

# Multivessel or Culprit-Only Stenting in Patients with Unstable Angina or NSTEMI

Shishehbor MH, Lauer MS, Singh IM, et al. In unstable angina or non-ST-segment acute coronary syndrome, should patients with multivessel coronary artery disease undergo multivessel or culprit-only stenting? *J Am Coll Cardiol* 2007;49:849–54.

## Study Overview

**Objective.** To evaluate nonculprit multivessel stenting compared with culprit-only stenting in patients with multivessel disease presenting with unstable angina or non-ST-segment elevation myocardial infarction (NSTEMI).

**Design.** Retrospective cohort study.

**Setting and participants.** Patients with unstable angina or NSTEMI were selected from an ongoing registry of patients undergoing percutaneous coronary intervention (PCI) at the Cleveland Clinic. All patients who had undergone PCI with bare-metal stents from January 1995 to June 2005 were included. Patients were excluded if they had chronic total occlusions or staged procedures or if they had undergone previous coronary artery bypass graft surgery. Unstable angina was defined as new-onset, progressive, or postinfarct chest pain at rest, and NSTEMI was defined as troponin elevation with electrocardiographic (ECG) changes or angina. A culprit lesion was defined by angiographic report, ECG, echocardiogram, and nuclear stress test. All patients had at least 2 vessels with  $\geq 50\%$  stenosis. Angiographic severity of coronary artery disease (CAD) was assessed using the Duke CAD Prognostic Score.

**Main outcome measures.** The primary endpoint was a composite of death, myocardial infarction (MI) requiring

hospitalization (excluding periprocedural MI), or any target or nontarget vessel revascularization (PCI or coronary artery bypass grafting). Adverse cardiovascular events were determined by telephone contact, hospital records, and the Social Security Death Index. Secondary endpoints included the composite primary endpoint plus periprocedural MI and decreased renal function.

**Main results.** 1240 patients met study criteria; 479 underwent multivessel stenting and 761 underwent culprit-only stenting. After adjusting for baseline and angiographic characteristics, patients in the multivessel stenting group had a lower incidence of the primary endpoint (death, MI requiring hospitalization, or revascularization; hazard ratio, 0.80 [95% confidence interval, 0.64–0.99];  $P = 0.04$ ). In propensity-matched analysis, the incidence of the primary endpoint was lower in the multivessel stenting group as compared with culprit-only stenting (hazard ratio, 0.67 [95% confidence interval, 0.51–0.88];  $P = 0.004$ ). However, findings were mainly driven by rates of revascularization, and no differences in mortality or MI requiring hospitalization were found.

**Conclusion.** Compared with culprit-only stenting, multivessel stenting was significantly associated with a lower revascularization rate and thus a lower composite endpoint in patients with unstable angina or NSTEMI.

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### Commentary

The American College of Cardiology/American Heart Association recommends multivessel stenting when “there is a high likelihood of success and a low risk of morbidity and the vessel(s) to be dilated subtend a moderate or large area of viable myocardium and have high risk by noninvasive testing” [1]. However, in reality not all NSTEMI patients undergo noninvasive testing before angiography, and this creates a dilemma for the invasive cardiologist of whether nonculprit lesions should be stented. Shishehbor et al compared multivessel stenting with culprit-only stenting for treating unstable angina and NSTEMI patients with multivessel CAD. In this study, inclusion criteria were clearly defined, and primary endpoints were clinically relevant. The authors accounted for baseline differences and angiographic severity, and patients were matched by a propensity score. Multivessel stenting led to a lower primary composite endpoint, but the findings were attributed to lower revascularization rates, not fewer deaths or MIs requiring hospitalization.

This study has significant limitations. All revascularization procedures used bare-metal stents, which are associated with a higher rate of restenosis than drug-eluting stents, and this may limit the generalizability of the study. Second, the investigators were unable to confirm why patients underwent revascularization. Was revascularization needed because of restenosis, ongoing chest pain, or worsening CAD? This information is important given that revascularization was the primary outcome in the study. Third, medical therapy with  $\beta$  blockers, angiotensin-converting enzyme (ACE) inhibitors, and statins was suboptimal in this study population. In the multivessel and culprit-only groups, respectively,  $\beta$ -blocker use was 44% and 47%, ACE inhibitor use was 25% and 21%, and the statin dose was not specified in either group. Finally, strategies could have potentially “crossed over,” with a culprit-only procedure becoming a multivessel procedure and vice-versa based on difficulty of the procedure, which could be a confounder.

Results from this study suggest that in unstable angina

and NSTEMI, patients with multivessel CAD have a lower revascularization rate after undergoing multivessel stenting compared with culprit-only stenting but derive no benefit in terms of mortality or rehospitalization. A question remains: would overall event rates or incidence of the primary endpoint in the culprit-only group decrease if the rates of  $\beta$ -blocker and ACE inhibitor use were improved? In addition, more information regarding the statin dose would have been informative because high-dose statins have been shown to cause regression of atherosclerosis [2] and may affect revascularization rates.

### Applications for Clinical Practice

Should significant coronary lesions ( $\geq 50\%$  stenosis) be stented when they are nonculprit vessels (ie, not causing angina or physiologically impeding coronary blood flow)? Some argue that nonculprit lesions will eventually require intervention and therefore should be stented during the initial procedure. On the other hand, why place a stent in a nonculprit lesion that is not physiologically causing angina or obstructing flow? More studies evaluating multivessel stenting versus culprit-only stenting are needed to clarify this debate, and given the limitations of this study, nonculprit vessel stenting in unstable angina or NSTEMI should not be recommended at this time.

—Review by Robert L. Huang, MD

### References

1. Braunwald E, Antman EM, Beasley JW, et al. ACC/AHA guideline update for the management of patients with unstable angina and non-ST-segment elevation myocardial infarction—2002: summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Patients With Unstable Angina). *Circulation* 2002;106:1893–900.
2. Nissen SE, Nicholls SJ, Sipahi I, et al. Effect of very high-intensity statin therapy on regression of coronary atherosclerosis: the ASTEROID trial. *JAMA* 2006;295:1556–65.

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