Journal of Cardiology Cases 30 (2024) 150–153

Contents lists available at ScienceDirect

Journal of Cardiology Cases

journal homepage: www.elsevier.com/locate/jccase

Case Report

Iatrogenic atrial septal closure for mitral stenosis after transcatheter edge-to-edge repair: A case report

Shuhei Tanaka (MD, PhD), Hiroshi Ueno (MD, PhD) *, Nobuyuki Fukuda (MD, PhD), Teruhiko Imamura (MD, PhD, FJCC), Koichiro Kinugawa (MD, PhD, FJCC)

The Second Department of Internal Medicine, University of Toyama, Toyama, Japan

ARTICLE INFO

Article history:
Received 13 March 2024
Received in revised form 19 June 2024
Accepted 1 July 2024

Keywords:
Mitral regurgitation
Heart failure
Iatrogenic atrial septal defect

ABSTRACT

Interventions for structural heart disease requiring a transvenous and transseptal approach, such as transcatheter edge-to-edge repair (TEER), cause iatrogenic atrial septal defect (IASD) after treatment. Its clinical impact remains uncertain. We present the case of an 84-year-old female patient with heart failure with preserved ejection fraction and stress-induced mitral regurgitation (MR), who was repeatedly hospitalized for acute pulmonary edema. Exercise stress echocardiography reproduced worsening MR. Intervention for MR by TEER was considered, while small mitral valve area was a concern. Mitral stenosis (MS) occurred at the time of TEER, but the clip was eventually implanted to prioritize improvement of regurgitation. IASD was subsequently a concern, as the volume of the left-to-right shunt was increasing, and cardiac output was decreasing. We decided to perform a percutaneous IASD closure, which successfully elevated her blood pressure and allowed her to be discharged home on foot. The coexistence of MS, as in the present case, may increase the negative hemodynamic impact of IASD. Percutaneous IASD closure may be a promising therapeutic strategy to stabilize hemodynamics in carefully selected cases.

Learning objective: Iatrogenic atrial septal defect (IASD) closure is rarely necessary after transcatheter edge-to-edge repair (TEER). Excessively narrowing mitral valve area after TEER can increase the hemodynamic impact of left-to-right shunt flow through IASD. IASD closure may increase cardiac output, but the indication should be carefully determined after confirming the hemodynamic impact, e.g. balloon closure studies.

Introduction

Transcatheter edge-to-edge repair (TEER) with the MitraClip™ system (Abbott Vascular, Abbott Park, IL, USA) for mitral regurgitation (MR) has been reported with favorable clinical outcomes in heart failure patients with a wide spectrum of ejection fraction [1]. TEER is also beneficial for exercise-induced MR, regardless of the severity of MR at rest [2].

However, TEER requires several specific anatomic conditions. Among these conditions, baseline small mitral valve orifice area (MVA) can sometimes be an Achilles heel. It has been reported that post-TEER smaller MVA or an increase in trans-mitral valve mean pressure gradient, indicating postoperative mitral stenosis (MS), does not have a significant prognostic impact, and management of MR by aggressive TEER is often a priority [3].

After the structural heart disease intervention via transseptal approach, iatrogenic atrial septal defect (IASD) occurs. However, it usually causes only a small amount of left-to-right shunt and does not have significant clinical implication.

Nevertheless, the hemodynamic impact of IASD in patients with MS after TEER has not been well studied. Thus, we do not know whether we can ignore IASD, especially in such a scenario.

Case report

The patient was an 84-year-old woman with a history of hypertension, chronic kidney disease, and post pacemaker implantation state for sick sinus syndrome, who had been repeatedly hospitalized for heart failure. Only 3 weeks after being discharged from another hospital, she continued to have chest discomfort, and she was taken to our hospital.

