Abciximab in Patients With Acute Coronary Syndromes Undergoing Percutaneous Coronary Intervention After Clopidogrel Pretreatment
The ISAR-REACT 2 Randomized Trial

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Context No specifically designed studies have addressed the role of the glycoprotein IIb/IIIa inhibitor abciximab in patients with non–ST-segment elevation acute coronary syndromes (ACS) undergoing percutaneous coronary intervention (PCI) after pretreatment with 600 mg of clopidogrel.

Objective To assess whether abciximab is associated with clinical benefit in high-risk patients with ACS undergoing PCI after pretreatment with 600 mg of clopidogrel.

Design, Setting, and Patients International, multicenter, randomized, double-blind, placebo-controlled study conducted from March 2003 through December 2005, enrolling 2022 patients (mean age, 66 years) with non–ST-segment elevation ACS undergoing PCI.

Interventions Patients were assigned to receive either abciximab (0.25 mg/kg of body weight bolus, followed by a 0.125-µg/kg per minute [maximum, 10 µg/min] infusion for 12 hours, plus heparin, 70 U/kg of body weight) or placebo (placebo bolus and infusion of 12 hours, plus heparin bolus, 140 U/kg). All patients received clopidogrel, 600 mg, at least 2 hours prior to the procedure, as well as 500 mg of oral or intravenous aspirin.

Main Outcome Measures The primary end point was a composite of death, myocardial infarction, or urgent target vessel revascularization occurring within 30 days after randomization; secondary end points were rates of in-hospital major and minor bleeding.

Results Of 2022 patients enrolled, 1012 were assigned to abciximab and 1010 to placebo. The primary end point was reached in 90 patients (8.9%) assigned to abciximab vs 120 patients (11.9%) assigned to placebo, a 25% reduction in risk with abciximab (relative risk [RR], 0.75; 95% CI, 0.58-0.97; P = .03). Among patients without an elevated troponin level, there was no difference in the incidence of primary end point events between the abciximab group (23/499 patients [4.6%]) and the placebo group (22/474 patients [4.6%]) (RR, 0.99; 95% CI, 0.56-1.76; P = .98), whereas among patients with an elevated troponin level, the incidence of events was significantly lower in the abciximab group (67/513 patients [13.1%]) compared with the placebo group (98/536 patients [18.3%]), which corresponds to an RR of 0.71 (95% CI, 0.54-0.95; P = .02) (P = .07 for interaction). There were no significant differences between the 2 groups regarding the risk of major and minor bleeding as well as need for transfusion.

Conclusions Abciximab reduces the risk of adverse events in patients with non–ST-segment elevation ACS undergoing PCI after pretreatment with 600 mg of clopidogrel. The benefits provided by abciximab appear to be confined to patients presenting with an elevated troponin level.

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degree of risk at presentation predicts potential benefit from an early invasive strategy,\textsuperscript{4} current guidelines recommend an early invasive strategy in high-risk patients.\textsuperscript{1,5} In addition, deferral of intervention for prolonged antithrombotic pretreatment does not improve the outcome compared with immediate intervention.\textsuperscript{6}

Although percutaneous coronary interventions (PCIs) are an established therapeutic approach in high-risk patients presenting with ACS,\textsuperscript{7} it is still unclear what the best adjunctive antithrombotic therapies are. There is increasing evidence that treatment with clopidogrel prior to PCI prevents postprocedural ischemic complications\textsuperscript{8,9} and may attenuate differences observed between 2 antithrombotic regimens given periprocedurally.\textsuperscript{10,11} Several studies have shown that a 600-mg loading dose of clopidogrel, compared with the usual 300-mg dose, is as safe and is significantly more rapidly acting and that maximal inhibition of platelet aggregation is achieved within 2 hours after administration.\textsuperscript{12-14} In the first Intracoronary Stenting and Anti-thrombosis Regimen: Rapid Early Action for Coronary Treatment (ISAR-REACT) trial, a 600-mg loading dose of clopidogrel was well tolerated and was associated with such a low frequency of early complications that the use of the glycoprotein (Gp) IIb/IIIa inhibitor abciximab was a useful therapy in patients with non–ST-segment elevation ACS undergoing PCI, even after pretreatment with a 600-mg loading dose of clopidogrel.

