Incessant non-sustained ventricular tachycardia immediately after MitraClip placement: a case report

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Background
MitraClip therapy has become an alternative therapy for primary and secondary mitral regurgitation (MR) in patients at high surgical risk. However, this procedure is associated with several complications.

Case summary
The patient was a 93-year-old male with severe MR caused by prolapse of the mid-posterior mitral leaflet (P2) and atrial enlargement. His heart failure (HF) continued to worsen, requiring hospitalization. Considering his high surgical risk, the heart team chose MitraClip treatment. After one clip was placed in the centre of the mitral valve (P2 lateral side), MR severity was reduced from severe to trivial. However, immediately after grasping, incessant non-sustained ventricular tachycardia (VT) with a heart rate of 150 beats/min occurred. Since there were no significant ST-T changes on electrocardiogram and no left ventricular (LV) wall motion abnormalities on echocardiography, ischaemic heart disease was ruled out, and pacing with a temporary pacemaker, potassium level correction, and intravenous amiodarone administration were performed. The frequency of VT decreased but it did not disappear. Diuretics were administered for HF, and VT disappeared within a few hours, with no recurrence, probably due to a decrease in the LV chamber size after diuresis.

Discussion
The VT waveform showed a right bundle branch block pattern with a superior axis. Furthermore, a negative lead I and a transition zone with an abrupt change from V4 to V5 indicated that PVC/VT arose from the posterior papillary muscle area. The probable cause was mechanical extension of the posterior medial papillary muscle as a result of leaflet grasping, with resolution following appropriate volume management.

Keywords
MitraClip • ventricular tachycardia • chordae tendineae • case report

ESC Curriculum
4.3 Mitral regurgitation • 5.6 Ventricular arrhythmia

Learning points
• Transient incessant ventricular tachycardia (VT) has rarely been observed immediately after leaflet grasping with the MitraClip.
• We speculated that the VT in this case was induced by leaflet grasping causing mechanical extension of the posterior medial papillary muscle.
• Heart teams should be aware that this complication can appear after grasping the MitraClip and may be resolved by appropriate volume management.
Introduction

Mitral regurgitation (MR) may occur in up to 50% of patients with heart failure (HF), and it is associated with adverse cardiac remodelling and myocardial stretch. Furthermore, HF with severe MR is associated with an increased risk of ventricular arrhythmia and sudden cardiac death. By decreasing MR severity, the degree of negative cardiac remodelling can be reduced, as can the risk of pump failure and ventricular arrhythmias.

Recently, MitraClip therapy has become an alternative treatment for patients with primary and secondary MR who are at high surgical risk. In this population, MitraClip has been established as a safe procedure with very low adverse event rates compared with mitral surgery. However, several complications of this therapy have been reported. Here, we describe a rare case of transient ventricular tachycardia (VT) occurring immediately after MitraClip grasping.

Timeline

| Day 0 | Mitra Clip procedure |
| Day 0 (Immediately after grasping) | incessant non-sustained VT with a heart rate of 150 beats/min |
| Day 0 (A few hours later) | PVC/VT disappeared |
| Day 0 (After transfer to the intensive care unit) | treated for heart failure with diuretics |
| Day 1 | PVC/VT resolved |

→ amiodarone discontinued, temporary pacemaker removed, sedation terminated, extubated

Case presentation

The patient was a 93-year-old male with severe MR caused by prolapse of the mid-posterior mitral leaflet (P2) and atrial enlargement. At the age of 69 years, he was diagnosed with Stanford A type acute aortic dissection (ADD) and underwent ascending and partial arch replacement. In addition, he had hypertension, chronic renal failure (estimated glomerular filtration rate: 33.7 mL/min/1.73 m²), and persistent atrial fibrillation. Despite his age, he was independent in terms of activities of daily living (Clinical Frailty Scale: 3–4/9) and had near-intact cognitive ability (Mini-Mental State Examination: 29/30). Two months before the MitraClip procedure, he developed dyspnoea on exertion. Severe MR was documented, and HF continued to worsen and required hospitalization.

Electrocardiogram (ECG) revealed atrial fibrillation with premature ventricular contraction (PVC). The origin of PVC was a right bundle branch block (RBBB) pattern with an inferior axis due to a high R wave and negative T wave in V1 (Figure 1A). Echocardiography in the stable stage showed severe MR due to P2 prolapse (Supplementary material online, Video 1, Figure 2A and B), with elongated chordae tendineae. Evaluation using the proximal isovelocity surface area method revealed an effective regurgitant orifice area of 0.47 cm² and a regurgitant volume of 71 mL. The mean mitral valve gradient was 2 mmHg and the mitral valve area as determined by three-dimensional planimetry was 4.6 cm². The left ventricular (LV) end-diastolic diameter was mildly increased at 57 mm, but the LV ejection fraction was normal with no wall motion abnormalities. Moderate post-capillary pulmonary hypertension was detected by right heart catheterization. In addition, coronary angiography showed no significant stenosis. Because of the patient’s history of AAD surgery, chronic renal failure, New York Heart Association (NYHA) Class III, and the need for urgent surgery, his operative mortality risk was determined to be 26.6% by the Society of Thoracic Surgeons score and 26.8% by EUROSCORE II. Considering the high surgical risk, our heart team decided to perform MitraClip therapy.

