

Editorial

Simple, Obvious, Forgotten

Sencillo, obvio, olvidado

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Heart failure (HF) complicating acute coronary syndromes is associated with increased in-hospital mortality and poor long-term survival.^{1,2} Appropriate aggressive treatment based on early recognition of this high-risk group of patients is recommended by current guidelines.^{3,4} The time-honored Killip classification of acute heart failure (AHF) in the setting of acute myocardial infarction⁵ is one of the components in the GRACE (Global Registry of Acute Coronary Events) score⁶—the guideline-recommended tool for prognostication of acute coronary syndrome patients. The adoption of guidelines by practising physicians eventually leads to improved outcomes.^{7,8}

In the article published in *Revista Española de Cardiología*, Masip et al.⁹ focus on the value of bedside pulse-oximetry (SpO₂) in the diagnosis of AHF in the setting of noncardiogenic shock acute myocardial infarction patients. The cumulative incidence of AHF in this small cohort was 35%, in agreement with previously reported data from the Israeli national HF survey,¹⁰ but was twice that observed in the GRACE cohort of non-ST-elevation myocardial infarction complicated with AHF (17%) on admission, and more than that reported by the Euro Heart Survey ACS-I (26%).^{11,12} Importantly, AHF was clinically silent in the majority of the patients—less than 20% had some degree of shortness of breath. Morning baseline oxygen saturation correlated well with Killip class and Battler et al.'s score¹³: -0.666 and -0.619 respectively ($P < .001$). In the univariate analysis for predicting 1-year death or rehospitalization for HF, SpO₂ showed the highest odds ratio (OR) (95% confidence interval [95%CI]), OR=5.5 (95%CI, 2.6–11.8)—among other measurements—left ventricular ejection fraction, OR=4.3 (95%CI, 1.9–9.6); heart rate >76 bpm, OR=2.5 (95%CI, 1.2–5), and respiratory rate >21 breaths/min, OR=2.5 (95%CI, 1.2–5), comparable with a prior history of HF—OR=5.5 (95%CI, 2.3–12.9)—, a well-known predictor of in-hospital HF in ACS patients.¹⁴ The SpO₂ cut-off level of <93% showed the greatest area under the curve with the highest test accuracy (83%). Importantly, among 206 enrolled patients, 29 were misclassified by the Killip score, 20 (10%) were false negative and 9 (4%) were false positive. The combination of SpO₂ with Killip class and respiratory rate increased the specificity and the positive predictive value of the models.

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The role of arterial hypoxemia in the cascade of HF complicating acute myocardial infarction was recognized in the classical works of Fillmore et al.,¹⁵ indicating that arterial hypoxemia roughly correlates with left ventricular dysfunction. The role of low oxygen tension as a powerful indicator of patients' severity is so obvious to clinicians that this parameter has not been evaluated systematically so far. The European Society of Cardiology guidelines on HF¹⁶ recommend arterial blood gas analysis as class I, but the level of evidence is C, indicating a lack of adequate evidence. SpO₂ is mentioned as a noninvasive replacement for arterial blood gas analysis with the known limitations of inability to assess CO₂ dynamics, as well as unreliable readings in low perfusion or decreased cardiac output states.

SpO₂ is a composite of different pathophysiologic processes expressing the balance between blood supply and the level of gas exchange in the lungs. This complex system involves cardiac output, regional blood redistribution and the level of gas exchange in the lungs. The lack of standardization of reporting (whether blood gases obtained by arterial puncture, mixed venous or peripheral venous sampling, measurement of SpO₂ during air or oxygen respiration) precluded the widespread recommendation of this important parameter in acute coronary syndrome/acute heart failure.

One of the first studies on the role of SpO₂ in determining the clinical profile of AHF was the single center registry of 340 patients,¹⁷ showing that simple assessment of impending respiratory and hemodynamic failure at admission by measuring the saturation of O₂ and systolic blood pressure enables rapid and accurate risk stratification of patients admitted for AHF. Unfortunately, important variables, such as the conditions during the sampling and the sampling techniques used, were not detailed. Despite the multiple limitations, the prognostic relevance of SpO₂ was highlighted.

The study by Masip et al.⁹ is the next step in the right direction—reporting SpO₂ in a standardized manner—morning hours, breathing room air, or several minutes after oxygen discontinuation. These authors point to the utility of baseline oxygen saturation as an additional tool for the diagnosis of AHF and the assessment of prognosis in patients with acute myocardial infarction even after stratification by Killip and Kimball⁵ and Battler et al.'s radiology scores.¹³

Despite the limitations of that study, described in the article itself, the main message is clear: noninvasive oxygen saturation is an easily obtainable, simple, and cheap indicator of HF status which deserves standardized reporting and inclusion in further clinical trials and registries dealing with AHF in the setting of acute myocardial infarction.

CONFLICTS OF INTEREST

None declared.

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