Therefore, the objective of the randomized, double-blind, placebo-controlled ISAR-REACT 2 trial was to assess the hypothesis that the Gp IIb/IIIa inhibitor abciximab is a useful therapy in patients with non-ST-segment elevation ACS undergoing PCI, even after pretreatment with a 600-mg loading dose of clopidogrel.

METHODS

Study Population

Patients were enrolled from March 2003 through December 2005. The inclusion criteria were an episode of angina (with an accelerating pattern or prolonged [>20 minutes] or recurrent episodes at rest or with minimal effort) within the preceding 48 hours, accompanied by an elevated troponin T level (>0.03 μg/L) or a new finding of ST-segment depression of at least 0.1 mV or transient (<20 minutes) ST-segment elevation of at least 0.1 mV or new or presumed new bundle-branch block; significant angiographic lesions in a native coronary vessel or venous bypass graft amenable to and requiring a PCI; and written informed consent from the patient.

The exclusion criteria were an ST-segment elevation acute MI (ST-segment ≥0.1 mV elevation in ≥2 contiguous electrocardiographic leads persisting for at least 20 minutes); hemodynamic instability; pericarditis; malignancies with life expectancy less than 1 year; increased risk of bleeding (stroke within the previous 3 months, active bleeding or bleeding diathesis, recent trauma or major surgery in the last month, suspected aortic dissection); oral anticoagulation with a coumarin derivative within the previous 7 days; receipt of a Gp IIb/IIIa inhibitor within the previous 14 days; systolic blood pressure of greater than 180 mm Hg unresponsive to therapy; a hemoglobin level less than 100 g/L or hematocrit less than 34%, or platelet count less than 100 × 10\(^3\) cells/μL or greater than 600 × 10\(^3\) cells/μL; known allergy to the study medication; and pregnancy (present or suspected).

All patients provided written informed consent prior to catheterization for participation in the study, and the study protocol was approved by the ethics committees of all participating centers in Europe and South America.

Study Protocol

Patients in both study groups received oral clopidogrel, 600 mg, at least 2 hours before PCI. They also received 500 mg of oral or intravenous aspirin. The recommended strategy was an early PCI with stenting within 6 hours from establishment of the diagnosis of ACS. After the decision to perform a PCI but before the guide wire had crossed the lesion, patients were randomly assigned in a double-blind manner using sealed envelopes containing the block randomization sequence for each participating center. Patients in the abciximab group received abciximab (0.25 mg/kg of body weight bolus, followed by a 0.125-μg/kg per minute [maximum, 10 μg/min] infusion for 12 hours), plus heparin, 70 U/kg of body weight. Patients in the placebo group received a placebo bolus and infusion of 12 hours plus heparin bolus, 140 U/kg. Double-blinding was achieved by using vials of similar appearance in the 2 groups.

All patients had troponin levels measured prior to randomization, though not necessarily after PCI. Post-PCI MI criteria were based on creatine kinase CK-MB levels (see below).

Coronary stenting was the preferred PCI according to protocol. Postinterventional therapy included 200 mg of as-
pirin indefinitely; 75 mg of clopidogrel twice a day until discharge but for no longer than 3 days, followed by daily administration of 75 mg for at least 6 months; as well as other cardiac medications believed to be appropriate by each patient’s physician. The protocol provided for the performance of electrocardiograms and collection of blood samples for determination of cardiac enzyme levels, hemoglobin levels, and platelet counts every 8 hours for the first 24 hours after the procedure and daily afterwards, until discharge. Three or more cardiac enzyme measurements were obtained in 87% of the patients; 2 or more measurements were obtained in 98%.

A telephone interview was conducted at 30 days, and patients with cardiac complaints were seen in the outpatient clinic for a complete clinical, electrocardiographic, and laboratory check-up. The local research coordinators collected the data and forwarded them to the data coordinating center. High-quality data were ensured by checking source documentation.

