Takotsubo syndrome co-existing with acute myocardial infarction: an overlooked phenomenon in clinical practice

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Over the past years, takotsubo syndrome (TS) has drawn a particular attention as a transient form of acute cardiomyopathy that usually mimics acute myocardial infarction (AMI) in clinical practice. Accordingly, definitive diagnosis of this phenomenon generally requires urgent coronary imaging to exclude an existing AMI mostly associated with a culprit coronary artery stenosis. In their recently published elegant report, Looi et al have compared general incidence along with demographic and clinical characteristics between TS and AMI patients admitted to five major tertiary hospitals in New Zealand. In particular, we agree with the authors on potential underdiagnosis of TS in clinical practice. However, we would like to make a few comments on this issue:

Underdiagnosis of TS might not be exclusively attributable to its misdiagnosis as AMI in the clinical setting. Accordingly, this might also be due to its potential co-existence with AMI in a portion of cases. Importantly, TS co-existing with AMI is usually masked by the rampant findings of AMI, and hence is largely overlooked in the clinical setting. In other terms, an existing culprit coronary stenosis on coronary imaging (suggestive of AMI and universally used as an exclusion criterion for TS) might not fully exclude an existing TS in certain cases. Certain risk factors for such a co-existence might include spontaneous coronary artery dissection (SCAD) on coronary imaging, substantial physical stressors, severe systemic inflammation, frailty, intractable chest pain (requiring further measures) and an existing extensive AMI.

Importantly, evolution of TS might precede or follow that of AMI in this co-existence, and might present with atypical patterns (other than classical apical ballooning) potentially arising as a further diagnostic challenge in this setting. Mechanistically, both conditions might stem from a common trigger (for instance; severe physical stressors or systemic inflammation).

However, more commonly, one condition might trigger the other on follow-up (for instance; AMI due to SCAD might induce TS as a result of intractable angina or severe wall motion abnormalities in TS might potentially trigger AMI due to SCAD formation associated with severe coronary torsion, etc). Of note, TS co-existing with AMI might be confirmed mostly through magnetic resonance imaging (MRI) that exhibits, in details, the tissue involvement patterns of specific pathologies including TS. Based on the huge number of AMI patients (n=11,900) in the study by Looi et al, it seems highly likely that a portion of these cases (particularly those with the aforementioned risk factors) might have also suffered a co-existing TS. Accordingly, we wonder if there were such probable or absolute TS cases confirmed clinically or on imaging modalities including MRI, etc in the AMI population. Were these cases (if any) recruited as TS or AMI patients for comparison or excluded beforehand?

In summary; despite the strict categorical definition of TS in the recent guidelines...
(suggesting the presence of culprit coronary lesion (and hence; AMI) as an exclusion criterion), a portion of TS cases might also emerge in the setting of AMI due to a variety of common triggers or more likely; due to the potential impact of each phenomenon on the evolution of other. However, diagnosis of TS in this co-existence might be easily overlooked, particularly leading to a TS underdiagnosis in AMI population. Importantly, such a co-existence might also serve as a potential confounding factor when making a clinical comparison between TS and AMI characteristics in clinical studies. Accordingly, this might suggest proper detection of cases with this co-existence in an effort to exclude these cases from the analysis beforehand or assign them as a distinct category (with potentially diverse features) in a given study.

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Nil.

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