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CASE REPORT
Heart failure

A case report of transcatheter mitral valve repair in patient with severe acute mitral regurgitation, cardiogenic shock, and left atrial appendage thrombus as a rescue therapy: facing all enemies at once!

Mhd Nawar Alachkar [1]*, Jörg Schröder[1], Rüdiger Autschbach[2], and Mohammad Almalla [1]

[1]Department of Cardiology, Angiology and Intensive Care, University Hospital RWTH Aachen, Pauwelsstrasse 30, 52074 Aachen, Germany; and [2]Department of Heart Surgery, University Hospital RWTH Aachen, Pauwelsstrasse 30, 52074 Aachen, Germany

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Background Transcatheter mitral valve repair (TMVR) in patients with severe acute mitral regurgitation (MR) and high surgical risk has been described. Moreover, the use of cerebral protection device (CPD) during TMVR in patients without evidence of intracardiac thrombus has been investigated. To the best of our knowledge, TMVR as a rescue therapy in a patient with acute ischaemic MR, cardiogenic shock, and left atrial appendage (LAA) thrombus with concurrent use of CPD has not been reported.

Case summary A 59-year-old female with subacute lateral myocardial infarction caused by subacute stent thrombosis after stent implantation in the left circumflex artery 3 weeks previously presented with acute heart failure due to acute severe MR at a peripheral hospital. The patient was transferred to our tertiary centre for operative mitral valve repair. Transoesophageal echocardiogram revealed the presence of LAA thrombus. During the admission, the patient developed an electrical storm and cardiogenic shock. Because of the extremely high surgical risk and the lack of other therapeutic options, the patient was treated with TMVR (MitraClip™, Abbott Structural Heart Devices, Santa Clara, CA, USA) with the use of CPD (Sentinel™; Boston scientific) as a rescue therapy. After the procedure, the clinical and haemodynamic conditions of the patient improved significantly, and she could be discharged home without any neurological sequelae.

Conclusion TMVR with concurrent use of CPD as a rescue therapy may be considered in non-operable patients with cardiogenic shock caused by acute severe MR and evidence of LAA thrombus when no other therapy options are possible.

Keywords Acute ischaemic mitral regurgitation • Transcatheter mitral valve repair • Cerebral protection device • Left atrial thrombus • Case report

* Corresponding author. Tel: +49 241 8038066, Email: nalachkar@ukaachen.de
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Learning points

- Transoesophageal echocardiogram to rule out left atrial appendage (LAA) thrombus should be always performed before transcatheter mitral valve repair (TMVR), even in patients without known atrial fibrillation.
- TMVR with concurrent use of cerebral protection device may be considered as a rescue therapy in non-operable patients with acute mitral regurgitation (MR) and with evidence of LAA thrombus when no other therapy options are possible.
- TMVR using MitraClip may present an alternative to surgical therapy in high surgical risk patients presenting with acute ischaemic MR and cardiogenic shock.

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Introduction

Acute ischaemic mitral valve regurgitation (MR) is a known complication of acute myocardial infarction (MI). Clinical presentation depends on the severity of MR and varies from asymptomatic to severe acute heart failure with cardiogenic shock.[1] Surgical mitral valve repair was traditionally considered as the standard therapy for severe acute ischaemic MR.[2] Recently, transcatheter mitral valve repair (TMVR) has been described as an alternative therapy for acute MR in patients with cardiogenic shock and high surgical risk.[3] However, TMVR is contraindicated in the presence of intracardiac thrombus.[4] The routine use of a cerebral protection device (CPD) during TMVR in patients without evidence of intracardiac thrombi has been previously investigated.[5] However, TMVR with the use of CPD, despite the presence of left atrial appendage (LAA) thrombus, as a rescue therapy in patients with acute ischaemic MR and cardiogenic shock has to our knowledge not been yet reported.

