Randomised placebo-controlled trial of abciximab before and during coronary intervention in refractory unstable angina: the CAPTURE study

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Randomised placebo-controlled trial of abciximab before and during coronary intervention in refractory unstable angina: the CAPTURE study

The CAPTURE investigators*

Summary

Background Platelet aggregation is a dominant feature in the pathophysiology of unstable angina. Percutaneous transluminal coronary angioplasty (PTCA) in patients with this disorder carries an increased risk of thrombotic complications. Abciximab (c7E3) blocks the platelet glycoprotein IIb/IIIa receptor, thus preventing platelet adhesion and aggregation. The CAPTURE study was a randomised placebo-controlled multicentre trial to assess whether abciximab can improve outcome in patients with refractory unstable angina who are undergoing PTCA.

Methods The study recruited patients with refractory unstable angina, defined as recurrent myocardial ischaemia under medical treatment including heparin and nitrates. Predefined stopping rules were met at a planned interim analysis of data for 1050 patients, and recruitment was stopped. Data for 1265 patients (of 1400 scheduled) are presented here. After angiography, patients received a randomly assigned infusion of abciximab or placebo for 18-24 h before PTCA, continuing until 1 h afterwards. The primary endpoint was the occurrence within 30 days after PTCA of death (any cause), myocardial infarction, or urgent intervention for recurrent ischaemia. Analyses were by intention to treat.

Findings By 30 days, the primary endpoint had occurred in 71 (11.3%) of 630 patients who received abciximab compared with 101 (15.9%) of 635 placebo recipients (p=0.012). The rate of myocardial infarction was lower in the abciximab than in the placebo group before PTCA (four [0-6%] vs 13 [2-1%], p=0.029) and during PTCA (16 [2-6%] vs 34 [5-5%], p=0.009). Major bleeding was infrequent, but occurred more often with abciximab than with placebo (24 [3-8%] vs 12 [1-9%], p=0.043). At 6-month follow-up, death, myocardial infarction, or repeat intervention had occurred in 193 patients in each group.

Interpretation In patients with refractory unstable angina, treatment with abciximab substantially reduces the rate of thrombotic complications, in particular myocardial infarction, before, during, and after PTCA. There was no evidence that this regimen influenced the rate of myocardial infarction after the first few days, or the need for subsequent reintervention.

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*Writing committee, study organisation, and investigators given at end of paper

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abnormalities compatible with myocardial ischaemia (ST-segment depression, ST-segment elevation, or abnormal T waves), and one or more episodes of typical chest pain, ECG abnormalities, or both, compatible with myocardial ischaemia during therapy with intravenous heparin and glyceryl trinitrate, started at least 2 h previously. The latest episode of ischaemia should have occurred within the 48 h before enrolment, corresponding to Braunwald class III “acute” unstable angina.3,4 All patients had undergone angiography and had significant coronary artery disease with a culprit lesion suitable for angioplasty. Patients were enrolled within 24 h of angiography, and angioplasty was scheduled 18–24 h after the start of study medication. If necessary because of recurrent ischaemia, angioplasty could be done earlier, at the discretion of the investigator.

Reasons for exclusion from the study were: recent myocardial infarction, unless creatine kinase values had returned to below two times the upper limit of normal; features of persisting ischaemia that would require immediate intervention; a greater than 50% occlusion of the left main coronary artery or a culprit lesion located in a bypass graft; bleeding risk factors such as surgery, gastrointestinal or genitourinary bleeding during the 6 weeks before enrolment, or a cerebrovascular accident within the previous 2 years; planned administration of oral anticoagulants, intravenous dextran, or a thrombolytic agent before or during the qualifying angiogram until 4–6 h after discontinuation of heparin and study drug. Special care was given to obtain complete haemostasis at the site of arterial access. During the hospital stay and 30-day follow-up all events and medication were recorded, with special attention to bleeding complications and recurrent ischaemic symptoms.

The primary endpoint in the trial was the occurrence, within 30 days after randomisation, of death (from any cause), myocardial infarction, or an urgent intervention for treatment of recurrent ischaemia (angioplasty, coronary artery bypass surgery, intracoronary stent placement, intra-aortic balloon pump). A Clinical Endpoint Committee reviewed all case-report forms, ECGs, and supporting documents for confirmation that patients met the study entry criteria for refractory unstable angina; the occurrence of endpoints; the frequency of recurrent ischaemia; and important adverse events (bleeding, thrombocytopenia, and stroke).

