioplasty procedure, with minimal risk of procedural complications. Therefore, we hypothesized that complete revascularization will determine lower rates of residual or recurrent ischemia, resulting in a reduced need of repeat revascularization and lower overall cost.

The aim of this multicentre, randomized controlled trial is to establish in patients treated with primary angioplasty with multivessel coronary artery disease the efficacy in reducing the need for repeated revascularization (any revascularization, infarct related artery as well as non infarct-related artery) of a complete revascularization strategy (primary endpoint) and its safety (in hospital MACEs) and overall cost over a period of 12 months (secondary endpoint).

Material and Methods

Patients

The main clinical inclusion criteria were the presence of ischemic chest pain started less than 12 hours before hospital admission and/or ST-segment elevation of at least 1 mm in two or more contiguous electrocardiographic leads (peripheral leads) or 2 mm in the precordial leads (Figure 1). Because an unbalanced randomisation strategy was used, 17 patients were treated with culprit lesion angioplasty and 52 patients underwent complete revascularisation. Informed consent was obtained in patients with these clinical characteristics but enrolment and randomisation was performed only if the pre-treatment angiogram showed multivessel disease with the technical possibility of coronary revascularization with stents of at least two lesions (infarct related artery and one or more – maximum 3 – lesions in a major non-culprit related artery). Balloon dilatation alone could be performed on other lesions located in vessels <2.5 mm in diameter provided at least 1 non-infarct related artery was treated with stents. The main clinical exclusion criteria were the presence of significant lesions in vein grafts or arterial conduits or in segments previously treated with angioplasty or stent implantation, recent thrombolysis (less than 1 week), cardiogenic shock, defined as hypotension with systolic blood pressure <90 mmHg and tachycardia >100 beats/min, not due to hypovolemia or requiring inotropic support or balloon counterpulsation. The main angiographic exclusion criteria were single vessel disease (single territory disease); the presence of left main stenosis of 50% or more; intention to treat more than one totally occluded major epicardial vessel; diffuse calcification or severe tortuosity in the culprit and non-culprit arteries preventing the implantation of the study stents. A side branch larger than 2.0 mm which required to be covered by the stent constituted an exclusion criteria, unless the operator was willing and technically able to maintain patency of this side branch with either further balloon angioplasty or stent placement.

Procedure

After informed consent for the study was obtained, diagnostic coronary arteriography was performed, in general starting with the non-infarct related artery. In the presence of multivessel disease with suitable lesions, randomization was performed. Elective use of abciximab was highly encouraged but left to the operator's discretion.

After wire crossing and/or predilatation, stent implantation in the culprit artery was performed using one or more heparin coated Bx Velocity stents (HepaCoat™ Cordis, Johnson & Johnson, Miami, FL). After stent implantation, noncompliant balloons were used to postdilate the stent, if required. In case of large thrombus burden, the use of distal protection devices was encouraged.

Only in the patients randomized to immediate multivessel treatment, the procedure was completed with revascularization of all suitable lesions, with the use also in these lesions of heparin coated Bx Velocity stents (HepaCoat™ Cordis, Johnson & Johnson, Miami, FL, USA).

In the group randomized to culprit lesion treatment only this artery was stented and subsequent interventions on the non-culprit lesions were performed at the

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Investigator's discretion. Need and timing of the subsequent interventions were decided according to clinical status (persistent or recurrent angina), evidence of ischemia in non-invasive tests (perfusion scintigraphy or stress echocardiogram), angiographic severity of non-culprit lesions and clinical relevance of the affected vessels as well as organization standards of the participating centres. In these subsequent procedures of elective treatment of new lesions or restenoses, the Investigator was also required to use the Hepacoat stents. All patients received aspirin $\geq$100 mg and clopidogrel 75 mg or ticlopidine 500 mg for at least 1 month after the procedure.

All data were prospectively entered into a dedicated case record form allowing definition of success rate and complications. All angiograms of the treated lesions were analysed in an independent Core Laboratory for quantitative angiography.

Statistical Plan

The primary end-point was the rate of repeat revascularization over a period of 12 months. The assumption was that, according to the results of the ARTS study,[5] repeated revascularization would occur in <20% of the patients who underwent complete multivessel revascularization (mainly because of restenosis). Although the patients with revascularization limited to the culprit vessel are all hypothetical candidates for a repeat procedure, it was foreseen that 20–30% of them were not undergoing a second procedure because of clinical stabilisation and absence of symptoms with medical therapy.

