CASE REPORT

Impella percutaneous left ventricular assist device for severe acute ischaemic mitral regurgitation as a bridge to surgery

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SUMMARY

Ischaemic papillary muscle rupture causing acute severe mitral regurgitation (MR) has a dramatic presentation and a very high mortality. Emergent surgical repair improves outcomes, which necessitates robust preoperative stabilisation. Here we discuss a patient with cardiogenic shock with an acute severe MR that was deemed very high risk for emergent valve replacement due to haemodynamic instability and respiratory failure. A percutaneous left ventricular assist device Impella 2.5 (Abiomed, Danvers, MA) drastically improved clinical status, and the patient underwent a successful surgical mitral valve replacement soon after placement of the temporary assist device. Our case highlights that percutaneous ventricular assist devices may help to stabilise patients with severe acute ischaemic MR, and it can serve as a bridge to surgery in high risk patients.

BACKGROUND

Mild-to-moderate mitral regurgitation (MR) is a common complication of acute myocardial infarction (AMI). Severe acute MR presenting with haemodynamic collapse is usually related to an exceedingly rare mechanical complication such as papillary muscle rupture (PMR) or chordae tendinae rupture.¹ PMR occurs in both ST-segment elevation myocardial infarction (STEMI) as well as non-ST segment elevation myocardial infarction (NSTEMI). It is an early complication of AMI, and it results in flail mitral leaflets that cause severe acute MR.² This life-threatening severe acute MR prompts emergent surgical valve replacement.³ Patients usually have an acute presentation with hypotension and acute pulmonary oedema. Preoperative stabilisation is complex due to concomitant haemodynamic collapse and hypoxic respiratory failure. Choosing a vasopressor or an inotropic agent involves careful evaluation of the pathophysiology and haemodynamics. An increase in either preload or inotropy can increase MR with subsequent worsening of pulmonary oedema and a minimal increase in forward flow across the aortic valve. Afterload reduction with vasodilators would be ideal, but it is frequently limited by hypotension in patients with severe MR. We report an unstable patient with acute PMR causing severe acute MR, who failed preoperative stabilisation with vasopressors, inotropic therapy and an intraaortic balloon pump (IABP). This patient was successfully stabilised with a temporary percutaneous ventricular assist device (pVAD) Impella 2.5

(Abiomed, Danvers, MA) for mitral valve replacement and coronary artery bypass grafting (mitral valve replacement-CABG).

CASE PRESENTATION

A 59-year-old man with medical history of hypertension and diabetes mellitus was brought to the emergency department by paramedics as a STEMI alert. The patient had an intermittent chest pain for the last 4 days but did not seek medical advice. On physical examination, the patient was awake and alert but he was diaphoretic, hypotensive, tachycardic and hypoxic. The patient had jugular venous distension, bilateral inspiratory crackles and apical soft systolic murmur on chest auscultation.

INVESTIGATIONS

Cardiac troponin level was elevated, and ECG showed an inferior ST-segment elevation. Chest X-ray showed asymmetric pulmonary oedema, right worse than left. Emergent cardiac catheterisation disclosed a completely occluded right coronary artery with diffuse multivessel disease (figure 1). A right heart catheterisation revealed pulmonary artery pressure of 57/29/38 mm Hg (systolic/diastolic/mean) and an elevated pulmonary artery wedge pressure with giant V waves (figure 2A). Fick's cardiac output was calculated at 3.57 L/min, and the cardiac index was 1.74 L/min/m². Transoesophageal echocardiography performed in the catheterisation laboratory showed posteromedial papillary muscle rupture and flail anterior mitral valve leaflet causing severe MR (figure 3).

DIFFERENTIAL DIAGNOSIS

Shock can be hypovolemic, distributive, obstructive or cardiogenic. This case presentation is consistent with cardiogenic shock. The differential diagnosis of mechanical complications of AMI includes rupture of left ventricular free wall, rupture of interventricular septum and acute MR. In this case, the patient had cardiogenic shock due to inferior STEMI complicated by acute severe MR due to posteromedial PMR.

TREATMENT

During the catheterisation, refractory hypoxia due to severe pulmonary oedema prompted emergent endotracheal intubation. Various ventilation modes were attempted without an improvement

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Figure 1 Cardiac angiogram left anterior oblique straight view of RCA shows tortuous RCA with diffuse disease (arrowhead points to an almost near-complete occlusion of the RCA). RCA, right coronary artery.

in oxygenation. Cardiovascular surgery was consulted emergently but deemed this patient's surgical risk for intraoperative mortality to be extremely high, and it was decided to revisit candidacy for surgical intervention for severe MR once haemodynamics and respiratory status improved. An IABP was placed for haemodynamic support, and the patient was transported to the intensive care unit (ICU). The patient's profound hypotension precluded the use of vasodilators. Haemodynamic instability and severe hypoxaemia persisted despite inotropes, vasopressors, IABP and mechanical ventilation. A decision was made to exchange the IABP for an Impella 2.5 pVAD (Abiomed, Danvers MA) with 2.5 L of cardiac output support (figure 4). The pVAD was placed about 6 hours after presentation. Following pVAD placement, the cardiac output improved to 4.5 L/min with a cardiac index of 2.1 L/min/m² and the amplitude of V wave decreased (figure 2B). Pulmonary oedema and hypoxia improved, and it became easier to oxygenate the patient with drastic improvements in PaO₂/FiO₂ ratio. Vasopressor requirements decreased drastically, and within 1 hour of pVAD placement, the patient underwent emergent mitral valve replacement with three-vessel coronary artery bypass grafting where the pVAD was removed intraoperatively.

