

EDITORIAL COMMENT

An Evolving Roadmap for Cardiogenic Shock Requiring Temporary Mechanical Circulatory Support*



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Cardiogenic shock (CS) contributes to significant global morbidity and mortality and can result from several causes.¹⁻³ These include acute myocardial infarction (AMI), heart failure (HF), arrhythmia, fulminant myocarditis (FM), valvular heart disease, and pulmonary embolism (PE). Although the use of temporary mechanical circulatory support (TMCS) for CS has been burgeoning over the past 2 decades,³ their specific use, timing, and impact on clinical outcomes in differing causes of CS remain poorly understood. Expert consensus recommends early consideration for TMCS in patients with refractory CS with certain caveats.²⁻⁴ The intra-aortic balloon pump (IABP)-shock trials have shown no real benefit in patients undergoing percutaneous coronary intervention after AMI-CS. Whether TMCS in ST-segment elevation myocardial infarction or for ventricular unloading pre- or post-percutaneous coronary intervention in AMI-CS patients is beneficial remains relatively unknown. Given the advent of other percutaneous ventricular assist devices (pVADs) that help with right, left, or biventricular failure as a bridge to recovery, decision, or advanced therapies, TMCS use for non-AMI forms of CS is ripe for exploration.

TMCS USE PATTERNS AND SURVIVAL IN VARIOUS CS ETIOLOGIES

In this issue of *JACC: Asia*, Kondo et al⁵ explore TMCS use and survival outcomes in 65,837 adult patients who had CS and required TMCS identified from a nationwide Japanese JROAD-DPC (Japanese Registry of All Cardiac and Vascular Diseases-Diagnosis Procedure Combination) database of 927 teaching acute-care hospitals over a contemporary 7-year period. More than 77% of patients had AMI-CS, 11% had HF-CS, and 2%-3% had other types of CS.⁵ Overall, the in-hospital mortality rate was 32%,⁵ due perhaps to temporally improved revascularization strategies, more hemodynamic tailoring of therapeutic interventions, and institutional or regional thresholds for TMCS use.

Interestingly, those with HF-CS or arrhythmia-CS had, respectively, similar (HR: 0.99; 95% CI: 0.92-1.05) or worse (HR: 1.14; 95% CI: 1.04-1.26) in-hospital mortality as compared with those with AMI-CS, suggesting these patients should be considered a vulnerable, critically ill cohort.⁵ By contrast, patients with CS due to valvular heart disease, FM, or PE were more likely to survive to hospital discharge.⁵ Moreover, TMCS use varied depending on the etiology of CS.⁵ The IABP was used in >75% of patients with AMI-CS or HF-CS, whereas veno-arterial extra-corporeal membrane oxygenation (VA-ECMO) was used alone in those with PE (72%) or concomitantly with the IABP in nearly half of the patients with CS from FM or arrhythmogenic causes (43%-56%).