Myocardial infarction during the index hospital stay was defined as values of creatinine kinase or its MB isoenzyme more than three times the upper limit of normal in at least two samples and increased by 50% over the previous value, or an ECG with new significant Q waves in two or more contiguous leads. Myocardial infarction after discharge was defined as concentrations of creatine kinase or its MB isoenzyme above two times the upper limit of normal, or new significant Q waves in two or more contiguous ECG leads.

Bleeding was classified as major, minor, or insignificant, by previously published criteria.13 Major bleeds were defined as intracranial bleeding or episodes associated with a decrease in haemoglobin of more than 3.5 mmol/L (5 g/L). Bleeding was defined as minor if it was spontaneous and observed as gross haematuria or haematemesis, if blood loss (spontaneous or not) was observed with a decrease in haemoglobin of more than 2.1 mmol/L, or if there was a decrease in haemoglobin of more than 2.8 mmol/L with no significant bleeding site identified. Blood loss insufficient to meet criteria for minor bleeding was

Figure 1: Trial profile
placelet count during or after administration of the study agent to
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Physicians.15 These guidelines state that normovolaemic anaemia
given according to the guidelines of the American College of

relieve symptoms.

symptoms should receive transfusions on a unit-by-unit basis to
is acceptable for patients without symptoms and that those with

Major bleeding

Minor bleeding

Stroke

Stent

Urgent intervention

Death and myocardial infarction

% of patients

Time since enrolment (days)

Myocardial infarction/death

Myocardial infarction

Urgent intervention

Repete PTCA

CABG

Abciximab

Placebo

*p values (two-sided) <0.1 are reported. One patient had both. EExcluding those in patients who underwent CABG.
Table 3: Clinical events, 30 days’ follow-up

CK=creatine kinase; CABG=coronary artery bypass graft.

Table 2: Angiography and PTCA results
classified as insignificant. To account for transfusion, packed-cell

transfusion of packed red blood cells or whole blood within the
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*p values (two-sided) <0.1 are reported. One patient had both. EXcluding those in patients who underwent CABG.
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Recommendation was followed by the Steering Committee. The Safety and Efficacy Monitoring Committee recommended that recruitment should cease. This decision was made earlier than planned in 23 patients (1.8%), 14 of whom were in the placebo group (table 2). According to the investigators, the procedure was not successful, with a residual stenosis greater than 50%, in 70 patients receiving placebo and in 37 receiving abciximab (11·2 vs 6·0%, p=0.001). T treatment with abciximab also resulted in lower rates of urgent repeat PTCA, urgent stent placement, and bypass surgery (table 3); however, these differences were not statistically significant.

The primary endpoint (death, myocardial infarction, or urgent intervention within 30 days of enrolment) occurred in 101 (15·9%) patients in the placebo group and 71 (11·3%) in the abciximab group (p=0·012; table 3, figure 2). This difference was due mainly to a difference in the proportion with myocardial infarction (52 [8·2%] vs 26 [4·1%], p=0·002; table 3). The findings were consistent in all subgroups studied and were independent of age, sex, ECG findings at enrolment, and the presence of diabetes, peripheral vascular disease, or renal dysfunction.

Progression to myocardial infarction during the first 18–24 h after enrolment was rare, despite the inclusion of patients with acute, refractory, unstable angina. Even so, the frequency of myocardial infarction before PTCA was significantly lower in patients receiving abciximab than in those receiving placebo (table 3, p=0·029). Most infarcts occurred during or within 24 h of PTCA (p=0·021, figure 3), whereas infarction rates were low in both groups 2–30 days after PTCA (table 3). The lower rate of myocardial infarction in patients receiving abciximab than in those receiving placebo was found for both Q-wave and non-Q-wave infarcts, and independently of the creatine kinase threshold used to define an infarct (table 3).

