it has been shown to decrease mortality (1). However, in a number of patients undergoing primary percutaneous coronary intervention, epicardial coronary artery reperfusion does not translate into myocardial reperfusion, a phenomenon referred to as no-reflow. No-reflow has important clinical ramifications because it is associated with a worse prognosis (2). Niccoli et al. (2) discuss several investigated strategies for prevention and treatment of no-reflow, including management of distal embolization, reducing reperfusion times, and use of drugs such as glycoprotein IIb/IIa antagonists. In this context, it is also clinically important to elaborate on the potential increased risk of no-reflow that is seen when deploying oversized stents. This notion was tested in 90 patients diagnosed with new ST-segment elevation (3). In this study, patients were divided into either the stent overexpansion group (n = 25 patients; stent-to-artery ratio >1.2) or the stent nonoverexpansion group (n = 65; stent-to-artery ratio <1.2). Notably, no-reflow was increased in the overexpansion group when compared with the nonoverexpansion group (32% vs. 11%, respectively, p = 0.031), and heart failure was seen more often (28% vs. 14%, respectively, p = 0.036), although there was less target lesion revascularization during follow-up in the overexpansion group (3). Thus, during coronary stenting, the risks of stent overexpansion should be weighed against the benefits to limit the potential for harm. Insofar as primary percutaneous coronary intervention has emerged as the preferred therapeutic method in ST-segment elevation myocardial infarctions, physicians should retain a heightened awareness of all clinically-relevant mechanisms implicated in the phenomenon of no-reflow, including overexpansion of coronary stents.

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REFERENCES


Reply

We thank Drs. John R. and Roger Kapoor for their interest in our article (1) and for their comments on the role of coronary stent overexpansion in no-reflow. Iatrogenic coronary microvascular dysfunction (type D in the Camici-Crea classification) (2) has emerged as an important cause of microvascular dys-function, especially in the setting of primary percutaneous coronary interventions (PCIs) (2). Thrombus and plaque material can mechanically plug microcirculation and further increase vasoconstriction due to the release of potent vasoconstrictors by platelets. Accurate planning of a primary PCI is of paramount importance including pharmacological prevention of distal embolization and vasoconstriction by administration of glycoprotein IIb-IIIa inhibitors and vasodilators, as well as mechanical prevention of thrombus dislodgment by thrombus aspiration, which has been shown to substantially improve microvascular function and survival in patients treated by a primary PCI (3). Another technical point to consider, whenever possible, is direct stenting, which seems to improve the microvascular function compared with stenting preceded by balloon pre-dilation (4). Furthermore, stent overexpansion as well as aggressive post-dilation should be avoided in this setting because of the higher risk of no-reflow, as shown by Maekawa et al. (5), probably due to the higher risk of distal embolization from unstable plaques compared with stable plaques (6). Finally, recent reports on the safety of the drug-eluting stent in the setting of a primary PCI (7) suggest its possible use in the attempt to reduce target lesion revascularization, as is the case in stable patients.