Timeline

Three weeks before presentation	Non-ST elevation myocardial infarction Percutaneous coronary intervention witd implantation of two drug-eluting stents in a left circumflex artery (LCX)/OM1 bifurcation stenosis
Presentation (Day 0)	Complaint: acute heart failure Acute management: intravenous diuretics, non-invasive ventilation, admission to the intensive care unit (ICU)
Diagnostic work-up	Electrocardiogram: Sinus rhythm, Q wave in I, avL Troponin T 1255 pg/mL (reference range < 14) NT-proBNP 1693 pg/mL (reference range < 287) Transthoracic echocardiogram: mild left ventricle dilation, akinesia of the lateral wall, left ventricular ejection fraction 35%. Severe mitral valve regurgitation (MR) (Effective Regurgitant Orifice Area 55 mm^2, Regurgitant Volume 72 mL), mild tricuspid regurgitation. Right ventricle, aortic valve, and pulmonary valve were normal Transoesophageal echocardiogram: severe MR, thrombus in left atrial appendage Coronary angiogram: total occlusion of LCX with retrograde collateralization from right coronary artery (RCA). Left anterior descending artery and RCA without significant stenosis Cardiac magnetic resonance tomography: akinesia of all lateral segments, transmural scar and no evidence of viable myocardium in territory supplied by the LCX
Heart Team decision	Surgical mitral valve repair or replacement
Day 3	Development of cardiogenic shock Haemodynamic instability (catecholamine therapy) Electrical instability: many episodes of sustained ventricular tachycardia with electrical cardioversion
Day 5	Progressive respiratory failure Intubation with mechanical ventilation
New Heart Team decision	Transcatheter mitral valve repair (TMVR) with concurrent use of cerebral protection device (CPD)
Day 6	Insertion of CPD (Sentinel™) TMVR, placement of two clips (MitraClip™ NTR/XTR) Reduction of severe MR to a mild residual MR
Day 7	Haemodynamic and respiratory improvement, extubating the patient, withdrawal of catecholamine therapy
Day 9	Discharge from ICU No observed neurological deficit, transient ischaemic attack, or stroke
Day 13	Discharge from hospital

Case presentation

A 59-year-old female presented to a peripheral hospital with progressive dyspnoea. Her past medical history included coronary artery disease, peripheral artery disease, chronic obstructive pulmonary disease, and type 2 insulin-dependent diabetes mellitus. Three weeks before presentation, she had received percutaneous coronary intervention with implantation of two drug-eluting stents in a bifurcation stenosis of the left circumflex and obtuse marginal arteries due to non-ST elevation myocardial infarction. Back then, she was discharged on dual antiplatelet therapy (DAPT) with aspirin and ticagrelor as well as ramipril, metoprolol, torasemide, spironolactone, atorvastatin, metformin in addition to insulin. She reported not being compliant with her medication within the last 2 weeks.