After one clip was placed in the centre of the mitral valve (P2 lateral side), MR severity decreased from severe to trivial. The trans-mitral gradient increased from 0.6 to 2.5 mmHg. The final mitral valve area after MitraClip therapy was 3.06 cm². However, immediately after grasping of the mitral leaflet, incessant non-sustained VT with a heart rate of 150/min occurred and systolic blood pressure dropped from 110 to 70 mmHg during VT (Supplementary material online, Video 2). The VT waveform showed an RBBB pattern with a superior axis (Figure 1B).

There were no significant ST-T changes on ECG and no LV wall motion abnormalities on echocardiography. Since PVC occurred frequently after long RR intervals, we performed pacing with a temporary pacemaker at a rate of 100 beats/min, with the expectation of suppressing VT. We also corrected the potassium level (K ≥ 4.5 mEq/L; before correction, 3.7 mEq/L) and administered amiodarone intravenously. While the frequency of VT decreased, it did not disappear. Because

Figure 1 Electrocardiography. (A) Baseline electrocardiogram demonstrates atrial fibrillation and a single premature ventricular contraction with a right bundle branch block and an inferior axis. (B) The ventricular tachycardia waveform is a right bundle branch block with a superior axis pattern, indicating a different origin from the premature ventricular contraction documented before procedure.
Incessant non-sustained ventricular tachycardia

The rigid shaft connected to the clip may have caused VT by increasing papillary muscle tension, we decided to release the clip. However, VT persisted after the clip release. The patient was transferred to the intensive care unit with continued intravenous injection of amiodarone and was treated for HF with diuretics. Surprisingly, PVC/VT disappeared a few hours later and did not recur, and MR remained mild (Figure 3A and B). In addition, there was no significant elevation of myocardial enzymes during follow-up. The patient was clinically stable without VT or HF recurrence during 2-year follow-up.

Discussion

Here, we report a case of incessant non-sustained VT observed immediately after the MitraClip procedure for primary MR. The VT waveform showed a RBBB pattern with a superior axis. Furthermore, a negative lead I and a transition zone with an abrupt change from V4 to V5 indicated that PVC/VT arose from the posterior papillary muscle area. Since this PVC/VT morphology was not documented before the procedure and began just after grasping of the mitral leaflet, we speculated that it was induced by mechanical extension of the posterior medial papillary muscle as a result of leaflet grasping (Figure 4).

The fact that VT resolved a few hours after the clip procedure was fortunate, but serious complications could have occurred. The patient’s clinical course may be explained by fluid balance changes during and after MitraClip procedure. Intraoperatively, the fluid balance was high due to efforts to maintain blood pressure and because of continued fluid input from the device flush port. In this case, the total sedation duration was over 4 h and during this time there was a positive fluid balance of slightly over 1.5 L. As a result, LV volume increased from 163 mL before MitraClip procedure to 191 mL just afterward. Left ventricular volume decreased to 134 mL due to reduced preload in the conscious state within a few hours after the procedure. This geometry change might have released the tension on the chordae tendineae and reduced the frequency of VT. This is the first report of transient incessant VT observed immediately after MitraClip procedure.

**Figure 2** Baseline echocardiography. (A) Baseline echocardiography demonstrated mal-coaptation of the mitral valve leaflets between A2 and P2. (B) Echocardiography showed severe mitral regurgitation caused by P2 prolapse with elongated chordae tendineae.

**Figure 3** Mitral regurgitation before and after the mitraClip procedure. After one clip was placed in the centre of the mitral valve (P2 lateral side), mitral regurgitation worsened from severe (A) to trivial (B).
Conclusions

We experienced a rare case of transient incessant VT observed immediately after leaflet grasping with the MitraClip. Heart teams should be aware that this rare complication can appear after MitraClip grasping and may resolve with appropriate volume management.

Lead author biography

Sayaka Funabashi graduated from Kyorin University and received the MD degree in 2011. She was working as a Cardiology senior resident at Kyorin university hospital from 2013. Then, she became a cardiovascular resident and fellow at Department of Cardiovascular Medicine, National Cerebral and Cardiovascular Center, Japan from 2016. From 2021, she has worked as a Cardiology fellow at the Kyorin university hospital.

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 authors confirm that written consent for the submission and publication of this case report, including images and associated text, was obtained from the patient in line with COPE guidance.

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