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2022 Symposium Presentation

Hemodynamic Variations in Cardiogenic Shock Phenotypes

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Abstract

Patients with cardiogenic shock are not all the same. They present with a variety of hemodynamic profiles and other features that may allow us to create specific phenotypes. It is possible that phenotyping these patients at presentation may then help us to identify the optimal and earliest therapies that will improve outcomes and, at the same time, help us to overcome some of the heterogeneity currently undermining clinical trials.

Keywords: phenotype, hemodynamics, heart failure, cardiogenic shock

Background

The guidelines for and definitions of cardiogenic shock are slowly evolving. Put simply, cardiogenic shock is a low cardiac output state that leads to end-organ hypoperfusion, tissue hypoxemia, and its sequelae. Ten years ago, a specific phenotype for hemodynamic shock was investigated, and separate phenotypes for cardiogenic hypovolemic shock were presented. However, we know now that not all cardiogenic shock is created equal. Patients in the cardiac intensive care unit with cardiogenic shock may have very different baseline characteristics, illness severity, shock presentations, and hemodynamic profiles.

Cardiogenic Shock Phenotypes

In 2017, the American Heart Association published a scientific statement that emphasized and reintroduced the concepts involved in understanding and managing cardiogenic shock.¹ There are multiple phenotypic and hemodynamic presentations for cardiogenic shock that include combinations of wet and dry volume status with warm and cold peripheral circulation. The classifications presented were extrapolated largely from the acute heart failure literature, but they were limited by the lack of research available at the time. Indeed, patients can present with classic cardiogenic shock in which

their cardiac filling pressures and systemic vascular resistance are elevated; this presentation is the classic cold and wet phenomenon. Other patients may present in vasodilatory cardiogenic shock and are relatively euvolemic with normal cardiac filling pressures. In fact, the seminal SHOCK Trial and Registry found that a quarter of patients presented with cardiogenic shock and a low systemic vascular resistance—an allusion to a sepsis-like phenotype for patients who are actually in cardiogenic shock.²

Thus, without question, cardiogenic shock has multiple phenotypes. The term *phenotype* means a set of observable characteristics of an individual brought about by the genotype interacting with an environment. Many clinicians in the field of heart failure have observed that patients with different phenotypes of cardiogenic shock may respond differently to the pharmacologic and mechanical support strategies that are available. Individual patient groups could be classified by phenotypic presentations and may have variable risk-benefit profiles, particularly when applying different therapeutic strategies. Further, completed clinical trials in the field have identified a marked heterogeneity in the cardiogenic shock population, which confounds the results. Thus, there is a high likelihood that the failure of the clinical trials to show demonstrable improvement in outcomes for certain therapeutic modalities, particularly mechanical circulatory

support modalities, may be due to a lack of understanding of hemodynamic variations and phenotypes.

To complicate matters, the definition of cardiogenic shock has also varied between trials and guidelines.¹ Most definitions have minimal hemodynamic data and qualifiers as inclusion or exclusion criteria, which makes comparing results across studies difficult, if not impossible. As a proof of concept, Seymour and colleagues looked at a noncardiac population and were able to ascertain sepsis phenotypes through cluster and multidimensional cluster techniques.³ Each phenotype had different immune responses and outcomes. More recently, the Cardiogenic Shock Working Group used a cluster demonstration to identify hemodynamic phenotypes.⁴ However, the question now is, how do we use phenotype knowledge in practice?

Treatment

When treating patients in the cardiac intensive care unit, pathophysiology should be used to guide therapy, especially for patients receiving mechanical circulatory support services. By understanding and leveraging the knowledge of physiology, one can begin to see the different hemodynamic effects of our pharmacologic agents. Invasive hemodynamic information can identify what the appropriate pharmacologic therapy should be. If the patient is in classic cardiogenic shock with high systemic vascular resistance, an agent that increases contractility but also vasodilates would be ideal. However, if the patient has a low systemic vascular resistance or a mixed shock picture, adding a vasodilator may not be appropriate. Instead, an agent that increases systemic vascular resistance with or without increasing contractility might be better.

Historically, the pulmonary artery (PA) catheter has not been recommended for this patient population. In heterogeneous critical care populations, using a PA catheter did not demonstrably change outcomes.⁵ The ESCAPE trial confirmed the meta-analysis in an acute heart failure population and did not show a demonstrable benefit in the use of PA catheters for routine therapy. Thus, the PA catheter was “put to rest” in the mid-2000s, but there has been a resurgence in recent times. Today, cardiogenic shock and the use of temporary mechanical circulatory support strategies may benefit from an understanding of the hemodynamic and metabolic profiles of these patients; thus, the American College of Cardiology/American Heart Association Guidelines now support the use of PA catheters in patients with cardiogenic shock.

Conclusion

Invasive hemodynamic assessment is important for cardiogenic shock management. To understand how to more precisely leverage invasive hemodynamic information, however, more data is needed. For any patient profile, complexity should be balanced with practicality. Etiology, cardiomyopathy characteristics, and hemodynamics are factors that can contribute to developing an optimal and informative phenotype. A better understanding of the utility of patient phenotyping in cardiogenic shock should help to advance the field and optimize patient outcomes.

Disclosures

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