With 75% of the patients in the culprit lesion group expected to need a second procedure at 1 year follow-up (mainly because of elective treatment of other lesions), a total of 70 patients randomized in a 3:1 ratio (53 patients assigned to 'multivessel treatment group' and 17 patients assigned to 'culprit vessel treatment group') were required in order to test for a 55% difference in incidence of new revascularization with a power of 0.80 and a two-sided alpha error equal to 0.05. This unbalanced enrolment strategy was adopted in order to increase the reliability of the main secondary end-point, the incidence of adverse in-hospital events in the two groups, testing the safety of the multivessel treatment strategy in acute myocardial infarction. The other secondary endpoint was procedural cost in-hospital and during the 12 month follow-up. The initial procedural cost covered the days spent in an acute coronary intensive care or middle intensive care or in the cardiology ward and the cost of all the material used in the procedure. For the follow-up cost, the Disease Related Group (DRG) based cost of a primary/complex angioplasty procedure (€6660, Region Lombardy, Italy) was used.

Comparisons between groups were performed using the conventional Chi-square test or the Fisher's exact test. All computations were performed using the SAS analysis package.

Results

The clinical characteristics of the two groups are indicated in Table 1. Diabetes was more frequent in the group assigned to treatment of the culprit vessel only. A trend to a shorter interval between onset of symptoms and hospital arrival was present in the group with multivessel treatment (122 ± 97 vs 167 ± 180 min, $p = 0.247$). As required in the protocol, none of the patients treated was in cardiogenic shock. The high prevalence of Q wave and anterior myocardial infarction and the rapid enzymatic wash-out after mechanical recanalisation explain the high maximal release of total and MB-component of creatine kinase. The angiographic and procedural data of Table 2 indicate that absent or minimal flow was present in 70.5 and 78.4% of the infarct related arteries in the culprit and complete treatment groups, respectively (NS). Preprocedural abciximab was used in the vast majority of patients (82.4 and 75%, respectively).

The number of stenosed vessels was 2.29 ± 0.59 vs 2.27 ± 0.53 in the culprit and multivessel treatment groups, with 2.36 ± 0.64 lesions treated in the multivessel treatment group and, by definition, only the culprit lesion in the second group. After the procedure TIMI 3 flow in the infarct related artery was achieved in 88% of patients in both groups.

As indicated in Table 3, in-hospital adverse events were rare, limited to a 89 year old female with a large anterior MI patient in the multivessel treatment group who died because of cardiogenic shock and multiorgan failure 10 days after the procedure and one patient in the same group who required a second urgent angioplasty because of recurrent angina 1 day after the index procedure due to subacute thrombosis in a non-culprit vessel successfully retreated without significant new enzymatic release. At 12 months, 1 recurrent myocardial infarction was observed in each of the two groups. As expected, the need of a new revascularisation procedure was greater, more than twice as frequent in the culprit than in the multivessel treatment group (35.3 vs 15.4%) but the difference was not statistically significant ($p = 0.092$).

Procedural cost was greater in the multivessel treatment (€13 328 $\pm$ €3489) than in the culprit artery group (€12 613 $\pm$ €4490), with no statistically significant difference ($p = 0.263$). This difference, driven by a higher use of consumables, especially stents, contrast and Cath Lab. time, was fully compensated at follow-up by the lower number of procedures required (follow-up cost €7054 $\pm$ €6678 versus €9717 $\pm$ €5220, $p = 0.185$). Thus, at 1 year the overall cost was similar in the two groups (€20 382 $\pm$ €11 671 versus €22 330 $\pm$ €13 653, $p = 0.323$).

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	Culprit (n = 17)	Complete (n = 52)	p
Clinical characteristics			
Age (years)	65.3 ± 7.4	63.5 ± 12.4	0.575
Male (%)	84.6	88.2	0.531
Pre-PTCA (%)	2.0	0	0.796
Pre-CABG (%)	9.6	23.5	0.144
LVEF (%)	48.9 ± 8.6	48.4 ± 9.9	0.883
Diabetes (%)	41.2	11.5	0.012
Hypercholesterolemia (%)	52.9	41.2	0.285
Smoke (%)	81.0	66.6	0.514
Hypertension (%)	58.8	36.5	0.092
Clinical presentation of MI			
Time onset of symptoms-hospital (min)	167 ± 180	122 ± 97	0.247
Time hospital-cath.-lab. (min)	69 ± 54	88 ± 90	0.423
Q-wave MI (%)	75.0	80.4	0.445
r-tPA (%)	5.9	7.7	0.641
IIb-IIIa GP (%)	82.4	75.0	0.397
Anterior MI (%)	58.8	51.9	0.491
Killip 2–3 (%)	18.8	20.0	0.318
SBP (mmHg)	141 ± 24	136 ± 25	0.474
DBP (mmHg)	85 ± 18	83 ± 15	0.528
Heart rate (b/min)	78 ± 18	76 ± 19	0.651
Enzymatic changes			
Total CK (max) (U/L)	2291 ± 1416	2608 ± 2031	0.577
CK-MB (max) (U/L)	202 ± 135	173 ± 147	0.540

CABG, coronary artery bypass grafting; CK, creatine kinase; DBP, diastolic blood pressure; LVEF, left ventricular ejection fraction; PTCA, percutaneous transluminal coronary angioplasty; SBP, systolic blood pressure.

