INTERVENTIONAL CARDIOLOGY AND SURGERY

Beneficial effects of abciximab in patients with primary percutaneous intervention for acute ST segment elevation myocardial infarction in clinical practice

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Objectives: To assess the safety and effectiveness of abciximab in patients with ST elevation myocardial infarction (STEMI) treated with primary percutaneous coronary intervention (PCI) in clinical practice.

Methods: Data were analysed of 2184 consecutive patients treated with primary PCI for acute STEMI and either concomitant abciximab or no glycoprotein IIb/IIIa inhibitor (control group), who were prospectively enrolled in the Acute Coronary Syndromes (ACOS) registry between July 2000 and November 2002.

Results: Patients who were treated with abciximab were younger than the control group, and fewer of them had a history of stroke/transient ischaemic attack and systemic hypertension, but more of them had three-vessel coronary artery disease and cardiogenic shock. Cumulated mid-term survival for patients treated with abciximab was significantly higher than in the control group (91% v 79%, log rank $p < 0.05$, median observational time 375 days, range 12–34 months). The Cox proportional hazards model of mid-term mortality after admission with adjustment for baseline characteristics showed that mortality was significantly lower in the abciximab group than in the control group (hazard ratio 0.68, 95% confidence interval 0.49 to 0.95). Whereas overall there was no difference in bleeding complications, patients older than 75 years had more major bleeding events with abciximab (12.5% v 3.4%, $p = 0.03$).

Conclusion: In clinical practice adjunctive treatment with abciximab in patients with primary PCI for acute STEMI was associated with a reduction in mid-term mortality. The subgroup of patients older than 75 years who were treated with abciximab had more major bleeding complications.

Glycoprotein IIb/IIIa inhibitors are potent suppressors of platelet-dependent thrombus formation and they have been shown to reduce cardiac complications in patients undergoing percutaneous coronary intervention (PCI) with acute coronary syndromes without persistent ST elevation.^{1–2} As acute myocardial infarction is due to platelet-dependent thrombus formation, there is a good theoretical background for the use of glycoprotein IIb/IIIa inhibitors in these patients.³

The role of glycoprotein IIb/IIIa inhibitors in patients with PCI for ST elevation myocardial infarction (STEMI) is still under investigation. The use of abciximab along with PCI with and without stents for reperfusion therapy of acute STEMI has been evaluated in five randomised trials: RAPPORT (ReoPro and Primary PTCA Organization and Randomized Trial),⁴ ISAR-2 (Intracoronary Stenting and Antithrombotic Regimen-2),⁵ ADMIRAL (Abciximab Before Direct Angioplasty and Stenting in Myocardial Infarction Regarding Acute and Long-term Follow-up),⁶ CADILLAC (Controlled Abciximab and Device Investigation to Lower Late Angioplasty Complications)^{7–8} and ACE (Abciximab and the Carbostent Evaluation)^{9–10} trial. An analysis of the pooled data of these five trials showed that abciximab was superior to a control arm for the primary end point of death, reinfarction and target vessel revascularisation, as well as for the composite end point of death and reinfarction at 30 days.^{11–12}

Little is known about the effectiveness of abciximab along with PCI for STEMI in clinical practice. We therefore analysed data from the ACOS (Acute Coronary Syndromes)

registry to determine the impact of abciximab on the clinical outcome of patients with STEMI undergoing urgent PCI. This registry reflects the “real world”, as there were no exclusion criteria and consecutive patients were included.

METHODS

The ACOS registry

The ACOS registry^{13–14} is a prospective, multicentre, observational study of current treatment for acute coronary syndromes (STEMI, non-STEMI and unstable angina pectoris). Consecutive patients were recruited from June 2000 to December 2002. The 115 participating hospitals were located throughout Germany and comprised university hospitals, community hospitals and tertiary care centres, all providing intensive care units and early reperfusion therapy. Of 115 hospitals, 108 took part in the follow up after discharge. During the entire study period all patients with acute coronary syndromes were registered prospectively over 12 months and followed up during their clinical course to

Abbreviations: ACE, Abciximab and Carbostent Evaluation; ACEI, angiotensin-converting enzyme inhibitor; ACOS, Acute Coronary Syndromes; ADMIRAL, Abciximab Before Direct Angioplasty and Stenting in Myocardial Infarction Regarding Acute and Long-term Follow-up; CADILLAC, Controlled Abciximab and Device Investigation to Lower Late Angioplasty Complications; ISAR-2, Intracoronary Stenting and Antithrombotic Regimen-2; OR, odds ratio; PCI, percutaneous coronary intervention; RAPPORT, ReoPro and Primary PTCA Organization and Randomized Trial; STEMI, ST elevation myocardial infarction

document patient characteristics, short-term treatment, hospital course and mid-term outcome.

