Letters to the Editor
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STE MI and NSTEMI are two distinct pathophysiological entities

Montalescot et al.1 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.2 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.3 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.4 Smoking cessation did not influence this finding.5 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 other type of MI, it is still important to consider STEMI and NSTEMI as distinct entities.

References

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STE MI and NSTEMI are two distinct pathophysiological entities: reply

We thank Dr Rott for his thoughtful analysis of our recently published OPERA study and we concur with him on the point that patients with STEMI and NSTEMI have different initial management almost exclusively related to the decision of emergent reperfusion which is provided more systematically to STEMI than NSTEMI patients and, we have clearly outlined this point in our paper. However, it does not mean that the two entities do not share the same pathophysiology. Indeed, risk factors, demographics, pathogenesis of plaque rupture, complications, prognosis, and secondary prevention are quite similar in STEMI and NSTEMI. By definition, occlusion of the culprit coronary artery is more frequently found in STEMI patients, but it does exist in a significant number of patients presenting with NSTEMI. Moreover, ST-elevation may be missed in patients with an occluded artery particularly when the culprit lesion is located in the circumflex artery. Not to mention patients with simultaneously multiple active plaques or occluded vessels, undetectable by the electrocardiogram but found with angiography or intracoronary ultrason. Even haemostasis factors such as PAI-1 or von Willebrand factor have been reported to be increased in both situations and cannot discriminate between the two syndromes and certainly not sustain that they are two distinct pathophysiological entities.1,4 So, in contrast to what Dr Rott suggests, the frontier is not that sharp between STEMI and NSTEMI. For now, we agree that immediate management is different when a patient presents with vs. without ST-elevation but everyone knows that ST-elevation is sometimes transitory transforming the case in a ‘non STEMI’ for the decision of reperfusion. The opposite is also true. Finally, ongoing randomized studies are examining the hypothesis that immediate reperfusion of NSTE-ACS is a better strategy than a delayed approach; if these studies are positive that will make the two entities even closer than what they already are.

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References