

Eclipsed mitral regurgitation successfully treated with a combination of surgical and pharmacological therapies: a case report

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Background

Eclipsed mitral regurgitation (MR), which is characterized by a transient and reversible massive functional MR, usually causes recurrent episodes of acute pulmonary oedema in patients with a preserved left ventricular ejection fraction. The pathophysiological mechanism and optimal treatment of eclipsed MR are not yet fully understood.

Case summary

A 72-year-old woman was hospitalized with cardiogenic shock and takotsubo cardiomyopathy. After hospitalization worsening dyspnoea again appeared, and urgent transthoracic echocardiography revealed severe MR, which spontaneously resolved in a few minutes. At this point, eclipsed MR was detected for the first time. Diagnostic examination revealed that the eclipsed MR was caused by a left ventricular afterload increase. Ultimately, the patient began medical therapy and underwent mitral valve replacement. The subsequent clinical course was favourable.

Discussion

This case illustrates the importance of early intervention for eclipsed MR. A combination of surgical and pharmacological therapies can serve as one treatment option for an eclipsed MR.

Keywords

Eclipsed MR • Transient acute mitral regurgitation • Mitral valve surgery • Takotsubo cardiomyopathy • Case report

Learning points

- Eclipsed mitral regurgitation (MR) can cause a life-threatening condition, such as cardiogenic shock; therefore, early intervention should be considered.
- A combination of surgical and pharmacological therapies can serve as one treatment option for an eclipsed MR.

Introduction

Eclipsed mitral regurgitation (MR) was first reported by Avierinos *et al.*,¹ since when only a few cases have been reported in the literature.^{2–4} Characterized by transient and reversible massive functional MR, the clinical presentation of eclipsed MR is usually recurrent

episodes of acute pulmonary oedema in patients with a preserved left ventricular ejection fraction (LVEF). Since it is a transient phenomenon rarely reported, the diagnosis is probably underestimated. In addition, the pathophysiological mechanism and optimal treatment of eclipsed MR are not yet fully understood.

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Timeline

Day	Events
1st Day	A 72-year-old woman was hospitalized with cardiogenic shock and takotsubo cardiomyopathy.
8th Day	Recurrence of symptoms was noted, and a physical examination revealed a systolic murmur at the cardiac apex. Transthoracic echocardiography revealed severe mitral regurgitation (MR), which spontaneously resolved within minutes. This transient and severe MR ('eclipsed MR') was detected for the first time.
22nd Day	A diagnostic examination revealed a left ventricular afterload increase as the cause of the eclipsed MR.
23rd Day	Medical therapy was initiated.
29th Day	The patient redeveloped severe dyspnoea and cardiogenic shock secondary to the eclipsed MR.
43rd Day	A surgical mitral valve replacement was performed.
104th Day	The patient was discharged.
Follow-up (6 months)	The eclipsed MR did not recur after the left ventricular afterload increased.

Case presentation

A 72-year-old Japanese woman was transferred to our hospital with dyspnoea. Over the past 2 years, she had been admitted four times for heart failure requiring intensive treatment. Her medical history included paroxysmal atrial fibrillation, hypertension, and chronic kidney disease. Seven months prior, a catheter ablation was performed because paroxysmal atrial fibrillation was suspected as a trigger of the heart failure. However, after the catheter ablation, she had repeated episodes of heart failure.

On arrival at the hospital, her blood pressure was 83/34 mmHg and pulse rate 98 b.p.m. The percutaneous oxygen saturation under room air was 70%. She was dyspnoeic with poor perfusion as evident by cold extremities and poor capillary refill. Respiratory system auscultation revealed coarse crackles and wheezes bilaterally. No heart murmurs were heard. Jugular venous distention and mild peripheral oedema were observed. Arterial blood gas analysis showed metabolic acidosis with a pH of 7.11 (normal range 7.35–7.45). An electrocardiogram showed significant ST elevation in leads V2–V6 (Figure 1A). A chest X-ray demonstrated acute pulmonary oedema (Figure 1B). A transthoracic echocardiogram revealed apical and mid-ventricular severe hypokinesis with an estimated LVEF of 20%, and her MR was mild. Emergent coronary angiography did not demonstrate any significant coronary artery disease. No left ventriculography was performed because her serum creatinine was 1.50 mg/dL (normal range

0.4–1.2 mg/dL). Right heart catheterization revealed a pulmonary artery pressure of 45/27/36 mmHg (normal range 15–30/4–12/9–19 mmHg), pulmonary capillary wedge pressure (PCWP) of 33 mmHg (normal range 4–12 mmHg), and cardiac index of 1.28 L/min/m² (normal range 2.5–4.0 L/min/m²). She was diagnosed with cardiogenic shock and acute heart failure caused by takotsubo cardiomyopathy. Intra-aortic balloon pumping (IABP) was initiated to stabilize her haemodynamic state.