On admission

On admission, she had a low blood pressure of 95/54 mmHg and a heart rate of 60/min in an atrial pacing rhythm. Chest X-ray showed severe pulmonary congestion, plasma B-type natriuretic peptide was elevated to 954 pg/mL, and transthoracic echocardiography (TTE)

* Corresponding author at: The Second Department of Internal Medicine, University of Toyama, 2630 Sugitani, Toyama, Toyama 930-0194, Japan.
E-mail address: hiroshi.ueno.md@gmail.com (H. Ueno).

https://doi.org/10.1016/j.jccase.2024.07.002

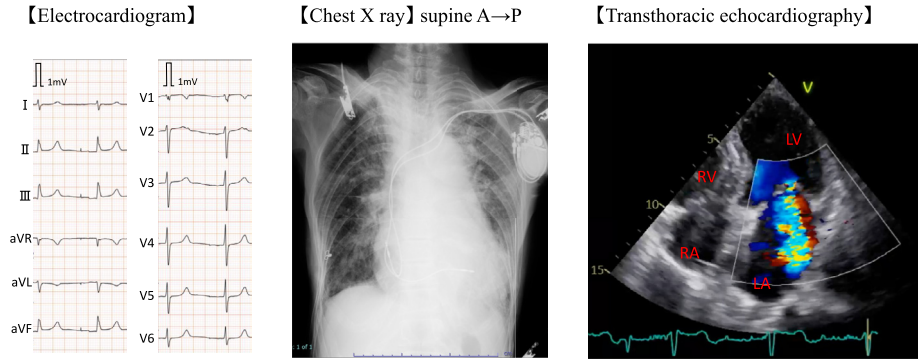

Fig. 1. Electrocardiogram, chest X-ray, and transthoracic echocardiography at admission to our hospital.
LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

showed severe MR from the A2-P2 region, although left ventricular systolic function was preserved (left ventricular end-diastolic diameter 37 mm and left ventricular ejection fraction 70 %) and left atrial (LA) size was almost normal (LA volume index 36 mL/m^2) (Fig. 1).

Small doses of intravenous diuretics quickly compensated for the heart failure and MR almost disappeared. Because MR worsened from 21 % of LA volume at rest to 32 % of that during exercise even after intensification of medical therapy including the introduction of angiotensin receptor neprilysin inhibitor or an increase in pacing rate to 70 bpm, a mitral valve intervention was considered. Due to her advanced age and high frailty, open heart surgery was not feasible, and TEER using the MitraClip™ system was considered.

