



# Multivessel revascularization for acute myocardial infarction and cardiogenic shock: when less is more

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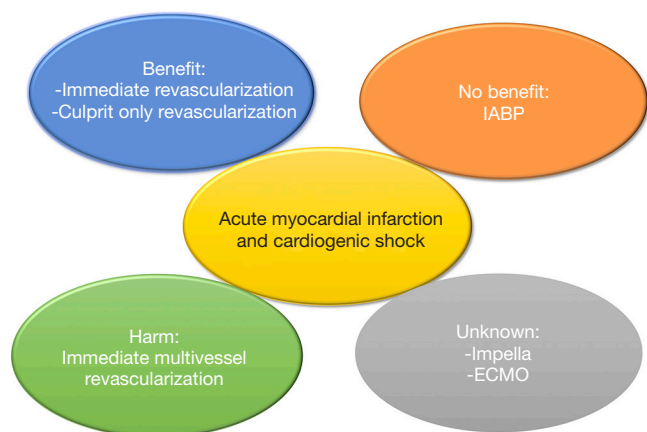
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Cardiogenic shock represents the most dreadful and the primary cause of in-hospital mortality in patients with acute myocardial infarction (AMI) (1). Cardiogenic shock (~80% of cases) occurs due to large infarction, pumps failure, infarct extension, re-infarction, or smaller infarction in preexisting left ventricular dysfunction with or without mechanical complication (2,3). Approximately 5–15% of patients with AMI are in cardiogenic shock at the time of presentation (4). Over the past 3 decades, the incidence of cardiogenic shock has been declining, a finding which has been attributed to widespread adoption of early revascularization and improvement in preventive measures. However, the prognosis of these patients remains poor. In the earlier studies such as the GUSTO-I study and the SHOCK registry, only 40% of the patients survived the hospitalization (5-8). The SHOCK trial, which randomized patients to immediate revascularization versus initial medical stabilization, showed a clear benefit of revascularization. Yet, the 30-day mortality in the patients who underwent revascularization was ~47% (5). In a recent report of the United States CathPCI registry for the years 2005–2013 showed that the in-hospital mortality has been ~30% despite increased adoption of prompt revascularization and the use of mechanical devices support as intra-aortic balloon pump (IABP) use (4), and the rates of 30 days readmissions remains high (9). Collectively, these findings suggest that we are in need for further interventions to improve the outcomes of this high-risk cohort.

Approximately 50% of patients with STEMI exhibit one or more non-culprit lesions at the time of presentation (i.e., multivessel disease). The presence of multivessel disease has been linked to worse outcomes as compared with those with culprit-only disease (10,11). Recent randomized trials have suggested that complete revascularization of non-culprit lesions either during the index procedure or as a staged procedure is associated with improved outcomes, due to a reduction in the risk of revascularization, but with no impact on hard outcomes as death and recurrent infarction (12,13). However, these trials have excluded patients with cardiogenic shock. It would be expected that those with cardiogenic shock might drive more benefit from a complete revascularization approach. In the 2015 American College of Cardiology Foundation/American Heart Association guidelines for non-culprit vessel revascularization in STEMI patients with multivessel disease were modified from Class III indication (harm), to a Class IIb indication suggesting that it is appropriate to intervene on non-culprit lesions when cardiogenic shock persists after treatment of the culprit lesion) (14), while the 2017 European Society of Cardiology STEMI guidelines gives a class IIa recommendation for complete revascularization (15). In the SHOCK trial, the rate of multivessel PCI increased over the trial period, which perhaps suggests improved operator experiences and improved technical handling in cardiogenic shock. However, this small subset had a worse



**Figure 1** Interventions that have been available for the management of patients with acute myocardial infarction and cardiogenic shock. ECMO, extracorporeal membrane oxygenation; IABP, intra-aortic balloon pump.

adjusted mortality compared with those who underwent culprit-only PCI (16). In a recent meta-analysis of 10 cohort studies with 6,051 patients with cardiogenic shock and multivessel disease, multivessel PCI was associated with higher early mortality (17). However, these data are driven from observational studies, which could be prone to unmeasured confounding and ascertainment bias. Thus, a randomized trial comparing a multivessel PCI versus a culprit-only revascularization for patients with multivessel disease would be eagerly needed.