The present study is a retrospective analysis of consecutive patients with STEMI and primary PCI as short term treatment during the first 48 h after hospital admission, who were treated either with abciximab or no glycoprotein IIb/IIIa inhibitor.

Data collection

Data on patient characteristics on admission were recorded, including age, sex, cardiovascular risk factors, concomitant diseases, prior myocardial infarction, prior stroke, prior cardiovascular interventions and chronic medical treatment. Data on symptoms and pre-hospital delay were also collected. Data on ECG findings, biochemical markers, reperfusion therapy and adjunctive treatment were documented. At discharge, outcome and major cardiovascular and cerebrovascular adverse events were recorded. Data on bleeding complications were documented on versions 2 and 3 of the ACOS registry form (925 of 2184 patients) but not on version 1.

Every participating centre was committed by written consent to include every consecutive patient with an acute coronary syndrome. All patients gave informed consent for processing their anonymous data. Data were collected on three record forms by the treating physicians. Completed data sheets were sent to the central data processing centre at the Institut für Herzinfarktforschung Ludwigshafen for uniform monitoring and registration. Source data were verified by comparison of the registry data with hospital records for randomly selected patients in 20 randomly selected participating centres. The study was approved by the ethics committee of the Landesärztekammer Mainz.

The Institut für Herzinfarktforschung Ludwigshafen planned a telephone follow up one year after discharge from hospital to document death, adverse events (recurrent infarction, coronary artery bypass graft, PCI and stroke), drug treatment and mid-term treatment. Patients were followed up after a median of 375 days with a range of 12–34 months. This delay was caused by problems in reaching all patients, surviving relatives or the usual physicians of some patients. Because of this delay, we termed the follow up and mortality “mid-term” instead of “one year”. All data were double keyed and regularly checked for inconsistencies and out-of-range errors.

Definitions

STEMI was diagnosed in the presence of the two following criteria: persistent angina pectoris for ≥ 20 min and ST segment elevation of > 1 mm in ≥ 2 standard leads or ≥ 2 mm in ≥ 2 contiguous precordial leads, or the presence of a left bundle branch block. The diagnosis was later confirmed by an enzyme rise (creatine kinase and its MB isoenzyme, aspartate aminotransferase and lactic dehydrogenase) to at least twice the normal value. The onset of chest pain or the latest intensified or prolonged sensation that led to the consultation of medical services was defined as the onset of STEMI. Pre-hospital delay was defined as the time between onset of STEMI and arrival at the hospital. Stroke was defined as the occurrence of persistent specific neurological deficits. Major bleeding was defined as bleeding that required blood transfusion, and minor bleeding was bleeding without blood transfusion. An absolute contraindication for aspirin was active haemorrhage; absolute contraindications for β blockers were cardiogenic shock, hypotension with systolic blood pressure ≤ 100 mm Hg, bradycardia < 50 beats/min, second- or third-degree atrioventricular block or advanced chronic obstructive pulmonary disease; and absolute contraindications for angiotensin-converting

enzyme inhibitors (ACEIs) were hypotension with systolic blood pressure ≤ 100 mm Hg or cardiogenic shock. Congestive heart failure was defined as New York Heart Association heart failure class II–IV in the presence of reduced left ventricular function on echocardiography (ejection fraction $< 40\%$).

Statistical methods

Data are presented as absolute numbers, percentage or medians with 25th and 75th centiles as appropriate. Whenever possible, percentages were used to describe patient populations. The frequencies of categorical variables in two populations were compared by χ^2 test and by calculating odds ratios (ORs) and 95% confidence intervals (CIs). Continuous variables were compared by two-tailed Wilcoxon rank sum test. The effect of abciximab treatment on mid-term mortality was evaluated by the Cox proportional hazards model by calculating the hazard ratios and the 95% CI. The following variables were entered in the Cox proportional hazards model and then eliminated by backward selection: age, prior myocardial infarction, diabetes mellitus, prior stroke/transient ischaemic attack, three-vessel disease, cardiogenic shock, renal insufficiency, clopidogrel, β blocker, aspirin, ACEI, pre-hospital delay, and stent implantation. Kaplan–Meier curves were used to analyse differences in the survival rates between patients with abciximab and no glycoprotein IIb/IIIa antagonist. Statistical comparison was made by log rank test. The survival functions were compared by log rank test. Values of $p < 0.05$ were considered significant. All p values are results of two-tailed tests. The data were analysed with the SAS system V.8.2 on a personal computer (SAS Institute, Inc, Cary, North Carolina, USA).

