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EDITORIAL COMMENT

Non-invasive ventilation for cardiogenic shock associated respiratory failure – Striking the perfect balance between risk and benefit

Ventilação não invasiva na insuficiência respiratória associada ao choque cardiogénico – encontrar o equilíbrio perfeito entre risco e benefício

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Respiratory failure is a common complication of cardiogenic shock (CS), requiring positive pressure ventilation (PPV) in about half of patients admitted to the cardiac intensive care unit (CICU).[1] Increased left ventricular (LV) filling pressure is the primary mechanism, generating hydrostatic transudation of fluid through the alveolar-capillary membrane, pulmonary blood shunting and low arterial oxygen content.[2] Secondary increases in ventilatory drive and work of breathing attempt to compensate both for hypoxemia and lactic acidosis. However, diverting a significant proportion of total systemic oxygen consumption to fuel the respiratory muscles further worsens the body's oxygen debt and over time leads to ventilatory pump fatigue and ensuing hypercapnia.[3] The persistence of impaired gas exchange and mixed acidosis aggravates myocardial ischemia and perpetuates cellular dysoxia and organ dysfunction that characterize the downward spiral of CS.[4] The question, then, of how best to approach respiratory failure during CS bears undeniable relevance.

In registries and trials on CS, the use of PPV for respiratory failure has ranged between 50 and 88% and although no strong evidence exists on which type and modality of ventilatory support is preferable, the presence of CS has generally been regarded as a contra-indication for non-invasive ventilation (NIV); it is a common exclusion criterion in randomized clinical trials in acute respiratory failure.[3,5] In acute cardiogenic pulmonary edema (ACPE), PPV addresses the hemodynamic problem by reducing both LV preload and afterload while simultaneously shifting Starling forces and redistributing fluid to the pulmonary capillaries through the use of positive end-expiratory pressure (PEEP), thus improving gas exchange.[1] In hemodynamically stable patients with ACPE, the use of NIV prevents endotracheal intubation (ETI) and reduces mortality (number needed to treat=29 and 28, respectively) when compared to standard oxygen therapy.[6] Preventing ETI and invasive mechanical ventilation (IMV) could provide additional benefits by decreasing the incidence of ventilator-associated pneumonia, sedation requirements, diaphragmatic weakness, delirium and duration of ICU stay, all of which may negatively impact outcomes.[1] Under these premises, NIV would be a logical and acceptable option even in CS. However, NIV efficacy is in turn influenced by patient cooperation, alertness and existing secretions; it does not completely abolish the work of breathing and presents a non-negligible failure rate that could be exacerbated in refractory CS.[1]

In this issue of the Journal, Carrillo-Aleman et al. attempt to answer ongoing doubts about NIV by presenting a ret-

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rospective analysis of 698 patients with CS admitted to a single center, 300 of whom requiring NIV (43%) were included and analyzed for predictors of NIV failure.[7] Mean patient age was 73.8±10.5 years, 57% were male and acute coronary syndrome (ACS) was present in 54.7% of cases. Based on propensity score, patients undergoing NIV were further matched with those requiring ETI and IMV (n=164 for both groups) and compared for outcomes. The authors found that NIV was associated with lower in-hospital (36% vs. 48.8%, p=0.025) and one-year mortality (52.4% vs. 64%, p=0.034). This effect was maintained in the subgroup of SCAI-C patients, while in SCAI-D and E patients, no statistically significant difference was found between the two strategies. In a field troubled by uncertainties, their work deserves a word of recognition for describing one of the largest patient series, while at the same time portraying a well-organized and protocolized implementation of NIV in a high-volume ICU with substantial accumulated experience in delivering different respiratory support modalities.