Table 1
Clinical characteristics.

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Discussion

The main finding of this study is that with modern stents treatment of all lesions in the setting of acute myocardial infarction can be accomplished without an increase of in-hospital adverse events and with an acceptable increase in procedural time and contrast utilization. In part, these results can be explained by the favourable mechanical characteristics of the study stent (flexibility, crossing profile, lesion coverage, secure crimping for direct stenting) and by the extremely low incidence of thrombosis observed in a previous large controlled study of acute myocardial infarction with this stable covalent binding of heparin.[6] However, the study failed to show an advantage in terms of prevention of new procedures in the culprit lesion treatment group, primary end-point of the study. We expected that most treatments would have been performed within 1–3 months after myocardial infarction, with some very severe life-threatening stenoses treated during the same hospitalization of the initial primary PTCA and some silent lesions treated at 4–6 months, prompted by the presence of residual ischemia in provocative tests. While the need of a second revascularisation in the multivessel treatment group was within the expected range (17%), in the culprit artery group the percentage of patients actually receiving a second procedure within the 12-month study period was less than half of the expected percentage of 75%. Possible causes include the diligent respect of the recommended strategy which required to limit a new treatment to patients with persistent or recurrent angina or signs of ischemia and the overestimation of lesion severity in non culprit arteries at the time of acute myocardial infarction, as previously described.[7]

Study limitations

The limited sample population is the most important study limitation, aggravated by the unbalanced enrolment in the two groups, with the consequence that the lower than expected difference in new revascularisation procedures did not reach statistical significance (primary endpoint not met). The anticipated 75% rate of revascularisation used to design the statistical plan was partially arbitrary as the available data on reintervention after primary angioplasty concentrate more on repeat culprit lesion revascularisation than on new lesion treatment. Still, the incidence of repeat revascularisation in the culprit treatment group was unexpectedly low (6/17 patients only) possibly because of a selection bias with enrolment of patients with non-culprit lesions which were not of great functional importance (i.e. located in small or distal vessels). Possibly patients with multiple lesions with an unstable appearance, more frequent during acute myocardial infarction,[8] were excluded from

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	Culprit (n = 17)	Complete (n = 52)	p
Two vessel disease	52.9	69.2	0.432
Three vessel disease	47.1	30.8	
Culprit lesion			
TIMI flow class PRE-PTCA 0 (%)	47.1	72.5	
TIMI flow class PRE-PTCA 1 (%)	23.5	5.9	0.141
TIMI flow class PRE-PTCA 2 (%)	11.8	7.8	
TIMI flow class PRE-PTCA 3 (%)	17.6	13.8	
LAD	57%	52%	
LCX	23%	32%	0.218
RCA	20%	16%	
PRE-PTCA reference diameter (mm)	2.92 ± 0.38	3.17 ± 0.48	0.181
PRE-PTCA MLD (mm)	0.21 ± 0.30	0.22 ± 0.41	0.354
PRE-PTCA diameter stenosis (%)	92 ± 10	93 ± 12	0.871
PRE-PTCA lesion length (mm)	11.4 ± 2.9	10.9 ± 4.6	0.289
POST-PTCA MLD (mm)	2.95 ± 0.48	2.87 ± 0.48	0.411
POST-PTCA diameter stenosis (%)	11 ± 7	12 ± 8	0.100
TIMI flow class POST-PTCA 0–1 (%)	0	2 (1/51)*	
TIMI flow class POST-PTCA 2 (%)	12 (2/17)*	10 (5/51)	0.791
TIMI flow class POST-PTCA 3 (%)	88 (15/17)	88 (45/51)	
Main angiographic characteristic non culprit lesions treated (n = 71)			
PRE-PTCA MLD (mm)	–	0.94 ± 0.41	
PRE-PTCA stenosis (mm)	–	66 ± 12	
PRE-PTCA reference diameter (mm)	–	2.79 ± 0.64	
PRE-PTCA lesion length (mm)	–	8.9 ± 7.6	
Procedural characteristics (all lesions)			
Treated lesion/patient	1.00 ± 0	2.36 ± 0.64	0.001
Stent/lesion	1.29 ± 0.61	1.12 ± 0.33	0.008
Stent/patient	1.29 ± 0.61	2.73 ± 0.78	0.001
Mean stent length (mm)	19.9 ± 8.4	16.4 ± 5.0	0.088
Max balloon pressure (atm)	13.6 ± 2.6	14.1 ± 2.5	0.561
Procedure duration (min)	53 ± 24	69 ± 38	0.032
Contrast used (ml)	242 ± 106	341 ± 163	0.025

