Journal of Intensive Medicine 3 (2023) 79–80

Contents lists available at ScienceDirect

Journal of Intensive Medicine

journal homepage: www.elsevier.com/locate/jointm

Editorial

From Acute heart failure to cardiogenic shock patients requiring admission in ICU

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We are talking about the most severe form of acute heart failure, i.e. cardiogenic shock (CS). Most epidemiological data on shock in critically ill patients focus on septic shock, which is considered the leading cause of mortality, with in-hospital mortality ranging from 20% to 60%.[1,2] Although less frequent, CS remains a genuine clinical challenge with similar or even higher mortality rates, and its outcome has seen little improvement.[1–3] CS is a critical syndrome of life-threatening peripheral hypoperfusion and organ dysfunction due to primary cardiac dysfunction.[4] It has a wide spectrum of presentation, ranging from preshock to refractory cardiogenic shock, and patients with refractory cardiogenic shock have the worst outcome.[1–5]

Using the rationale that morbidity and mortality from CS stem directly from complications associated with tissue hypoperfusion, intensivists play a key role in managing this multifaceted and complex pathology.[2,5,6]

Indeed, CS is a pathophysiologically complex and phenotypically heterogeneous clinical syndrome with multiple etiologies.[1,4,5] It can be caused by an acute cardiac disease or a systemic illness that decompensates a chronic cardiac condition associated with minimal cardiac reserve. A decade ago, 81% of CS was due to underlying acute coronary syndrome. However, the contribution of ACS has declined over the past two decades, in parallel with an increase in CS of other etiologies.[1,3] Thus, Eftychiou et al.[7] explored the limitations of commonly used historical CS classifications, including limited applicability to non-ACS populations and the inability to account for serial assessments. They also focused on *de novo* subtypes of CS (fulminant myocarditis, right ventricular failure, Takotsubo syndrome, postpartum cardiomyopathy, CS due to valve lesions, and other cardiomyopathies) and acute-on-chronic heart failure-CS.[7]

The diagnostic of CS is clinical but current guidelines emphasize the initiation of basic monitoring since the first hours of shock that should be completed by an advanced one in more complicated and refractory shock.[2,4,8] Hamzaoui et al.[9] detailed the different parameters relevant to each monitoring approach and how they can be used to support the optimal management of these patients. In fact, recent analyses of North American registries suggested that outcomes might be improved through early shock recognition and the use of standardized treatment algorithms.[6,9] Echocardiography is pivotal to diagnosing, classifying, and escalating CS management but the choice and the management of pharmacologic and mechanical circulatory support (MCS) therapies often require advanced physiological information derived from pulmonary artery catheters to guide MCS selection, therapeutic response, and device weaning.[9] Although the primary focus of treatment in CS should address the underlying primary insult, the mainstay of CS management first relies on inotropic agents and vasopressors to restore oxygen delivery and maintain normal ventricular-arterial coupling.[2,4] The practice and refinement of the selection of vasoactive agents is supported by limited clinical outcome data.[2,4] Lescroart et al.[10] reviewed current medical treatments of CS addressing excitation–contraction coupling and specific physiology on applied hemodynamics. The use of inotropes, vasopressors, and immunomodulating drugs is discussed according to the current international guidelines. Actually, the medical therapy of CS has barely changed over the past decade and primarily relies on inotropic drugs and vasopressor agents.[10] As Lescroart et al.[10] reported, dobutamine should first be considered to restore cardiac output while norepinephrine is used to restore end-organ perfusion pressure targeting MAP > 65 mmHg, as well as tissue perfusion pressure. Thus, dobutamine and norepinephrine should be introduced concomitantly when CS is associated with low MAP. Of note, the above-mentioned catecholamine-based management of CS has mainly been validated in an ischemic shock population. These guidelines are exported to other CS, but two conditions deserve a specific mention since catecholamine infusion could worsen outcomes: namely, Takotsubo syndrome and obstruc-

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https://doi.org/10.1016/j.jointm.2023.03.001
Received 4 March 2023; Accepted 9 March 2023. Managing Editor: Jingling Bao
Available online 31 March 2023

tive hypertrophic cardiomyopathy. For Takotsubo syndrome, clinicians should first consider using catecholamine-free therapy based on phosphodiesterase inhibitors. Caution is required in obstructive hypertrophic cardiomyopathies, as any increase in increased obstruction of the inotropism could result in LV outflow tract and worsen hemodynamics. New treatments targeting inflammation, vasoplegia, or inotropism are currently proposed to improve outcomes but their use deserves more studies.[2,4,10]

Despite an improvement in hemodynamic parameters, vasopressors and inotropes increase myocardial metabolic demand, impair tissue perfusion, increase the risk of arrhythmias, and may lead to complications.[10] The use of these drugs should therefore be minimized where possible and escalation of inopressors should be signal consideration of mechanical circulatory support (MCS) strategies in selected patients.

Early short-term MCS initiation may prevent the toxic use of catecholamines and is recommended in international guidelines, but the optimal timing remains uncertain and is complicated by the complexity of CS phenotypes.[2,4,6] The validated SCAI shock classification helps refine short-term MCS selection based on the stage of CS, as the outcome of each MCS will vary depending on the acuity stage at which MCS is initiated.[6] Stavros Eftychiou et al.[7] reviewed all short-term MCS focusing on the treatment of *de novo* subtypes of CS as well as of CS in cancer patients. The device selection recommendations are supported by limited evidence and are mainly guided by the pathophysiology of the type of CS, local expertise, and device availability/cost.[7] Among short-term MCS, veno-arterial extracorporeal membrane oxygenation devices (VA-ECMO) can provide partial or complete biventricular circulatory support and respiratory support. Despite the limitations, VA-ECMO should be the first-line MCS. Combining Impella or intraaortic balloon pump support with ECMO might decrease left ventricular pressure and improve outcomes.[4,7]

These short-term MCS devices are used as bridge-to-recovery, bridge-to-decision, bridge-to-bridge, and bridge-to-transplant.[4,6] Regarding long-term MCS, left ventricular assist device (LVAD) therapy is well-established in the treatment of patients with end-stage heart failure, although heart transplantation remains the gold standard.[4] Due to a shortage of donor organs, as well as the number of patients ineligible for transplantation, LVAD therapy has gained importance and intensivists need to understand the physiology of the devices, potential complications and their management.[4,6] Thus, Morshuis et al.[11] proposed a review of long-term MCS, the most frequent complications occurring in patients with LVAD after the post-operative period (bleeding, driveline infections, thrombosis, device malfunction, right ventricular failure, and arrhythmias) and their management.

So enjoy reading the special issue on CS. Hopefully, it will allow us to make progress in the management of CS patients.

Conflicts of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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