The careful interpretation of such an apparent benefit should nonetheless consider the study's observational design, long timeframe (1997–2021) and the specific setting in which it took place. Of note, elderly patients in a non-cardiac ICU with a high percentage of ''do not intubate'' (DNI) orders were included. This may have contributed to the higher rate of NIV when compared to other published cohorts and limits its external validity.[8] Due to the non-randomized design, patients offered immediate ETI could have been so on the basis of a clinician perceived higher clinical severity, dictating a worse outcome due to selection bias, which propensity score matching mitigates but does not fully eliminate. Conversely, the same clinical reasoning, coupled with properly applying and monitoring CS patients under NIV for signs of either non-improvement or clinical deterioration may have decisively contributed to the conceivable benefit of NIV in patients that subsequently improved. Still, in the patients that fail NIV, the possibility that delayed ETI contributes to increased morbidity and mortality is plausible and supported by the authors' own results and other observational data. In a subanalysis of the TRIUMPH trial, Van Diepen et al. showed a stepwise 30-day mortality increase for each hour delay in CS patients requiring IMV.[9] In the present study, NIV failure rate was 51%, which was almost double the rate of the one found in the CardShock study.[8] Mortality in patients failing NIV was 86.1%, a prohibitively high value that is only slightly attenuated by excluding patients with a DNI order. Admittedly, the *a priori* clinical severity of NIV candidates (e.g., SCAI-D/E vs. C, higher HACOR score or SAPS score) of which respiratory failure can be considered a surrogate, will influence not only the choice of respiratory support but the very success of NIV. A higher NIV failure rate in SCAI-D and E is therefore not surprising and although one cannot easily emerge from the ''chicken and egg'' conundrum, and unless a patient has a DNI order, NIV is likely unsuitable for most advanced CS stages.

Accepting these limitations, the clinician faced with a patient in CS that displays hypoxemia and/or signs of increased work of breathing and who considers NIV needs to make an expeditious decision. Based on the work by Carrillo-Aleman et al., alert CS patients without ACS who are not deteriorating (i.e. SCAI-C stage), that present with non-severe degrees of hypoxemia or signs of imminent respiratory exhaustion would be the ideal candidates for a trial of NIV under close bedside surveillance for markers of clinical worsening. By providing inspiratory assistance, bilevel NIV may be preferable (>90% use in the authors' cohort), with adaptive degrees of PEEP and fraction of inspired oxygen. If signs of increased worked of breathing do not quickly improve, ETI and IMV should not be delayed.

For CS patients requiring respiratory support, other relevant issues on initial management and therapeutic strategy will remain unsolved and add more complexity to the decision algorithm. Among them stands the approach to respiratory failure in patients eligible for mechanical circulatory support (MCS). Whether by directly unloading the LV, ensuring adequate systemic and myocardial oxygen delivery or a combination of both, these devices entail the possibility of precluding EIT and IMV in patients without signs of impending respiratory exhaustion, at the expense of MCS-related complications.[10] While NIV can be an option in this scenario (20 patients received extracorporeal membrane oxigenation and 39 patients an intra-aortic balloon pump during the study period), the reduced sample size hampers any meaningful conclusions. Secondly, in the ∼30% of patients presenting in classic CS without signs of pulmonary congestion[4] (typically chronic heart failure patients on diuretics but also after acute myocardial infarction) who persist with respiratory distress and hyperlactatemia after fluids and/or catecholamines, the decision of whether or not to initiate any kind of respiratory support and its interaction with MCS are even more difficult to untangle. Finally, the role of high-flow nasal oxygen therapy in CS is not clearly defined although it could be an option in the absence of severely increased work of breathing or at de-escalating from NIV or IMV.[5]

Even though it is unlikely that definitive evidence will appear any time soon, the results presented by Carrillo-Aleman et al. are a step forward in the task of guiding clinicians and their teams to deliver the correct respiratory support at the right time. Successfully navigating this delicate path will require frequent reassessment of patient hemodynamic severity as well as the predictable and actual response to NIV to strike the best balance between risk and benefit.

Conflicts of interest

The authors declare no conflicts of interest.

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