Figure 2 Haemodynamic tracings of the pulmonary artery wedge pressure (PAWP). (A) PAWP tracing before Impella placement shows giant V waves. (B) PAWP tracing after Impella placement shows decrease amplitude of V waves.

OUTCOME AND FOLLOW-UP

The patient did well postoperatively but had a prolonged hospital course complicated by renal failure, respiratory failure with prolonged mechanical ventilation and gastrointestinal bleeding. He was eventually discharged after a 26-day hospitalisation.

DISCUSSION

Preoperative stabilisation of cardiogenic shock with severe acute MR is complex. The cornerstone of therapy is surgical intervention after haemodynamic stabilisation.⁴ Patients should directly be transferred from the catheterisation laboratory to the operating room since the mechanical complication can only be fixed surgically. However, since these patients have immense intraoperative mortality, preoperative stabilisation may improve outcomes in these patients. A pVAD serves the purpose of mechanically off-loading the ventricle thereby preventing end-organ injury from hypoperfusion prior to surgical repair. The 2013 ACCF/AHA guidelines on the management of STEMI have a class IIa recommendation for IABP in patients with cardiogenic shock who do not quickly stabilise with pharmacological therapy and a class IIb recommendation for left ventricle assist devices for circulatory support in patients with refractory cardiogenic shock.⁵

There is little data on the effect on mortality with early versus late surgical repair. The SHOCK trial showed a lower unadjusted mortality in patients who underwent emergent surgical mitral valve replacement than those who did not.³ Although patients in cardiogenic shock from other reasons may benefit from vasoconstrictors or inotropic agents, the mechanism of action of these agents can be counterproductive in severe acute MR. They may worsen haemodynamics in patients with acute severe MR by increasing flow across the mitral valve with a ventricle that is already hyperdynamic. The use of vasodilators may decrease preload and augment forward flow across the aortic valve but is commonly precluded due to hypotension. IABP placement may help myocardial perfusion but does not help forward flow across the aortic valve or reduce the severity of MR. In our case, the patient continued to worsen despite vasopressors, inotropes, mechanical ventilation and IABP. By directly unloading the left ventricle, the Impella pVAD not only increases cardiac output but also improves pulmonary oedema due to the severely elevated left-sided filling pressures by decreasing retrograde mitral valve flow.

Although the use of pVAD has been described in ventricular septal defects post-AMI with good outcomes, there is little data about usage of pVAD for acute severe MR complicating AMI.^{6–9}

Harmon *et al*⁹ described a case in which Impella 2.5 pVAD was used to stabilise a patient with cardiogenic shock secondary

Figure 3 (A) Mid-oesophageal long-axis two-chamber view shows the papillary muscle (PM) and the flail anterior mitral valve leaflet (MVL) in the left atrium. (B) Three-dimensional reconstruction of a view of the left atrium shows the PM and the flail anterior mitral valve leaflet (MVL) in the left atrium. (C) Colour Doppler enhancement of the long-axis two-chamber view of the left ventricle shows eccentric severe mitral regurgitation.

Figure 4 (A) Placement of Impella 2.5 under fluoroscopy. (B) Chest X-Ray shows Impella 2.5 in place.

to acute ischaemic MR. In that case, despite MR there was no evidence of PMR or flail leaflet. The patient's haemodynamics improved after revascularisation and starting Impella support that was weaned after 20 hours. The patient was supported initially by non-invasive mechanical ventilation and intubation was avoided. Renard *et al*¹⁰ reported using biventricular mechanical support as a bridge to surgical mitral valve repair with Impella 5.0 and Impella RP in a patient with cardiogenic shock.

MitraClip therapy can be used in treatment of patients with degenerative MR who are deemed high risk for surgical repair. Emerging data show promise in cases with acute MR complicating myocardial infarction.^{11 12} MitraClip therapy was not used in our patient due to the presence of triple vessel coronary artery disease which would eventually require surgical intervention.

We demonstrated that acute stabilisation with mechanical left ventricular off-loading via a pVAD served as a bridge to surgery in setting of cardiogenic shock secondary to ischaemic MR, and thus directly contributed to good outcome. Currently, there is no prospective data on the use of pVADs in the setting of preoperative stabilisation of severe acute ischaemic MR. This case highlights the need for prospective studies to evaluate the value of pVAD in this setting and its effect on mortality.

Learning points

- ▶ Acute MR is an early mechanical complication of acute myocardial infarction (2–7 days postinfarction).
- ▶ Despite high intraoperative mortality, emergent surgery is the treatment of choice for papillary muscle rupture due to dismal outcome without surgery.
- ▶ Impella 2.5 percutaneous ventricular assist device may be considered in highly selected patients with acute ischaemic MR for stabilisation as a bridge for surgical intervention.

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