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**CASE REPORT**
*Valvular heart disease*

# Structures on the ventricular side of the prosthetic valve in extremely late mitral paravalvular leak: a case report

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**Background** Mechanisms of paravalvular leak (PVL) after mitral valve replacement have not been fully delineated. Herein, we report a case of structures on the ventricular side of the mitral valve in a patient with an extremely late PVL.

**Case summary** A 68-year-old female underwent aortic and mitral valve replacement with a mechanical valve 29 years ago. She was in good health for 28 years. However, exertional dyspnoea appeared 8 months ago. She was admitted to our hospital for congestive heart failure and haemolytic anaemia. Echocardiography showed severe regurgitation due to PVL of the mitral valve. The fluoroscopy showed that a circular calcification was found below the mitral prosthesis. The operation was performed through a median sternotomy. After the aortic cross-clamp, the aortic mechanical valve was removed. The ventricular side of the mitral valve was inspected with the endoscope through the aortic annulus before manoeuvers were performed in the mitral valve. A gap was seen between the prosthetic valve and annular tissue and subvalvular calcification. A bioprosthetic valve was placed with a modified collar-reinforcement technique using a xenopericardium strip. The postoperative course was uneventful. PVL and haemolysis completely disappeared.

**Discussion** The ventricular side of the prosthetic valve could be observed before the mitral valve was removed. Not only the protruding circular calcification and displacement of the prosthetic valve to the atrial side but also the loss of adhesion and adhesive nature of the annular tissue played a definitive role in the late PVL occurrence and recurrence after percutaneous or surgical repair.

**Keywords** Case report • Mitral valve replacement • Paravalvular leak • Haemolysis • Intraoperative endoscope

**ESC curriculum** 4.3 Mitral regurgitation • 4.9 Multivalvular disease • 4.10 Prosthetic valves • 7.5 Cardiac surgery

## Learning points

- To understand the specific findings in the preoperative imaging, which must be considered by the multidisciplinary heart team in deciding the optimal therapeutic modality.
- To learn the presumed mechanisms of late paravalvular leak, considering clinical manifestations and the details of annular and subvalvular structures.
- To understand the anatomical features of the ventricular side of a prosthetic valve, which might inhibit the procedural success of surgical or percutaneous repair and cause its recurrence after repair.