On Day 3, her haemodynamic condition improved and IABP was removed successfully. A transthoracic echocardiogram demonstrated complete recovery of the left ventricular function. However, 8 days later worsening dyspnoea again appeared, and a physical examination revealed a 3/6 systolic murmur at the cardiac apex. Urgent transthoracic echocardiography revealed severe MR with tenting of the mitral leaflets, which was absent on admission. In addition, severe tricuspid regurgitation was observed, and the estimated right ventricular systolic pressure increased to 68 mmHg. A few minutes later, the MR spontaneously resolved and was only graded as mild with a normal leaflet coaptation. At this point in time transient and severe MR, termed 'eclipsed MR' in a few past reports, was suspected for the first time as the cause of the repeated heart failure episodes.

We decided to perform a diagnostic examination to investigate the cause of the eclipsed MR in the catheterization laboratory. During the test, the PCWP and central arterial pressure were monitored. At baseline, MR was mild and PCWP was in the normal range (Figures 2A and 3A). Initially, a coronary spasm provocation test was performed. Coronary angiography showed no significant stenosis after a methylergonovine infusion, and the haemodynamic parameters remained unchanged. Next the handgrip manoeuvre was performed, but this could not induce any sufficient increase in the central blood pressure. We decided to use norepinephrine, which selectively increased the systemic vascular resistance and afterload. After the norepinephrine infusion (0.07 µg/kg/min), the central arterial pressure increased dramatically. A transthoracic echocardiogram in the supine position revealed severe MR, which was absent before the test. Echocardiographic characteristics changed significantly (Table 1). A transoesophageal echocardiogram was performed immediately and revealed severe MR with extreme apical tenting of both leaflets, resulting in a total lack of coaptation (Figure 2B). In addition, dynamic MR led to dramatic changes in the cardiovascular haemodynamics. Right heart catheterization revealed large V waves in the pulmonary wedge pressure (Figure 3B). With the decreasing central arterial pressure, the MR was spontaneously found to be mild on the echocardiogram.

Other investigations were also performed. Renal echography revealed the absence of any renal artery stenosis. Pheochromocytoma was ruled out by a normal plasma catecholamine level. Based on the aforementioned findings, we diagnosed that the eclipsed MR was caused by a left ventricular afterload increase. With the diagnosis and aetiology confirmed, the patient began medical therapy including beta-blockers and angiotensin-converting enzyme inhibitors aimed at decreasing the cardiac afterload. However, 6 days after starting the medications, she redeveloped severe dyspnoea and cardiogenic shock with severe acidosis secondary to eclipsed MR. Since previous reports also have described that eclipsed MR often recurs despite

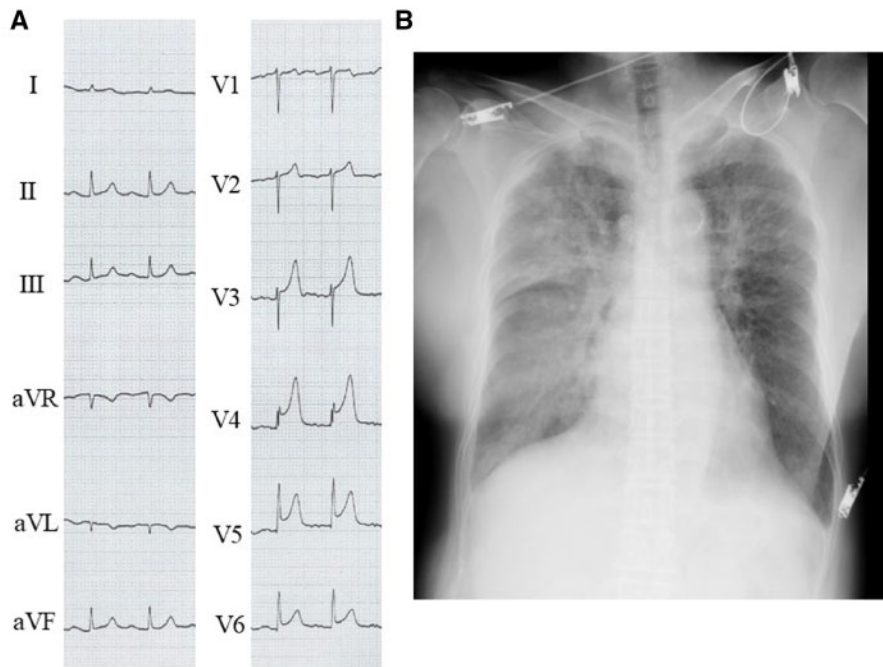


Figure 1 (A) An electrocardiogram showing significant ST elevation in leads V2–V6 (25 mm/s, 10.00 mm/mV). (B) Chest X-ray demonstrating acute pulmonary oedema with cardiac enlargement.

