



Case series

Risk stratification to guide management in cardiogenic shock due to acute myocardial infarction. An illustrative case series and review of the literature

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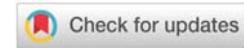
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Abstract

In patients who sustain an Acute Myocardial Infarction (AMI), Cardiogenic Shock (CS) is the most common cause of inpatient death. Despite significant advances in medical and surgical management, mortality rates approaching 80% have been described in some cohorts. However, the severity of CS and outcomes after AMI vary widely. Management requires a rapid, well-organised response and accurate risk stratification must guide complex decisions on ceilings of therapy in the acute setting. Whilst validated risk scores (e.g. the IABP-SHOCK II score and the CardShock score) are available, as highlighted in the present illustrative case series, their use must be guided by clinical judgement.

In the setting of CS due to AMI, it is the author's opinion that, the IABP-SHOCK II score should be used for risk stratification after PCI. It may also be appropriate to use the IABP-SHOCK II score in conjunction with the operator's opinion on the likelihood of restoration of TIMI 3 flow if the coronary anatomy and targets for PCI are known. However, in patients with AMI who develop CS prior to angiography the author recommends use of the CardShock score for risk stratification rather than consider the pre-PCI IABP-SHOCK II score. However, more data are required to validate this approach.

Introduction

Cardiogenic Shock (CS) occurs when inadequate tissue perfusion results from cardiac dysfunction. It is characterized by both systolic and diastolic dysfunction. In patients who sustain an Acute Myocardial Infarction (AMI), CS is the most common cause of inpatient mortality [1]. Indeed, despite significant therapeutic advances, mortality rates approaching 80% have been described in some cohorts with CS [2,3].

Management therefore requires a rapid, well-organised response [4]. A mechanical complication of AMI which could cause CS should be excluded immediately [4]. Bedside echocardiography can reveal acute mitral regurgitation, large RV infarction or rupture of the interventricular septum or left

ventricular free wall, for example [4]. Angiography is then required to assess the anatomy of the coronary arteries and the need for urgent revascularisation whilst medical therapy (i.e. vasopressor and inotropes) are initiated [4]. If CS fails to improve with medical therapy mechanical circulatory support or cardiac transplantation are the only therapeutic options [4]. The Intra-Aortic Balloon Pump (IABP) is the most widely used device for circulatory support [4,5].

However, meta-analysis has found that IABP do not reduce mortality after MI with or without CS and may increase risks of haemorrhage and recurrent ischemia [6].

The severity of CS after AMI and clinical outcome vary greatly [1-6]. However, increasing age is associated with worse



outcomes. Indeed, up to half of all patients admitted to hospital with AMI are over 70 years old [7]. Furthermore, 80% of the deaths due to AMI occurs in those over 65 years of age and 60% of the mortality in this cohort occurs in those over 75 years old [8]. Increasing age is associated with higher risk of developing CS after AMI [7,9]. Although elderly patients are at higher risk, they may still benefit from aggressive treatment of CS [9]. So, age alone should not define management strategies [9].

Accurate risk stratification is therefore required to guide complex treatment decisions (e.g., use of cardiac assist devices and other advanced therapies) in the acute setting. Whilst validated risk scores are available [2,3,5], as highlighted in the present illustrative case series, their use must be guided by clinical judgement.

Illustrative case 1

A 76-year-old man with hypertension and hypercholesterolaemia treated with perindopril 4mg daily and atorvastatin 20mg daily developed severe, sudden onset, breathlessness with central crushing chest pain at rest. He called for an ambulance and the paramedics performed an ECG at the scene. This demonstrated a heart rate of 60bpm (sinus rhythm) with marked ST elevation in leads I, aVL and V1–V5.

So, the ECG was transmitted to the Coronary Care Unit (CCU) and the cardiologist on call was pre-alerted to prepare the cardiac catheter laboratory. Aspirin and high flow oxygen were administered by the paramedics and the patient was then transferred to hospital. On admission to the CCU approximately one hour after the onset of his symptoms, the patient was alert, orientated and haemodynamically stable. Cardiovascular examination was unremarkable. Clopidogrel, intravenous morphine and sublingual GTN were given in CCU and the patient was transferred to the cardiac catheter laboratory.

Coronary angiography was performed. This revealed that the left main stem was short and the Left Anterior Descending (LAD) artery was occluded at the first diagonal origin. The circumflex and right coronary arteries had non-flow limiting minor atheroma.