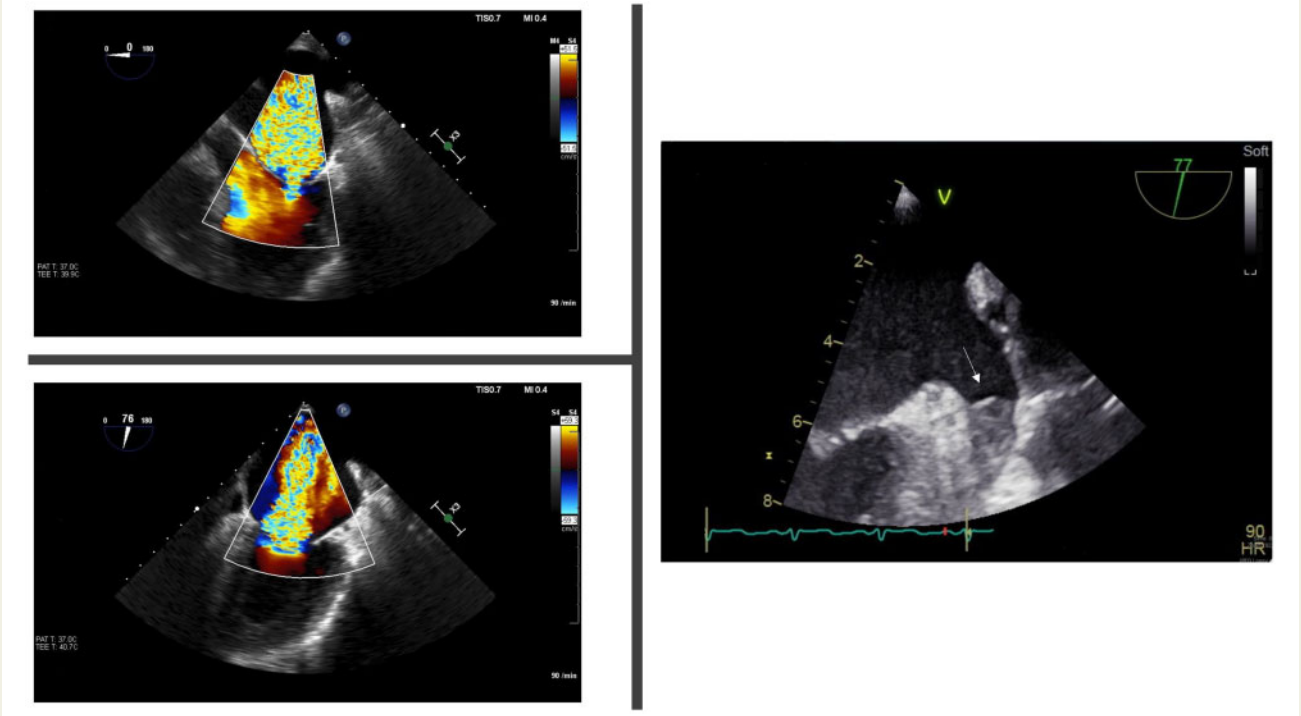


Figure 1 Left: mid-oesophageal four-chamber view (upper panel) and two-chamber view (lower panel) showing severe mitral regurgitation. Right: thrombus in left atrial appendage (white arrow).

On admission, her blood pressure was 100/70 mmHg with a heart rate of 100 beats/min and oxygen saturation of 83% under application of oxygen 8 L/min. Clinical examination showed bilateral fine pulmonary crackles, compatible with an acute pulmonary oedema and a holosystolic apical murmur. Electrocardiogram revealed sinus rhythm with Q wave formation in lateral leads (I, avL). Transthoracic echocardiogram (TTE) showed mild left ventricle dilation (left ventricular end-diastolic diameter 56 mm, left ventricular end-systolic diameter 48 mm), akinesia of the lateral wall with moderately reduced left ventricular ejection function (35%), atrial dilation (LA volume index 36 mL/m^2), severe MR (Effective Regurgitant Orifice Area 55 mm^2, Regurgitant Volume 72 mL/beat), and mild tricuspid regurgitation. No further abnormalities were noted on TTE.

The patient was stabilized with intravenous diuretics and application of non-invasive ventilation and was referred to our centre for further management. Upon admission in our intensive care unit (ICU), the patient was haemodynamically and respiratory stable. Transoesophageal echocardiogram confirmed the presence of severe MR, due to restrictive motion of the posterior mitral leaflet because of the lateral akinesia without any evidence of papillary muscle rupture. Furthermore, it demonstrated the presence of a 1.5 cm × 1.5 cm thrombus in LAA, potentially due to previously undetected atrial fibrillation (*Figure 1*). Anticoagulation with unfractionated heparin was initiated.

