Intraoperative myocardial infarction in non-cardiac surgery: is a universal definition feasible?

Diagnostic criteria for the redefinition of acute myocardial infarction (AMI) have been recently published.1 In this new classification, intraoperative (IPEMI) or post-operative (POMI) AMI, in the setting of non-cardiac surgery, were not included.

It is well known that this category of AMI occurs under particular circumstances while the diagnostic criteria have not been fully elucidated.2

Patients with pre-operative cardiac risk undergoing intermediate or high surgical risk operations are more prone to IPEMI and POMI. Meanwhile, stent thrombosis in patients with coronary artery disease undergoing non-cardiac surgery could pose a specific risk factor, because the time for complete endothelization varies among subjects. This risk increases in case of pre-operative anticoagulation therapy alteration for fear of bleeding.3

Mortality from IPEMI and POMI varies between 40 and 70%, especially because these are frequently underdiagnosed. On top, patients during the perioperative period cannot easily complain of angina. During surgery, it is not necessary to have hemodynamic instability, for an AMI to occur. Subtle ST-segment changes in electrocardiogram are not always appreciated, unless there is a computerized monitor analysis. The majority of perioperative AMIs are non-Q-wave and electrocardiographic changes are therefore non-specific.

Transoesophageal echocardiogram, although not routinely used, could reveal segmental wall motion abnormalities, in patients with ‘unusual intraoperative cardiac behaviour’.

Laboratory values of CPK and CPKMB are not reliable, because of co-existing muscle damage. Troponins need to be measured frequently in the immediate post-operative period. However, the 99th percentile of the upper reference limit, above which troponin levels become diagnostic of IPEMI and POMI, is still unknown.

Apart from high mortality, increased surgical morbidity (sepsis, wound infection, pneumonia, and deep vein thrombosis) is common, when IPEMI–POMI occur.

In 50% of patients who die of perioperative AMI, neither intracoronary thrombi nor atherosclerotic plaque rupture are found in autopsy.4

On the basis of the above, the majority of IPEMI and POMI should be classified as type 2, while there would be a few that could belong to anywhere between 3 and 4b, according to the new reclassification, but still a certain percentage remains unclassified.

In conclusion, non-cardiac surgery IPEMI–POMI consists of a particular category with high morbidity and mortality, and not fully clarified diagnostic criteria. They deserve to be included in the universal redefinition of AMI as a separate type-category.

References


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The Joint ESC/ACCF/AHA/WHF Task Force appreciates the letter of Dr Siniorakis and colleagues, which allows us to clarify its recommendations. We agree that there is a great deal to learn about peri- and post-operative myocardial infarction as the pathophysiology of these differs somewhat from that of myocardial infarction occurring in the usual setting. However, there are substantial data to guide us.1,2 We also agree that many of these infarctions are likely type 2. This fact raises several important considerations and some caveats as well. Specifically:

1. Studies of patients undergoing non-cardiac surgery strongly support the concept that many of the infarctions diagnosed in this connection are caused by prolonged imbalance between myocardial oxygen supply and demand on the background of coronary artery disease,1,2 which together with rise and fall of cardiac markers points towards myocardial infarction type 2.

2. The fact that many such patients have type 2 infarctions should not obscure the likelihood that some of the infarctions are type 1 as well. Pathology of fatal peri- or post-operative myocardial infarctions shows plaque rupture and platelet aggregation leading to thrombus formation in approximately half of these events.3 Given the differences that likely exist in the therapeutic approaches to each, close clinical scrutiny to identify this group is essential.

3. Some patients may not have myocardial infarction at all. Careful clinical evaluation including a detailed history, examination, and evaluation of further investigations to identify and treat those with pulmonary embolism, sepsis and/or the many other conditions associated with myocyte necrosis and troponin elevations also is strongly advocated.4

4. The available data suggest not only the need for short-term strategies to improve prognosis but also the need to develop ways of evaluating those who do well short term but who are at accentuated risk longer term.5,6

Although there is still the need for data in specific subgroups of patients with peri-