

infarction.<sup>3</sup> Irrespective of these considerations, we agree that, with respect to the prognosis of patients with TTS, the underlying trigger is also an important factor. In fact, in a previous work by our group,<sup>4</sup> we reported that the clinical course during hospitalization (length of stay and complications) and follow-up (recurrences) was worse in secondary than in primary TTS. This is why we proposed to extend this simple nomenclature. Primary TTS has no identifiable trigger, or is triggered by major psychological stress, while secondary TTS is triggered by physical factors (such as respiratory exacerbation, surgery, and trauma).<sup>5</sup>

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#### Definition of Myocardial Infarction Type 4a: Can We Define Its Diagnosis and Systematize Clinical Practice?



#### Definición de infarto tipo 4a: ¿podemos definir mejor su diagnóstico y sistematizar la práctica clínica?

#### To the Editor,

After reading the article “Comments on the 2018 ESC Fourth Universal Definition of Myocardial Infarction”,<sup>1</sup> we are prompted to comment on the diagnosis of type 4a acute myocardial infarction (AMI), which describes AMI occurring after percutaneous coronary intervention (PCI). We note a major discrepancy between the definition put forward by the European Society of Cardiology (ESC)<sup>2</sup> and the concept of “clinically relevant myocardial infarction” proposed in 2014 by the Society for Cardiovascular Angiography and Interventions (SCAI)<sup>3</sup>; we furthermore believe that this discrepancy generates conspicuous and undesirable variability in clinical practice. Guideline documents should examine this issue in greater depth, in order to establish a consensus definition and thereby eliminate this variability.

In the previous and current ESC guidelines, the key diagnostic criterion for type 4a AMI is a post-PCI elevation  $\geq 5$  times the 99<sup>th</sup> percentile upper reference limit for myocardial injury markers. A confirmed diagnosis requires this to be concurrent with one of the following factors: new ischemic electrocardiogram changes, new Q waves, imaging evidence of the loss of viable myocardium, or angiographic evidence of a vascular complication explaining the marker elevation.

The SCAI uses distinct criteria, defining “clinically relevant myocardial infarction” as a biomarker elevation  $\geq 70$  times the local laboratory upper limit of normal or  $\geq 35$  times that limit if accompanied by new pathological Q waves in 2 contiguous leads or new persistent left bundle branch block.<sup>3</sup> The SCAI authors argue

that the AMI definition adopted by the ESC is not clearly linked to subsequent events such as death or heart failure; widespread adoption of this biomarker threshold may therefore have serious consequences for the appropriate assessment of devices and treatments, potentially affecting clinical care pathways and leading to misinterpretation of physician competence. Thus, in place of an AMI definition sensitive to mild myonecrosis, the SCAI consensus document recommends the use of a higher biomarker elevation threshold that has shown strong links to subsequent adverse events in clinical studies.<sup>3</sup>

Likely as a consequence of this lack of consensus, current clinical practice shows an alarming variability. Moreover, in health care settings where cost concerns are more pressing, the lack of consensus and the resulting uncertain applicability of recommended thresholds to decision-making have resulted in low rates of biomarker measurement. This is evident from the US National Cardiovascular Data Registry, which shows that post-PCI biomarkers were measured in only 26% of 157 825 Medicare patients undergoing elective PCI at 711 hospitals between 2004 and 2008; the registry furthermore shows that the likelihood of postprocedure biomarker surveillance was significantly dependent on the treating hospital.<sup>4</sup>

In light of these observations, further efforts should be directed at improving the diagnosis of type 4a AMI and systematizing clinical practice. A more precise knowledge base would provide needed clarity, helping to identify those patients truly in need of biomarker analysis and providing health care savings by avoiding unnecessary biomarker determinations. Such savings are especially advisable in the current climate of escalating costs, which places a priority on dispensing with measures that do not provide value.<sup>5</sup>

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## Definition of Myocardial Infarction Type 4a: Can We Define Its Diagnosis and Systematize Clinical Practice? Response



### Definición de infarto tipo 4a: ¿podemos definir mejor su diagnóstico y sistematizar la práctica clínica? Respuesta

To the Editor,

We thank Lozano et al. for their interest in our article.<sup>1</sup>

It is true that scientific societies differ in the criteria they use to define myocardial infarction (MI). Type 4a MI is that occurring after percutaneous coronary intervention and is defined by the European Society of Cardiology as an elevation in high-sensitivity cardiac troponin (hs-cTn)  $\geq 5$  times the 99<sup>th</sup> percentile upper reference limit (URL) if this is accompanied by electrocardiogram changes, the appearance of new Q waves, and imaging or angiographic evidence of myocardial ischemia.<sup>1</sup> In contrast, the Society for Cardiovascular Angiography and Interventions (SCAI) defines “clinically relevant” postrevascularization MI as an hs-cTn increase  $\geq 70$  times the 99<sup>th</sup> percentile URL in the presence of new pathological Q waves or new persistent left bundle branch block.<sup>2</sup> These divergent definitions are based on different scientific evidence. The European Society of Cardiology definition of type 4a MI is based on optimal hs-cTn thresholds that have been validated for the prediction of cardiovascular events in recent studies.<sup>3</sup> The SCAI definition is based on the assumption that the optimal biomarker for defining clinically relevant MI after percutaneous coronary intervention is the serum creatine kinase MB fraction (CK-MB)<sup>3</sup>; the proposed hs-cTn threshold of  $\geq 70$  times the 99<sup>th</sup> percentile URL is calculated from the 7:1 ratio between troponin and CK-MB and was shown in a previous study to be a reliable proxy for elevated CK-MB.<sup>4</sup>

Clinical practice guideline recommendations should be the servants, not the masters, of clinical judgment. Adherence to this guiding principal will help us to improve the quality of care for our patients and balance the costs and benefits of the techniques used.

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## Validity of the Minimum Basic Data Set for Research Into Outcomes of the Care of Acute Coronary Syndrome



### Validez del Conjunto Mínimo Básico de Datos para la investigación de resultados en la atención al síndrome coronario agudo

To the Editor,

We have read with interest the article by Bernal et al.<sup>1</sup> published in *Revista Española de Cardiología* and would like to make several comments.

First, we would like to congratulate the authors on their study and on the research topic chosen. In the era of big data, new opportunities to use large databases have greatly enhanced prospects for research into health care outcomes. The study by Bernal et al. is a clear example of the usefulness of the minimum