RESULTS

Baseline characteristics

From 2000 through to the end of 2002 a total of 16 817 consecutive patients with acute coronary syndromes from 115 hospitals were enrolled into the ACOS registry. For the present analysis 2184 patients with acute STEMI and treatment with primary PCI during the first 48 h were included. Exclusion criteria were pre-treatment with thrombolysis and treatment with glycoprotein IIb/IIIa inhibitors other than abciximab. In the final group, 946 patients (43%) received abciximab and 1238 (57%) no glycoprotein IIb/IIIa inhibitor.

Presenting symptoms and signs

Patients who were treated with abciximab were younger than the control group but the groups did not differ in incidence of prior myocardial infarction, PCI or coronary artery bypass grafts. The abciximab group had a lower prevalence of a history of systemic hypertension, but diabetes mellitus, hypercholesterolaemia and obesity were not different. There were more smokers in the group of patients who were treated with abciximab. No differences were found in initial heart rate and rate of atrial fibrillation on the initial ECG. The incidence of cardiogenic shock was higher in the abciximab group (table 1).

Short term treatment and angiographic findings

More stents were implanted in patients who were treated with abciximab (84.4% *v* 77.5%, $p < 0.001$, OR 1.57, 95% CI 1.26 to 1.96). More of the patients treated with abciximab concomitantly received aspirin, clopidogrel, β blockers, ACEIs and statins during the first 48 h (table 2). Three-vessel coronary artery disease was more common in the abciximab group (table 3).

Table 1 Baseline characteristics of patients with acute ST elevation myocardial infarction treated with primary PCI and either abciximab or no glycoprotein IIb/IIIa inhibitor

	Abciximab (n = 946)	No Gp IIb/IIIa inhibitor (n = 1238)	p Value	Odds ratio (95% CI)
Mean age (range) (years)	62.9 (52.1–70.8)	64.7 (56.0–73.0)	<0.001	
Age ≥75 years	136 (14.4%)	242 (19.5%)	<0.01	0.69 (0.55 to 0.87)
Women	260 (27.5%)	333 (26.9%)	0.76	1.03 (0.85 to 1.25)
Prior MI	118 (12.5%)	171 (13.8%)	0.36	0.89 (0.69 to 1.14)
Prior PCI or CABG	109 (11.5%)	114 (9.2%)	0.08	1.28 (0.97 to 1.69)
Prior stroke/TIA	37 (3.9%)	79 (6.4%)	0.01	0.60 (0.40 to 0.89)
Systemic hypertension	511 (54.0%)	738 (59.6%)	<0.01	0.80 (0.67 to 0.94)
Diabetes mellitus	215 (22.7%)	310 (25.0%)	0.21	0.88 (0.72 to 1.07)
Hypercholesterolaemia*	590 (62.4%)	765 (61.8%)	0.78	1.02 (0.86 to 1.22)
Smoking	42.7 (404%)	34.7 (430%)	<0.01	1.40 (1.18 to 1.67)
Obesity (BMI ≥30 kg/m ²)	208 (23.2%)	242 (22.0%)	0.52	1.07 (0.87 to 1.32)
Renal failure†	24 (2.5%)	33 (2.7%)	0.85	0.95 (0.56 to 1.62)
Prehospital delay >3 h	420 (46.2%)	673 (57.1%)	<0.001	0.64 (0.54 to 0.77)
Cardiogenic shock	93 (7.7%)	74 (4.0%)	<0.001	1.71 (1.25 to 2.36)
Heart rate >100 beats/min	114 (12.1%)	182 (14.7%)	0.07	0.79 (0.62 to 1.02)
Atrial fibrillation	35 (3.7%)	51 (4.1%)	0.62	0.89 (0.58 to 1.39)

*Low density lipoprotein cholesterol >3.37 mmol/l, history of hypercholesterolaemia or drug treatment for hypercholesterolaemia; †creatinine >180 µmol/l.

