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PROCEDURAL COMPLICATIONS: PART 1

INTERMEDIATE

CASE REPORT: CLINICAL CASE

Atrial Thrombosis Caused by a Dislocated Left Atrial Appendage Closure Device After Mitral Valve Replacement

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ABSTRACT

A patient with a left atrial appendage occlusion device underwent mitral valve replacement. Later, the patient developed a left atrial thrombosis with thromboembolic myocardial infarction caused by a dislocation of the occlusion device. Exclusion of the device and non-device-based appendage occlusion may have prevented the patient from experiencing postoperative complications (**Level of Difficulty: Intermediate.**). (J Am Coll Cardiol Case Rep 2020;2:2327–30)

HISTORY OF PRESENTATION

A 68-year-old female patient was admitted with palpitations and dyspnea. Clinical examination indicated a non-ST-segment elevation myocardial infarction as well as tachyarrhythmia.

PAST MEDICAL HISTORY

Five years earlier, she had received a percutaneous left atrial appendage occlusion (LAAO) implant (WATCHMAN, Boston Scientific, Marlborough, Massachusetts) because of atrial fibrillation (AF) and warfarin intolerance. About 1 year earlier, she underwent percutaneous catheter intervention with stenting of the right coronary artery because of coronary artery disease. A current coronary angiography did not show any relevant coronary stenosis. Owing to severe functional mitral valve regurgitation, she underwent minimally invasive mitral valve surgery via right anterolateral mini-thoracotomy 4 months prior. A primary attempt for repair failed to restore the valve function, and a biological valve prosthesis (29-mm Perimount Magna Mitral Ease, Edwards Lifesciences, Irvine, California) was implanted with good result. Intraoperative and postoperative echocardiography showed a correct placement of the LAAO implant. After regular postoperative recovery, the patient was discharged. Because of warfarin intolerance, the patient was treated with enoxaparin for 3 months as postoperative anticoagulation for the bioprosthesis and with acetylsalicylic acid for the coronary artery disease.

LEARNING OBJECTIVES

- To identify cardiac thrombosis, its symptoms, origins, and complications.
- To understand the potential risks of LA appendage devices for patients undergoing cardiac surgery and to develop patient specific treatment alternatives.

DIFFERENTIAL DIAGNOSIS

Palpitation and dyspnea of the patient could have been caused by an exacerbation of the AF and a

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ABBREVIATIONS AND ACRONYMS

AF = atrial fibrillation

LA = left atrium

LAA = left atrial appendage

LAAO = left atrial appendage occlusion

recurrence of the mitral valve pathology. Troponin indicated an acute myocardial infarction.

