Prognosis after AMI-related cardiogenic shock: myocardial blush score is one piece of the puzzle

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Although early primary percutaneous coronary interventions (PCI) have dramatically improved survival in patients with ST-segment elevation myocardial infarction (STEMI), patients who develop cardiogenic shock (CS) after acute MI are often not managed with guideline-recommended therapies and mortality rates remain high. In addition, among patients who receive PCI for CS, the mortality rates appear to be increasing over time, perhaps due to treatment of higher acuity patients[1]. These observations underscore the need for a better understanding of the pathology and more effective treatments.

We know that PCI success is an important determinant of survival. Primary PCI in early presenting, non-shock STEMI patients achieves Thrombolysis In Myocardial Infarction (TIMI) 3 flow in more than 90% of cases. Conversely, in patients with acute myocardial infarction (AMI) complicated by CS, PCI success rates are much lower, and the lack of restoration of flow in both shock and non-shock groups is associated with worse survival[1,2]. However, studies have suggested that myocardial perfusion, as determined by blush scores, is a stronger predictor of survival after STEMI.

In this issue of EuroIntervention, Overtchouk et al report core laboratory-assessed TIMI flow and myocardial perfusion grade (TMPG) in 665 patients with CS enrolled in the CULPRIT-SHOCK trial[3].

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As expected, both TIMI flow grade and TMPG after PCI correlated with 30-day mortality, but in a multivariate model only myocardial perfusion was associated with mortality (adjusted odds ratio 0.38 [0.20-0.71], p=0.002). The authors are to be congratulated on completing this provocative trial looking at some of the angiographic criteria that affect mortality in CS patients. However, the article is entitled "Angiographic predictors ......", yet only TIMI flow and TMPG are discussed. For interventional cardiologists, it would be useful to know if there were other angiographic predictors of poor outcome, such as significant disease proximal or distal to the treated lesion or residual thrombus. In addition, it is surprising that pre-PCI TIMI flow was not important given that it predicts smaller infarct size and survival in other studies[4] and it is believed that early vessel patency reduces the risk of developing CS. It should be pointed out that 33% of the patients in this trial had TIMI 3 flow at baseline (much higher than expected from prior STEMI trials), probably because only 63% of patients in this report had STEMI. How the culprit vessel was determined in NSTEMI patients is not defined and one may expect NSTEMI patients to have greater coronary flow at baseline. Moreover, there are no data provided regarding the non-culprit vessel. Surprisingly, the multivariate analysis did not appear to consider data proven to be of prognostic importance in the investigators' prior trial[5].

Table 1 lists risk factors that have been identified as being associated with short- and/or long-term mortality in CS[1,5-10]. Importantly, early identification and treatment of patients at risk but not hypotensive[9] may prevent development of end-stage CS; however, we need randomised trials in Society for Cardiovascular Angiography and Interventions (SCAI) Shock Classification categories A and B in order to be certain[10].

Finally, we agree that myocardial perfusion is an important prognostic indicator; however, what can be done to improve perfusion?

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The opinions expressed in this article are not necessarily those of the Editors of EuroIntervention or of the European Association of Percutaneous Cardiovascular Interventions.

Numerous pharmacologic agents have failed to improve infarct size or prognosis. In a recent study, the TIMI 3 flow grade following PCI was surprisingly good (91%) despite patients being in CS[11]. It is intriguing to think that this may be due, in part, to the improvement in cardiac output and reduction of left ventricular end-diastolic pressure (LVEDP) with mechanical circulatory support placed prior to PCI, thus improving the coronary perfusion pressure gradient. Ongoing studies, such as the Door to Unload (DTU)-STEMI trial, will hopefully provide more answers.

Table 1. Predictors of mortality in STEMI-related cardiogenic shock.

Baseline demographics
Advanced age
Diabetes or elevated glucose level at admission
Abnormal renal function
Anaemia
Prior stroke or peripheral vascular disease
Frailty
Acute index event
Altered mental status
Cardiac arrest with anoxic brain injury
Late development of shock
Delayed or no reperfusion therapy
Acute revascularisation of non-culprit vessels
Lower EF
Mitral regurgitation severity
Mechanical complications
NSTEMI
Number and dose of inotropic and vasopressor agents
Higher lactate levels
Impaired lactate clearance
Systemic inflammatory response
Using mechanical circulatory support after reperfusion (compared to prior)
Lack of protocol-driven collaborative management
Haemodynamic parameters
Low systolic blood pressure (BP)
High shock index (heart rate/systolic BP)
Cardiac power output (cardiac output x cardiac output/451) <0.6
Pulmonary artery pulsatility index (PaPI) <1.0
High right atrial pressure
Ejection fraction
Right ventricular dysfunction
Angiographic predictors
Left main, proximal LAD or vein graft culprit
Chronic occlusion of major non-culprit vessel
3-vessel disease
Baseline culprit TIMI flow <3
Post-PCI culprit TIMI flow <3
Post-PCI myocardial blush <3

Conflict of interest statement

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