Study End Points and Definitions

The primary end point was the combined cumulative incidence of death from any cause, MI, and urgent target vessel revascularization (coronary artery bypass graft surgery or PCI) due to myocardial ischemia within 30 days after randomization. The diagnosis of MI was made according to the Thrombolysis In Myocardial Infarction (TIMI) criteria20 and based on development of new abnormal Q waves (≥30 ms in duration and ≥0.1 mV in depth) in 2 or more contiguous precordial leads or in 2 or more adjacent limb leads, considered to be distinct from the evolution of the index MI; elevation of CK-MB isoenzyme levels (or total CK levels if CK-MB levels were not available) to 3 times the upper limit of normal or greater and, if the pre-PCI CK-MB (or total CK) level was higher than the upper limit of normal, both an increase by at least 50% over the previous value and documentation that the level of CK-MB (or total CK) was decreasing prior to the suspected recurrent MI; recurrent anginal symptoms or new electrocardiographic changes compatible with MI, associated with an elevation of CK-MB level to 50% or more above the peak level prior to randomization for patients in whom there had been no documented decrease of initially elevated CK-MB level prior to randomization; or a CK-MB level more than 10 times the upper limit of normal for patients undergoing coronary artery bypass graft surgery.

The secondary (safety) end points were rates of in-hospital major and minor bleeding. Major and minor bleeding were defined according to the TIMI criteria.20 The criteria for the diagnosis of major bleeding included intracranial or clinically significant overt signs of hemorrhage associated with a greater than 30-g/L decrease in hemoglobin level or an absolute decrease in hemocrit of greater than 15% (when hemoglobin level was not available). The diagnosis of intracranial bleeding required confirmation by computed tomography or magnetic resonance imaging of the head. The criteria for the diagnosis of minor bleeding included observed blood loss and a decrease in hemoglobin level of 30 to 50 g/L (or, when hemoglobin level was not available, a decrease in the hematocrit of 9 to 15 percentage points) or a decrease in hemoglobin level of 40 g/L or greater (or ≥12% in hematocrit) if no bleeding site was identifiable. We also monitored for the occurrence of profound thrombocytopenia (<20 × 10^3 platelets/µL) or need for transfusion of blood products.

All events were adjudicated and classified by an event adjudication committee blinded to the assigned treatment.

Statistical Analysis

We assumed a 30-day incidence of the primary end point of 8% in the abciximab group and of 12% in the placebo group, which corresponds to a risk reduction of 33% with abciximab. With 900 patients included in each group, the trial had 80% power to detect this reduction at a 2-sided α error of .05.

All analyses were performed in a blinded manner regarding the random assignment treatment. Unblinding of the study groups was performed after completion of the statistical analyses. Data are presented as mean (SD) or as counts or proportions (%). Categorical data were compared with the χ² test or Fisher exact test when expected cell values were less than 5. Continuous data were compared with the t test.

The main analysis used a simple binomial rate ratio to calculate the relative risk (RR) and 95% confidence interval (CI) associated with the use of abciximab compared with placebo regarding the primary end point. The Kaplan-Meier method was used to graphically describe cumulative event incidence with differences checked by means of the log-rank χ² test.

A secondary analysis addressed the comparison of abciximab with placebo in the prespecified subsets defined by the presence of an increased troponin level at baseline (>0.03 µg/L) and of diabetes, as well as by the duration of clopidogrel pretreatment. In a multivariable logistic regression model including the baseline characteristics of
the patients, we formerly assessed the interaction of these subset-defining variables (troponin level, diabetes, and duration of clopidogrel pretreatment) with assigned treatment regarding the primary end point. Statistical significance was accepted for a 2-sided \( P < .05 \). S-PLUS version 4.5 (Insightful Corp, Seattle, Wash) was used for all statistical analyses.

### RESULTS

**FIGURE 1** shows the flow of study participants. Patient characteristics are displayed in Table 1 and are notable for a mean age of 66 years and for high proportions of patients with an elevated troponin level (n = 1049 [52%]), multivessel disease (n = 1503 [74%]), and complex lesions (n = 1632 [81%]). Nearly all patients (1960 [97%]) were treated with coronary stents, which were drug-eluting in approximately half (995 [49%]). There were no differences regarding concomitant drug therapy at discharge between the 2 groups. Thirty-day follow-up was complete in all patients.