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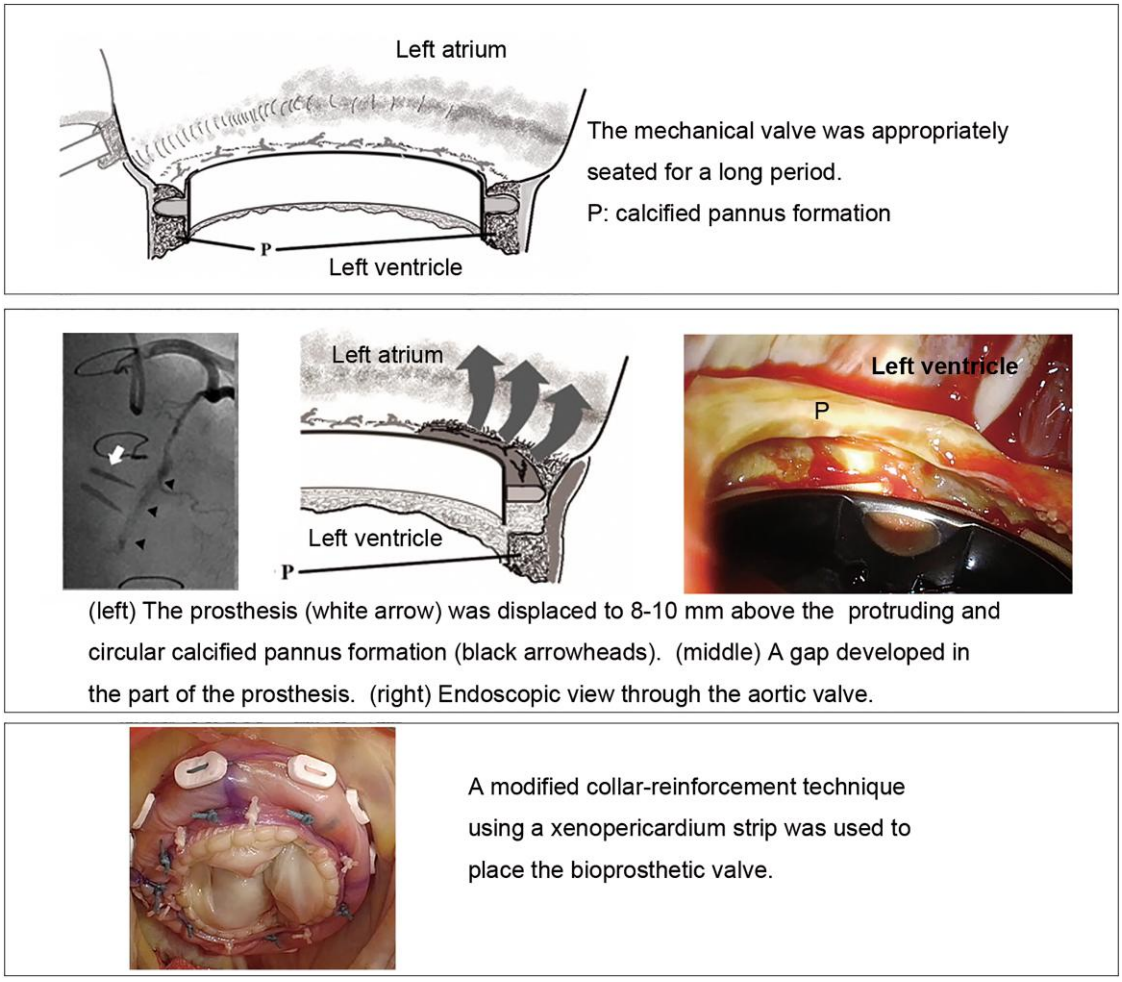
## Introduction

Paravalvular leak (PVL) is a serious complication after mitral valve replacement. PVL occurs a decade or more after implantation despite appropriate manoeuvres and the absence of bacterial infection. Surgical repair is effective for eliminating PVL and concomitant haemolysis in these patients,[1] while early mortality ranged from 5.0% to 13% in the previous reports.[1–5]

Percutaneous repair is safe, less invasive, and effective for symptom relief. The early mortality rate was reported as 2.5–3.1%.[1,4] The procedural success rate was reported as 55–70%[1,4] and residual PVL was strongly associated with adverse clinical outcomes.[6] In addition, PVL recurrence was later detected in 11.3% of patients 6.2 months after percutaneous repair and in 17.2% of patients 42.8 months after surgical repair.[4] Moreover, Said and colleagues revealed multiple recurrences in 15% of their patients.[2] Understanding the structural details around PVL,[7] advanced multimodality imaging,[8] and appropriate patient selection[6] are important for successful percutaneous and surgical repair.

## Summary figure




## Case presentation

A 68-year-old female patient with rheumatic valve disease underwent aortic and mitral valve replacement with St. Jude Medical mechanical valves (Abbott Laboratories, Chicago, IL) 29 years ago. Echocardiography was annually conducted during the follow-up period. There were no cardiac events and no abnormal findings on the follow-up echocardiogram for a long period. She was in good health and had no history of infective endocarditis or bacteraemia for 28 years.

Exertional dyspnoea appeared 8 months prior. PVL of the mitral valve was first detected by echocardiography 5 months prior. At that time, the flow velocity across the aortic valve was 3.5 m/s and the mean pressure gradient was calculated as 22 mmHg. Haemolytic anaemia and dyspnoea gradually developed. She was admitted to our hospital for congestive heart failure. A systolic heart murmur was detected, and the patient presented with orthopnea. The haemoglobin and lactate dehydrogenase levels were 6.8 g/dL and 1379 U/L, respectively. Lung congestion and pleural effusion were seen in the chest roentgenogram. Echocardiography revealed severe mitral regurgitation due to PVL, causing pulmonary hypertension. The transoesophageal echocardiogram revealed severe PVL from the lateral side (see Supplementary material online, *Video S1*). Subvalvular pannus formation caused prosthetic valve stenosis in the aortic position. The peak

flow velocity across the aortic valve was 4.0 m/s, and the aortic valve area was calculated as 0.8 $cm^2$. She was diagnosed with congestive heart failure associated with PVL and aortic valve stenosis. The coronary angiogram revealed no significant stenosis. The mitral prosthesis was displaced 8–10 mm higher than where a circular calcification was present (*Figure 1* and Supplementary material online, *Video S2*). Our multidisciplinary heart team concluded that surgical repair of PVL concomitant with replacement of the aortic valve would be feasible.

