EDITORIAL

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Can improvements in our physiological understanding yield information on the utility of endothelial progenitor cell capture stents?

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At the 2006 meeting of the European Society of Cardiology (ESC), Camenzind et al. [1] reported that patients were at risk of very late stent thrombosis when first-generation drug-eluting stents (DES) were placed. This warning introduced a new era of controversy; the clinical implications of re-endothelialization after stent implantation remain intensively debated today. Topics include the adequate duration of dual antiplatelet treatment, stent-related endothelial dysfunction, and the safety and efficacy of bare-metal stents, as compared to DES. The "endothelial progenitor cell (EPC) capture stent" is an attractive concept, being strongly supported by the notion of "accelerated endothelial healing" in the present era of the "ESC firestorm." However, the clinical performance of EPC capture stents is far from satisfactory [2]. Such stents have not reduced the need for restenosis, and even stent safety has been challenged; reports of stent thrombosis have appeared. However, the concept of accelerated endothelial healing remains attractive.

Choi et al. [3], in an observational study, compared the effects of DES and EPC capture stents on microvascular dysfunction in post-percutaneous intervention (PCI) patients. The index of myocardial resistance (IMR) was significantly lower in the latter than in the former group 6 to 12 months after index PCI. IMR can be used to diagnose microvascular dysfunction [4], which is known to be associated with an increased risk of adverse cardiovascular events [5]. Previous studies on the effects of DES implantation on endothelial function have yielded mixed results. The effects of stent type on microvascular function have not been well-studied. EPC capture stents were associated with a lower IMR than were DES in the cited work, but the coronary flow reserve (CFR) did not differ between the two groups. Although the observed difference in IMR is interesting, any clinical implication of a lower IMR without any concomitant change in CFR in the EPC capture stent group remains unclear. The cited study was small and the patient groups heterogeneous. It is difficult to compare CFR and IMR between two small groups with mixed infarct- and non-infarct-related arterial lesions. Microvascular dysfunction is caused by multiple pathogenic mechanisms in patients with coronary artery disease, especially after coronary intervention [5]. No potential causal relationship between IMR and stent type can be

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inferred in the absence of baseline information that was not provided in the cited study. And paucity of data suporting clinical benefit of EPC capture stent compared with DES limit the clinical value of cited work. As the authors thereof indeed discussed, the study was observational in nature and had associated limitations. However, the efforts of the cited work to elucidate the biological differences between DES and EPC capture stents, using modern means of physiological evaluation, were valuable; the topic deserves further attention.

Conflict of interest

No potential conflict of interest relevant to this article was reported.

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