However, shortly after completion of the angiogram the patient developed further chest pain and then became confused and drowsy. He was peripherally cool and diaphoretic. His BP was 85/30 and HR was 110bpm (sinus rhythm). Echocardiography demonstrated hypokinesia of the lateral wall of the LV and the Ejection Fraction (EF) was visually estimated to be 30%. Pericardial effusion, acute mitral regurgitation and ventricular septal rupture were excluded. The patient had developed cardiogenic shock due to the acute ST elevation Myocardial Infarction (MI).

The CardShock score was 7 (age 76 years, confusion, EF 30%, AMI aetiology, lactate 5.5mmol/l and estimated glomerular filtration rate 38mL/min/1.73m²). This high CardShock score is associated with 77% mortality [3]. A change in the focus of therapy, from active management to palliation, was therefore considered.

However, the Intra-Aortic Balloon Counterpulsation in Acute Myocardial Infarction Complicated by Cardiogenic Shock (IABP-SHOCK II) was then calculated. Prior to Percutaneous Coronary Intervention (PCI) the IABP-SHOCK II score was 4 (age 76 years, creatinine 150µmol/l and lactate 5.5mmol/l). This intermediate risk IABP-SHOCK II score suggested that the associated mortality would be 49.2% if TIMI 3 flow could be restored to the LAD [2]. The operator was hopeful that this could be achieved. However, if TIMI 3 flow was not restored, the IABP-SHOCK II score would increase to 6. This would be associated with a mortality of 76.6% [2]; similar to that predicted by the CardShock risk score.

A central venous catheter was inserted and infusions of dobutamine and noradrenaline were started but failed to improve organ perfusion. Whilst these attempts were made to stabilise the patient the situation was rapidly discussed with the cardiac surgeon on call and the patient's son. They agreed that full active management was in the patient's best interests and appropriate despite the high CardShock score. It was agreed that PCI should be attempted as the initial revascularisation strategy.

However, reperfusion injury may occur during PCI and exacerbate CS. As the patient's condition was critical, the cardiac surgical team were asked to consider the place of emergency Coronary Artery Bypass Graft surgery (CABG). Although in this setting emergency CABG is associated with a very high morbidity and mortality it was agreed that CABG would be performed if PCI was unsuccessful.

So, the cardiac anaesthetist on call was asked to attend. An IABP was placed percutaneously through the left femoral artery. Balloon counterpulsation was initiated at a 1:1 ratio with 100% augmentation. The blood pressure improved to 98/47, the heart rate fell to 90bpm and the patient's level of consciousness improved.

Anaesthesia was then induced and invasive mechanical ventilation was initiated prior to angioplasty. Thrombolysis in MI (TIMI) grade 3 flow was restored after aspiration of thrombus from the LAD, which was subsequently stented. The IABP-SHOCK II score therefore remained 3 after PCI. The patient remained haemodynamically stable throughout and was recovered and extubated in the cardiac catheter laboratory before being transferred back to the CCU with the IABP in situ.

The IABP remained in situ for 48 hours after the PCI. During this time the dobutamine and noradrenaline were weaned. The IABP support was then gradually weaned and stopped 24 hours after the inotropes were stopped. Weaning involved gradually decreasing the ratio of augmented to non-augmented beats from 1:1 to 1:2 to 1:3 (over 12 hours). He remained haemodynamically stable and the next day was transferred from the CCU to the cardiology ward.

The patient did not experience any further chest pain but he was short of breath on exertion (New York Heart Association; NYHA class III). He was started on bisoprolol and ramipril. These were both slowly titrated to 5mg daily over the next 10 days.



On discharge his heart rate and BP were 60bpm and 105/55mmHg respectively. His ECG showed sinus rhythm with poor R wave progression and T wave inversion in leads I, aVL and V1-V5.

On review in clinic 3months later the patient's breathlessness had improved and there were no clinical signs of heart failure. On echocardiography the lateral wall of the LV remained mildly hypokinetic but the LV function had improved (EF 50%).

Illustrative case 2

An 84-year-old man with hypertension treated only with perindopril 4 mg daily developed severe, sudden onset, breathlessness with central crushing chest pain at rest. He called for an ambulance and the paramedics performed an ECG at the scene. This demonstrated a heart rate of 80bpm (sinus rhythm) with marked ST elevation in leads I, aVL, III, aVF and V1-V5.

