Acute posteromedial papillary muscle rupture secondary to aortic valve endocarditis: a case report

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**Contributors:** AM wrote the manuscript. AM and TS performed surgery and were involved in the patient’s care. MO performed the echocardiography. SH organized the patient’s care.

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Abstract

Background: Acute papillary muscle rupture due to infective involvement has been recognized as a complication of infective endocarditis. However, there is very limited literature describing the rupture of the posteromedial papillary muscle in primary aortic valve endocarditis without aortic root abscess. This report highlights the etiology of the papillary muscle rupture in the setting of primary aortic valve endocarditis and the importance of a multidisciplinary approach.

Case summary: An 81-year-old man without any heart failure symptoms presented with fever and loss of vision in his left eye. Initial echocardiography revealed moderate aortic valve regurgitation due to a perforated right coronary cusp without aortic root abscess, and his blood cultures were positive for Group G Streptococci. During adequate antibiotic therapy, he developed acute severe mitral regurgitation secondary to posteromedial papillary muscle rupture. Following emergent aortic and mitral valve replacement using bioprosthetic valves, he made excellent progress on a 6-week course of intravenous antibiotics.

Discussion: The echocardiography and the histological findings suggested that the main cause of papillary muscle rupture was most likely a metastatic focus of infection from the aortic valve via a regurgitant jet. Successful treatment of this fatal complication includes
early diagnosis and prompt surgical intervention by a multidisciplinary approach.

Keywords: Papillary muscle rupture, Aortic valve regurgitation, Infective endocarditis, Metastatic foci of infection, Multidisciplinary approach, Case report
Learning Points

1. Physicians should recognize the eccentric aortic regurgitant jets as a cause of the metastatic focus of infection on the left ventricular aspect in the aortic valve endocarditis.

2. We have demonstrated the key role of the “time-to-therapy” concept using a multidisciplinary approach in the prompt management of infective endocarditis and related catastrophic complications.
| Time                | Events                                                                                                                                 |
|---------------------|----------------------------------------------------------------------------------------------------------------------------------------|
| Two days before     | The patient began to experience left eye pain and worsening eyesight, and fever and general fatigue developed. Patient was taken to another hospital and was diagnosed as endogenous endophthalmitis and blood sample was sent for culture. |
| admission           |                                                                                                                                      |
| Day 0               | The patient was admitted to our hospital without any signs or symptoms of heart failure. A funduscopic examination of the left eye revealed a Roth spot. Transthoracic echocardiography (TTE) revealed a perforation on the right coronary cusp of the aortic valve with moderate aortic regurgitation. A diagnosis of aortic valve endocarditis was made on the basis of the modified Duke criteria. Daptomycin was started as an empirical therapy. |
| Day 1               | The blood cultures turned positive for streptococcus Group G that was sensitive for penicillin G. Antibiotic treatment was switched to penicillin G and gentamicin. |
| Day 4               | No evidence of aortic root abscess was found, and the mitral leaflets were intact with mild mitral regurgitation (MR) in the transesophageal echocardiography. |
| Day 5               | Blood cultures returned negative.                                                                                                                                                              |
| Day 7 | Patient’s condition suddenly deteriorated with symptoms of dyspnoea. Follow-up TTE demonstrated a newly severe MR with normal left ventricular wall motion. Emergent aortic and mitral valve replacement was completed successfully. |
|-------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Day 34 | Left ophthalmectomy was required because of left panophthalmitis |
| Day 50 | The patient completed 6 weeks of postoperative penicillin G. |
| Day 65 | The patient made a full recovery from surgeries and was discharged home in a stable afebrile state. |
| 3 years after discharge | The patient was regularly checked every year. There was no recurrence of infection. |
Introduction

Medical management with antibiotics under strict clinical and echocardiographic observations is a good option for patients with infective endocarditis (IE) with well-tolerated severe valvular regurgitation \(^{(1)}\). However, surgical treatment in the active phase despite adequate antibiotic treatment can strongly influence the outcome, if particularly when the infection spreads below the primary infected valve. Here, we report an unusual case of aortic valve endocarditis that developed acute posteromedial papillary muscle (PM) rupture during adequate antibiotic therapy.