Maj or bleeding complications occurred in only 3·8% of patients, although both major and minor bleeding events were more common during treatment with abciximab than during placebo treatment (table 3). No excess strokes were observed with abciximab. In the placebo group, two patients had non-haemorrhagic stroke and one had an intracranial haemorrhage (1, 5, and 7 days after enrolment, respectively). Stroke occurred in a single patient treated with abciximab (15 days after enrolment), but the type of stroke could not be determined. Most bleeding complications occurred at arterial puncture sites. In both treatment groups, bleeding was more common in groups. Event rates were calculated for patients in each treatment group by the Kaplan-Meier method. Fisher’s exact test was used to make pairwise comparisons between the groups for binary measurements. Logistic regression analysis was used to verify the association between bleeding complications, body weight, and heparin dose. Analyses were by intention to treat.

Results

The CAPTURE trial was discontinued after the third interim analysis of 1050 patients. Complete data, fully reviewed by the Clinical Endpoint Committee, were available for 976 patients, and 74 patients had been reviewed partially. By that point, 87 (16·4%) of 532 patients in the placebo group and 56 (10·8%) of 518 in the abciximab group had had a primary endpoint (death, myocardial infarction, or urgent intervention within 30 days). Since the p value for the difference (p=0·0064) was below the prespecified stopping criterion (p=0·0072), and since the data were consistent among all subgroups analysed, the Safety and Efficacy Monitoring Committee recommended that recruitment should cease. This recommendation was followed by the Steering Committee, after consultation with regulatory authorities.

Figure 1 shows the flow of patients through the trial. 1266 patients were enrolled, of 1400 scheduled. Follow-up data were complete for all but one patient (placebo) who withdrew consent after randomisation. Five other patients in the placebo group did not receive placebo (two refused but allowed follow-up and three for logistic reasons). Eight patients did not receive abciximab (one received other therapy, five withdrew consent but allowed follow-up, two for logistic reasons).

The two treatment groups were similar in terms of baseline characteristics (table 1): 73% were male, 50% had a history of angina, and 41% had had a previous myocardial infarction. 72% of patients were enrolled within 6 h of the first (diagnostic) angiogram, 60% had experienced myocardial ischaemia within the 12 h before treatment, and 95% had an ischaemic episode after a minimum of 2 h treatment with nitrates and intravenous heparin. Study drug was started in 1253 patients. It was discontinued early (before 30 min after PTCA) in 86 patients (45 placebo, 41 abciximab) for various reasons, including bleeding (one vs nine), bypass surgery (five vs one), and stent placement (eight vs three). Angioplasty was attempted in 1241 patients (98%). The procedure was done earlier than planned in 23 patients (1·8%), 14 of whom were in the placebo group (table 2). According to the investigators, the procedure was not successful.
patients who received a high dose of heparin during PTCA, and in patients with low body weight. For patients receiving less than 100 IU/kg heparin, the bleeding rates were 1·2% and 4·4% in the placebo and abciximab groups, respectively. The corresponding rates were 2·7% and 6·6% in those receiving 100–149 IU/kg and 7·9% and 14·8% in patients receiving 150 IU/kg heparin or more. In logistic regression analysis both heparin dose per kg (p=0·0001) and use of abciximab (p=0·0008) were significantly related to bleeding risk. By contrast, the reduction in primary endpoint was related only to use of abciximab (p=0·016) and not to heparin dose (p=0·70).

Thrombocytopenia (<100 × 10^9/L) occurred in 5·6% of the abciximab group and 1·3% of the placebo group. Ten patients receiving abciximab had platelet counts below 50 × 10^9/L within 24 h; no placebo recipient had this complication. None of these patients had bleeding complications. Two patients had platelet counts below 20 × 10^9/L. Treatment with study drug (abciximab) was discontinued in five patients, who all received platelet transfusions. Full recovery of platelet counts (to more than 100 × 10^9/L) occurred within 24 h in three patients, within 48 h in three, and within 5 days in three. Follow-up measurements were not available in one patient.

At follow-up 6 months later, death or myocardial infarction had occurred in 56 (9·0%) abciximab-treated patients and 69 (10·9%) placebo recipients (p=0·19, figure 4). Bypass surgery had been required by 33 (5·4%) and 44 (7·1%), respectively (p=0·20). PTCA was needed for similar proportions of patients in both groups, mainly because of restenosis (table 4). Also, medication up to 6 months of follow-up was similar in the two groups (table 5). At 6 months, 242 events had occurred in 193 abciximab-treated patients compared with 274 events in 193 placebo recipients. Thus, the number of events per patient was lower after abciximab (p=0·067).