The operation was performed through a median sternotomy. Cardiopulmonary bypass was started with ascending aorta cannulation and bicaval drainage. The aorta was transversely incised after an aortic cross-clamp and cardioplegia infusion. The aortic mechanical valve and subvalvular pannus tissue were removed. The ventricular side of the mitral valve was inspected with an endoscope passing through the aortic annulus before the mitral valve manoeuvers (*Figure 2A*, Supplementary material online, *Video S3*). A gap was seen between the prosthetic valve and annular tissue and subvalvular calcification, as seen on fluoroscopy (*Figure 2A*). The adhesion of the prosthetic valve to the annular tissue had been presumed to be loosened. All stitches and pledgets are normally involved in the annular tissue. However, these were discovered on the bare under surface of the mechanical valve's sewing cuff in this patient.

Next, the mitral valve was inspected through the right-sided left atriotomy. A gap between the annulus and sewing cuff was located at the lateral portion (*Figure 2B*). Neointima covered over the cuff, except for the gap. The annulus-cuff adhesion was easily dissected, and the mechanical valve with all threads and the pledgets were removed (*Figure 2C*). Calcified subvalvular tissue was resected, and a modified collar-reinforcement technique using a xenopericardium strip was used to place the Epic bioprosthetic valve (Abbott Laboratories, Chicago, IL).[9] Finally, aortic valve replacement was performed.

The postoperative course was uneventful. The haemolysis completely disappeared. The haemoglobin level was recovered to >11.0 g/dL, and the lactate dehydrogenase level became almost normal. The patient was discharged from the hospital without any complications 14 days postoperatively. She is now in New York Heart Association class I without the recurrence of PVL after 3 years.

## Discussion

The treatment for PVL should be determined with a multidisciplinary approach. Indications and patient selection for the percutaneous or surgical repair have not been fully established, although the percutaneous repair is less invasive and should be currently considered a preferred therapy. Goel and colleagues mentioned a dehiscence of more than one-third of the circumference of the prosthesis as a contraindication for percutaneous treatment.[7] Millan and colleagues reported improved haemolytic anaemia in only 25% of patients who were treated with the percutaneous repair.[1] In particular, the long duration between the valve replacement and PVL repair is considerably associated with moderate or severe residual PVL after percutaneous repair.[10] Late PVL, in addition to a mechanical valve, haemolytic anaemia,[1] large or multiple PVL,[2,7] and recurrent PVL, could be indications for surgical repair. Previous reports revealed that patients who underwent surgical repair had 10- or 13-year durations after valve replacement.[3,4,11] Surgical repair was chosen in this case because the PVL in our patient was considerably large and causing significant haemolysis, the duration between prior surgery and the manifestation of PVL was extremely long, and the patient had concomitant aortic valve stenosis and no considerable risk for surgery.

The presumed mechanism of late PVL is as follows: The valve was stably fixed at an adequate position for a long time. The circular pannus formation protruded on the ventricular side of the cuff, and the atrial surface of the sewing cuff was covered with neo intima (*Figure 3A*). Calcification of the pannus formation and annular tissue had progressed. However, the adhesion to the annular tissue became progressively weak at some point, and the threads and pledgets were no longer fixed or adhered to the surrounding annular tissues. The prosthetic valve concomitant with the threads and pledgets started to deviate to the left atrium and a gap developed in the part of the prosthesis, which took the largest stress or had the weakest adhesion (*Figure 3B*).

