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Detection of increased temperature of the culprit lesion after recent myocardial infarction: the favorable effect of statins
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BACKGROUND: Increased thermal heterogeneity has been demonstrated in atherosclerotic plaques, with the higher temperature recorded in acute myocardial infarction (MI). Dietary or treatment interventions reduce heat production. The purpose of the present study was to investigate whether increased plaque temperature is maintained for a prolonged period after MI and the role of statin administration. METHODS: We enrolled 55 patients, 29 with recent MI and 26 with chronic stable angina (CSA). Total cholesterol, C-reactive protein (CRP), interleukin-6 (IL-6) and soluble adhesion molecules were measured in the study population. All patients underwent coronary plaque temperature measurements. Temperature difference (DeltaT) was designated as the temperature of the culprit atherosclerotic plaque minus the temperature of the proximal healthy vessel wall. RESULTS: Under treatment with statins were 19 patients with recent MI and 14 with CSA. In patients with recent MI DeltaT was 0.19 +/- 0.18 degrees C, while in patients with CSA was 0.10 +/- 0.08 degrees C (P =.03). Patients treated with statins had lower DeltaT compared to untreated patients (0.10 +/- 0.11 versus 0.20 +/- 0.18 degrees C, P =.01). Treated patients with recent MI had similar DeltaT compared to CSA patients treated with statins (0.13 +/- 0.13 versus 0.07 +/-.06 degrees C, P =.14), while untreated patients with recent MI had substantially increased DeltaT compared to untreated patients with CSA (0.28 +/- 0.22 versus 0.14 +/- 0.10 degrees C, P =.04). DeltaT was positively correlated with CRP (R = 0.50, P <.01), IL-6 (R = 0.58, P <.01), and intercellular adhesion molecule-1 (R = 0.40, P =.03) levels. CONCLUSION: Increased plaque temperature is observed for an extended period after myocardial infarction, indicating that the inflammatory process is sustained after plaque rupture. Statins have a beneficial effect after MI on plaque temperature.
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Assessment of plaque vulnerability by angioscopic classification of plaque color
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BACKGROUND: The disruption of yellow plaque and subsequent thrombosis is regarded as the mechanism of acute coronary syndrome. However, there are limited reports on the assessment of plaque vulnerability. Therefore, we tested whether the angioscopically determined yellow color intensity of plaque is associated with the prevalence of thrombosis on the plaque. METHODS: The angioscopic images of 843 patients who underwent catheterization and angioscopic examination from November 1999 to July 2003 for the diagnosis of coronary artery diseases were analyzed. Suspected culprit vessel was observed by angioscopy, and the yellow color intensity (1, light yellow; 2, yellow; 3, intensive yellow) of all yellow plaques (n = 1253) detected in the nonstenotic (diameter stenosis <50%) coronary segments was determined, as well as whether there was thrombosis on the plaques. RESULTS: The number of detected

yellow plaques was 345, 721, and 187 for color grade 1, 2, and 3, respectively. The prevalence of thrombosis detected by angioscopy (15%, 26%, and 52% on the plaque of color grade 1, 2, and 3, respectively, P <.0001) was significantly higher on the plaque of higher yellow color grade. CONCLUSIONS: The yellow color intensity of plaque determined by angioscopy was strongly related with the prevalence of thrombosis on the plaque. The yellow color intensity may be a marker of plaque vulnerability.
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Color of culprit lesion at 6 months after plain old balloon angioplasty versus stenting in patients with acute myocardial infarction
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BACKGROUND: Although the healing process of disrupted yellow plaques at myocardial infarction (MI) culprit lesions has been reported, the effect of stenting on this process has not been clarified. Stenting has been reported to deteriorate the endothelial function after percutaneous coronary intervention (PCI). Therefore, we compared the angioscopic morphology of culprit lesions at 6 months after plain old balloon angioplasty (POBA) and stenting to clarify the effect of stenting on the healing of disrupted culprit plaques of acute MI. METHODS: Patients with acute MI who had yellow culprit plaque, successful reperfusion therapy with POBA (n = 21) or stenting (n = 22), and a successful 6-month follow-up angioscopic examination were included in this study. Oral ticlopidine (200 mg/day) was administered for 3 to 6 months after stenting. RESULTS: At 6 months after reperfusion therapy, the color of the culprit lesion became white in significantly more patients treated with stenting than treated with POBA (50% vs 14%; P =.01). However, the prevalence of thrombus appeared to be higher in patients treated with stenting than in patients treated with POBA (27% vs 5%; P =.04). Although there was some difference in the patients' characteristics in the groups, logistic regression analysis revealed no significant influence of those factors on the color of or on the prevalence of thrombus at the culprit lesion. CONCLUSIONS: Coronary stenting in patients with acute MI leads to the disappearance of yellow color at a significantly higher rate than POBA; however, whether it stabilizes the plaque requires further investigation.
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Intravascular thermography: Immediate functional and morphological vascular findings
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AIMS: To investigate safety, feasibility, and injurious effect on endothelial cells of a thermography catheter as well as effect of flow on measured temperature in non-obstructive arteries. METHODS AND RESULTS: Safety and feasibility were tested in both rabbit aortas and pig coronary arteries. Evaluation of endothelial damage by the catheter (acute, 7 and 14 days) was performed in pig coronaries using Evans Blue,

scanning electron microscopy (SEM) and Factor-VIII antibody and compared with normal arteries and arteries that underwent intravascular ultrasound (IVUS). The effect of flow on temperature heterogeneity was analysed both in vitro and in vivo conditions. All procedures were successful without any adverse events; intra- and inter-operator variability was low. Intracoronary use of the catheter was associated with acute but reversible de-endothelialization, paralleling the findings associated with IVUS use. Changes in flow velocities under physiologic flow conditions did not significantly influence the temperature differences measured both in vitro and in vivo; temperature heterogeneity was more pronounced in absence of flow. CONCLUSIONS: Intracoronary thermography using a dedicated catheter is safe and feasible with a similar degree of de-endothelialization as IVUS. Temperature heterogeneity remained unchanged under normal physiologic flow conditions allowing clinical use of thermography.
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