**Efficacy Analysis**

**FIGURE 2** shows the frequency with which the primary end point was reached in the 30 days after randomization. The primary end point was reached in 90 patients (8.9%) assigned to abciximab vs 120 (11.9%) assigned to placebo. Thus, there was a significant 25% reduction of the risk with abciximab (RR, 0.75; 95% CI, 0.58–0.97; \( P = .03 \)). **TABLE 2** shows the incidence of ischemic events in the 2 study groups, as well as the RR associated with the use of abciximab. Most of the risk reduction caused by abciximab resulted from a reduction in the occurrence of death and MI. **FIGURE 3** shows the frequency with which the primary end point was reached in the subsets of patients defined by the presence or absence of an elevated troponin level. As expected, the incidence of ischemic events was much higher in the subset of patients with elevated troponin levels, irrespective of the study group. There was no difference in the incidence of ischemic events between the abciximab group (23/499 patients [4.6%]) and the placebo group (22/474 patients [4.6%]) among patients without an elevated troponin level (RR, 0.99; 95% CI, 0.56–1.76; \( P = .98 \)). However, among patients with an elevated troponin level, the incidence of ischemic events was significantly lower in the abciximab group (67/513 patients [13.1%]) compared with the placebo group (98/536 patients [18.3%]) (RR, 0.71; 95% CI, 0.54–0.95; \( P = .02 \)) (\( P = .07 \) for interaction). **FIGURE 4** shows the incidence of the primary end point and the relative risk associated with the use of abciximab in various subgroups. Neither diabetes (\( P = .37 \)) nor duration of clopidogrel pretreatment (\( P = .34 \)) showed a significant interac-