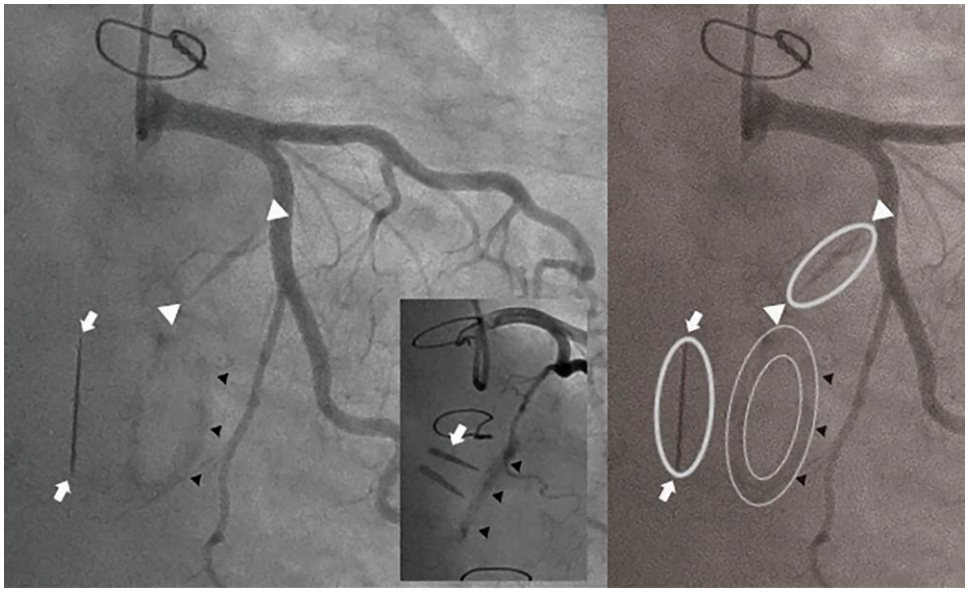


**Figure 1** The mitral valve is normally located at the ventricular side of the aortic valve and with a 'subaortic curtain' between these valves. In this patient, the mitral prosthesis (a circle with white arrows) was displaced to 8–10 mm above the circular calcification (double circle with black arrowheads), which indicated the original position where the mitral valve had been implanted (see Supplementary material online, *Video S2*). A circle with white arrowheads indicates the aortic prosthetic valve.

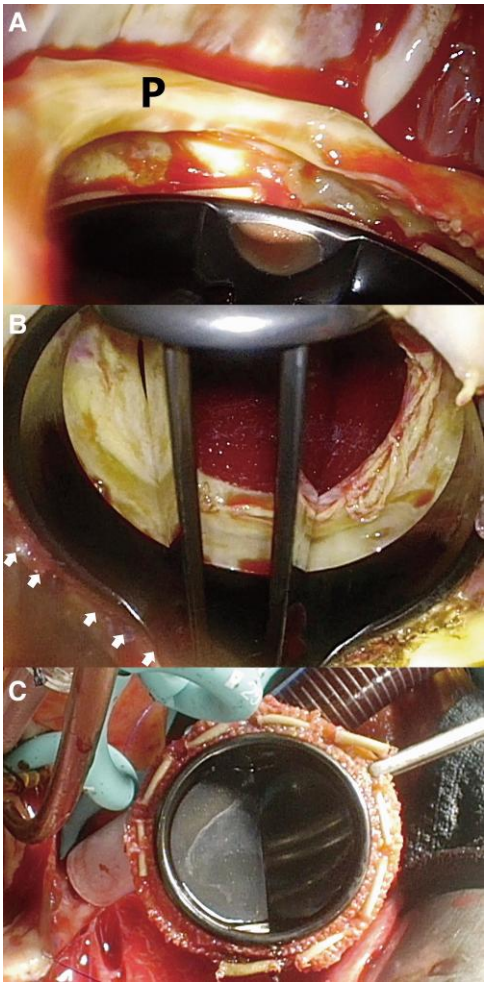



**Figure 2** First, the aortic prosthesis was removed under cardiac arrest. (*A*) The mitral valve was inspected through the aortic annulus using an endoscope. Some gaps were seen between the mitral prosthesis and the circular subvalvular pannus formation, which was protruding and calcified. All threads and pledgets were found on the cuff of the valve. P: calcified and protruding pannus tissue. (*B*) View from the left atriotomy. The cuff fabric was not covered with the neointima in the lateral portion causing PVL. White arrows indicate the gap causing PVL (see Supplementary material online, *Video S3*). (*C*) The prosthesis with threads and pledgets could be easily removed.