Case presentation

An 81-year-old man with a history of hypertension was admitted to our hospital with a 4-day history of fever and rapid visual deterioration of the left eye. On presentation, his body temperature was 38.7°C and blood pressure was 132/49 mmHg, with a regular pulse rate of 60 beats/min, respiratory rate of 20 breaths/min, and oxygen saturation of 97% on room air. On physical examination, a grade 2 aortic diastolic murmur was noted. Funduscopic examination revealed a Roth spot in the left eye. His white blood cell count (normal range: 3,800–8,600 cells/mm\(^3\)) was and procalcitonin level (normal value: <0.5 ng/mL) were 17,720 cells/mm\(^3\) and 14.08 ng/mL, respectively. Transthoracic echocardiography (TTE) revealed mild mitral regurgitation (MR), moderate aortic
regurgitation (AR) with a 347 msec of pressure half time, and no retrograde holodiastolic
flow in the abdominal aorta (Fig. 1, Video 1, Supplementary material online Video S1b–
S1d). The left ventricular (LV) ejection fraction (LVEF) was 53% with diffuse global
hypokinesis of the LV wall and mild LV dilation (LV diastolic dimension: 55 mm; LV
systolic dimension: 38 mm). Transesophageal echocardiography revealed 8 mm
thickening and a perforation on the right coronary cusp (RCC) that caused moderate AR
(Fig. 2a, 2b, Video 2a, Supplementary material online, Video S2b–S2e). There was no
evidence of an aortic root abscess, septal wall abscess that may cause a decreased heart
rate regardless of the actual sepsis, or abnormal mitral apparatus. His vitreous and blood
cultures were positive for Group G Streptococci that were sensitive to penicillin G. Aortic
valve endocarditis complicated with endogenous endophthalmitis was diagnosed on the
basis of the modified Duke criteria. Both the 2015 ESC and 2015 AHA/ACC guidelines
recommend early surgery for patients with IE who present with symptoms or signs of
heart failure (HF), persistent infection, and recurrent emboli. Early surgery, which is a
class I level B recommendation according to both the guidelines, is recommended for
patients with persistent bacteremia or fever lasting for >7 days after the initiation of
appropriate antimicrobial therapy \(^{(1, 2)}\). The management plan was discussed with a
multidisciplinary care team, and the decision to perform surgery was made after the
initiation of antibiotic therapy and relevant observations. Intravenous penicillin G was
started based on the susceptibility study findings after empirical therapy. On day 5, the
blood cultures were negative for Streptococci, and the white blood cell count and
procalcitonin level were decreased to 10,100 cells/mm$^3$ and 1.80 ng/mL, respectively. No
symptoms of HF were observed, and the vital signs other than low-grade fever were stable.
Follow-up TTE demonstrated no remarkable changes from the initial findings. However,
on day 7, his condition suddenly deteriorated and he developed HF symptoms consistent
with those of New York Heart Association functional class III. Follow-up TTE
demonstrated a newly diagnosed severe MR with normal LV wall motion and 69% LVEF
(Fig. 3, Video 3a, Supplementary material online, Video S3b–S3c), and
electrocardiography revealed sinus tachycardia without ischemic changes. The
differential diagnosis for acute MR in patients with IE included destructive endocarditis
extending to the mitral valve and coronary emboli.

The patient was immediately transferred to the operating room. Intraoperatively,
vegetations were observed on the perforated RCC (Fig. 4a–4c) without any evidence of
aortic annulus infection. Close inspection of the mitral apparatus revealed complete
rupture of the posteromedial PM head with presence of vegetations. No gross evidence
of infection was found in the mitral leaflets (Fig. 4d–4f). After resecting the aortic valve
leaves, the anterior mitral leaflet and a part of the P2 and P3 scallops, debridement of all infected tissues were performed. Mitral valve replacement was completed with a 27-mm Epic bioprosthesis (St. Jude Medical Inc., St. Paul, MN, USA) followed by aortic valve replacement using a 23-mm Crown RPT bioprosthesis (Sorin Group, Burnaby, Canada).

Although the postoperative course was complicated by persistent fever due to panophthalmitis requiring left ophthalmectomy on postoperative day 17, the patient was discharged in a stable condition after completing antibiotic therapy. The culture of the resected PM was positive for Group G Streptococci. The histopathological examination of the PM indicated numerous neutrophilic leukocytes suggesting myocardial infection (Fig. 5). A postoperative follow-up after 3 years did not reveal any sign of a recurrent infection.
Discussion

PM rupture is an unusual life-threatening complication in patients with IE, and only few reports have described this complication in aortic valve endocarditis settings (3-5). The clinical course of our patient was similar to those of three other cases occurring during adequate antibiotic management. However, the main difference was that our case involved endophthalmitis and that PM rupture occurred without the presence of aortic root abscesses. To the best of our knowledge, this is the only case of acute posteromedial PM rupture in the setting of primary aortic valve endocarditis without the presence of aortic root abscesses. The causes of PM rupture in aortic valve endocarditis may be caused by the direct invasion of the endocarditis into the aortic-mitral fibrous continuity, leading to a distraction of the mitral apparatus, coronary emboli, or metastatic foci of infection from the aortic valve via regurgitant jets. Initial echocardiography revealed a unique presentation of an eccentric AR jet via an RCC perforation impinged on the posteromedial PM that swirled in the LV impinging against P3 with a competent mitral valve with normal morphology. These characteristics and the discontinuous infected areas of the RCC and posteromedial PM strongly suggest that the possible pathogenic mechanisms underlying PM rupture are the translocation of infective organisms to the PM and mechanical damage by the AR jet via the RCC perforation.
Persistent fever during antibiotic therapy may be a critical sign of PM infection; however, early diagnosis is difficult via follow-up TTE. Multislice computed tomography scan is possibly superior to TTE in providing information regarding the extent of any perivalvular extension \(^{(1)}\), and positron emission tomography/computed tomography may serve as an alternative diagnostic method for the detection of secondary infection sites \(^{(6)}\). Taken together, identifying an optimal time for elective surgery in patients with IE requires new alternative diagnostic data and multidisciplinary approaches. This report highlighted the importance of these aspects in the management of IE to avoid any potential complications and improve the patient’s prognosis.

