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EDITORIAL COMMENT

Cardiogenic shock in acute myocardial infarction: Stratify to prevent

Choque cardiogénico no enfarte agudo do miocárdio: estratificar para prevenir

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Cardiogenic shock (CS) is defined as persistent hypotension (systolic blood pressure <90 mmHg) secondary to myocardial dysfunction, associated with signs of organ hypoperfusion. CS may be present in 10% of patients with ST-segment elevation myocardial infarction (STEMI) and is associated with 30-day mortality of about 50%.[1] In the majority of STEMI patients, hemodynamic deterioration occurs after hospital admission, which means that there may be room for preventive measures and highlights the importance of early recognition of those most likely to evolve to CS.[2]

Scores such as Controlled Abciximab and Device Investigation to Lower Late Angioplasty Complications (CADILLAC), Thrombolysis in Myocardial Infarction (TIMI), the Global Registry of Acute Coronary Events (GRACE) and the Zwolle risk score are used to stratify patients and enable the adoption of different levels of clinical monitoring, therapeutic care and post-discharge strategies.[3,4] However, the search for simpler and more accurate scores has continued.

The shock index (SI) is defined as the ratio of heart rate to systolic blood pressure, and was introduced in 1967 by Allgower and Burri to assess the degree of hypovolemia in hemorrhagic or infectious shock states.[5] The SI, which is easy to calculate, is an objective measure of cardiovascular performance and a marker for predicting the onset of hypotension. Assessment of SI in the context of acute myocardial infarction was only used more recently, and a first meta-analysis, of eight studies enrolling 20 404 patients, was published last year.[6] A high SI was associated with increased in-hospital mortality and higher risk of short- and long-term adverse outcomes compared to low SI.

An important limitation of SI is the lack of information about systemic vascular resistance status. Mean arterial pressure (incorporating both systolic and diastolic blood pressure) best represents tissue perfusion status. The modified shock index (MSI), which is the ratio of heart rate to mean arterial pressure, has been shown to be a better predictor of mortality than heart rate, systolic blood pressure, diastolic blood pressure and SI alone in trauma patients.[7]

Shangguan et al. were the first to assess the predictive value of MSI in the context of STEMI.[8] In a retrospective study of 160 consecutive patients, they found that MSI $\geq$1.4, assessed in the emergency department, was an independent factor for major adverse cardiac events and seven-day all-

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cause mortality, with a stronger association than SI. Yu et al. retrospectively studied 1864 STEMI patients undergoing primary coronary angioplasty to assess whether admission age SI (age multiplied by SI) and MSI were useful clinical parameters to predict long-term prognosis, with both showing good prognostic performance.[9] The cutoff value of MSI for the prediction of all-cause mortality was 0.71.

In this issue of the Journal, Abreu et al.[10] assess the prognostic value of MSI to predict six-month mortality in a large retrospective observational study of 1158 STEMI patients without cardiogenic shock on admission. They found that MSI $\geq$0.93 was present in about a quarter of the patients and was associated with worse in-hospital clinical course. Adverse events, acute heart failure and cardiogenic shock were significantly more frequent in this subgroup. MSI was also an independent predictor of overall six-month mortality. The cutoff of 0.93 identified by the authors is between those in the above studies, which presumably reflects methodological differences, such as population selection and the timing and method for assessing hemodynamic parameters. However, their approach of using MSI in patients with no shock at admission, and assessing heart rate and blood pressure in the hemodynamic laboratory, seems to be the most appropriate and practical way to apply this index in clinical practice.

Their study has limitations, some of which are acknowledged by the authors, including its single-center and retrospective design, the lack of a control group to effectively test their hypothesis, and the lack of comparison with other hemodynamic indices or risk scores. Nevertheless, the authors should be congratulated for their important contribution to an issue that is still poorly defined and that needs further investigation, since a simple risk stratification of these patients remains an unmet clinical need. They have paved the way for future studies that may validate this strategy.

Conflicts of interest

The author has no conflicts of interest to declare.

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