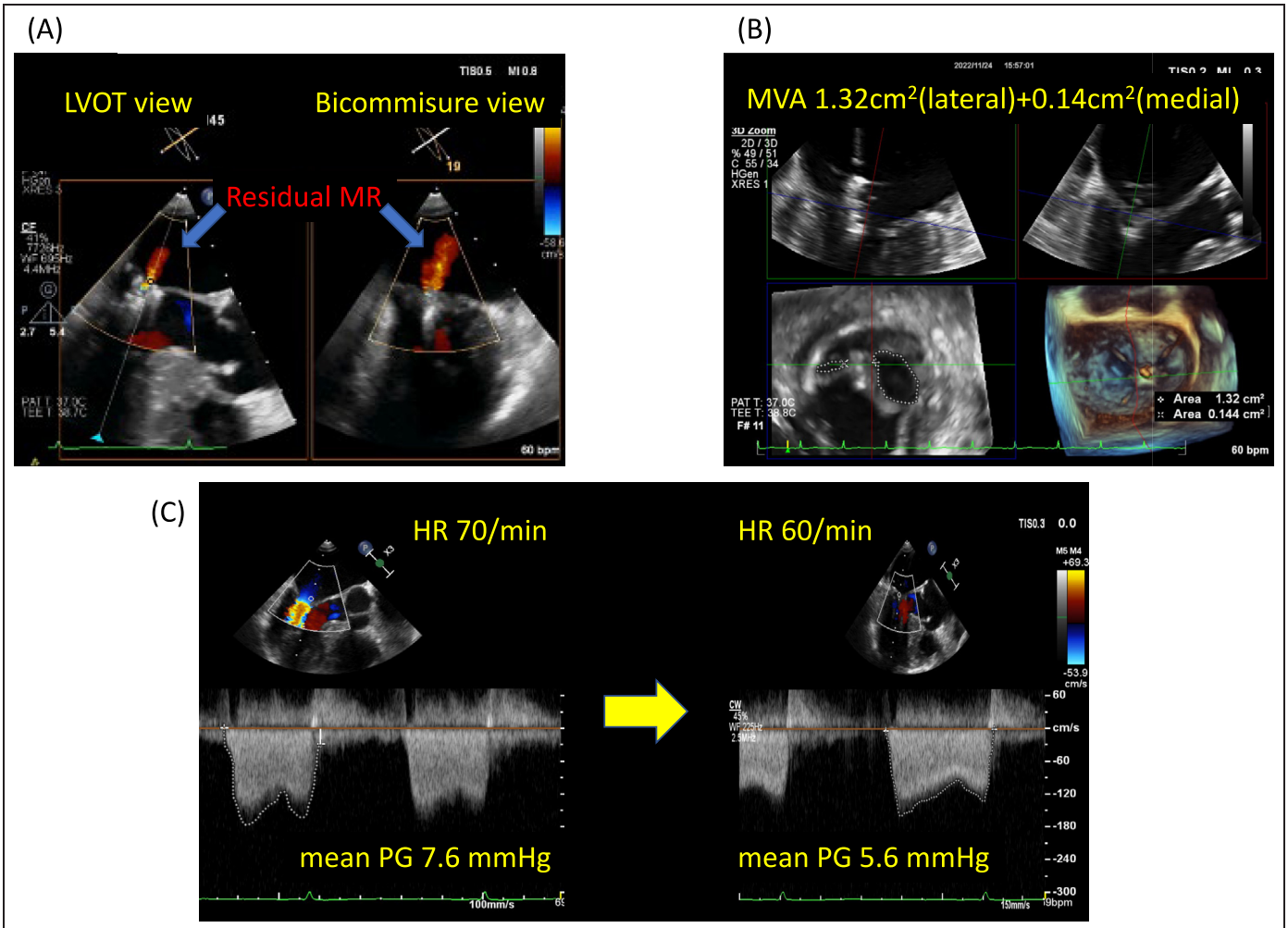

Fig. 2. TEE findings after TEER. (A) Residual regurgitation following TEER, (B) 3D measurement of MVA after TEER, (C) change in mean PG of transmitral flow after 10/min less pacing rate.
HR, heart rate; TEE, transesophageal echocardiography; TEER, transcatheter edge-to-edge repair; MVA, mitral valve area; PG, pressure gradient; LVOT, left ventricular outflow tract; MR, mitral regurgitation.

Indication of TEER

Transesophageal echocardiography revealed thickening of the valve leaflets, but no huge degenerative change such as prolapse or billowing. Although there was no evidence of commissural fusion, a certain degree of restricted motion was particularly observed during the diastolic phase. Based on these findings, the etiology of MR was considered to be Carpentier type IIIa, indicating primary MR.

Other anatomic factors were suitable for TEER, but the small MVA measured 3D planimetry of 2.0 cm^2 was a strong concern for iatrogenic MS after clipping. However, she had repeatedly been hospitalized for heart failure in the short term and always had severe MR on admission. Intervention for MR was considered effective in preventing acute pulmonary edema. Since she had a very small body surface area of less than 1.2 m^2 and only trivial organic changes in the mitral valve, we thought it was worth trying TEER, despite an MVA of 2.0 cm^2.

TEER procedure

TEER was performed on day 29 with a plan not to implant if post-clipping MS was unacceptably severe. MitraClip™ NT was placed in the A2-P2 region of the mitral valve, and the mean trans-mitral pressure gradient (PG) of trans-mitral flow increased from 1.1 mmHg to 7.6 mmHg. When the pacing rate was decreased from 70/min to 60/min, the mean PG decreased to 5.6 mmHg (Fig. 2).

