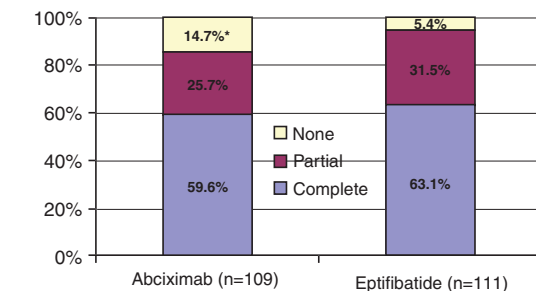


### EVA-AMI Trial: Eptifibatide Not Inferior to Abciximab in Primary PCI for Acute MI

A new study shows that a double bolus and infusion of eptifibatide is not inferior to abciximab when used during primary PCI for acute MI.<sup>1</sup> A total of 429 patients were randomized after MI who were about to undergo planned primary PCI within 12 h to receive abciximab (bolus and 12-h infusion) or eptifibatide (2 boluses and 24-h infusion), along with aspirin, clopidogrel, and either enoxaparin or unfractionated heparin. The primary end point was a surrogate for reperfusion and mortality—greater than 70% resolution of ST-segment elevation between baseline and 1 h after PCI in the per-protocol population.<sup>2,3</sup> The 95% confidence interval for the difference between treatment arms in the incidence of the primary end point fell within the boundary for noninferiority (-15% difference). A significantly greater number of patients in the abciximab group than in the eptifibatide group showed persistent ST-segment elevation at 1 h after PCI (Fig. 1). Preliminary analyses of in-hospital clinical events showed no major differences in the incidence of death, reinfarction, heart failure, target-vessel revascularization, bypass surgery, or stroke. Major bleeding occurred in 0% of patients given abciximab and in 1.8% of patients given eptifibatide; the corresponding rates of minor bleeding were 4.5% and 4.1%, respectively. The researchers conclude that eptifibatide may be a safe and effective alternative to abciximab in the prevention of ischemic events after primary PCI for acute MI.

**Figure 1. Per-Protocol Analysis: Incidence of ST-Segment Resolution 1 Hour After PCI in the EVA-AMI Trial.**



P = .021 vs eptifibatide.

### BRIEF-PCI: Shorter Eptifibatide Infusion Safe, Effective After Nonemergency PCI

The standard regimen for eptifibatide treatment in patients undergoing PCI carries several disadvantages, including prolonged hospitalization, increased costs, and possible excess bleeding. Furthermore, the regimen was established before the advent of routine dual antiplatelet therapy (with aspirin and clopidogrel) and stenting. A study now shows that a shorter eptifibatide infusion can be safe and effective in patients undergoing nonemergency PCI.<sup>4</sup> After receiving the standard eptifibatide boluses during nonemergency PCI (with stenting in ~41% of cases), 624 patients with acute coronary syndromes (> 48 hours for MI) or stable angina were randomized in a double-blind, placebo-controlled fashion to receive either a < 2-hour or 18-hour eptifibatide infusion. Most patients also received clopidogrel before PCI. The primary end point was postprocedural myonecrosis within 24 hours. Adjudicated secondary end points included 1) the composite of death, MI, or target-vessel revascularization (TVR) at 30 days, and 2) the composite of the triple end point plus in-hospital major bleeding. The brief eptifibatide infusion was shown to be not inferior to the standard regimen in terms of the primary end point, overall and among patients who did or did not receive "adequate" pretreatment with clopidogrel. The rates of the triple and quadruple end points likewise did not differ significantly, although the rate of major bleeding alone was significantly higher in the standard-infusion group (4.2% vs 1% for the brief-infusion group; P = 0.02). Researchers conclude that eptifibatide infusion can safely be shortened to < 2 hours after successful nonemergency PCI with no increase in postprocedural myonecrosis. This approach also may reduce the risk of major bleeding.

### Strong Platelet Response to Clopidogrel Equals Less MI Risk During Stenting

Bliden and colleagues recently studied the correlation between inhibition of platelet aggregation induced by adenosine diphosphate (ADP) and inhibition after stimulation with other agonists.<sup>5</sup> They then assessed how such correlations related to the release of cardiac biomarkers in 50 patients undergoing elective stenting who received a 600 mg loading dose of clopidogrel plus aspirin and bivalirudin. Patients were classified as low responders (LR), defined as ≤ 10% relative platelet inhibition (RPI); moderate responders (MR), defined as > 10% to < 30% RPI; or high responders (HR), defined as > 30% RPI. None of the patients had elevated biomarker levels at 2 hours after stenting. By 24 hours after stenting, 3 patients in the LR group (n = 7) had an elevated troponin 1 level, 2 had a creatinine kinase-MB level > 3 times the upper normal limit, and 1 had a CK-MB level > 1-3 times the upper normal limit. The corresponding patient numbers in the MR group (n = 20) were 1, 1, and 1. No patient in the HR group (n = 23) had biomarker release at any time. Patients who show a strong response to clopidogrel appear to be protected from periprocedural MI.

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