Balloon Mitral Commissurotomy for Mitral Stenosis After Resolution of a Large Mobile Left Atrial Thrombus by 2-year Warfarin Treatment

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We report on a patient who was referred for percutaneous transvenous mitral commissurotomy for symptomatic mitral stenosis. However, transthoracic and transesophageal echocardiography both revealed a large mobile left atrial thrombus, occupying almost half of the left atrial body. Because the patient strongly declined surgical mitral treatment, he was administered warfarin for 2 years. Complete resolution of left atrial thrombus was confirmed by follow-up transesophageal echocardiography. Successful percutaneous transvenous mitral commissurotomy was undertaken without complications. [*J Chin Med Assoc* 2009;72(12):646–649]

Key Words: heart atrium, mitral valve stenosis, thrombosis, warfarin

Introduction

Percutaneous transvenous mitral commissurotomy (PTMC) is an effective and less invasive treatment than surgical treatments for symptomatic mitral stenosis. 1-3 Left atrial body thrombus is considered to be an absolute contraindication to the procedure due to embolic risks. It has also been reported that intensive warfarin therapy may lead to dissolution of left atrial thrombus, after which successful PTMC can be performed.^{4,5} In this report, we describe a patient with rheumatic mitral stenosis who was referred for PTMC. However, he was found to have a large, floating, left atrial thrombus, occupying almost half of the left atrial body as revealed by transthoracic echocardiography and transesophageal echocardiography (TEE). The patient strongly declined surgical mitral commissurotomy and left atrial thrombectomy treatment. He was then referred back to the cardiologist at the local hospital. After 2 years of warfarin therapy, complete resolution of the left atrial thrombus was confirmed by follow-up TEE. Successful PTMC was undertaken without complications.

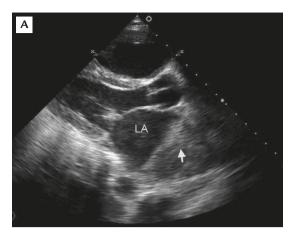
Case Report

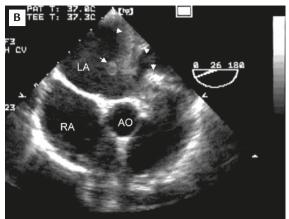
A 41-year-old man with known rheumatic mitral stenosis was referred to our institution for PTMC because of increasing dyspnea on exertion and orthopnea. He had only recently been discharged from a local hospital for pulmonary edema. Physical examination revealed mild pulmonary rales at the bases, an opening snap, and a diastolic rumbling murmur at the cardiac apex. His electrocardiogram showed atrial fibrillation at 70 beats/min. Transthoracic echocardiography was performed and disclosed severe mitral stenosis with mild mitral regurgitation and a large left atrial thrombus, occupying almost half of the left atrial body (Figure 1A). The calculated mitral valve area was 0.6 cm² with a mean diastolic transmitral pressure gradient of 13 mmHg. TEE demonstrated a large mobile left atrial thrombus, occupying almost half of the left atrial body, with parts of the thrombus appearing fresh, protruding and mobile (Figure 1B). There was severe spontaneous echo contrast in the left atrial body and a left atrial appendage thrombus was present as well (Figure 1B). The mitral leaflets were moderately thickened



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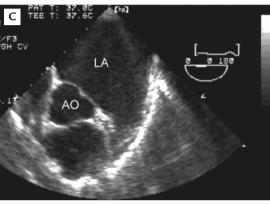


Figure 1. (A) Transthoracic echocardiography (parasternal long-axis view) shows a large echogenic thrombus (arrow) in the left atrium, occupying almost half of the left atrial body. (B) Transesophageal echocardiography shows a large left atrial thrombus (arrowheads), occupying almost half of the left atrial body. Parts of the thrombus look mobile and fresh (arrow). (C) After 2 years of warfarin treatment, complete resolution of the thrombus was confirmed by transesophageal echocardiography. LA=left atrium; RA=right atrium; AO=aortic orifice.

and had restricted mobility, but there was no leaflet calcification or distortion of the subvalvular structure. The total mitral score was 5. Because the patient strongly declined surgical mitral commissurotomy and left atrial thrombectomy treatment, he was then referred back to the cardiologist at a local hospital.

The patient was placed on warfarin therapy with a dosage ranging between 1.25 mg and 3.75 mg per day to maintain an international normalized ratio between 1.3 and 4.0 for the following 2 years. In the meantime, he was twice admitted for pulmonary edema at the local hospital. His cardiac rhythm was intermittently sinus with episodes of atrial fibrillation. He was transferred back to our institution for recurrent pulmonary edema and for a reassessment of his mitral disease status 2 years after his first referral. A repeat TEE was performed with no evidence of the left atrial thrombus (Figure 1C). The cardiac rhythm was sinus, and he was placed on amiodarone to prevent atrial fibrillation. He underwent PTMC with a 26-mm Inoue balloon up to 23-mm inflation (Figure 2A). The mitral valve area increased from $0.6\,\mathrm{cm}^2$ to $1.85\,\mathrm{cm}^2$. The mean transmitral pressure gradient decreased from 16 to 3 mmHg (Figure 2B) and pulmonary artery systolic pressure declined from 91 to 52 mmHg (Figure 2C). At subsequent follow-ups, the patient was symptomfree and there was no evidence of emboli.

