Giant Thrombus Formation Immediately After Mitral Valvuloplasty

Yoshihiro Aizawa, MD, Toshiko Nakai, MD, Takafumi Kurosawa, MD, Yuki Saito, MD, Koyuru Monno, MD, Takumi Hatta, MD, Takafumi Hiro, MD, Munehito Arimoto, MD, Shunji Osaka, MD, Hiroaki Hata, MD, Motomi Shiono, MD, and Atsushi Hirayama, MD

Summary

Patients with atrial fibrillation (AF) are at risk of cardioembolism. Atrial thrombus formation associated with AF typically occurs in the left atrial appendage (LAA); therefore, transesophageal echocardiography (TEE) is important for detection of such a thrombus and measurement of LAA flow velocity. LAA closure is routinely performed during mitral valve surgery in patients with AF to prevent cardiogenic stroke. We report the case of a 65-year-old woman with severe mitral regurgitation (MR) and AF in whom a giant thrombus formed almost immediately after mitral and tricuspid valvuloplasty and concurrent LAA resection. No atrial thrombus or spontaneous echo contrast (SEC) was detected by TEE before the surgery. However, a giant intramural thrombus was detected in the left atrium 7 days after surgery. It was thought that the atrial dysfunction as well as the change in morphology of the left atrium resulting from the severe MR complicated by AF and congestive heart failure produced a thrombotic substrate. This case suggests that careful surveillance for thrombus formation and careful maintenance of anticoagulation therapy are needed throughout the perioperative period even if no SEC or thrombus is detected before surgery. (Int Heart J 2015; 56: 000-000)

Key words: Atrial thrombus, Mitral regurgitation, Mitral valve surgery

A 65-year-old woman was referred to our hospital because of severe MR and dyspnea on effort. Her medical history included chronic AF, hyperuricemia, and a previous myocardial infarction. A systolic murmur was heard at the apex, and coarse crackles were heard in all lung fields. Chest radiography revealed an increased cardiothoracic ratio (71%) with enlarged atria. Electrocardiography showed AF of 75 bpm. Transthoracic echocardiography revealed enlargement of the left atrium (74 × 67 mm), severe MR, and tricuspid regurgitation (Figure 1). Left ventricular (LV) systolic function was normal with an LV ejection fraction (EF) of 72%, and LV wall thickness was within normal range. There was no chorda tendinea tear or mitral valve prolapse; however, the anterior and posterior leaflets of the mitral valve showed myxomatous degeneration. Despite the enlarged atrium and chronic AF, TEE showed no thrombus or spontaneous echo contrast (SEC) in any cardiac chamber. It did show, however, massive mitral regurgitant flow reaching the roof of the left atrium (Figure 2). LAA peak emptying velocity (LAAEV) was 28 cm/sec, and LAA peak filling velocity (LAAFV) was 51 cm/sec. The patient was treated with warfarin (warfarin sodium), ubidecarenon (Neuquinon), digoxin (Digosin), and ferrous fumarate (Ferrum). Her plasma NT-pro BNP level was 3691 pg/mL. Coronary angiography showed no coronary artery stenosis, and left ventriculography showed normal motion; however, the MR was severe and classified as Sellers grade 2. The severe MR was considered the main cause of the heart failure, and mitral valvuloplasty (MVP) was indicated.

MVP, tricuspid valvuloplasty, and LAA resection were performed concurrently as an open heart procedure and were completed without complications. The postoperative course went well. The patient was extubated on postoperative day (POD) 1 and started rehabilitation on POD 5. Warfarin, which had been substituted with heparin (drip infusion) for 3 days before the surgery, was restarted on POD 4 once the absence of bleeding was confirmed. One week after the surgery, transthoracic echocardiography was performed to assess the re-
paired valve. A giant intramural thrombus (30 × 17 mm) was seen in the left atrium (Figure 3). The warfarin dosage was increased to maintain the prothrombin time-international normalized ratio (PT-INR) within the therapeutic range, and 20 days later, TEE showed that the thrombus had disappeared. Fortunately, cerebral infarction did not occur and the patient was discharged on a regimen of warfarin (3.5 mg), aspirin (100 mg), furosemide (20 mg), eplerenone (50 mg), and rabeprazole (10 mg). A timeline of the clinical course is shown in Figure 4.

**Discussion**

We encountered thrombus formation almost immediately after mitral valve surgery in a patient with MR complicated with AF and heart failure. Because patients with AF are at risk of cardioembolism, the CHADS2 or CHA2DS2-VASc score is widely used for stratifying the risk of stroke. The risk was evaluated in our patient, who had a CHADS2 score of 3 and a CHA2DS2-VASc of 5, indicating the need for anticoagulation therapy. TEE performed before the mitral valve surgery detected no thrombus or SEC; however, the LAAEV was low (28 cm/second) and put the patient at risk for thrombus formation. An LAAEV of < 40 cm/sec is considered a risk factor for thrombus formation as well as an indication of LAA dysfunction. LAAEV was relatively high (51 cm/second), but it could have been overestimated due to the massive regurgitant flow.

The LAA is a well-recognized site of thrombus formation in patients with MR, and it is logical to assume that the risk of thrombus formation decreases with LAA resection performed at the time of MVP. Despite the LAA resection in our patient, a huge thrombus was newly detected by transthoracic echocardiography 1 week after the surgery. The anticoagulation therapy, which was used to control postoperative bleeding, was not sufficiently effective at an INR of 1.58. After MVP, blood flow was slow enough to allow thrombus formation. In addition, the left atrial dysfunction was temporarily exacerbated by the atrial incision. The three abnormalities comprising Virchow’s triad, which contributes to thrombus formation, were aggravated after the surgery. Previous studies have shown that severe MR is protective against thrombus formation because it reduces LA blood stasis. The MR jet agitates blood stasis in the LA cavity, reducing SEC and increasing LA blood flow, and thus prevents thrombus formation. Miyasaka, et al reported an as-
association between mild MR and thromboembolic events in patients with nonrheumatic AF. They compared the incidence of thromboembolic events between patients with no MR, grade 1 MR, and ≥ grade 2 MR and found the incidence to be significantly higher among patients with grade 1 MR than among those with ≥ grade 2 MR and those with no MR, also suggesting that the higher grade mitral regurgitant flow prevents the blood stasis. Tse, et al showed a correlation between the severity of MR and elevated platelet activation. They speculated that the platelet activation associated with MR is a result of the hemodynamic irregularity (turbulent flow in the left atrium) caused by the regurgitant jet in the presence of an abnormal valvular surface, independent of the underlying etiology. The degree of platelet activation was positively correlated with the severity of MR and was independent of the underlying etiology of mitral valve disease, age, and left atrial size. Watanabe, et al reported association between LAA flow velocity, LVEF, right ventricular systolic pressure, LV mass index, and LA dimension and LA thrombus formation under warfarin therapy.

Our patient had an enlarged left atrium, suggesting a high degree of platelet activation and a tendency toward thrombus formation. Moreover, it is possible that atrial endothelial dysfunction existed as a result of both the MR and long-lasting AF. The endothelial dysfunction would have provided a substrate for thrombus formation. Fortunately, in our case, the thrombus resolved with a boost in the anticoagulation therapy and did not cause a stroke.

Massive regurgitant flow might prevent thrombus formation, but this protection will be lost once the MR is corrected. Therefore, it is very important to exercise constant vigilance through echocardiographic assessment and to maintain effective anticoagulation therapy especially during the perioperative period.

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