Coronary angiogram showed a total occlusion of the left circumflex artery (LCX), potentially due to stent thrombosis because of the interrupted DAPT (*Figure 2*). Left anterior descending artery and right coronary artery were without significant stenosis. Cardiac magnetic resonance tomography (C-MRI) revealed akinesia of all lateral segments, with a transmural scar and no evidence of vital myocardium in the territory supplied by the LCX (*Figure 3*). The case was discussed in our Heart Team. Considering the relatively young age of the patient and the presence of LAA thrombus as a contraindication for TMVR, it was decided to proceed with operative repair of the mitral valve. Due to the lack of viable myocardium detected with C-MRI, it was decided against a concomitant revascularization of LCX.

On Day 3 of admission, the patient developed pulmonary oedema and hypotension with reduced urinary output and clinical signs of hypoperfusion. Due to progressive respiratory failure, she was intubated and mechanically ventilated. The patient was managed with vasopressor (Norepinephrine 0.035–0.01 µg/kg/min.) and dobutamine (5–10 µg/kg/min). Under this therapy, an adequate mean arterial pressure was achieved with improved organ perfusion and normalized lactate value, so that the application of mechanical circulatory system did not have to be considered. Meanwhile, she developed many episodes of atrial fibrillation with tachycardic ventricular response in addition to several episodes of haemodynamically significant sustained ventricular tachycardia which required electric cardioversion. Until the time of intubation, the patient did not exhibit any neurological deficits.

A new multidisciplinary Heart Team evaluation was performed. The patient was deemed to have a prohibitive risk for a surgical mitral valve intervention (EuroSCORE II 26%). However, because of the ongoing haemodynamic compromise and inability to withdraw the

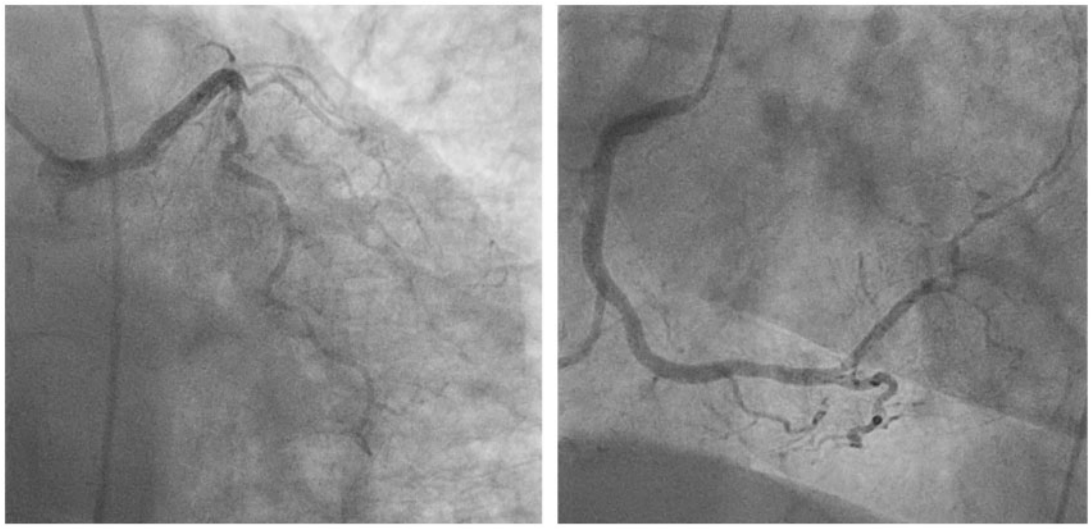


Figure 2 Left: coronary angiography showing a total occlusion of the left circumflex artery. Right: coronary angiography showing the right coronary artery.

