Editorial

CULPRIT-SHOCK: towards a "simplified" decision making of cardiogenic shock with multivessel coronary artery disease

Pedro A. Villablanca[1], Pablo Flores[2], Humberto Torres[2], Salvador Villablanca[2], Oneglio Pedemonte[2]

[1]Division of Cardiology, New York University School of Medicine, NY, USA; [2]Division of Cardiovascular Disease, Universidad de Valparaiso, Viña del Mar, Chile

Correspondence to: Pedro A. Villablanca, MD, MSc. Division of Cardiology Diseases, New York University School of Medicine, Cardiac Catheterization Laboratory NYU Langone Medical Center 560 First Avenue, HCC 14. New York, NY 10016, USA.
Email: pedrovillablanca@hotmail.com.

Provenance: This is a Guest Editorial commissioned by the Executive Editor Zhongheng Zhang, MD, MM (Department of Emergency Medicine, Sir Run-Run Shaw Hospital, Zhejiang University School of Medicine, Hangzhou, China).

Comment on: Thiele H, Akin I, Sandri M, *et al.* PCI Strategies in Patients with Acute Myocardial Infarction and Cardiogenic Shock. N Engl J Med 2017;377:2419-32.

Received: 24 January 2018; Accepted: 01 February 2018; Published: 03 March 2018.
doi: 10.21037/jeccm.2018.02.02
View this article at: http://dx.doi.org/10.21037/jeccm.2018.02.02

The incidence of cardiogenic shock (CS) as complication of acute myocardial infarction (AMI) involves 5% to 10% of cases and remains the main source of death in patients hospitalized with AMI (1), especially in those with multivessel (MV) coronary artery disease (CAD) (2). The landmark study SHOCK trial established a significant survival benefit with early revascularization as compared to initial medical stabilization in patients with AMI and CS (3). Despite lack of randomized controlled trials (RCT) and meta-analyses showing higher early mortality with MV percutaneous coronary intervention (PCI) than with culprit-lesion-only PCI (4), major societies endorse MV revascularization for CS Shock with MV CAD (5,6). Even with advances in PCI technology and shock management, mortality in rates in CS remains high, with up to one-half of all patients dying before hospital discharge (7).

The results of the CULPRIT-SHOCK (Culprit Lesion Only PCI versus Multivessel PCI in Cardiogenic Shock) trial, which was a RCT to assess whether PCI of the culprit lesion only with the option of staged revascularization of non-culprit lesions would confer better clinical outcomes when compared with immediate MV PCI among patients who have MV CAD and AMI with CS (8). The trial randomized 706 patients to either culprit-lesion-only PCI, with the option of staged revascularization, or immediate MV PCI. At 30 days, the major findings from this RCT were a significantly lower rate of death or renal-replacement therapy among those who initially underwent PCI of the culprit-lesion-only than among those who underwent MV PCI [RR, 0.83; 95% confidence interval (CI), 0.71–0.96; P=0.01], driven mainly by significant lower mortality among patients who underwent culprit-lesion-only PCI (RR, 0.84; 95% CI, 0.72–0.98; P=0.03), while the difference in the rates of renal replacement therapy was not statistically significant. There were no statistically significant differences between the two groups with respect to recurrent myocardial infarction, rehospitalization for heart failure, bleeding, or stroke, Additionally, no significant differences were found in the time to hemodynamic stabilization and support, length of intensive care unit stay, or requirement for and duration of catecholamine therapy.

This is a very well done RCT, which is a remarkable accomplishment in this population. The authors provide compelling evidence that a strategy of culprit-lesion-only PCI is preferred over initial MV PCI for patients CS. While findings from a meta-analysis of uncomplicated STEMI with MV CAD who received MV PCI showed mortality benefit (9); all of the RCTs excluded CS patients (10-12). The potential mechanisms of this increased risk remain notional (13). The increased rate of adverse events seen with MV PCI strategy in CS patients may be explained by the prothrombotic and proinflammatory milieu associated

with endothelial dysfunction and high catecholaminergic state as well as the longer and more complex procedure performed under unstable conditions (14). Though not significant, the higher etiology of AKI after MV PCI, is likely multifactorial related to embolization from catheter manipulation and contrast-induced nephropathy along with hemodynamic impact of CS. Despite major advances in PCI technique and antithrombotic pharmacology for AMI, the routine use of an intra-aortic balloon pump (IABP) and other percutaneous mechanical circulatory support (MCS) have not improved outcomes (15,16), The entry criteria of CULPRIT-SHOCK trial allowed patients to be treated up to 12 hours after the start of CS. The later PCI is initiated, the less likely the patient will recover, regardless of the PCI strategy. MCS was used in about 28% of patients in both groups. Though time to hemodynamic stabilization didn't differ significantly between groups, there were intergroup differences in the use of different MCS devices. When hemodynamic imbalance persists in CS, ongoing ischemia, and venous congestion, leads to multiorgan dysfunction and lactate accumulation. This creates a more complex metabolic derangement that may not respond to treatment of the underlying cause (17). For this reason, early unloading of the left ventricle before revascularization in shock might make a difference as an emerging target of therapy to improve outcomes associated with CS (18,19). Future studies quantifying the optimal timing of MCS in CS are required. Another potential factor to explain this high mortality observed in the MV PCI group may have been the inclusion of chronic total occlusion (CTO). Henriques *et al.* (20) in their RCT reported that there was no benefit in recanalizing CTO of the coronary tree in patients with AMI and MV CAD (20). CTOs are probably beyond what most operators do with MV PCI. Treating CTOs in the trial likely prolonged the time of the procedure and increased the contrast dose and the risk of renal failure. If the operators hadn't intervened on the CTOs, there may have been fewer imbalances in terms of renal failure.

