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Beyond Revascularization: "The Second Golden Hour" in Post-Infarction Cardiogenic Shock

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Introduction: Acute myocardial infarction complicated by cardiogenic shock remains associated with high mortality despite advances in revascularization and mechanical circulatory support (MCS). Venoarterial extracorporeal membrane oxygenation (VA-ECMO) provides temporary hemodynamic stabilization, allowing myocardial recovery or bridging to advanced heart failure therapies.[1] We present a case highlighting the importance of timely escalation of care in refractory cardiogenic shock.

Case report: 63-year-old man presented to a regional PCI center with acute anterior ST-elevation myocardial infarction. Proximal left anterior descending (LAD) artery occlusion was treated with drug-coated balloon angioplasty, achieving TIMI II flow and requiring additional tirofiban administration. Post-procedurally, his condition deteriorated rapidly, from decompensated heart failure to systemic hypoperfusion despite vasoactive support. He was promptly referred to a tertiary center with MCS capabilities. Upon transfer, echocardiography demonstrated severely reduced left ventricular (LV) systolic function and cardiac index (CI), preserved right ventricular systolic function, and severe mitral and tricuspid regurgitation. Despite escalating vasoactive support, shock progressed from SCAI stage C to D, requiring MCS escalation. Pulseless electrical activity cardiac arrest occurred in the catheterization laboratory, requiring cardiopulmonary resuscitation. Peripheral VA-ECMO was established, rapidly restoring hemodynamic stability and end-organ perfusion. Repeat coronary angiography excluded significant coronary obstruction, with persistent TIMI II LAD flow. The subsequent course was complicated by recurrent ventricular tachycardia requiring multiple defibrillations and atrial fibrillation, while echocardiography on day two revealed LVEF 10% with LV apical thrombosis. MCS dependency, low CI, and elevated LV filling pressures prompted heart transplant listing on day four. However, on day ten, gradual improvement in ventricular contractility supported ECMO decannulation. It was urgently performed percutaneously due to uncontrolled cannulation-site bleeding. Following decannulation, LVEF improved to 20–25% with sustained hemodynamic stability without vasoactive support, marking a shift from bridge-to-transplant toward bridge-to-recovery strategy. Neuropsychiatric complications additionally suspended transplant eligibility. Rehabilitation was initiated with significant clinical improvement. However, on day nineteen, progressive pneumonia and systemic inflammatory deterioration precipitated sudden circulatory collapse, consistent with septic shock. Despite maximal intensive care, recurrent cardiac arrest resulted in death.

Conclusion: This case emphasizes early recognition of post-PCI hemodynamic deterioration and timely referral to specialized shock centers. VA-ECMO provided rescue support during cardiac arrest, enabling stabilization and partial myocardial recovery. Complications such as severe infections may precipitate hemodynamic collapse during the vulnerable recovery period. Successful management of cardiogenic shock requires continuous reassessment and timely escalation or de-escalation of circulatory support. In this rapidly evolving clinical setting, even a matter of hours can determine the window of opportunity for life-saving interventions.

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LITERATURE

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