So, the ECG was transmitted to the Coronary Care Unit (CCU) and the cardiologist on call was pre-alerted to prepare the cardiac catheter laboratory. Aspirin and high flow oxygen were administered by the paramedics and the patient was then transferred to hospital. Within minutes of arrival the patient deteriorated.

Although the patient remained alert, he was peripherally cool and diaphoretic. His BP was 75/20 and HR was 130bpm (sinus rhythm). Echocardiography demonstrated hypokinesia of the apex, septum and inferior and lateral walls of the LV and the EF was estimated to be 10%. Pericardial effusion, acute mitral regurgitation and ventricular septal rupture were excluded. The patient had developed CS due to the AMI.

The CardShock score was 7 (age 84 years, EF 10%, AMI aetiology, lactate 8mmol/l and estimated glomerular filtration rate 29.5mL/min/1.73m²). This CardShock score is associated with 77% mortality [3]. However, IABP-SHOCK II score was 4 (age 84 years, creatinine 200µmol/l and lactate 8mmol/l) prior to PCI. This intermediate risk IABP-SHOCK II score suggested that the associated mortality would be 49.2% if TIMI 3 flow could be restored [2]. However, if TIMI 3 flow was not restored, the IABP-SHOCK II score would increase to 6. This would be associated with a mortality of 76.6% [2]; similar to that predicted by the CardShock risk score.

A central venous catheter was inserted and infusions of dobutamine and noradrenaline were started but failed to improve organ perfusion. Whilst these attempts were made to stabilise the patient the situation was rapidly discussed with the cardiac surgeon on call and the patient in the presence of his son. It was agreed that the prognosis was poor and that proceeding to angiography was not in the patient's best interests. The patient concurred with this decision.

The patient was transferred to the intensive therapy unit where vasopressor and inotropic support were continued. However, the patient sadly passed away shortly thereafter.

Discussion

The diagnosis of CS requires sustained hypotension (systolic BP <90mm Hg for over 30min) with a reduced cardiac index (<2.2L/min/m²) but raised pulmonary capillary wedge pressure (>15mmHg) [4]. It may be diagnosed clinically by observing hypotension and clinical signs of poor tissue perfusion (e.g. oliguria, cyanosis, cool peripheries, prolonged capillary refill time and confusion). These signs usually persist despite correction of hypoxia, hypovolemia, acidosis and arrhythmias [4].

Cardiogenic shock is the most common cause of inpatient mortality in patients who have an AMI [1]. Despite significant advances in the technologies available for organ support (e.g. IABP) and therapeutic intervention (e.g. PCI), mortality rates approach 80% in some cohorts [2,3].

However, the severity of CS after AMI and clinical outcome vary greatly [1-6]. Whilst patients over 75 years of age are at highest risk of CS and death [7,9], this cohort may still benefit from aggressive management when appropriate [9]. In the acute setting, complex treatment decisions must be made within minutes. Accurate risk stratification is therefore required to guide complex decisions on management and ceilings of therapy.

The IABP-SHOCK II [2] and CardShock risk scores [3], have recently been externally validated as good predictors of inpatient mortality in CS after AMI [5]. However, the present case highlights the challenges with their use in the acute setting.

The IABP-SHOCK II score was developed using only patients with AMI-related CS undergoing PCI, and included data on TIMI flow after PCI [2]. Thus the outcome of PCI is required to complete risk stratification using the IABP-SHOCK II score. So intuitively it would seem more appropriate to use the CardShock risk score in patients who have not had PCI. However, the CardShock risk score was developed from the whole CardShock study population [3]. The CardShock cohort included patients with a broad range of aetiologies (i.e., not only AMI) [2,3].

The present cases highlight the potential significant discrepancy between the final CardShock risk score and the predicted IABP-SHOCK II score if TIMI 3 flow can be restored after AMI. In the first case the patient developed CS after diagnostic angiography had delineated the coronary anatomy. It was therefore known that the patient had a single flow limiting lesion in the LAD. The operator was confident that this thrombus could be treated by PCI and that TIMI 3 flow could be restored. So, after rapid discussion with the multidisciplinary team on call and the patient's next of kin it was decided that active management was appropriate.