INVESTIGATIONS

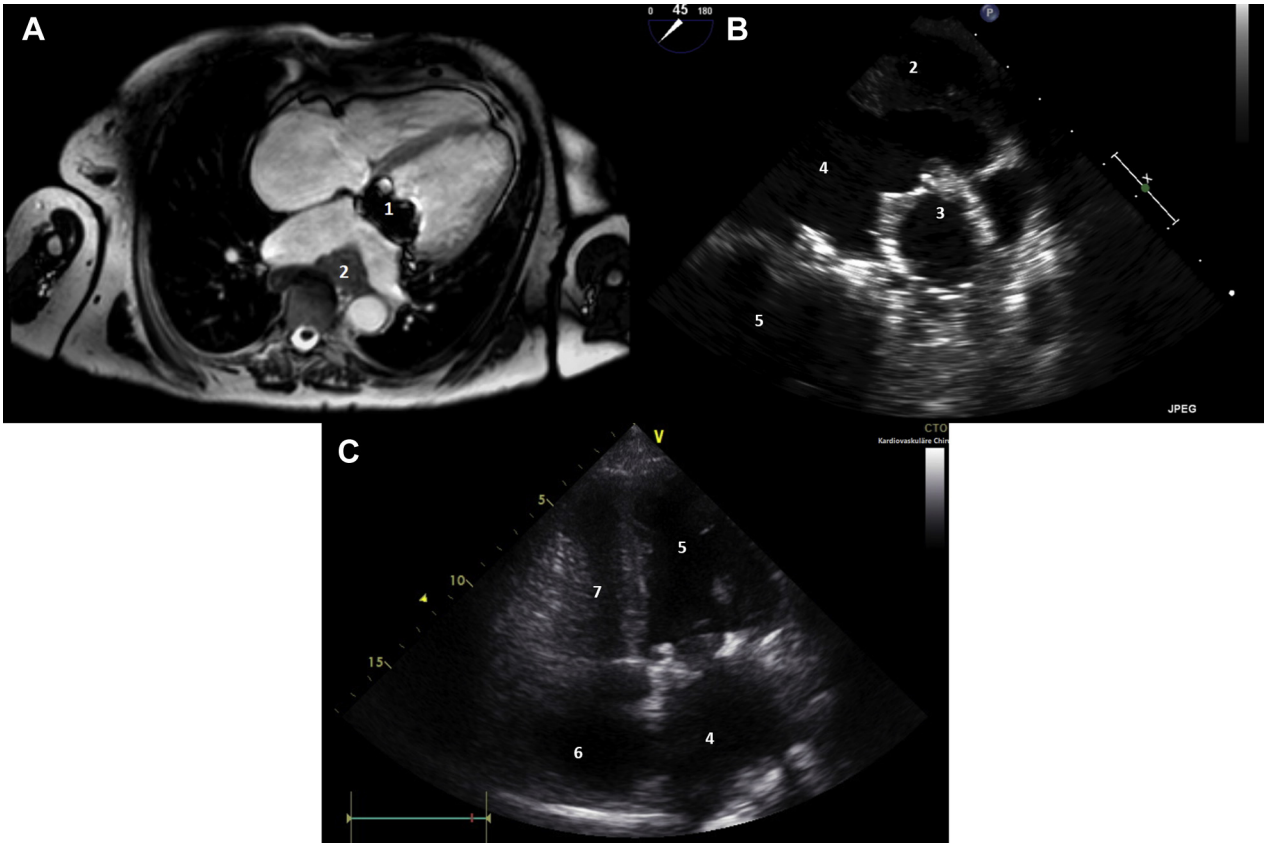
Coronary catheterization showed no relevant stenosis or acute embolism of the coronary arteries and a cardiac magnetic resonance was performed. Cardiac magnetic resonance displayed posterior wall infarction, most likely caused by previous transient thromboembolism. In addition, a new onset impairment of left ventricular function was found. The LAAO device was dislocated with a large adherent thrombus in the LA (**Figures 1A and 1B**).

MANAGEMENT

The patient was immediately heparinized, and rhythm control for AF was achieved by beta-blockage. Afterward, the patient was transferred for surgical removal of the LAAO implant as well as LA thrombectomy.

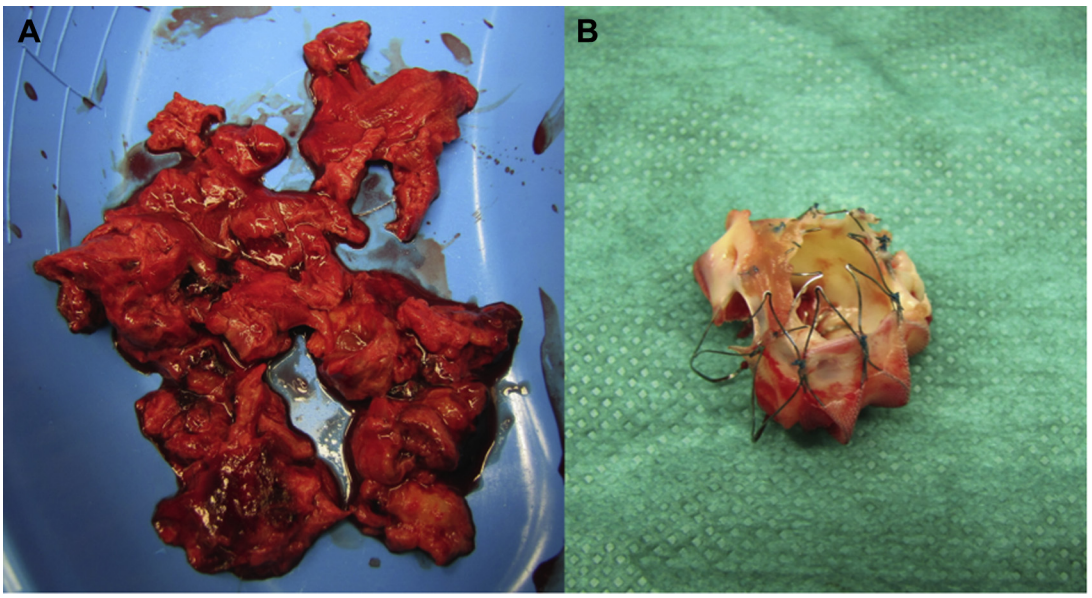
The second operation was performed with median sternotomy. The LA was exposed under cardioplegic arrest. An extensive thrombosis of more than 50% of the LA cavity was found, with different stages of thrombogenesis indicating a still ongoing process. The thrombotic mass was carefully removed until the atrium was cleared (**Figure 2**). LAAO device was explanted and the LAA manually amputated at the level of its base, using a double-layered suture line in order to prevent rethrombosis. The mitral valve prosthesis and left ventricle were examined, showing no signs of pathologies. Postoperatively, the patient was transferred to the intensive care unit, on the third postoperative day further to the peripheral ward, and was discharged four days later. In accordance with the Department of Hemostaseology and