Discussion
In this trial, patients treated with abciximab had a 29% lower rate of the primary endpoint of death, myocardial infarction, or urgent repeat intervention up to 30 days after PTCA than patients who received placebo. The corresponding reduction in the rate of myocardial infarction was 50%. These findings accord with those of other clinical studies of abciximab and studies with other inhibitors of the platelet glycoprotein IIb/IIIa receptor. In the EPIC (Evaluation of c7E3 for the Prevention of Ischemic Complications) trial, administration of a bolus of abciximab followed by 12 h infusion at the same dose as in our study reduced the rate of death or myocardial infarction from 9·6% (placebo) to 6·1% (p=0·015) and reduced the need for urgent reintervention. A bolus-only regimen was less effective. In our study, pretreatment with abciximab reduced the rates of these events both before and during and immediately after the intervention. In the EPILOG (Evaluation of PTCA to Improve Long-term Outcome by ReoPro GPIIb/IIIa receptor blockade) study, which was also stopped early by the Safety and Efficacy Monitoring Committee, similar results were obtained in patients undergoing elective PTCA. In all three studies with abciximab, the initial treatment effects were maintained for at least 30 days. Most reductions in the same events, although not statistically significant, were observed in patients undergoing PTCA and treated with eptifibatide (p=0·06) or tirofiban (p=0·16). Two of these patients differ from abciximab in that they are small molecules with short half-lives and with more reversible binding to the IIb/IIIa receptor. Nevertheless, these studies consistently support the efficacy of platelet glycoprotein IIb/IIIa receptor blockers in preventing thrombotic complications before and during coronary intervention.

In contrast with other studies, patients in CAPTURE with more severe, refractory, unstable angina were treated during the 18–24 h before planned PTCA. Abciximab resulted in a reduction of events during this period as well as of procedure-related events (figure 2). Apparently, some stabilisation of the unstable plaque was achieved during this treatment period. Since most infarctions occurred during or after the intervention, further event reduction might have been achieved by a longer treatment period before PTCA. PTCA might even have been avoided in some of these patients after stabilisation of the plaque had been achieved. Thus, further studies can be justified to investigate the efficacy of abciximab and related drugs in patients with unstable angina but no planned coronary revascularisation procedure.

As in the EPIC trial11 patients treated with abciximab in CAPTURE had higher rates of complications than those in the placebo group. However, the rate of major bleeding complications was much lower in CAPTURE than in the previous study (3·8 vs 10·6%), by the same definitions. Minor bleeding rates were also lower in our study (4·8 vs 18·8% in EPIC). This reduction was achieved by reduction of the heparin dose, and greater attention to the site for vascular access. However, we also observed a significant relation between heparin use and bleeding risk. A further reduction of heparin doses and early sheath removal in the EPILOG study avoided excess bleeding episodes in patients receiving abciximab. Heparin dose should be monitored closely in patients treated with abciximab. During PTCA, heparin dose should be restricted to 70 IU/kg.

Complications during coronary angioplasty are of either mechanical or thrombotic origin. Balloon angioplasty results in a disruption or dissection of the arterial wall, which leads to exposure of plaque contents, collagen, and other components of the vascular wall to the blood.
resulting in platelet activation and thrombosis. M
echanical complications from large dissection flaps can
now be treated by stents.23-25 Stents may also reduce the
area of exposure of thrombogenic components of the
vascular wall. M any of the thrombotic complications and
associated myocardial infarctions can be avoided when
abciximab is given, whether for 18–24 h before the
procedure as in CAPTURE, or for 10–30 min before and
12 h after intervention as in the other studies.11,12,13 In
CAPTURE, treatment with abciximab was effective both
in patients with thrombus visible on the angiogram and in
those without visible thrombus. Angiography is not,
however, an adequate method to detect thrombus,
particularly when it is adherent to the vessel wall.

In view of the costs of abciximab, some physicians may
decide to use this drug only or mainly to treat thrombotic
complications when these occur during an intervention.
Such use may be effective, but it has not been tested
rigorously in randomised trials. Currently available data
indicate that pretreatment with abciximab is warranted in
all patients undergoing PTCA, and particularly in patients
with refractory unstable angina.

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