Iatrogenic Left Circumflex Coronary Occlusion Following Mitral Valve Replacement Surgery: A Case-report

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Abstract

Injury to epicardial coronary arteries following mitral valve replacement surgery, albeit rare, could have fatal complications. In this case, we suggest conservative medical treatment as a safe approach in patients who are not suitable to undergo revascularization.

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ABSTRACT

Injury to epicardial coronary arteries following mitral valve replacement surgery, albeit rare, could have fatal complications. In this case, we suggest conservative medical treatment as a safe approach in patients who are not suitable to undergo revascularization.

Key words: left circumflex coronary artery; mitral valve replacement; occlusion

Key Clinical Message

In patients undergoing valve surgery who present with symptoms such as ventricular tachycardia, ECG changes, or elevated cardiac biomarkers should prompt the possible injury to epicardial coronary arteries and should immediately undergo angiography.

BACKGROUND

Progress in the techniques of mitral valve replacement (MVR) surgery and the resultant high efficacy and safety have led to its widespread use. (1-3) However, MVR may be rarely associated with serious complications. Acute myocardial infarction following MVR is amongst these serious complications that result from the compressive effect of the mitral valve prosthesis on the adjacent coronary arteries, particularly the left circumflex coronary artery (LCX) and the posterior artery of the left ventricle (LV). (3-6) Iatrogenic damage to the LCX following MVR is rare, but it may constitute a complicated and life-threatening injury. (1, 2, 7) The existing literature contains a dearth of information on post-MVR iatrogenic LCX injury; accordingly, we herein present a case of LCX injury secondary to MVR, managed with conservative medical treatment. Additionally, we discuss the causes of this infrequent complication and the treatment of choice. (4)

CASE PRESENTATION

In August 2018, a 59-year-old woman with a history of severe rheumatismal mitral stenosis and balloon mitral valvulotomy in 2010 and 2012, respectively, was admitted to our center (Tehran Heart Center) with a complaint of dyspnea on exertion (functional Class III). The patient had atrial fibrillation rhythm with an appropriate ventricular response. Transthoracic and subsequently transesophageal echocardiographic examinations showed a normal-sized LV with preserved systolic function (left ventricular ejection fraction [LVEF] =50–55%), a severely enlarged left atrium, a mildly enlarged right atrium, and a normal-sized right ventricle with preserved systolic function. Also visualized were severe rheumatismal mitral stenosis (mean gradient = 13.6 mm Hg, mitral valve area = 1.3 cm^2 , and Wilkins score = 8-10), mild-to-moderate tricuspid regurgitation, mild mitral regurgitation, mild pulmonary insufficiency with a pulmonary arterial pressure of 32 mm Hg, and a left atrial appendage thrombus ($8 \times 10 \text{ mm}$). The patient was scheduled for an elective MVR, and she underwent a routine preoperative coronary angiography (CA) (Fig. 1).

Three days after admission, the patient underwent MVR through mid-sternotomy. The left atrium was opened with an incision parallel to the interatrial groove, the aorta was cross-clamped, and cardioplegia was infused for the replacement of the mitral valve. Next, the anterior leaflet of the atrium was resected and the valve was replaced. A Regent St Jude mechanical valve (# 29) was used. About 6 hours after surgery and before extubation, she developed a sustained monomorphic ventricular tachycardia, which was instantly cardioverted with direct-current shock (200 joules biphasic). Thereafter, intravenous amiodarone was started with a bolus dose of 150 mg and 1 mg/min infusion. She remained in a stable condition for 10 hours before extubation. About an hour after extubation, a sustained monomorphic ventricular tachycardia reoccurred, which was successfully cardioverted to atrial fibrillation rhythm with direct-current shock (200 joules biphasic). Electrocardiography (ECG) revealed ST-elevation in the inferior leads during the second episode of ventricular tachycardia which disappeared after cardioversion. Urgent transthoracic echocardiography showed a diminished LVEF of 45% with significant hypokinesia in the LCX territory. The troponin level exhibited a rise to 1386 ng/L. On suspicion of iatrogenic occlusion in the coronary arteries, the patient underwent emergency angiography, which revealed occlusion in the LCX at the mid-part, most probably due to the compressive effect of the prosthetic valve on the artery (Fig. 2).

There were 2 options for the treatment of the patient: either to perform redo surgery for the removal of the valve, revascularization of the LCX, and reimplantation of the valve or to perform percutaneous coronary intervention (PCI). Time constraints precluded the second option; therefore, PCI was performed to place a stent, which was unsuccessful. A decision was then made to place her on conservative medication. While receiving inotrope agents and anti-ischemic therapy, she was maintained on the balloon pump for 3 days

because of LV hypokinesia and a low LVEF.

The patient was discharged from the hospital 3 days after the removal of the balloon pump in a stable condition and with improved cardiac function. Needless to say, generally, the occurrence of such complications renders the removal of the balloon pump extremely difficult. Our patient remained stable, with her follow-up echocardiography on April 27, 2019, demonstrating LVEF of 50%; mild mitral regurgitation; mild aortic stenosis; a normal functioning prosthetic mitral valve with good bileaflet motion, acceptable gradient (mean gradient = 2.5 mm Hg), and minimal paravalvular leak from the anterior side of the swing ring; and a pulmonary arterial pressure of 27 mm Hg. The study conforms to the ethical guidelines of the 2013 Declaration of Helsinki as reflected in a prior approval by ethics committee of the Tehran Heart Center.