FIGURE 1 Cardiac Imaging of the Patient



(A) Cardiac magnetic resonance imaging of the patient prior to the second surgery. **(B)** Transesophageal echocardiography of the patient prior to the second surgery. **(C)** Transthoracic echocardiography of the patient prior to the second hospital discharge. 1 = mitral valve prosthesis; 2 = left atrial thrombosis; 3 = left atrial appendage closure device; 4 = left atrium; 5 = left ventricle; 6 = right atrium; 7 = right ventricle.

FIGURE 2 **Intraoperative Findings**



Thrombotic mass filled more than 50% of the whole left atrium and was attached to the atrial wall as well as to the left atrial appendage closure device. **(A)** Thrombus; **(B)** left atrial appendage closure device.

Transfusion Medicine of our university hospital, the patient was treated with rivaroxaban as well as clopidogrel (for 12 months, followed by lifetime treatment with acetylsalicylic acid).

DISCUSSION

AF is the most common form of arrhythmia and increases the risk for embolic strokes by more than 5 times (1). The LAA is known as the primary origin of thrombogenesis in nonvalvular AF patients (2). First-line therapy with pharmaceutical blood thinners increases the risk of bleeding and can cause allergies and intolerance (3). Therefore, closure of the LAA is recommended for patients with AF and increased risk for bleeding (4). Percutaneous LAAO is a common procedure for AF patients to decrease the risk of thrombogenesis and stroke (5). Although seldom reported, failed endothelialization or dislocation of the implant may increase the risk of device-related thrombosis (5). However, 5 years after the device implantation, failed endothelialization is not a plausible explanation in this particular patient. Abnormal hemodynamic conditions in LAA (e.g., stasis in mitral stenosis or after prosthetic valve replacement) may further contribute to thrombogenesis (6). In most cases, reporting of a dislocation of LAAO devices happens peri-interventionally and appears within the first months after the implantation (7,8), and often devices tend to migrate to the left ventricular outflow tract and the aorta (7,8). Most patients experience acute heart failure because of dysfunction of the mitral or aortic valve as well as device thrombosis (7,8).

Although a direct correlation between mitral valve replacement and the dislocation of the device cannot be confirmed, without a doubt these events are most likely interrelated, as it appeared to be in the typical time period (7,8). Though a postoperative echocardiographic examination after the first operation did not reveal any movement or mechanical destabilization of the LAAO, intraoperative manipulation appears possible. Surgical intervention may have destabilized the fibrotic tissue fixing the LAAO device and provoked a slow dislocation during the following months. In addition, mitral valve surgery can provoke reverse remodeling of the LA, leading to smaller atrial volumes and altered atrial topography (9). This might

have further affected the positioning of the LAAO in a multifactorial process. Moreover, mitral valve replacement decreases flow velocities in the LAA and can also increase the risk of thrombogenesis in AF patients (6). After dislocation of the LAAO device, this effect may have amplified the risk for LAA thrombosis. As implanted LAAO devices could fail, in patients undergoing cardioplegic cardiac surgery, we recommend an advanced intraoperative examination of the implanted LAAO in order to decide whether it is possible to explant the device, regardless of the potential risk of device dislocation by the performed procedure. Surgical amputation of the LAA as well as clip exclusion are safe and feasible procedures to improve the outcome of AF patients by reducing the risk for stroke and other thromboembolic events, with minimal general risk of device-related thrombosis (5,10).

FOLLOW-UP

Echocardiography showed no recurrence of thrombosis as well as good valve prosthesis function at hospital discharge (**Figure 1C**) as well as 3 months later. No neurological symptoms were observed at any time.

CONCLUSIONS

In this special case, explantation of the LAAO device and non-device-based LAA exclusion during mitral valve surgery may have prevented the patient from myocardial infarction, development of ischemic cardiomyopathy, and exposure to the risk of a second cardiac operation. However, current literature provides little evidence for decision finding in such a situation. As the number of patients with LAAO devices steadily increases, a comparable scenario is likely to be encountered in a higher frequency. Further studies are needed to address the gap in knowledge regarding the optimal LAAO strategy in patients with valvular heart diseases.

AUTHOR DISCLOSURES

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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