Circadian Variations of Infarct Size
in STEMI

To the Editor:

We read with interest the recent article by Reiter et al regarding the circadian variations of infarct size in acute myocardial infarction (AMI).

This article confirms that there is a circadian variation of infarct size in ST-segment elevation myocardial infarction (STEMI) patients according to the time of the day of AMI onset, something recently shown by our group (Suarez-Barrientos et al). More important, a third independent series by Arroyo Ucar et al has been reported almost simultaneously with the current paper by Reiter and colleagues showing similar results. The fact that 3 independent series report a circadian variation of infarct size in humans provides robust evidence that this spontaneous phenomenon is real.

Despite both studies (Reiter et al and Suarez-Barrientos et al) having found a circadian variation in infarct size, the time of the day of STEMI onset resulting in larger infarcts varies between both series: Reiter et al found that enzyme release is higher when STEMI onset is between 1 and 5 AM, while our previous study showed that the largest infarcts are from patients with STEMI onset in the dark-to-light transition (ie, between 6 AM and noon). The latter data are in agreement with previous animal studies.

There are several potential reasons for this apparent disparity:

1. Patients excluded from the final analysis: Reiter et al initially evaluated a population of 1031 STEMI patients, and excluded from the analysis ~85% of the population. Of note, they excluded 568 patients because of TIMI flow >0 or collateral flow, and 104 additional patients for preinfarction angina or postconditioning. In contrast, in our previous study we excluded from the analysis only patients with previous infarctions or not revascularized by any means (~15% of exclusions in comparison with ~85% by Reiter et al). The final study population in Reiter et al was 165 patients, and that of Suarez-Barrientos et al was 811 patients. We believe that such a high rate of exclusions in the work by Reiter et al represents a source of selection bias in the work by Reiter et al.

2. Statistical adjustment for factors influencing per se infarct size: the statistical analysis in our previous work took into consideration infarct size modifiers (such as time of ischemia, culprit artery, previous treatment with β-blockers, and ACE inhibitors), while Reiter et al did not adjust the peak enzyme release for any of these modifiers, being therefore another potential source of bias in the work by Reiter et al.

3. Reiter et al followed the same methodology we described for infarct size estimation: peak concentration of cardiac injury biomarkers. We reported the whole set of CK and troponin-I data. However, Reiter et al reported only CK data. They claim that troponin data were similar, but they decided not to show this critical piece of information that would have reinforced the CK results.

From all the above, we wonder how the results from Reiter et al would have been if they had performed such a comprehensive statistical adjustment for infarct size modifiers and with a more reasonable rate of patient exclusion from the actual analysis.

Disclosures

None.

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