MR almost disappeared and pulmonary vein flow, originally systolic dominant, showed further elevation in the S wave. The LA pressure remained following TEER, but the v-wave improved from 20 mmHg to 18 mmHg. Although the MVA measured 3D planimetry was approximately 1.5 cm^2, the procedure was successfully completed with clip implantation as it was thought to be of great benefit in controlling MR during exacerbation. IASD after TEER was only L-R shunt.

Clinical course after TEER procedure

A few days after TEER, her urine output decreased, and her general discomfort worsened. TTE showed trivial MR but a high transmitral pressure gradient of 7 mmHg and a marked L-R shunt. Right heart catheterization showed a low pulmonary artery wedge pressure of 9 mmHg, but the cardiac index was also low at 1.9 L/min/m^2 and the oxygen saturation was 61.4 % in the superior and inferior vena cava and 70.9 % in the pulmonary artery, indicating a significant O_2 step-up, and the Qp/Qs was 1.3.

We decided to perform an urgent percutaneous IASD closure, as MS after TEER contributed to the significantly increased L-R shunt volume and caused low cardiac output. The balloon occlusion test of the IASD showed a rapid increase in blood pressure of more than 10 mmHg, indicating that the IASD closure was considered effective.

A 10.5 mm Figulla Flex II (Occlutech Holding AG, Schaffhausen, Switzerland) was used for occlusion (Fig. 3). After the successful procedure, her blood pressure further increased and was maintained around 100 mmHg. She was discharged to home on foot after the addition of an angiotensin-converting enzyme inhibitor and diuretics.

Discussion

Therapeutic strategies for load-induced dynamic MR

Clinically, it is common to see cases in which MR is no longer significant after heart failure compensation, despite the coexistence of severe MR during heart failure worsening. In this way, the severity of MR varies dynamically depending on the cardiac load.

Its mechanisms in patients with reduced ejection fraction are considered as follows. The increase in afterload caused by the rapid increase in blood pressure during exercise leads to a worsening coaptation of the mitral valve leaflet. In the presence of increased volume, significant increases in systemic resistance lead to exercise-induced ventricular dilatation in the absence of adequate arterial dilatation [4]. An increase in left ventricular sphericity, which correlates with greater papillary muscle distance, may have a pronounced effect on mitral geometry even without a significant change in volume.

In patients with preserved ejection fraction, the mechanisms are not clear, but the first two theories may be true. TEER is also a therapeutic option for load-induced MR and has shown favorable results compared to medical therapy alone [2].