BMI, body mass index; CABG, coronary artery bypass graft; Gp, glycoprotein; MI, myocardial infarction; PCI, percutaneous coronary intervention; TIA, transient ischaemic attack.

In-hospital outcome

Peak creatine kinase was higher in the abciximab group than in the control group (1068 U/l (464–2225) *v* 870 U/l (359–1898), *p* < 0.001). Hospital mortality did not differ between the abciximab and the control group (table 4). The rate of non-fatal reinfarction and the composite end points of non-fatal reinfarction and non-fatal stroke, the composite end point of death and reinfarction, and the composite end point of death, reinfarction and stroke were significantly lower in the abciximab group than in the control group. Minor and major bleeding complications did not differ between the two groups. Patients older than 75 years, however, had more major bleedings in the abciximab group than in the control group (12.5% *v* 3.4%, OR 4.1, 95% CI 1.03 to 15.92, *p* = 0.03).

Mid-term mortality

The cumulated mid-term survival rate was significantly higher in the abciximab-treated group than in the control group (91% *v* 88%, log rank *p* < 0.05, Kaplan–Meier curve). The Kaplan–Meier survival curves were nearly identical in the first month and progressively diverged during the following 11 months as a consequence of the greater increase in mortality in the control group (fig 1). Cox regression analysis of mid-term mortality after admission with adjustment for baseline characteristics (age, diabetes mellitus, renal insufficiency, three-vessel disease, cardiogenic shock, clopidogrel, β blocker, pre-hospital delay, aspirin, ACEI, stent, prior stroke/transient ischaemic attack and prior myocardial infarction) showed that mortality was lower in abciximab-treated patients than in the control group (hazard ratio 0.68, 95% CI 0.49 to 0.95) (fig 2).

Subgroup analysis of mid-term mortality

After adjustment for baseline characteristics treatment with abciximab was associated with a significantly lower mid-term mortality in men, patients with diabetes mellitus and patients with creatinine concentration < 180 µmol/l. In almost all other subgroups there was a trend towards lower mortality in the abciximab group. The only subgroup that did not benefit from abciximab was the group with creatinine ≥ 180 µmol/l (fig 2).

Mid-term event rate

Non-fatal events did not differ in follow up (reinfarction: 2.2% in the abciximab group *v* 3.3% in the control group, OR 0.66, 95% CI 0.35 to 1.25; stroke: 1.3% *v* 1.2%, OR 1.15, 95% CI 0.46 to 2.84). The rate of repeat PCI (6.8% *v* 7.4%, OR 1.04, 95% CI 0.75 to 1.45) and coronary artery bypass graft surgery (6.8% *v* 7.4%, OR 0.92, 95% CI 0.63 to 1.36) also did not differ between the two groups during follow up.

DISCUSSION

Mid-term mortality of patients with PCI for STEMI in clinical practice, who were treated with abciximab, was significantly lower than that of patients who were not treated with any glycoprotein IIb/IIIa inhibitor. Our analysis is the first evaluation of a large multicentre registry for primary PCI, which confirms the results of randomised trials on the beneficial effect of abciximab. The pooled data of five randomised trials of PCI with or without abciximab showed a reduction of the composite end point of death, reinfarction and target vessel revascularisation and of the composite end point of death and reinfarction at 30 days, but not of 30 day mortality.¹¹ The three-year follow up in ADMIRAL¹⁵ and the one-year follow up in ACE¹⁰ showed that the initial clinical

Table 2 Concomitant treatment during the first 48 h

	Abciximab (n = 946)	No Gp IIb/IIIa inhibitor (n = 1238)	p Value	Odds ratio (95% CI)
Aspirin	899 (95.0%)	1106 (89.3%)	<0.001	2.28 (1.62 to 3.22)
Clopidogrel	820 (86.7%)	934 (75.4%)	<0.001	2.12 (1.69 to 2.66)
β blocker	803 (84.9%)	956 (77.2%)	<0.001	1.66 (1.33 to 2.07)
ACEI	683 (72.2%)	783 (63.2%)	<0.001	1.51 (1.26 to 1.81)
Statin	759 (80.3%)	793 (64.3%)	<0.001	2.13 (1.63 to 2.78)

ACEI, angiotensin-converting enzyme inhibitor; Gp, glycoprotein.