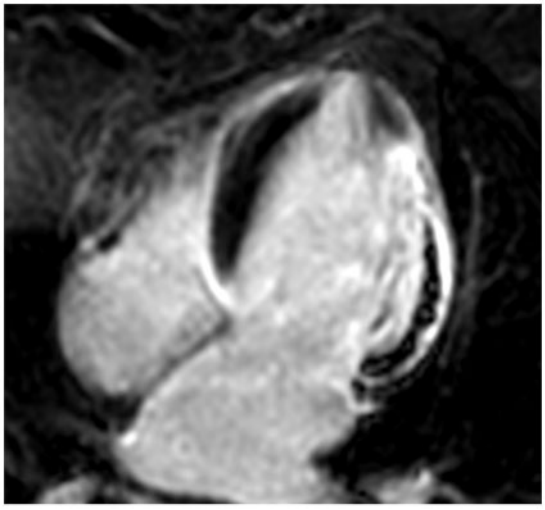


Figure 3 Cardiac magnetic resonance tomography in four-chamber view showing transmural late gadolinium enhancement with evidence of microvascular obstruction of the mid and basal segments of the lateral wall.

vasopressors therapy, a therapeutic intervention was urgently required. Despite the presence of LAA thrombus, and due to the lack of other options, Heart Team decided to proceed with urgent TMVR under employment of CPD as a rescue therapy. Pre-intervention cerebral computer tomography revealed many cerebral and cerebellar old infarcts with chronic occlusion of the basilar artery.

The procedure was conducted on Day 6. Initially, a CPD (Sentinel™; Boston scientific) was inserted through the right radial artery. The proximal filter was placed in the brachiocephalic trunk and the distal one was placed in the left common carotid artery. Then, TMVR procedure was performed with the placement of two clips (MitraClip™ NTR/XTR) (*Figure 4*). The previously severe MR was reduced to a mild residual regurgitation (*Figure 5*). Postintervention, the CPD was removed. Thrombotic debris was visually identified in both filters of the device.

The patient could be extubated the next day (Day 7) and weaned off vasopressor support. After the procedure, no further episodes of VTs were documented. Two days after the procedure (Day 9), patient was discharged from the ICU without neurological sequelae. After receiving appropriate respiratory care and physiotherapy on the normal ward, the patient was completely mobile and she was discharged from the hospital 4 days later in a good clinical situation. An admission in a cardiac rehabilitation facility within 10 days after discharge was arranged. Six months later, we contacted the patient by telephone. She reported feeling well without any heart failure symptoms. Further routine follow-up will be performed by her cardiologist.

Discussion

Severe acute ischaemic MR is associated with a poor short- and long-term prognosis.[6] Surgical treatment of acute ischaemic MR in patients with acute or subacute MI is associated with high mortality.[7] The feasibility of TMVR as an alternative to surgery in treatment of acute ischaemic MR after MI in patients with cardiogenic shock has been reported.[3,8,9] Our patient presented with acute pulmonary oedema and developed cardiogenic shock due to severe MR without any option for revascularization. The calculated EuroSCORE II for operative mitral valve repair was 26% and the surgical risk was deemed extremely high. However, the presence of LAA thrombus precluded the use of TMVR considering the risk of distal embolization. Even in the absence of intracardiac thrombi, which is considered a contraindication for TMVR, stroke occurred in 2.6% of patients undergoing

