STEMI and NSTEMI: the dangerous brothers

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This editorial refers to ‘STEMI and NSTEMI: are they so different? 1 year outcomes in acute myocardial infarction as defined by the ESC/ACC definition (the OPERA registry)’ by G. Montalescot et al., on page 1409

Ever since the redefinition of myocardial infarction (MI) in the year 2000,1 a new entity has entered the field: non-ST-elevation MI (NSTEMI). STEMI and NSTEMI share the release of specific myocardial necrosis markers which define them clinically as acute MI (AMI) and set them apart from unstable angina, an acute coronary syndrome which does not qualify as MI.

NSTEMI: getting to know the younger brother

With the creation of the NSTEMI as a new clinical entity, the need for data regarding prognosis and treatment options arose. By definition, STEMI and NSTEMI are only different with respect to the reflection of acute myocardial ischaemia and necrosis in the ECG. Although this difference may be triggered by the size of the infarcted area, it may also be only the location of the infarct—an occluded circumflex artery does not project equally well on the ECG as does an occluded right coronary artery. Furthermore, specificity and sensitivity of ECG changes are influenced by several other factors including prior MI, bypass surgery, variation of coronary anatomy, bundle-branch block, and others. Are these differences meaningful—should they lead to different clinical approaches—as the guidelines currently recommend?

Are treatment strategies that have been tested before the redefinition of AMI, such as thrombolytic therapy, and that have shown to be beneficial in STEMI and non-beneficial in other acute coronary syndromes applicable to NSTEMI patients? Certainly not without further research.

The OPERA registry

Montalescot et al.,2 are to be congratulated for extending our knowledge about the relative prognosis and treatment reality of STEMI and NSTEMI significantly. OPERA is a multicentre, countrywide registry in France that compared outcome and treatment of 2151 patients from 56 centres at hospital discharge and at 1 year. The results show that patients with NSTEMI and STEMI have comparable in-hospital and long-term prognosis. They also have similar independent correlates of adverse outcome. However, NSTEMI patients undergo less and later reperfusion and also less consequent secondary prevention.

The majority of previous registries have shown similar results, OPERA being of special interest because it was performed in a country with aggressive reperfusion policy and widely available cath-lab facilities.

Clinical consequences

A pattern emerging from registries is that STEMI patients are younger and have more myocardium at stake and less cardiac and non-cardiac concomitant diseases. NSTEMI patients are older and have had more prior cardiac damage and also non-cardiac disease. Even though their acute cardiac damage is less, NSTEMI can be considered as a ‘last straw’ that pulls an already overburdened heart down.

Progress in the treatment of MI has been rapid in the past years; however, most studies focused on STEMI. Most recently, individual trials and meta-analyses3 have been published that address NSTEMI as a unique entity. In fact, there are now numerous studies underway that will define the short- and long-term benefits of various antithrombotic approaches as well as those of an early invasive vs. conservative strategy.

As a clinical consequence, we would go along with the conclusions that Montalescot et al. offer: NSTEMI patients appear to be undertreated with respect to reperfusion and also after discharge from hospital. The similar prognosis of NSTEMI and STEMI patients should lead to a more aggressive in-hospital and secondary prevention treatment of both groups, particularly the NSTEMI population. A primary research goal should be to find out which individuals derive most benefit from such a more aggressive approach. Even after ISAR-COOL and ICTUS, this remains somewhat controversial.4,5 However, all of these strategies derived from well-designed registries still need to be confirmed in large, randomized, clinical outcome studies. Until proven otherwise, STEMI and NSTEMI are no identical twins, but equally dangerous.

Conflict of interest: none declared.

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Clinical vignette

Superior vena cava obstruction due to markedly enlarged right pulmonary artery in Eisenmenger syndrome

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A 24-year-old man, with Down syndrome, partial atrioventricular septal defect, and non-restrictive patent ductus arteriosus, had previously documented dilatation and thrombosis of his right pulmonary artery (RPA) secondary to his Eisenmenger syndrome. His clinical situation had been reasonably stable when he presented with increasing dyspnoea and cough. Chest X-ray (CXR) on admission (Panel B) showed significant increase in the size of the RPA when compared with a film obtained 5 years earlier (Panel A).

CT pulmonary angiography (CTPA) was performed on a Siemens Somatom 64 scanner. This revealed massive dilatation of the RPA (Panel C) with extensive thrombus (asterisk) in the RPA. This markedly enlarged RPA impinged on the right main bronchus (Panel D) posteriorly (black arrowhead), as well as on the superior vena cava (SVC) anteriorly (white arrowhead). The SVC was severely compressed, but this compression involved only that portion of the SVC caudal to the insertion of the azygos vein (Panel E). The azygos vein was noted to be dilated, as were the intercostal veins (Panel E). We submit that, unlike previous cases of SVC obstruction by an enlarged RPA, there was no SVC syndrome in this case, because the SVC compromise occurred caudal to the insertion of the azygos vein and the expanding RPA had not yet obstructed the azygos arch. The upper body venous drainage could be accommodated, therefore, through the azygos system.

Panels A and B. CXRs from 2001 and 2006, respectively. The white arrows point to a very prominent MPA segment on the left of the cardiac silhouette. The white arrowheads indicate the massively dilated RPA, which is markedly larger in 2006.

Panels C and D. CTPA transaxial images obtained after injection of contrast into the right antecubital vein. Panel C was obtained at the level of the carina and demonstrates the enlarged MPA and RPA. Most of the RPA is obliterated by the thrombus (asterisk). Panel D is more caudal. It demonstrates the degree of extension of the thrombus in both anterior–posterior and right–left directions. The small black arrowhead indicates some compression of the right main bronchus and the large white arrowhead (in both panels) shows the marked flattening and lumen compromise of the SVC. The azygos vein (white arrow) is unusually well opacified. A row of small white arrowheads denotes a line of calcification within the thrombus.

Panel E. Three-dimensional volume rendered image showing the SVC from a right oblique view and its entry into the right atrium. The dilated RPA and thrombus have been removed for clarity. The SVC has been markedly attenuated (white arrowhead) by the mass effect of the massively enlarged RPA. The azygos vein and its arch are very well seen decompressing the SVC. The small white arrows point to the prominent intercostal veins. Ao, aorta; DA, descending aorta; LPA, left pulmonary artery; MPA, main pulmonary artery.