We thank Dr Spiegel for his comments on our article that was recently published in the Journal.1

In the field of coronary artery disease, an increase in cardiac enzyme is reported to occur in 5–30% of patients after angiographically successful percutaneous coronary intervention (PCI), and periprocedural myocardial infarction (PMI) is generally defined as an elevation of cardiac enzyme more than 3-fold above the upper limit of normal value within 24 h after PCI.2 Short- and long-term prognoses in patients with PMI are demonstrated to correlate with the extent of enzyme elevation in the acute phase.3 The major causes of PMI after angiographically successful PCI are assumed to be a distal embolization and/or the effect of bioactive molecules on the down-stream microcirculation (these are released from injured culprit plaques during PCI and include cholesterol particles, thrombus, necrotic/apoptotic tissue, and vasoactive humoral factors).

Recent study with intravascular ultrasound showed that a large necrotic/lipid core area at the culprit coronary lesion was an independent predictor of post-PCI enzyme elevation.4 We found in our coronary CT study that intensive rosuvastatin therapy stabilized vulnerable plaque by reducing the ratio of lipid core volume to plaque volume, a significant decrease of 9.0%, as well as increasing the minimal CT value in the target plaque by 7.4-fold, during 6 months treatment.5 Although baseline LDL cholesterol levels differ between the cohort of the SAMMPRIS trial and our study population (97.0 vs. 151.6 mg/dl, respectively), it is possible that intracranial arterial stenotic lesions of the SAMMPRIS population consisted of a lipid core at baseline as in our study population. Percutaneous transluminal angioplasty and stenting (PTAS) within 3 business days after randomization possibly induced plaque laceration to such vulnerable lesions, and released intraplaque contents into the cerebral circulation, resulting in a significantly higher incidence of stroke within 30 days, especially within 1 day, compared with the medical management alone group in the SAMMPRIS trial.

It has been demonstrated by several clinical studies that statin treatment prior to PCI significantly decreases PMI in both stable and unstable coronary artery disease.6,7 If PTAS had been performed after adequate duration of medical management in the SAMMPRIS trial, the event rate in the PTAS group may have been comparable or even lower than in the aggressive medical therapy alone group. We agree with the comment from Dr Spiegel that plaque modification with prior optimal statin treatment should be applied in cases of PTAS for atherosclerotic intracranial arterial diseases, especially in patients with stable clinical presentation and high LDL levels. Target LDL levels and the duration of treatment prior to PTAS need to be determined through future prospective studies.

References


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