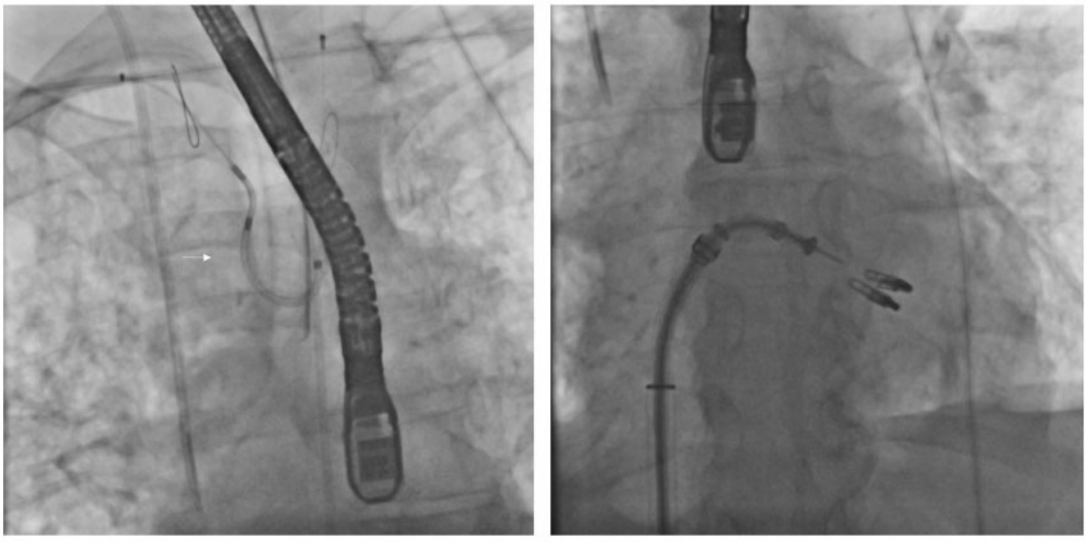


Figure 4 Fluoroscopic images showing Sentinel™ cerebral protection device in place (arrow) and two MitraClips™ (NTR and XTR).

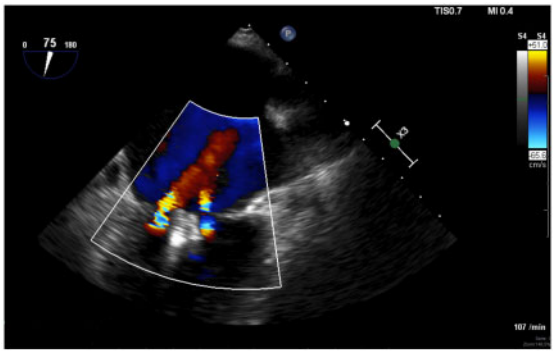


Figure 5 Mid-oesophageal two-chamber view showing the result of the procedure with residual mild mitral regurgitation.

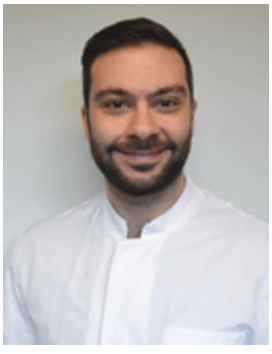
TMVR in the EVEREST II study.[10] The use of CPD has been described to reduce stroke in patients undergoing transcatheter aortic valve implantation.[11] The use of Sentinel™ CPD during TMVR was reported to be safe and feasible in a small cohort of 14 high-risk surgical patients undergoing TMVR.[5] Furthermore, Calcagno *et al.*[12] reported the use of CPD because of acute thrombus formation during TMVR. Moreover, a case of elective TMVR in a patient with filamentous structure of unknown aetiology in LAA under the use of CPD was also reported.[13] Despite lack of evidence supporting the routine use of CPD in TMVR, Pagnesi *et al.*[14] reported that its use may be considered in patients with high risk for cerebral embolization. Our patient presented with thrombus in the LAA prior to the intervention, which certainly exhibits an extremely high risk of cerebral embolization. However, because of the persistent cardiogenic shock and high surgical mortality risk, our Heart Team decided to proceed with TMVR as a rescue therapy with employment of CPD.

To the best of our knowledge, this is the first reported case of TMVR in a patient with acute ischaemic MR, electrical storm, persistent cardiogenic shock, and pre-interventional evidence of LAA thrombus under employment of CPD.

Conclusion

TMVR under the use of CPD may be considered as a rescue therapy in high surgical risk patients with acute ischaemic MR and cardiogenic shock complicating an acute MI who have evidence of LAA thrombus, when no other therapy option is possible.

Lead author biography



Mhd Nawar Alachkar is a cardiology resident at the University Hospital in Aachen, Germany.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

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