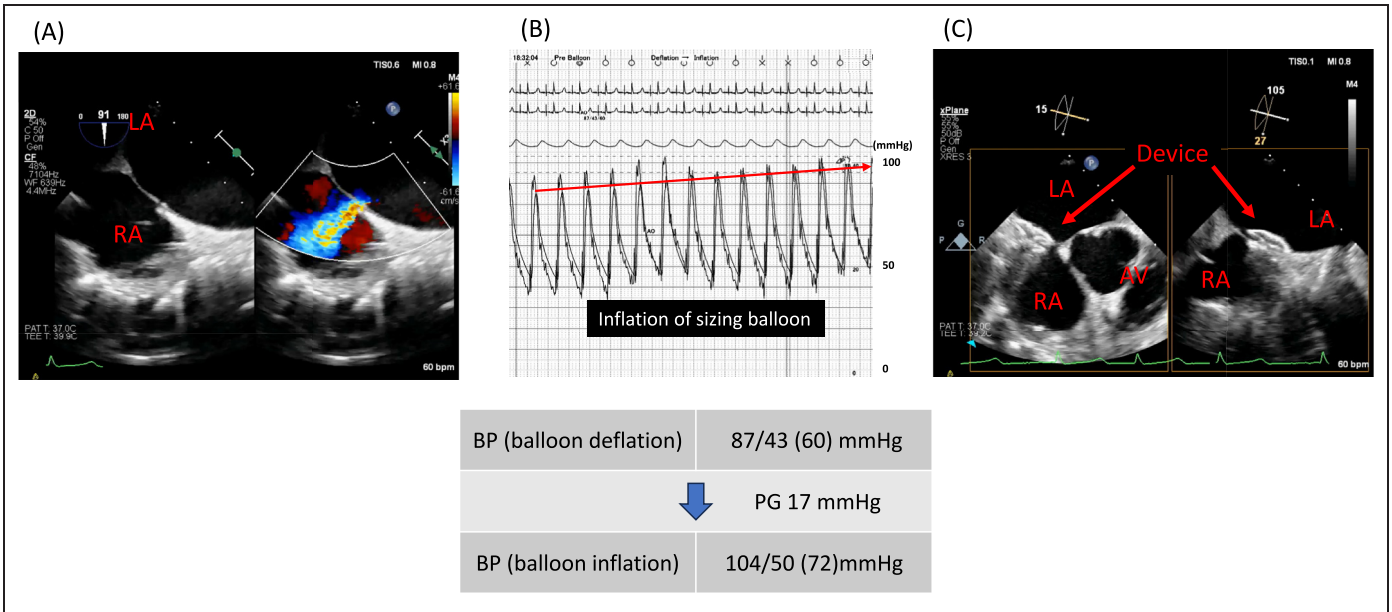

Fig. 3. TEE findings and hemodynamic change during IASD closure. (A) Left-to-right shunting from IASD in the absence of PEEP. (B) Increased blood pressure during IASD closure with sizing balloon. (C) TEE finding after Figulla Flex II implantation.
BP, blood pressure; TEE, transesophageal echocardiography; IASD, iatrogenic atrial septal defect; PEEP, positive end-expiratory pressure; PG, pressure gradient; LA, left atrium; RA, right atrium; AV, aortic valve.

Anatomic complication after TEER

TEER always causes iatrogenic MS and IASD. Regarding iatrogenic MS, it is certain that there will be more stenosis than before TEER, regardless of the degree. Whether iatrogenic MS is associated with prognosis is controversial [3,5], but in general it is important to prioritize MR reduction [6].

IASD that occurs after the removal of the steerable guiding catheter (25Fr) in the MitraClip system is also occasionally a problem. The percentage of residual IASD remaining at 6 months following TEER was 40 % in our cohort, similar to the other literature. In the TVT registry, only 1.5 % of cases required IASD closure, mostly because of O_2 desaturation due to R-L shunting through an IASD [7].

In this case, we prioritized improving MR due to its exacerbation during decompensated heart failure. As a result of the clinical impact of iatrogenic MS, the influence of IASD on hemodynamics could also be significant. Furthermore, after TEER, there was concern about load-induced MS with PH. However, a clip was implanted after confirming that slowing the pacing rate would minimize the hemodynamic impact of MS.

Additionally, the LA emptying fraction and LA peak systolic strain were markedly reduced in this case, at 18.3 % and 12.8 %, respectively, indicating LA dysfunction and decreased LA compliance. The decreased LA compliance may have contributed to the increased L-R shunt volume in the IASD, as well as in iatrogenic MS.

Features and treatment of iatrogenic Lutembacher syndrome

Lutembacher is credited with the first comprehensive description of ASD with MS in 1916 [8], which is named Lutembacher syndrome. Lutembacher syndrome is well-tolerated for a longstanding symptom of pulmonary venous hypertension, such as hemoptysis, paroxysmal nocturnal dyspnea, and orthopnea due to LA decompression from L-R shunting by an ASD. Symptoms such as palpitations and fatigue occur early in patients with Lutembacher syndrome due to the increased L-R shunt and decreased systemic cardiac output.