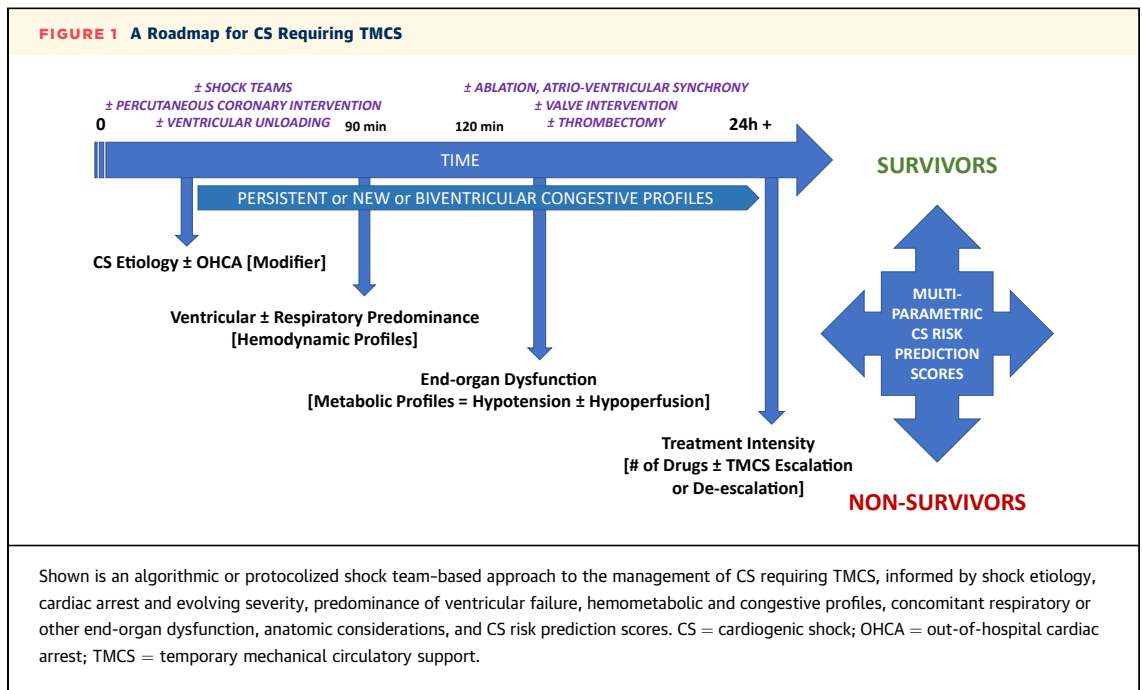
SHAPING THE ROADMAP FOR CS MANAGEMENT

These study findings underscore several important concepts surrounding the early recognition of shock etiology to facilitate targeted management toward better survival. Not surprisingly, the need for TMCS

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use considers the CS etiology, the congestive profile, the ventricular failure phenotype, as well as the presence of respiratory and other end-organ dysfunction. Isolated left ventricular failure may benefit from primarily left ventricular support devices, whereas cardiorespiratory or biventricular failure may require VA-ECMO or other combinations of TMCS. This study highlighted other interesting findings. First, the proportion of CS patients >80 years of age, HF vs AMI cases, pVAD vs IABP use particularly in the FM-CS group, along with in-hospital mortality from CS all increased over time in Japan.⁵ Moreover, the CS etiology, type of TMCS use, and survival outcomes differed by age, sex, and CS severity.⁵ The time to and duration of TMCS use, along with the hospital length of stay and cost were also influenced by the CS etiology.⁵ Finally, the predictors of poor survival included older age especially in the AMI and FM groups, low or high body mass index, annual number of TCMS used, need for cardiopulmonary resuscitation and intubation, and lack of right heart catheterization.⁵ Mechanistically these make sense—older, frail, more comorbid, or sick patients and certain vascular anatomy and body habitus can place a patient at more risk of vascular, coagulopathic, or end-organ complications that impact the provision of adequate circulatory support and decongestion. In addition, more volume of TMCS can equate with fewer complication rates due to operator or institutional experience, a volume-outcome relationship that has been previously observed in other

diseases across the surgical and interventional literature. Finally, the Cardiogenic Shock Working Group, the Society of Cardiovascular Angiography and Interventions (SCAI), and the National Cardiogenic Shock Initiative all highlight the implications on clinical trajectory and survival of different hemometabolic profiles, evolving SCAI stages of CS and longitudinal congestive profiles, shock teams, and shock protocols (Figure 1).⁶⁻¹²

THE UNMAPPED ROADWAYS OF CS CARE

There are several limitations of this study. The JROAD-DPC database reflects only observational data from the teaching hospitals within Japan with acute-care cardiovascular beds, where some missing data was in fact imputed. Data was analyzed by fiscal year and lacked information regarding the hemometabolic profiles, congestive states, and SCAI stages of CS, as well as of TMCS escalation or de-escalation or the use of right-sided pVAD support beyond VA-ECMO, which makes it difficult to compare these study findings with published CS data or generalizable to other geographic regions, institutions, or health care systems. As well, selection bias excluded certain populations (eg, those undergoing cardiac surgery or needing TMCS post-cardiotomy or renal replacement therapy), which could have provided insights into those CS states, hemometabolic or congestive profiles, and end-organ management. Randomized trials are

evaluating VA-ECMO or pVAD use but only for certain causes of CS.¹³⁻¹⁶ Understanding better how TMCS escalation and de-escalation strategies can be tailored to the patient's hemometabolic profile, congestive state, and end-organ dysfunction perhaps informed by CS risk prediction scores may allow for optimizing patient survival and morbidity for each CS etiology, or palliation where there is futility due to irreversible sequelae.¹⁷ Future work will need to tackle these and other important knowledge gaps in the diagnosis and management of CS through precision medicine that identifies endotypes of CS amenable to targeted therapies.

CONCLUSIONS

This study highlights the ever-changing landscape of CS worldwide. Older patients are presenting with more severe CS and congestive profiles increasingly due to various nonischemic causes of decompensation. The importance of tailoring the TMCS to the patient's clinical presentation by CS etiology and

severity, cardiac arrest, ventricular failure predominance, hemometabolic and congestive profiles, concomitant end-organ dysfunction, and anatomic considerations impacts the patient's clinical trajectory. Future studies will need to explore these elements more closely for each etiology and phenotype of CS to help clinicians match the right device to the right patient at the right time.

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