The anatomical details of our patient confirmed that percutaneous repair was unsuitable. The presence of circular and protruding calcification, which could be commonly seen in patients who have had prosthetic valves for ≥10 years, will interfere with the fitting of a plug, and expanding a plug may worsen the deviation of the valve to the atrial side. In addition, the adhesion to the annular tissue was loosened not only at the site of PVL but also at the other site of the prosthetic valve. Therefore, even if the gap could be successfully filled with a plug, recurrence of PVL is highly likely, because the annular tissue should have lost the nature of generating tight adhesion.

A critical issue regarding surgical repair may be a relatively high incidence of recurrence during follow-up.[4,11,12] Stiff and collagenised adhesion between the prosthetic valve and the annular tissue should be regenerated to achieve durable repair. Re-replacement with collar reinforcement is technically easy and provides durable repair without

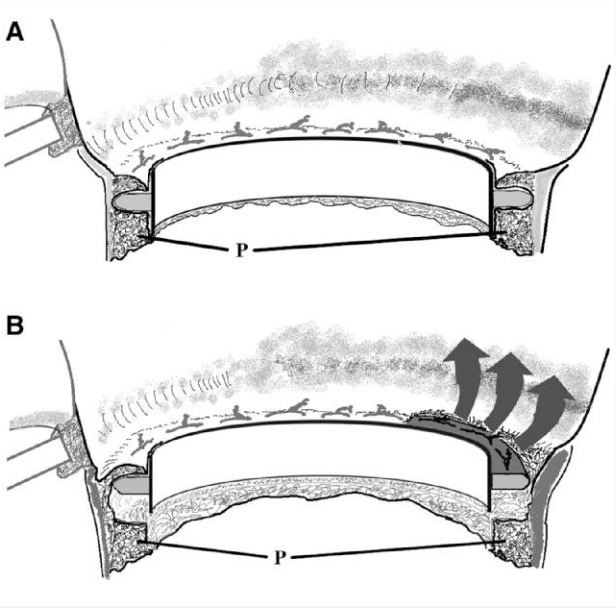



**Figure 3** The mechanism of PVL is presumed as follows: (*A*) the protruding and circular calcified subvalvular tissue was considered as the calcified pannus formation. It should prove that the mechanical valve was appropriately seated for a long period. P: calcified and protruding pannus tissue. (*B*) At some point, the valve and the annular tissue adhesion became loose. Threads and pledgets no longer contributed to the fixation of the valve. The neointima from the left atrium covering the cuff fabric was not stiff enough to maintain fixing the mechanical valve. Finally, the valve deviated to the left atrial side. PVL occurred and developed first at the lateral portion of the prosthesis. P: calcified and protruding pannus tissue.

broad annular debridement, which may cause fatal complications, such as ventricular rupture, aortic valve regurgitation, annular disruption, and circumflex artery obstruction.[9,11]

## Conclusion

The structures on the ventricular side of the mitral valve in a late PVL could be visualized in an untouched condition. Not only the protruding circular calcification and displacement of the prosthetic valve to the atrial side but also the loss of adhesion and adhesive nature of the annular tissue played a definitive role in the late PVL occurrence and recurrence after percutaneous or surgical repair.

## Lead author biography

Hiroyuki Nakajima is a professor in the Department of Cardiovascular Surgery of International Medical Center Saitama Medical University. He completed his residency training at the National Cerebral and Cardiovascular Center in Osaka. He has high expertise in coronary surgery, valvular surgery, re-operation cardiac surgery, and minimally invasive surgery.

# Supplementary material

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

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**Consent:** The authors confirm that written informed consent for the publication of this case report including images, videos, and associated text has been obtained from the patient in accordance with COPE guidelines.

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## Data Availability

All data related to this case report are presented in the published manuscript.

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