The CULPRIT-SHOCK trial "simplified" the decision making of CS with MV CAD. Interventional cardiologists should not be forced to do MV PCI in the setting of CS. Current recommendations from guidelines to consider initial MV PCI in patients with CS should be revised and updated accordingly.

Acknowledgements

None.

Footnote

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

References

1. Goldberg RJ, Samad NA, Yarzebski J, et al. Temporal trends in cardiogenic shock complicating acute myocardial infarction. N Engl J Med 1999;340:1162-8.
2. Sanborn TA, Sleeper LA, Webb JG, et al. Correlates of one-year survival inpatients with cardiogenic shock complicating acute myocardial infarction: angiographic findings from the SHOCK trial. J Am Coll Cardiol 2003;42:1373-9.
3. Hochman JS, Sleeper LA, Webb JG, et al. Early revascularization in acute myocardial infarction complicated by cardiogenic shock. SHOCK Investigators. Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock. N Engl J Med 1999;341:625-34.
4. de Waha S, Jobs A, Eitel I, et al. Multivessel versus culprit lesion only percutaneous coronary intervention in cardiogenic shock complicating acute myocardial infarction: A systematic review and meta-analysis. Eur Heart J Acute Cardiovasc Care 2018;7:28-37.
5. Levine GN, Bates ER, Blankenship JC, et al. 2015 ACC/AHA/SCAI Focused Update on Primary Percutaneous Coronary Intervention for Patients With ST-Elevation Myocardial Infarction: An Update of the 2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention and the 2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction. J Am Coll Cardiol 2016;67:1235-50.
6. Ibanez B, James S, Agewall S, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J 2018;39:119-77.
7. Jeger RV, Radovanovic D, Hunziker PR, et al. Ten-year trends in the incidence and treatment of cardiogenic shock. Ann Intern Med 2008;149:618-26.
8. Thiele H, Akin I, Sandri M, et al. PCI Strategies in Patients with Acute Myocardial Infarction and Cardiogenic Shock. N Engl J Med 2017;377:2419-32.
9. Villablanca PA, Briceno DF, Massera D, et al. Culprit-

lesion only versus complete multivessel percutaneous intervention in ST-elevation myocardial infarction: A systematic review and meta-analysis of randomized trials. Int J Cardiol 2016;220:251-9.
10. Wald DS, Morris JK, Wald NJ, et al. Randomized trial of preventive angioplasty in myocardial infarction. N Engl J Med 2013;369:1115-23.
11. Gershlick AH, Khan JN, Kelly DJ, et al. Randomized Trial of Complete Versus Lesion-Only Revascularization in Patients Undergoing Primary Percutaneous Coronary Intervention for STEMI and Multivessel Disease: The CvLPRIT Trial. J Am Coll Cardiol 2015;65:963-72.
12. Engstrom T, Kelbaek H, Helqvist S, et al. Complete revascularisation versus treatment of the culprit lesion only in patients with ST-segment elevation myocardial infarction and multivessel disease (DANAMI-3-PRIMULTI): an open-label, randomised controlled trial. Lancet 2015;386:665-71.
13. Reyentovich A, Barghash MH, Hochman JS. Management of refractory cardiogenic shock. Nat Rev Cardiol 2016;13:481-92.
14. Hanratty CG, Koyama Y, Rasmussen HH, et al. Exaggeration of nonculprit stenosis severity during acute myocardial infarction: implications for immediate multivessel revascularization. J Am Coll Cardiol 2002;40:911-6.
15. Thiele H, Zeymer U, Neumann FJ, et al. Intraaortic balloon support for myocardial infarction with cardiogenic shock. N Engl J Med 2012;367:1287-96.
16. Agarwal S, Sud K, Martin JM, et al. Trends in the Use of Mechanical Circulatory Support Devices in Patients Presenting With ST-Segment Elevation Myocardial Infarction. JACC Cardiovasc Interv 2015;8:1772-4.
17. Reynolds HR, Hochman JS. Cardiogenic shock: current concepts and improving outcomes. Circulation 2008;117:686-97.
18. Basir MB, Schreiber TL, Grines CL, et al. Effect of Early Initiation of Mechanical Circulatory Support on Survival in Cardiogenic Shock. Am J Cardiol 2017;119:845-51.
19. O'Neill WW, Schreiber T, Wohns DH, et al. The current use of Impella 2.5 in acute myocardial infarction complicated by cardiogenic shock: results from the USpella Registry. J Interv Cardiol 2014;27:1-11.
20. Henriques JP, Hoebers LP, Ramunddal T, et al. Percutaneous Intervention for Concurrent Chronic Total Occlusions in Patients With STEMI: The EXPLORE Trial. J Am Coll Cardiol 2016;68:1622-32.

doi: 10.21037/jeccm.2018.02.02
Cite this article as: Villablanca PA, Flores P, Torres H, Villablanca S, Pedemonte O. CULPRIT-SHOCK: towards a "simplified" decision making of cardiogenic shock with multivessel coronary artery disease. J Emerg Crit Care Med 2018;2:27.