However, patients with AMI who develop CS often have diffuse coronary artery disease. So, the decision to deploy an IABP and proceed to PCI or cardiac surgery would have been much more complex if the patient had diffuse, severe, triple



vessel disease or indeed had the patient deteriorated prior to angiography. This situation is demonstrated by the second case in the present series. The patient was too unstable to attempt angiography. However, the widespread ECG changes and extensive hypokinesia on echocardiography suggested that severe, triple vessel disease was likely. So, it was believed that the CardShock score was more likely to reflect this patient's prognosis than the pre-PCI IABP-SHOCK II score. So, after rapid discussion with the multidisciplinary team on call and the patient it was decided that active management was extremely unlikely to improve the outcome.

It is the author's opinion that it is better to use the IABP-SHOCK II score than the CardShock score for risk stratification after PCI in the setting of CS due to AMI. It may also be appropriate to use the IABP-SHOCK II score in conjunction with the operator's opinion on the likelihood of restoration of TIMI 3 flow if the coronary anatomy and targets for PCI are known. However, in patients with AMI who develop CS prior to angiography it is the authors practice to use the CardShock score for risk stratification rather than consider the pre-PCI IABP-SHOCK II score. However further, ideally, randomised data are required to validate this approach.

Conclusion

Although meta-analysis has found that IABP does not reduce mortality due to CS after MI [6], the severity of CS and clinical outcomes vary greatly in this setting [1-3]. It has therefore been suggested that risk stratification be used to guide the advanced management of CS. Whilst validated risk scores are available [3-5], as highlighted in the present illustrative case, their use must be guided by clinical judgement.

References

- Goldberg RJ, Spencer FA, Gore JM, Lessard D, Yarzebski J (2009) Thirty-year trends (1975 to 2005) in the magnitude of, management of, and hospital death rates associated with cardiogenic shock in patients with acute myocardial infarction: a population-based perspective. *Circulation* 119: 1211-1219. [Link: https://bit.ly/33sr6WS](https://bit.ly/33sr6WS)
- Pöss J, Köster J, Fuernau G, Eitel I, de Waha S, et al. (2017) Risk Stratification for Patients in Cardiogenic Shock after Acute Myocardial Infarction. *J Am Coll Cardiol* 69: 1913-1920. [Link: https://bit.ly/2QsMfeu](https://bit.ly/2QsMfeu)
- Harjola VP, Lassus J, Sionis A, Køber L, Tarvasmäki T, et al. (2015) Clinical picture and risk prediction of short-term mortality in cardiogenic shock. *Eur J Heart Fail* 17: 501-509. [Link: https://bit.ly/3dcrla](https://bit.ly/3dcrla)
- Rajendram R, Ehtisham J, Forfar JC (2011) *Oxford Case Histories in Cardiology*. Oxford University Press, UK. [Link: https://bit.ly/2J0iSvE](https://bit.ly/2J0iSvE)
- Rivas-Lasarte M, Sans-Roselló J, Collado-Lledó E, García-Fernández V, Noriega FJ, et al. (2020) External validation and comparison of the CardShock and IABP-SHOCK II risk scores in real-world cardiogenic shock patients. *Eur Heart J Acute Cardiovasc Care*. [Link: https://bit.ly/2UhFQ6S](https://bit.ly/2UhFQ6S)
- Su D, Yan B, Guo L, Peng L, Wang X, et al. (2015) Intra-Aortic Balloon Pump May Grant No Benefit to Improve the Mortality of Patients With Acute Myocardial Infarction in Short and Long Term. An Updated Meta-Analysis. *Medicine (Baltimore)* 94: e876. [Link: https://bit.ly/3d87goi](https://bit.ly/3d87goi)
- Rask-Madsen C, Jensen G, Kober L, Melchior T, Torp-Pedersen C, et al. (1997) Age-related mortality, clinical heart failure, and ventricular fibrillation in 4259 Danish patients after acute myocardial infarction. *Eur Heart J* 18: 1426-1431. [Link: https://bit.ly/2IUts06](https://bit.ly/2IUts06)
- Gurwitz JH, Col NF, Avorn J (1992) The exclusion of the elderly and women from clinical trials in acute myocardial infarction. *JAMA* 268: 1417-1422. [Link: https://bit.ly/2wafrQw](https://bit.ly/2wafrQw)
- Acharya D (2018) Predictors of Outcomes in Myocardial Infarction and Cardiogenic Shock. *Cardiol Rev* 26: 255-266. [Link: https://bit.ly/33BpFWk](https://bit.ly/33BpFWk)

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