Treatment of structural heart disease through the transseptal approach such as percutaneous transseptal mitral commissurotomy can cause a so-called iatrogenic Lutembacher syndrome [9]. In this case, the progression of MS through TEER is thought to cause a high-volume L-R shunt, resulting in a decrease in systemic output.

Traditionally the gold-standard treatment has been open heart surgery. However, with the advancement of percutaneous interventional techniques, hardware, and more expertise, device closure for IASD has become the preferred treatment choice [10]. Percutaneous IASD closure is not technically so challenging but rather a detailed evaluation to determine the indication for treatment is of great importance.

Consent

Written informed consent was obtained from the patient.

Funding

None.

Declaration of competing interest

There is no conflict of interest related to this manuscript.

References

[1] Gröger M, Scheffler JK, Schösser F, Schneider LM, Rottbauer W, Markovic S, Keßler M. Percutaneous edge-to-edge mitral valve repair for mitral regurgitation improves heart failure symptoms in heart failure with preserved ejection fraction patients. ESC Heart Fail 2021;8:5010–21.
[2] Izumo M, Kuwata S, Ishibashi Y, Suzuki T, Ohara H, Watanabe M, Sato Y, Nishikawa H, Okuyama K, Kamijima R, Takai M, Kou S, Harada T, Akashi YJ. Prognostic impact of transcatheter mitral valve repair in patients with exercise-induced secondary mitral regurgitation. Eur Heart J Cardiovasc Imaging 2021;22:530–8.
[3] Halaby R, Herrmann HC, Gertz ZM, Lim S, Kar S, Lindenfeld J, Abraham WT, Grayburn PA, Naidu S, Asch FM, Weissman NJ, Zhang Y, Mack MJ, Stone GW. Effect of mitral valve gradient after MitraClip on outcomes in secondary mitral regurgitation: results from the COAPT trial. JACC Cardiovasc Interv 2021;14:879–89.
[4] Sullivan MJ, Knight JD, Higginbotham MB, Cobb FR. Relation between central and peripheral hemodynamics during exercise in patients with chronic heart failure. Muscle blood flow is reduced with maintenance of arterial perfusion pressure. Circulation 1989;80:769–81.
[5] Hadjadj S, Freitas-Ferraz AB, Paquin A, Rouleau Z, Simard S, Bernier M, O'Connor K, Salaun E, Pibarot P, Clavel MA, Rodés-Cabau J, Paradis JM, Beaudoin J. Echocardiographic variables associated with transvalvular gradient after a transcatheter edge-to-edge mitral valve repair. J Am Soc Echocardiogr 2022;35:86–95.
[6] Neuss M, Schau T, Isotani A, Pilz M, Schöpp M, Butter C. Elevated mitral valve pressure gradient after Mitraclip implantation deteriorates long-term outcome in patients with severe mitral regurgitation and severe heart failure. JACC Cardiovasc Interv 2017;10:931–9.
[7] Chhatriwalla AK, Vemulapalli S, Szerlip M, Kodali S, Hahn RT, Saxon JT, Mack MJ, Ailawadi G, Rymer J, Manandhar P, Kosinski AS, Sorajja P. Operator experience and outcomes of transcatheter mitral valve repair in the United States. J Am Coll Cardiol 2019;74:2955–65.
[8] Lutembacher R. De la sténose mitrale avec communication interauriculaire. Arch Mal Coeur 1916;9:237–60.
[9] Aminde LN, Dzudie A, Takah NF, Ngu KB, Sliwa K, Kengne AP. Current diagnostic and treatment strategies for Lutembacher syndrome: the pivotal role of echocardiography. Cardiovasc Diagn Ther 2015;5:122–32.
[10] Phan QT, Nguyen HL, Le TD, Lee W, Won H, Shin S, Sharmin S, Nguyen TQ, Kim S. Combined percutaneous procedure in patient with Lutembacher syndrome: a case report and real-world experience review. Cardiol Res 2018;9:385–91.