LAD, left anterior descending artery; LCX, left circumflex artery; MLD, minimal luminal diameter; RCA, right coronary artery; TIMI, thrombolysis in acute myocardial infarction. *In 1 patient in each group culprit vessel TIMI flow could not be determined because of a too short angiographic acquisition. PTCA, percutaneous transluminal coronary angioplast.

Table 2
Angiographic and procedural characteristics.

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randomisation because immediate treatment was performed. Although only the treated lesions were analyzed with quantitative angiography, this hypothesis appears unlikely when the reference diameter, percent diameter stenosis and lesion length of the lesions treated in the second randomised group are considered (Table 2).

A 12 months follow-up is probably insufficient to detect a long lasting benefit offered by the stabilisation of other significant lesions in patients prone to further disease progression. Still a 12-month interval fully covers the process of restenosis and provocative tests were recommended at 4–6 months after myocardial infarction. The similar incidence of death and reinfarction suggest that complete revascularization of all the apparently significant lesions was not clinically beneficial at 1 year but no systematic data are available to assess functional capacity, freedom from angina and left ventricular function in the two groups. In the hospitals involved in this trial, often tertiary referral centres, the clinical decision-making in the follow-up period is normally left to a cardiologist different from the angioplasty operator, often working in a different district hospital. Still, the presence of angiographically significant lesions and of the possible need of a second revascularization procedure, were certainly mentioned in all the patients' discharge summary after angioplasty. The preminence of clinical reasons to guide the final decision to repeat angiography or perform angioplasty was certainly in accordance with the study protocol.

After the significant reduction of restenosis achieved with stents with antiproliferative coatings, the interest of all studies with conventional stents appears limited. Lemos and co-workers reported marked reduction (1.1% at 300 days) of new target lesion revascularisation in 186 consecutive patients undergoing PTCA for acute myocardial infarction with the use of sirolimus eluting stents.[9] A complete or almost complete disappearance of new target lesion revascularisation procedures for

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	Culprit (n = 17)	Complete (n = 52)	p
In hospital			
Death (%)	0	1 (1.9)	0.754
Repeat MI (%)	0	0	–
Urgent PTCA (%)	0	1 (1.9)	0.675
CABG (%)	0	0	–
Any MACE (%)	0	2 (3.8)	0.164
Procedural cost (€)	12613 ± 4490	13328 ± 3489	0.263
1–12 month follow-up			
Death (%)	0	0	0.754
MI (%)	1 (5.9)	1 (1.9)	0.435
PTCA or CABG (%)	6 (35.3)	8 (15.4)	0.092
Any MACE (%)	6 (35.3)	8 (15.4)	0.331
Follow up cost (€)	9717 ± 5220	7054 ± 6678	0.185
12 months cumulative AE			
Death (%)	0	1 (1.9)	0.754
MI (%)	1 (5.9)	1 (1.9)	0.435
PTCA or CABG (%)	6 (35.3)	9 (17.3)	0.174
Any MACE (%)	6 (35.3)	11 (21.1)	0.331
Total Cost (€)	22330 ± 13653	20382 ± 11671	0.323

AE, adverse events; CABG, coronary artery bypass grafting; MACE, major adverse cardiac events; MI, myocardial infarction; PTCA, percutaneous transluminal coronary angioplasty..

Table 3
Clinical events and cost at follow-up.

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restenosis would affect both groups and is unlikely to significantly modify the percent difference in the 12 month incidence of new revascularisation procedures. The fourfold cost increase of drug eluting stents in comparison with other stents is bound to be a significant disincentive to a complete multivessel treatment during the same procedure.

In conclusion, multivessel treatment during primary PTCA was safe but the need for subsequent clinically driven revascularisation was so low that no clinical or economical advantage were obtained when all lesions were treated at the time of the angioplasty of the infarct related artery. The equivalence observed between the two strategies suggests that a symptom driven staged approach to multivessel revascularisation during primary angioplasty for acute myocardial infarction is preferrable as it avoids unnecessary non clinically beneficial treatments.

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