Letters to the Editor
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STEMI and NSTEMI are two distinct pathophysiological entities

Montalescot et al. recently demonstrated that patients with STEMI and NSTEMI have similar in-hospital and long-term prognoses as well as similar independent correlates of outcome, despite very different in-hospital management. As per accepted guidelines, whereas most STEMI patients underwent emergent reperfusion treatment (e.g. primary PCI or thrombolysis), NSTEMI patients hardly ever received this kind of therapy. This different treatment strategies, however, are justified by evidence-based medicine as thrombolytic therapy in non-Q wave MI patients showed no benefit over standard therapy. The reason for the failure of intravenous thrombolytic therapy to improve clinical outcomes in the absence of AMI with ST-segment elevation is most likely related to the fact that in STEMI the culprit artery is usually occluded by a thrombus, whereas in NSTEMI the culprit artery is usually patent with a non-occlusive thrombus.

The development of STE vs. NSTEMI does not appear to be coincidental. We have demonstrated that most patients with recurrent MI episodes will have either repeated episodes of STEMI or NSTEMI but not both, suggesting predilection of some patients to repeated episodes of occlusive thrombi and others to repeated episodes of non-occlusive thrombi. Smoking cessation did not influence this finding. Individual differences in endogenous tissue plasminogen activator levels/activity as well as fibrinogen VII and PAI-1 levels, may explain these differences which suggest STEMI and NSTEMI are in fact different entities. Therefore, while we certainly agree with the authors that secondary prevention such as aggressive lipid lowering, antiplatelet therapy, etc. are critical with either type of MI, it is still important to consider STEMI and NSTEMI as distinct entities.

References


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