DISCUSSION

We herein presented a case of iatrogenic occlusion in the LCX after MVR, resolved with medical treatment. The LCX is located near the commissure of the mitral valve, and the coronary sinus is adjacent to the annulus of the posterior mitral valve leaflet; both vessels lie within the left atrioventricular groove. (3, 4) The LCX courses along the left atrioventricular groove; it ends near the margin of the LV in 85% to 95%of individuals, whereas it continues to the crux of the heart to develop the posterior descending artery (the dominant left coronary artery) in 5% to 15%. (3) Suggested mechanisms of injury to the epicardial coronary vessel following MVR include air embolism, the encirclement of the artery by suture, coronary spasm, the use of oversized prosthetic values, the excessive removal of the anterior leaflet, and the close proximity of the LCX to the mitral annulus, especially in the left dominant ones. (2, 3, 8) The mechanism of injury, in our patient, was probably the pressure of the prosthetic valve onto the LCX, resulting in the occlusion of the artery. In such cases, symptom onset is usually intraoperative or immediately after surgery and common presentations are ventricular tachycardia or arrhythmia, ECG changes, elevated cardiac biomarkers such as troponin, and new wall motion abnormalities. (1, 9) Our case presented with ventricular tachycardia a few hours after surgery, with ST-segment changes in the inferior leads during tachycardia and an elevated level of troponin (1389 ng/L). Imaging can identify the cause of ventricular tachycardia, and angiography helps to evaluate the location of the injury and whether any further invasive procedure is required. (4, 10, 11) The hemodynamic stability of our patient enabled us to identify the presence and extent of the coronary lesion via angiography. (3)

There are only a few methods that are favored for the treatment of LCX injury, (9) with the choices limited to either redo surgery aimed at removing the prosthetic valve for replacement with a smaller one or aortocoronary bypass grafting onto the LCX distal to the site of the injury.(12) Initially, we performed PCI on our patient for stent implantation, which proved unsuccessful. Subsequently, of the 2 options of either valve removal for LCX revascularization followed by MVR or conservative medical treatment, we decided that, given the exigencies of time, the latter would be more advisable.

CONCLUSION

In conclusion, despite the low incidence rate of introgenic damage to the LCX caused by the excision and removal of the valve in MVR surgery, the complication can prove complex and life-threatening. The prevention of such a complication is of utmost importance and requires CA on all patients undergoing valve surgery with a view to verify the location of the LCX. (12) In patients without preoperative preliminary angiography that present with symptoms such as ventricular tachycardia, ECG changes, or elevated cardiac biomarkers, we recommend that postoperative CA be performed forthwith to detect any possible injury to the LCX.

ACKNOWLEDGMENTS

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CONFLICT OF INTEREST

The authors declare that they have no competing interests.

AUTHOR CONTRIBUTIONS

SHAT, SM and MJM contributed to the study conception and design. Material preparation was performed by MA, MRB and MJM. The first draft of the manuscript was written by NY, MGHL, and ATT and all authors commented on previous versions of the manuscript. All authors read and approved the final version of the manuscript.

ETHICAL APPROVAL

The patient provided verbal consent for the publication of his case and it was approved by the ethics committee of the Tehran Heart Center

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Figure Legends

Figure 1: Preoperative coronary angiogram shows an intact left circumflex coronary artery (arrow).

Figure 2: Postoperative coronary angiogram shows occlusion in the left circumflex coronary artery (arrow)

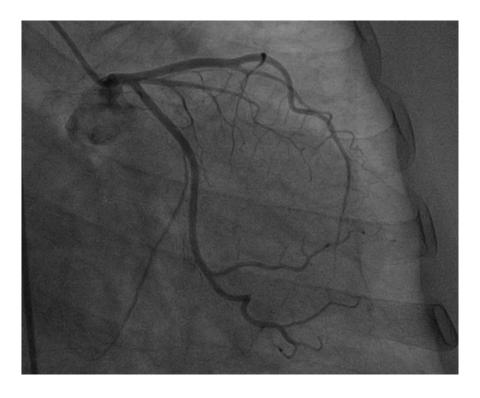


Figure 1: Preoperative coronary angiogram shows an intact left circumflex coronary artery (arrow).

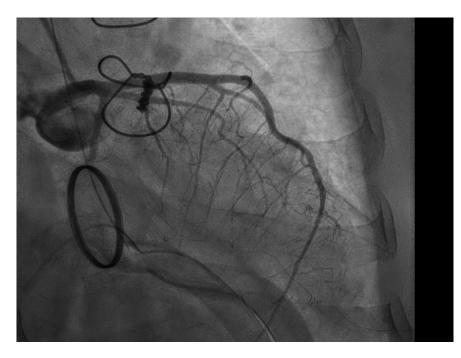


Figure 2: Postoperative coronary angiogram shows occlusion in the left circumflex coronary artery (arrow)