optimal medical therapy,^{3,5} we considered that pharmacological therapy alone was difficult for management of the eclipsed MR. In addition, the eclipsed MR was considered to be more critical than we expected because the patient experienced a life-threatening condition repeatedly throughout the clinical course: in the worst case, this could lead to cardiac death. The severity of the clinical course urged us to undertake surgical therapy for our patient. After much discussion between the patient and our heart team, we decided to perform mitral surgery. On Day 43, the patient underwent mitral valve replacement using a bioprosthetic valve. The surgical findings revealed no organic changes in the mitral valve itself and mitral valve complex. The post-operative clinical course was favourable, and the patient was discharged uneventfully. Five months after the surgery, she was readmitted to our hospital for follow-up. As before, the haemodynamic parameters were monitored. We confirmed that the central blood pressure became more difficult to increase than previously after the same amount of norepinephrine infusion (0.07 µg/kg/min) (Figure 2C). At that time, the MR was not exacerbated (Figure 3C). One year and 3 months after surgery, the patient has been living with no recurrence of symptoms.

Discussion

Here, we have presented a case of eclipsed MR, which is characterized by transient and reversible severe MR. The clinical course of eclipsed MR usually includes recurrent unexplained acute pulmonary oedema. However, our patient repeatedly experienced a life-

Table 1 Changes in echocardiographic characteristics

	At baseline	During eclipsed MR
LVEDD (mm)	44	49
Mitral tenting area (cm ²)	0.88	1.94
Mitral annulus diameter (mm)	30	34
sPAP (mmHg)	29	59
Tricuspid regurgitation	Trivial	Severe

LVEDD, left ventricular end-diastolic diameter; MR, mitral regurgitation; sPAP, systolic pulmonary artery pressure.

threatening condition, such as cardiogenic shock. Therefore, this case informed us that eclipsed MR is much more serious than might be expected, so early intervention should be considered when the cause of repeated heart failure episodes or cardiogenic shock is diagnosed as eclipsed MR.

The pathophysiological mechanism of eclipsed MR is not yet fully understood. In past reports, some authors have suggested that coronary spasms or microvascular dysfunction represent the mechanism. It has been theorized that eclipsed MR appears when coronary spasms trigger transient left ventricular dysfunction and mitral apparatus ischaemia.⁶ Yet in the present case, no coronary spasms were induced and the haemodynamic parameters were unchanged after the administration of methylergonovine. However, we were able to reproduce the eclipsed MR by the administration of pharmacological

