Short Communication

Interventional Cardiology

Current management of cardiogenic shock from stress-induced cardiomyopathy: Consideration of catecholamine excess and left ventricular outflow tract obstruction

Sun-Joo Jang[1], Abhinav Aggarwal[2], Carlos D. Davila[1*]

[1]Department of Internal Medicine, Section of Cardiovascular Medicine, Yale University School of Medicine, New Haven, CT, USA;

[2]Department of Medicine, Yale New Haven Health Bridgeport Hospital, Bridgeport, CT, USA

*Author for correspondence:
Carlos D. Davila, Section of Cardiovascular Medicine, Department of Internal Medicine, Yale University School of Medicine, New Haven, CT, USA, E-mail: carlos.davila@yale.edu

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Abstract

Stress-Induced Cardiomyopathy (SIC) or Takotsubo cardiomyopathy is usually a reversible myopathy precipitated by emotional or physical stress. The important pathophysiology underlying SIC includes excessive catecholamine leading to myocardial stunning and in certain occasions dynamic Left Ventricular Outflow Tract (LVOT) obstruction. Thus, cardiogenic Shock (CS) related to SIC (SIC-CS) would need a tailored strategy considering catecholamine excess and LVOT obstruction. Invasive hemodynamic monitoring is important to minimize the use of inotropics/vasopressors and decide the timing of Mechanical Circulatory Support (MCS). Early use of MCS in advanced SCAI shock stages may be important. The choice of MCS devices should be carefully assessed in the presence of severe LVOT obstruction. Further studies are needed to understand the safety, efficacy, and timing of MCS devices in patients with SIC-CS.

Keywords: Stress-induced cardiomyopathy • Takotsubo cardiomyopathy • Cardiogenic shock • Mechanical circulatory support

Introduction

Epidemiology and diagnosis

Stress-Induced Cardiomyopathy (SIC), also referred to as Takotsubo cardiomyopathy or apical ballooning syndrome is an important disease requiring differentiation from Acute Coronary Syndrome (ACS) [1]. The incidence of SIC has been increasing over time with the incidence of Cardiogenic Shock (CS) ranging between 6%-20%, however, these patients with SIC-CS experience a high in-hospital mortality of 15% [2]. It is a non-ischemic cardiomyopathy triggered by catecholaminergic excess from psychological or physical stress [3,4]. However, SIC often presents ST-segment elevation on electrocardiogram and elevated cardiac enzymes mimicking acute myocardial infarction [5]. The Mayo Clinic criteria can be helpful for the diagnosis of SIC which includes four criteria: transient regional left ventricular wall dysfunction with deficits beyond a single epicardial contribution, new ST elevation, T-wave inversion or troponin elevation, absence of angiographic evidence of plaque or coronary obstruction, absence of myocarditis or pheochromocytoma. Diagnostic coronary angiogram is important to exclude any possible ACS. Right heart catheterization is also important for the diagnosis and management of CS.

Pathophysiology

Two important pathophysiological characteristics of SIC that need attention are

catecholamine excess from stressors and dynamic Left Ventricular Outflow Tract (LVOT) obstruction. High levels of epinephrine with stress switch β2-receptor coupling in the apical ventricular cardiomyocytes from Gs protein to Gi protein pathway, resulting in a negative inotropic effect in the apex where the beta receptor density is the highest [6]. Ansari et al, reported that the catecholamine use in patients with SIC can increase short-term and long-term mortality [7]. Dynamic LVOT obstruction is a common finding in SIC with an incidence of 10% to 50% [8,9]. Intraventricular and LVOT pressure gradient is generated by anterior-apical akinesis and inferior-posterior hyperkinesis which would be a leading mechanism of CS in SIC. Excessive catecholamine can also worsen this gradient by promoting further posteroinferior wall contraction.

Management

The most important treatment of SIC is to relieve emotional or physical stress. However, patients with SIC who develop CS (SIC-CS) require further intensive cardiac critical care. Stepwise consideration of LVOT obstruction and SCAI shock stages and customized usage of pharmacological and mechanical circulatory support would be important for the optimal management of SIC-CS [10].

SIC-CS without LVOT obstruction

It is recommended to follow the guidelines of CS in SIC patients without significant LVOT obstruction [11]. Fluid resuscitation can be carefully performed to maintain blood pressure. A trial of temporary inotropics such as dopamine, dobutamine, or milrinone is beneficial when the hypotension or shock is not resolved with fluid resuscitation. Close monitoring of invasive hemodynamics and early use of Mechanical Circulatory Support (MCS) may improve survival [12]. As MCS devices are increasingly used in patients with CS and are recommended for SCAI shock stages C, D, and E [13], they are also frequently being used in patients with SIC-CS. In advanced SCAI shock stages, refractory to pharmacological therapy, early use of MCS would be beneficial to reduce the dose and duration of inotropic therapy. Different types of MCS, such as Intra-Aortic Balloon Pump (IABP), micro-axial pump (Impella), or Extracorporeal Membrane Oxygenation (ECMO) have been utilized as a temporary bridge-to-recovery in SIC-CS. A recent study showed an increasing trend toward the use of Impella and decreasing trend toward the use of IABP [10,14]. However, differential risks and benefits of different MCS types are very limited in patients with SIC-CS with data only available with case series, observational data, or single-center studies. Considering lower cardiovascular mortality than ACS and reversible characteristics of SIC, vascular and bleeding complications related to the invasive devices and cost-effectiveness should be thoroughly compared.

SIC-CS with LVOT obstruction

For patients with SIC-CS who develop LVOT obstruction, increasing the preload with fluid resuscitation may help to reduce this gradient. The use of inotropic/vasopressors can be challenging since it can worsen the LVOT gradient [15,16]. Low-dose short-acting β-adrenergic receptor blockers (e.g., esmolol) can be attempted if the patient is not bradycardiac or is maintaining blood pressure [17,18]. However, this can be only useful when blood pressure is elevated during the hyperacute phase of SIC because of the high serum catecholamine level. There is no data showing the long-term efficacy of this therapy. The use of peripherally acting vasopressors (such as phenylephrine or vasopressin) can be beneficial to increase blood pressure avoiding further LVOT gradient generation from traditional inotropics [19]. However, close invasive hemodynamic monitoring should be considered since these agents may worsen the cardiac output. If LVOT obstruction is resolved, the patient can be treated following conservative management according to the guidelines for CS and heart failure. For persistent LVOT obstruction or shock despite careful medical treatments, early use of MCS should be considered. Devices that do not provoke LVOT gradient (such as Impella) can be beneficial in this scenario [20,21]. IABP use in SIC-CS would aggravate LVOT obstruction [22,23]. If shock or hypoxia progress, veno-arterial ECMO can be considered, however, it can increase afterload with limited myocardial recovery.

Conclusion

Management of SIC-CS should consider its specific pathophysiology of excessive catecholamine and dynamic LVOT obstruction. Early MCS use in SIC-CS and close hemodynamic monitoring may be beneficial in the management of SIC-CS. Non-LVOT gradient-provoking MCS devices, such as Impella have been more frequently used in SIC-CS. Further prospective studies are necessary to compare the safety and efficacy of different MCS devices in patients with SIC-CS.

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