Table 3 Angiographic findings during index hospital stay

	Abciximab (n = 946)	No Gp IIb/IIIa inhibitor (n = 1238)	p Value	Odds ratio (95% CI)
Number of diseased vessels				
1	441 (47.1%)	609 (49.2%)	0.15	0.88 (0.74 to 1.05)
2	254 (27.1%)	383 (30.9%)	0.77	1.03 (0.85 to 1.25)
3	231 (24.7%)	246 (19.9%)	0.02	1.29 (1.05 to 1.58)
Culprit vessel stenosis				
LAD	400 (42.3%)	507 (41.0%)	0.99	1.00 (0.84 to 1.19)
RCA	409 (43.2%)	469 (37.9%)	0.05	1.19 (1.00 to 1.42)
LCX	137 (14.5%)	262 (21.2%)	<0.01	0.70 (0.55 to 0.89)

Gp, glycoprotein; LAD, left anterior descending coronary artery; LCX, left circumflex artery; RCA, right coronary artery.

benefit was preserved. Our study supports these findings in a real-world scenario. Our data are in line with the single-centre registry report of Brener *et al*¹⁶ that abciximab was independently associated with improved survival of patients with acute coronary syndromes.

Overall bleeding complications did not increase. This is in concordance with most of the randomised trials. ISAR-2, CADILLAC and ACE found no difference in bleeding complications between the treatment groups, whereas in ADMIRAL the abciximab group had more minor bleeding events. Only in RAPPORT, the first study published, was there an excess of major bleeding complications in the abciximab cohort, most of them confined to the access site. In the ACOS registry, however, the subgroup of patients older than 75 years had more major bleeding events in the abciximab group than in the control group. This might be explained by co-morbidity in this age group, especially renal insufficiency, with different metabolism of drugs that may not have been adapted to raised creatinine concentration.

In the ACOS registry consecutive patients were enrolled, including high-risk patients with advanced age, renal insufficiency and cardiogenic shock, and resuscitated patients. The mean age of the patients in the ACOS registry and the percentage of female patients were similar to those in other acute coronary syndrome registries.^{13 17 18} Only patients in ACE had a comparable mean age; in all other randomised trials they were significantly younger (59–62 years). CADILLAC had multiple angiographic exclusion criteria eliminating the anatomically difficult cases, and so the patients in that study constituted a rather low-risk population.

In-hospital mortality in the ACOS registry was 5.6% in the abciximab group, compared with lower mortality at 30 days in the randomised trials, ranking from 1.9% and 2.0% (CADILLAC and ISAR-2) to 3.4% and 3.5% (ADMIRAL and ACE). The higher mortality in our population was due to their

higher risk profile. In accordance with the randomised trials, in our population in-hospital mortality did not differ between the abciximab group and the control group. The composite end point of reinfarction and stroke and the composite end point of death, reinfarction and stroke were lower in the abciximab group. The pooled data of the five randomised trials showed a reduction of the composite end point of death and reinfarction at 30 days, whereas in every single trial this difference was not significant. This is in line with our data showing a trend towards a reduction in the composite end point of death and reinfarction with abciximab. Mid-term survival in the ACOS registry population was significantly higher in the abciximab group than in the control group, although there were more patients in cardiogenic shock in the abciximab group and peak creatine kinase was higher. This finding was consistent even after adjustment for baseline variables in the Cox regression model.

Our results should be compared with the results of the ACE trial, which used broad inclusion criteria, including patients in cardiogenic shock and patients with high-risk coronary anatomy. At one year the survival rate in ACE was $95 \pm 2\%$ in the abciximab group and $88 \pm 2\%$ in the control group ($p = 0.017$).¹⁰ This difference in mortality is similar to the results in our population. In subgroup analyses by age (binary cut off 75 years), sex, stent, diabetes, renal insufficiency (creatinine $> 180 \mu\text{mol/l}$) and cardiogenic shock, all point estimates were consistent with the overall result. However, the CI for patients > 75 years of age, for female patients, for patients with and without stents, for patients without diabetes, for patients in cardiogenic shock and for patients with renal insufficiency crossed the line of unity. In patients with creatinine concentration $> 180 \mu\text{mol/l}$ abciximab had no beneficial effect. Our subgroup analysis is limited by small numbers in the subgroups and significance is lost due to the small sample size in some subgroups.