In this context, the CULPRIT-SHOCK trial randomized 706 patients to either immediate multivessel PCI versus culprit lesion only with the option of staged revascularization of non-culprit lesions in the setting of cardiogenic shock and AMI (18). The crossover rate was relatively low (12.5% in the culprit-lesion-only PCI group and 9.4% in the multivessel PCI group). The primary end point was the composite of death or severe renal failure leading to renal-replacement therapy within 30 days after randomization. At 30 days, the composite primary end point of death or renal-replacement therapy occurred in 45.9% in the culprit-only group versus 55.4% in the multivessel PCI group [relative risk (RR) 0.83; 95% confidence interval (CI): 0.71–0.96,  $P=0.01$ ], which was driven mainly by lower death in the culprit-only group (RR 0.84; 95% CI: 0.72–0.98,  $P=0.03$ ). The RR of renal-replacement therapy was 0.71 (95% CI: 0.49–1.03,  $P=0.07$ ). Adding renal replacement therapy as an endpoint has its relevant clinical implications since those undergoing a multivessel PCI

approach are expected to receive a higher contrast volume; however, the trial showed that this does not increase the risk of renal replacement therapy. Despite the criticism that might arise from the low frequency of radial approach (i.e., <20% in both intervention groups), this trial is well conducted. The CULPRIT-SHOCK trial supports that there was a lower risk of death in patients who only had the culprit lesion treated with no difference in intensive care unit stay or duration of pressor use. This could be related to the complex interplay of various mechanistic pathways in cardiogenic shock with accelerated platelet aggregation and coagulation cascades when extra time is exerted for PCI of non-culprit lesions with further impact on ventricular function. Despite the large number of unknown deaths in the multivessel PCI group; the reported deaths were driven mainly by cerebral related deaths, despite similar stroke rates between the two groups. While complete revascularization might be of benefit in the non-shock population, this trial suggested a potential harm from this approach in the shock state. In this trial, the most common cause of death was brain injury so a plausible mechanism could be catheter manipulation.

There has been an increased interest in the use of percutaneous mechanical support devices, which has been encouraged by the poor outcomes in the cardiogenic shock population. IABP has been shown to be of no benefit in patients with cardiogenic shock who are planned for immediate revascularization (19). While other devices have been gaining interest such as continuous flow pumps (Impella) and extracorporeal membrane oxygenation (ECMO) (20). Small studies have compared IABP with new percutaneous support devices but these studies were all underpowered for hard end points. In the absence of data from randomized trials, the 2015 SCAI/ACC/HFSA/STS clinical expert consensus statement on mechanical support did not provide specific direction on device selection in cardiogenic shock (21). In the CULPRIT-SHOCK trial, the use of mechanical support device was nearly 30% in both arms. Impella was mainly used in the culprit lesion group while ECMO was the mainly used in the multivessel group. IABP utilization was ~25% in both arms. Despite the use of these devices, the rate of 30-day mortality in the CULPRIT-SHOCK trial (~50%) did not remarkably change from the group of patients who underwent immediate revascularization in the SHOCK trial 2 decades ago (5), these findings suggest that we are in need for further efforts to impact the outcomes of this high risk population.

In summary, the findings of the CULPRIT-SHOCK suggest that a culprit-only revascularization strategy should be the revascularization strategy of choice in patients with AMI and cardiogenic shock. This trial will impact our daily revascularization decisions, and suggests that sometimes by doing “less”, we might have “more” impact on our patients.

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## Footnote

*Conflicts of Interest:* The authors have no conflicts of interest to declare.

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