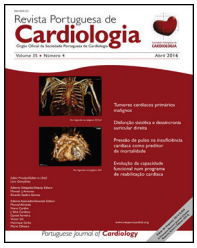
Rev Port Cardiol. 2018;37(3):225–226

Revista Portuguesa de
Cardiologia
Portuguese Journal of Cardiology
www.revportcardiol.org



EDITORIAL COMMENT

Is the monocyte to high-density lipoprotein cholesterol ratio important in risk stratification after myocardial infarction?

O rácio Monocitos/HDL colesterol é importante na estratificação de risco após um enfarte do miocárdio?

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Even with the best therapies, ST-elevation myocardial infarction (STEMI) is still associated with a high risk for complications, including mortality, sometimes in middle age.

When a patient suffers acute STEMI, action must be taken rapidly to save myocardium, and in parallel, it is important to perform early risk stratification to predict complications and to determine prognosis.

Risk assessment in acute myocardial infarction (MI) has long been an urgent concern of attending physicians. The first classification was the simple clinical Killip class, followed by the hemodynamic Forrester classification. A series of single risk factors then emerged: high-sensitivity C-reactive protein,[1] red blood cell distribution width,[2] hemoglobin level,[3] central obesity,[4] homocysteine levels,[5] and others, each of which was shown by different individual studies to influence or modulate prognosis after MI.

A more complete and comprehensive assessment, including clinical variables like age, low weight, late intervention and diabetes, and hemodynamic variables such as low blood pressure and high heart rate, as well as the old Killip classes, were included in the TIMI risk score, which is frequently used in clinical practice to predict mortality in patients with STEMI.[6] The GRACE[7] and ProACS[8,9] risk scores, the latter based on a large sample of Portuguese patients, are alternative comprehensive scores with potential clinical utility.

In a paper published in this issue of the *Journal*, Sercelik et al. present a study assessing the relationship between the monocyte to high-density lipoprotein cholesterol (HDL-C) ratio (MHR) and TIMI risk score in the prognosis of STEMI.[10]

The thinking behind this ratio is clear: monocytes are linked with inflammation and cytokines associated with the extent of the MI; and HDL-C protects tissues by removing cholesterol and suppressing monocyte activation. MHR may therefore be a new tool for STEMI risk stratification.

On the basis of this rationale, the authors analyze a sample of 111 patients with STEMI and 50 patients with angiographically normal coronary arteries. When the two groups were compared in multivariate analysis, MHR was the only independent predictor of STEMI. In correlation analysis, the authors found a significant positive correlation between MHR and TIMI risk score ($r=0.479$, $p<0.001$).

However, this interesting paper presents some limitations. First, the sample is too small to draw firm conclusions. Second, some questions arise concerning the selection of

DOI of original article: https://doi.org/10.1016/j.repc.2017.06.021
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the controls: if patients underwent coronary angiography, even if the conclusion was that their arteries were normal, it is difficult to accept that the patients were completely normal, or they would not have undergone this invasive procedure.

The results are interesting, but the correlation coefficient between MHR and TIMI score is relatively low (r=0.479, which implies a determination coefficient of 0.229). It is statistically significant, but the clinical significance is less clear.

As an example, if we assess height and weight in a population, we will obtain a large and significant correlation: taller people are on average heavier than smaller ones. But in individual terms, there will be both thin and obese persons in both groups. In other words, the correlation may be significant in an overall assessment but weak for an individual decision. However, when treating a STEMI patient, it is necessary to take individualized decisions and to prescribe individualized treatment.

Finally, the study endpoint assessed for validation of MHR, the TIMI risk score, is an intermediate one. To prove the real importance of MHR in clinical settings, it should be assessed in terms of the complications of STEMI (mortality or major adverse cardiovascular events). In the present context, at best, it can be as useful as the TIMI risk score. The study's results, revealing a correlation coefficient of 0.479, show it is far from achieving even this aim. One interesting alternative would be to assess the relevance of MHR in addition or as an alternative to the TIMI score. Of course, to reach significance with clinical endpoints, a larger study with a longer follow-up would be required.

MHR is a new circulating biomarker. Like other novel biomarkers, it is affected by publication bias: positive studies tend to be published and negative ones do not. In these circumstances, the strength and potential value of any new biological marker, including MHR, tend to be overestimated.

In conclusion, this is an interesting paper which raises the possibility of a new simple and early marker of STEMI prognosis: MHR. MHR has a good overall correlation with the TIMI risk score. It is an indicator of mechanisms (inflammation and the 'cleaning' process) that are not usually assessed. It may thus function as a new tool for assessing patients with STEMI, or an additional tool to increase the discriminative power of other risk scores.

To gain clinical application, this marker must be evaluated in randomized prospective studies with hard endpoints and appropriate samples to reach solid conclusions. I believe that, until the results of such studies are available, conventional assessment, including immediate Killip class and TIMI or other risk scores, will maintain its importance in clinical practice.

A final comment: With today's practice of immediate coronary revascularization for virtually all patients with acute STEMI, whatever the risk, risk stratification is not the cornerstone of the approach to STEMI. However, risk stratification can be specially useful in patients with non-ST-elevation MI, in whom identification of high risk can change the approach.

Conflicts of interest

The author has no conflicts of interest to declare.

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