Table 4 In-hospital outcome

	Abciximab (n = 946)	No Gp IIb/IIIa inhibitor (n = 1238)	p Value	Odds ratio (95% CI)
In-hospital mortality	53 (5.6%)	71 (5.7%)	0.89	0.98 (0.68 to 1.41)
Non-fatal reinfarction	18 (2.0%)	50 (4.3%)	<0.01	0.46 (0.27 to 0.79)
Congestive heart failure	26 (2.9%)	31 (2.7%)	0.72	1.10 (0.65 to 1.86)
Non-fatal stroke	1 (0.1%)	6 (0.5%)	0.12	0.22 (0.03 to 1.81)
Non-fatal MACCE	19 (2.1%)	55 (4.7%)	<0.01	0.44 (0.26 to 0.75)
MACCE	71 (7.5%)	121 (9.8%)	0.06	0.75 (0.55 to 1.02)
MACCE	72 (7.6%)	126 (10.2%)	0.04	0.73 (0.54 to 0.98)
All bleeding events*	37/461 (8.0%)	27/464 (5.8%)	0.19	1.41 (0.84 to 2.36)
Major bleeding events*	12/461 (2.6%)	8/464 (1.7%)	0.36	1.52 (0.62 to 3.76)

*Bleeding rate was documented only in versions 2 and 3 of ACOS (Acute Coronary Syndromes) registry, not in version 1.

Gp, glycoprotein; MACCE, major adverse cardiac and cerebrovascular events (death, reinfarction, stroke); MACE, major adverse cardiac event (death and reinfarction).

Figure 1 Kaplan-Meier curve for the first 1.5 years (550 days) after admission of patients with ST elevation myocardial infarction (STEMI) treated with percutaneous coronary intervention (PCI) and either abciximab or no glycoprotein (Gp) IIb/IIIa inhibitor.

As ACOS is a clinical registry for acute coronary syndromes, reasons for our findings are only speculative. One reason for the long-term effect of abciximab may be an anti-inflammatory effect. Such an effect may be particularly prominent in thrombus-containing lesions.⁵ Another reason may be the improved myocardial salvage in the acute phase with preserved left ventricular function by facilitated urgent reperfusion.¹⁹ This may explain why the survival curves were nearly identical in the first month and then progressively diverged as a consequence of greater increase in mortality in the control group. In addition, abciximab improves the recovery of microvascular perfusion and concomitantly improves the regional wall motion compared with conventional heparin treatment.²⁰ Abciximab may attenuate ischaemia-reperfusion injury at the microcirculation level and enhance the degree of myocardial salvage. The primary goal

of treatment for patients with acute myocardial infarction should be to ameliorate myocardial salvage, because the clinical outcome after acute myocardial infarction is mostly determined by infarct size.²¹ In addition, abciximab reduces platelet-leucocyte interaction by reducing the platelet mass in platelet-monocyte aggregates. The long lasting effect of abciximab may be related to plaque stabilisation due to adhesion of platelets and blocked glycoprotein IIb/IIIa receptors.²²

Limitations

The present study was not a randomised, controlled study evaluating the effect of abciximab in patients with PCI for STEMI. In the ACOS registry treatment with abciximab was left to the discretion of the physician. This could have resulted in selection bias, making patients at risk and patients with problems during the intervention more likely to receive abciximab.²³ However, such a bias would most likely be to the disadvantage of abciximab. As abciximab was given at the operator's discretion, patients at higher risk of bleeding were probably not given the drug, which would be a bias in favour of abciximab. Furthermore, the ACOS registry reflects the real world and all patients with PCI for STEMI were included, so therapeutic consequences can be deduced from the results of this study.

Conclusion

In conclusion our data support the findings of randomised trials that abciximab not only reduces the early hazard of ischaemic complications but also offers additional mid-term benefit.¹¹⁻¹⁹ This benefit is not restricted to a certain subgroup. Overall the real-world patient, who is often at high risk, seems to gain even more benefit from treatment with abciximab than do patients in randomised trials with partly low-risk patients. In clinical practice, therefore, adjunctive treatment with abciximab in patients with primary PCI for acute STEMI is associated with a reduction in mid-term mortality.

Figure 2 Subgroup analysis of abciximab treatment versus no glycoprotein (Gp) IIb/IIIa inhibitor and hazard ratio for mid-term mortality in patients with ST elevation myocardial infarction treated with percutaneous coronary intervention. Analysis adjusted for age, prior myocardial infarction, diabetes mellitus, prior stroke/transient ischaemic attack, three-vessel disease, cardiogenic shock, renal insufficiency, clopidogrel, β blocker, aspirin, angiotensin-converting enzyme inhibitor, pre-hospital delay and stenting.

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The participants of ACOS registry are listed in Lorenz *et al.*¹⁴

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