Discussion

Stroke is one of the most serious complications of PTMC and is likely secondary to embolism from a left atrial thrombus. The presence of left atrial thrombi is a common finding in rheumatic mitral valve disease, and the incidences reported in previous studies ranged from 7.4% to 32%.^{4,5} Left atrial thrombus is considered to be an absolute contraindication to the procedure due to the risk of embolism.

Warfarin depletes the reduced form of vitamin K, which is an important cofactor for post-translation modification (carboxylation) of coagulation factors II, VII, IX and X, thus inhibiting the coagulation pathway. 6–8 If coagulation is inhibited by warfarin and no more thrombus is formed, gradual dissolution of the thrombus by tissue-type plasminogen activator-related fibrinolysis can be expected.

Some reports have shown that left atrial thrombi may resolve in some patients after anticoagulant therapy, and PTMC can be performed subsequently.^{4,5,9}

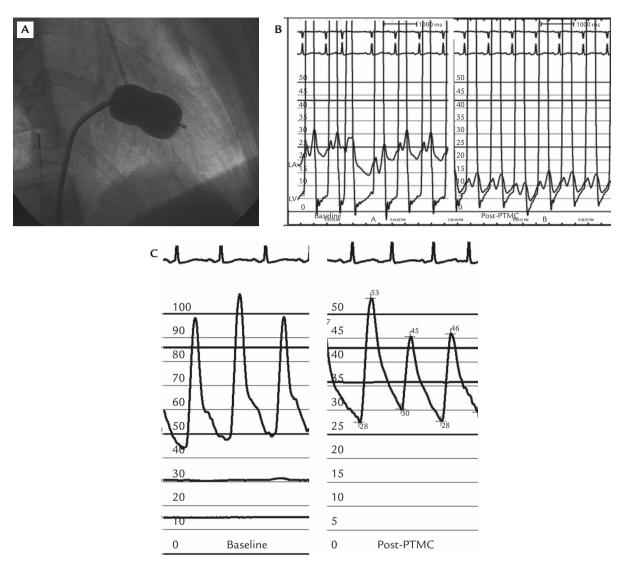


Figure 2. (A) An Inoue balloon was inflated during percutaneous transvenous mitral commissurotomy. (B) Mean transmitral pressure gradient decreased from 16 to 3 mmHg after percutaneous transvenous mitral commissurotomy (left panel: baseline; right panel: post-percutaneous transvenous mitral commissurotomy [PTMC]). (C) Pulmonary artery systolic pressure declined from 91 to 52 mmHg after PTMC (left panel: baseline, top scale bar 100 mmHg; right panel: post-PTMC, top scale bar 50 mmHg).

A report by Silaruks et al showed that of 219 PTMC candidates with left atrial thrombus, complete resolution of the thrombus was demonstrated in 53 patients (24.2%) after 6 months of warfarin treatment.⁴ In another prospective study, Lin et al showed that the resolution of left atrial thrombi was observed in 8 of 14 patients (57.2%) after warfarin therapy over a period of 1–6 months.⁵ Only 1 patient obtained benefit from longer warfarin therapy (>6 months), while in the other 7 patients, thrombus resolution occurred within 3 months.⁵ Therefore, it is not wise to delay PTMC for an extended period to wait for complete thrombus resolution, because stroke and refractory heart failure might occur during the interval. In our case, because

the patient strongly declined surgical treatment, he was placed on warfarin treatment for 2 years and monitored periodically at a local hospital. Complete resolution of left atrial thrombus was proved by TEE 2 years later.

Lin et al reported that the resolution of left atrial thrombi was more frequently observed in patients either with a smaller left atrial dimension or with their thrombi located inside their left atrial appendage.⁵ An analysis by Silaruks et al⁴ showed that the significant predictors of thrombus resolution were a New York Heart Association class of 2 or less, a left atrial appendage thrombus size of 1.6 cm² or less, a left atrial spontaneous echocardiographic contrast grade of 1 or

less, and an international normalized ratio of at least 2.5. Patients with all of these predictors had a 94.4% chance of complete thrombus resolution. Our case had several unfavorable characteristics for predicting the thrombus resolution, including a large baseline left atrial size of 5.9 cm, a large thrombus occupying almost half of the left atrial body, a New York Heart Association class 3–4 symptom, and an erratic international normalized ratio range.

In conclusion, left atrial thrombi can be resolved completely after optimal anticoagulation in a considerable proportion of patients with rheumatic mitral stenosis. Subsequent PTMC can be performed safely without an increased risk of systemic embolization. Left atrial thrombus should not be an absolute contraindication for PTMC. However, we believe surgery should be the first-line therapy for patients with large mobile left atrial thrombus. Furthermore, we do not recommend such a long waiting period because there is a potential risk of cardiac emboli during the follow-up interval.

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