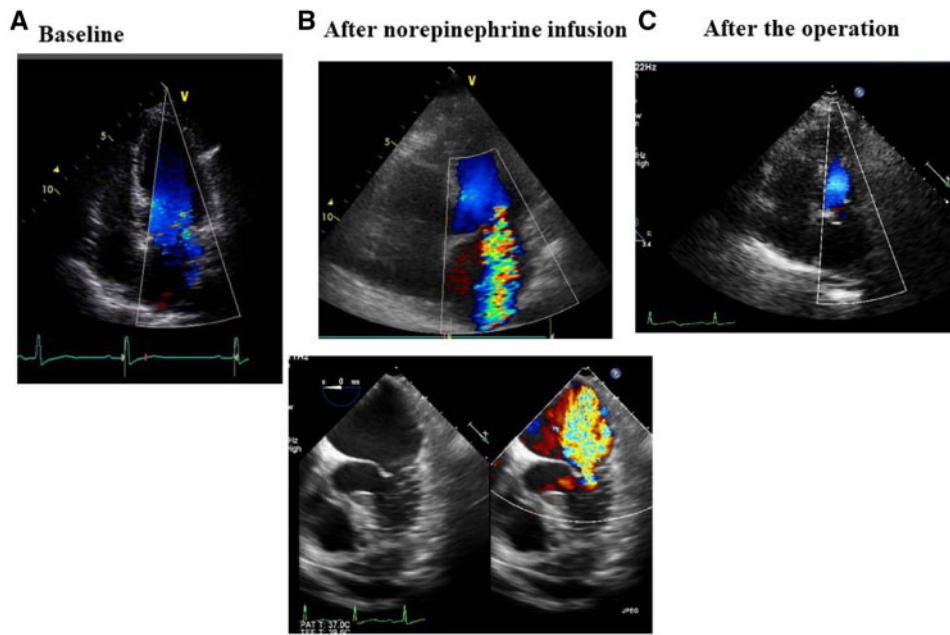
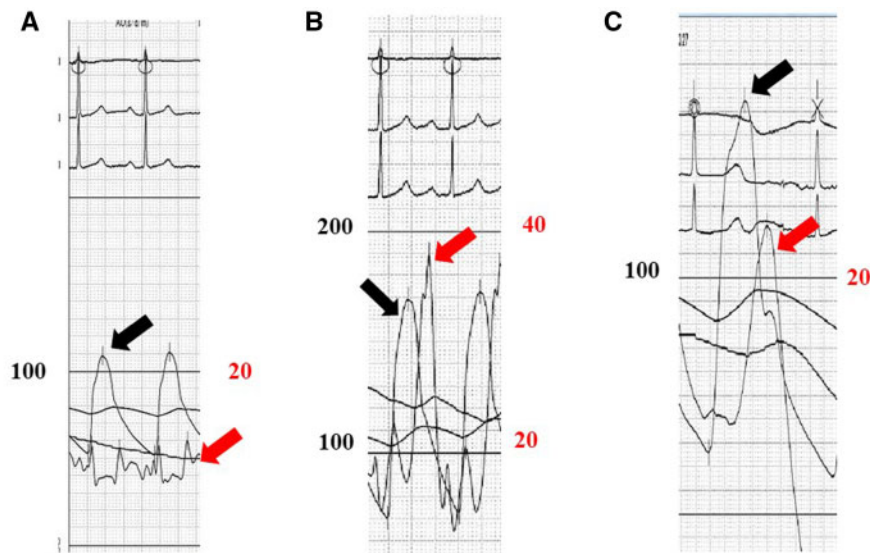


Figure 2 Transthoracic echocardiography and transoesophageal echocardiography. At baseline, mitral regurgitation was mild (A). However, severe mitral regurgitation occurred after the norepinephrine infusion. A transoesophageal echocardiogram revealed severe mitral regurgitation with extreme apical tenting of both leaflets, resulting in a total lack of coaptation (B). After surgery, the mitral regurgitation was not exacerbated despite the increase in central blood pressure (C).



	Baseline	after norepinephrine infusion	After the operation
cBP s/d(m) mmHg	110/52(71)	167/76(106)	136/62(91)
PCWP a/v(m) mmHg	6/7(5)	21/39(25)	14/21(16)

Figure 3 Haemodynamic changes. Central blood pressure (black arrows) and pulmonary capillary wedge pressure (red arrows). a, a wave; cBP, central blood pressure; d, diastolic; m, mean; PCWP, pulmonary capillary wedge pressure; s, systolic; v, v wave.

agents that increased the afterload. Given this finding, we hypothesized that transient left ventricular and mitral annulus dilatation would have occurred in the process of responding to the increased afterload caused by endogenous catecholamine secretion, which would have led to sudden extreme tenting and a leaflet coaptation defect. In this case, the clinical presentation of eclipsed MR included cardiogenic shock following takotsubo cardiomyopathy. Takotsubo cardiomyopathy has also been reported to be associated with increased concentrations of plasma catecholamines, suggesting that there are common mechanisms underlying takotsubo cardiomyopathy and eclipsed MR.⁷

The optimal treatment of eclipsed MR is also unclear. The past case series described that eclipsed MR recurs despite optimal medical treatment. These patients underwent surgical therapy besides medical therapy, and most of them experienced no cardiac events after surgery. In the present case, an afterload increase was prevented by medical therapy and the MR exacerbation was expected to be suppressed by the surgical therapy. However, it remains unclear whether the MR could be suppressed in a situation whereby the left ventricular afterload increases suddenly in daily life. Since the clinical course of our patient was favourable after the treatment, we surmised that the combination of surgical and pharmacological therapies represents one treatment option for eclipsed MR.

Lead author biography



Keishi Ichikawa graduated from Okayama University, Medical School in 2012. He was a Junior Resident in Hiroshima City Hospital from 2012 to 2014 and a Medical Staff in the Department of Cardiology, Iwakuni Clinical Centre, from 2014 to 2017. He has been Medical Staff in the Department of Cardiology, Okayama University Hospital since 2017.

Supplementary material

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

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

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

Conflict of interest: none declared.

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