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**Table 1. Characteristics of Study Patients**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Abciximab (n = 1012)</th>
<th>Placebo (n = 1010)</th>
<th>( P ) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), y</td>
<td>66.0 (11.0)</td>
<td>66.5 (11.3)</td>
<td>.33</td>
</tr>
<tr>
<td>Women</td>
<td>236 (23.3)</td>
<td>262 (25.9)</td>
<td>.17</td>
</tr>
<tr>
<td>Arterial hypertension*</td>
<td>632 (62.5)</td>
<td>649 (64.3)</td>
<td>.40</td>
</tr>
<tr>
<td>Diabetes*</td>
<td>252 (24.9)</td>
<td>284 (28.1)</td>
<td>.10</td>
</tr>
<tr>
<td>Insulin-treated diabetes</td>
<td>92 (9.1)</td>
<td>86 (8.5)</td>
<td>.65</td>
</tr>
<tr>
<td>Current smoking*</td>
<td>230 (22.7)</td>
<td>219 (21.7)</td>
<td>.57</td>
</tr>
<tr>
<td>Hypercholesterolemia*</td>
<td>623 (61.6)</td>
<td>609 (60.3)</td>
<td>.56</td>
</tr>
<tr>
<td>Prior myocardial infarction</td>
<td>245 (24.2)</td>
<td>243 (24.1)</td>
<td>.94</td>
</tr>
<tr>
<td>Prior aorto-coronary bypass surgery</td>
<td>102 (10.1)</td>
<td>109 (10.8)</td>
<td>.60</td>
</tr>
<tr>
<td>Body mass index, mean (SD)†</td>
<td>27.2 (5.9)</td>
<td>27.3 (5.2)</td>
<td>.51</td>
</tr>
<tr>
<td>Elevated troponin (&gt;0.03 µg/L)</td>
<td>513 (50.7)</td>
<td>536 (53.1)</td>
<td>.28</td>
</tr>
<tr>
<td>Elevated creatine kinase MB (&gt;24 U/L)</td>
<td>226 (22.3)</td>
<td>229 (22.7)</td>
<td>.85</td>
</tr>
<tr>
<td>Hours from clopidogrel loading, mean (SD)</td>
<td>6.1 (9.3)</td>
<td>6.1 (10.2)</td>
<td>.94</td>
</tr>
<tr>
<td>Multivessel disease</td>
<td>753 (74.4)</td>
<td>750 (74.3)</td>
<td>.94</td>
</tr>
<tr>
<td>Left ventricular ejection fraction, mean (SD), %</td>
<td>53.3 (12.3)</td>
<td>53.3 (12.5)</td>
<td>.98</td>
</tr>
<tr>
<td>Target vessel</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left main artery</td>
<td>24 (2.4)</td>
<td>22 (2.2)</td>
<td>.35</td>
</tr>
<tr>
<td>Left anterior descending artery</td>
<td>424 (41.9)</td>
<td>408 (40.4)</td>
<td></td>
</tr>
<tr>
<td>Left circumflex artery</td>
<td>241 (23.8)</td>
<td>262 (26.0)</td>
<td></td>
</tr>
<tr>
<td>Right coronary artery</td>
<td>285 (28.1)</td>
<td>265 (26.2)</td>
<td></td>
</tr>
<tr>
<td>Bypass graft</td>
<td>38 (3.8)</td>
<td>53 (5.2)</td>
<td></td>
</tr>
<tr>
<td>Complex lesions‡</td>
<td>812 (80.2)</td>
<td>820 (81.2)</td>
<td>.59</td>
</tr>
<tr>
<td>Lesion length, mean (SD), mm</td>
<td>14.6 (7.8)</td>
<td>14.7 (8.0)</td>
<td>.79</td>
</tr>
<tr>
<td>Vessel size, mean (SD), mm</td>
<td>2.9 (0.52)</td>
<td>2.9 (0.55)</td>
<td>.80</td>
</tr>
<tr>
<td>Diameter stenosis, mean (SD), %</td>
<td>68.8 (16.5)</td>
<td>69.4 (16.1)</td>
<td>.41</td>
</tr>
<tr>
<td>Type of intervention</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drug-eluting stent</td>
<td>501 (49.5)</td>
<td>494 (48.9)</td>
<td>.58</td>
</tr>
<tr>
<td>Bare-metal stent</td>
<td>484 (47.8)</td>
<td>481 (47.6)</td>
<td></td>
</tr>
<tr>
<td>Balloon angioplasty</td>
<td>27 (2.7)</td>
<td>35 (3.5)</td>
<td></td>
</tr>
<tr>
<td>Concomitant drug therapy at discharge</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Statins</td>
<td>938 (92.7)</td>
<td>921 (91.2)</td>
<td>.22</td>
</tr>
<tr>
<td>β-Blockers</td>
<td>957 (94.6)</td>
<td>950 (94.1)</td>
<td>.62</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>885 (87.5)</td>
<td>905 (89.6)</td>
<td>.13</td>
</tr>
<tr>
<td>Nitrates</td>
<td>33 (3.3)</td>
<td>22 (2.2)</td>
<td>.13</td>
</tr>
<tr>
<td>Calcium antagonists</td>
<td>92 (9.1)</td>
<td>96 (9.5)</td>
<td>.75</td>
</tr>
</tbody>
</table>

Abbreviation: ACE, angiotensin-converting enzyme.

*Arterial hypertension defined as systolic blood pressure ≥140 mm Hg and/or diastolic blood pressure ≥90 mm Hg at least on 2 separate occasions; diabetes mellitus, as the presence of an active treatment with insulin or an oral antidiabetic agent; current smoking, as regular smoking in the prior 6 months; hypercholesterolemia, as documented total cholesterol value of ≥240 mg/dL (≥6.2 mmol/L).

†Calculated as weight in kilograms divided by the square of height in meters.

‡Defined as lesions of type B2 or C according to the modified lesion morphology classification of the American College of Cardiology/American Heart Association.16

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tion with assigned treatment regarding the primary end point.

**Safety Analysis**

TABLE 3 displays the number of patients who experienced bleeding and those who required blood transfusions or developed profound thrombocytopenia. There were no significant differences between the 2 groups regarding the risk of major and minor bleeding as well as need for transfusion. Profound thrombocytopenia was observed only in the group receiving abciximab (8 patients vs none in the placebo group, $P=.008$).

**COMMENT**

We assessed the role of the Gp IIb/IIIa inhibitor abciximab in patients with ACS undergoing PCI after pretreatment with a 600-mg loading dose of clopidogrel. Abciximab reduced the risk of adverse ischemic events; the benefit provided by this drug was confined to patients with an elevated troponin level. This benefit was not offset by a significant increase in bleeding.