In conclusion, the rupture of the PM via AR jets due to the translocation of vegetations to the PM in an isolated aortic valve endocarditis is a potential complication. The adverse effects of AR jets with respect to LV aspects must be carefully considered, and the assessment of AR jets by initial echocardiography and strict follow-up examination for early diagnosis and prompt surgery with a multidisciplinary approach are imperative for the treatment of this rare complication.
Consent: The authors confirm that written consent for submission and publication of this case report, including the images and associated text, are in agreement with the COPE guidelines.

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Figure and Video Legends

Figure 1. Initial transthoracic echocardiography images
(a) The parasternal long-axis view of the diastole shows eccentric aortic regurgitation (AR). The parasternal long-axis view of the systole shows mild mitral regurgitation (MR). (b) The parasternal short-axis view of the diastole shows that the AR jets (red arrow) swirling along the basal posterior wall and reaching the P3 with normal mitral morphology. (c) Apical three-chamber view shows that AR jets impinge against the posteromedial papillary muscle (PM).

Figure 2. Transesophageal echocardiography images
Short axis (a) and long-axis views (b) show moderate AR and a perforation on the right coronary cusp (yellow arrow). The eccentric AR jets reach the P2 (red arrow). (c) Midesophageal four-chamber view (d) shows the intact A1 and P1 of the mitral leaflets. Midesophageal commissural view (e) shows the intact posterior mitral leaflet and AR jets impinging on the P3 (red arrow).

Figure 3. Follow-up transthoracic echocardiography images just before surgery
Parasternal long-axis view (a) shows newly developed severe MR. Parasternal
short-axis view (b) shows P3 prolapse (yellow arrow). Apical four-chamber view (c) shows a prolapsed leaflet with a mobile mass (red arrow).

Figure 4. Macroscopic images of the specimens.

(a) Fresh aortic valve specimen, (b) right coronary cusp (RCC) in the aorta side view, and (c) formalin-fixed aortic valve specimen showing a perforation (arrow) and vegetation on the RCC. (d) Fresh specimen of the anterior mitral leaflet, (e) fresh specimens of the ruptured posteromedial papillary muscle (PM) (red arrow) and partial P2 (white arrow) and P3 (yellow arrow), and (f) formalin-fixed specimen demonstrating infective PM destruction and no evidence of infectious change on the mitral leaflets.

Figure 5. Histological images of the posteromedial papillary muscle (PM).

(a) Hematoxylin and eosin (H&E) staining (original magnification×40) and (b) H&E staining (original magnification×100) reveal the neutrophil infiltration in the PM, suggesting suppurative myocarditis.

Video 1a. Parasternal long-axis view shows eccentric aortic regurgitation (AR) and mild mitral regurgitation (MR).
Video 2a. Midesophageal short axis view shows moderate AR and a perforation on the right coronary cusp.

Video 3a. Parasternal long-axis view shows newly developed severe MR in follow-up transthoracic echocardiography just before surgery.

Supplementary material online, Video S1b. Parasternal short-axis view shows that the AR jets swirl along the basal posterior wall and reach the P3 with normal mitral morphology.

Supplementary material online, Video S1c. Apical three-chamber view shows that the AR jets impinge against the posteromedial papillary muscle.

Supplementary material online, Video S2b. Long-axis view shows moderate AR and a perforation on the right coronary cusp.

Supplementary material online, Video S2c. Midesophageal long-axis view shows that the eccentric AR jets reach P2.
1 Supplementary material online, Video S2d. Midesophageal four-chamber view shows the intact A1 and P1 of the mitral leaflets.

2 Supplementary material online, Video S2e. Midesophageal commissural view shows the intact posterior mitral leaflet and AR jets impinging on the P3.

3

4 Supplementary material online, Video S3b. Parasternal short-axis view shows P3 prolapse.

5

6

7 Supplementary material online, Video S3c. Apical four-chamber view shows a prolapsed leaflet with mobile mass.
Figure 3.
Figure 4.