The patients in the present trial were characterized by a high proportion of elderly patients (mean age, 66 years) with complex lesions (81%) and with elevated troponin levels (52%). An important difference between this and prior trials of Gp IIb/IIIa inhibitors in patients with ACS is that, in the prior trials, randomization was performed before angiography. As a result, in each of those trials some patients without coronary artery disease—and, consequently, with lower risk—were enrolled. In contrast, in the present trial, patients were randomly assigned after the diagnosis of obstructive epicardial coronary artery disease was established angiographically. These characteristics and the trial design increased the risk profile of the patients in this trial and should be taken into account when making comparisons between this and other trials in patients with ACS.7,22,23

Special consideration should be given to the C7E3 Fab Antiplatelet Therapy in Unstable Refractory Angina...
Major bleeding although none reached statistical significance when considered individually. The adverse event rate observed in patients without an elevated troponin level was similar to that seen previously in the first ISAR-REACT trial, an earlier trial of abciximab in stable patients undergoing elective PCI. In that subset of patients without elevated troponin levels, as in prior studies, abciximab did not provide added clinical benefit to patients pretreated with 600 mg of clopidogrel. In contrast, among patients with an elevated troponin level, the risk of recurrent ischemic events was considerably higher than that in those without elevated troponin levels and was reduced by 29% by abciximab. This is in line with previous studies that have shown that patients with ACS and elevated troponin levels derive the greatest benefit from therapy with Gp IIb/IIIa inhibitors. Pretreatment with a loading dose of clopidogrel has become standard therapy in patients undergoing PCI. Recent studies have shown that not only faster but greater platelet inhibition can be achieved by use of a loading dose of 600 mg of clopidogrel as compared with 300 mg. This has also been associated with better protection from myocardial injury during PCI. In stable patients undergoing elective PCI, pretreatment with 600 mg of clopidogrel provides platelet inhibition sufficient to enable a safe procedure without the need of Gp IIb/IIIa inhibitors.

We found a 25% reduction of the risk of recurrent ischemic events among patients assigned to abciximab. Notably, the gradient in favor of abciximab was seen for all components of the primary end point—death, MI, and urgent target vessel revascularization—although none reached statistical significance.

Table 3. In-Hospital Bleeding Events, Need for Transfusion, and Thrombocytopenia

<table>
<thead>
<tr>
<th>Event</th>
<th>Abciximab (n = 1012)</th>
<th>Placebo (n = 1010)</th>
<th>RR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major bleeding*</td>
<td>14 (1.4)</td>
<td>14 (1.4)</td>
<td>1.00 (0.50-2.08)</td>
</tr>
<tr>
<td>Intracranial bleeding</td>
<td>1 (0.1)</td>
<td>1 (0.1)</td>
<td>1.00 (0.01-78)</td>
</tr>
<tr>
<td>Minor bleeding*</td>
<td>42 (4.2)</td>
<td>33 (3.3)</td>
<td>1.27 (0.81-1.99)</td>
</tr>
<tr>
<td>Transfusion of blood products</td>
<td>25 (2.5)</td>
<td>20 (2.0)</td>
<td>1.25 (0.70-2.23)</td>
</tr>
<tr>
<td>Profound thrombocytopenia†</td>
<td>8 (0.8)</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

*See “Methods” section for definitions of major and minor bleeding.
†Platelet count <20 x 10⁶ cells/L.

Error bars indicate 95% confidence intervals.
and received unfractionated heparin during the procedure. We used a larger heparin bolus than is traditionally used in the United States, but, at the same time, there was no activated clotting time guidance and no additional heparin doses were administered during the procedure. Alternative strategies based on the direct thrombin inhibitor bivalirudin administered with or without Gp IIb/IIIa antagonists are currently being evaluated in patients with ACS who are not routinely pretreated with 600 mg of clopidogrel. In addition, novel, rapidly acting P2Y12 antagonists such as prasugrel can be assessed as alternatives to clopidogrel loading and may modify the benefit profile of abciximab in patients with ACS treated invasively.

CONCLUSION

Abciximab reduces the risk of adverse events in patients with non–ST-segment elevation ACS who are undergoing PCI after pretreatment with 600 mg of clopidogrel. The benefits of abciximab appear to be confined to patients with an elevated troponin level.

REFERENCES


