Acute thrombus formation on the delivery sheath during left atrial appendage occlusion: case reports with placement of cerebral protection devices

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Abstract

Acute thrombus formation on the delivery sheath is rare condition during percutaneous left atrial appendage occlusion. We presented two cases that TEE showed a floating thrombus attached to the tip of delivery sheath during the procedure. Cerebral embolic protection devices were used to prevent neurological events after thrombus was detected. The

Introduction

Left atrial appendage occlusion (LAAO) is an alternative to oral anticoagulants for prevention of stroke in patients with atrial fibrillation who are not optimal candidates for long-term anticoagulation. Acute thrombus formation on the delivery sheath during left atrial appendage occlusion is rare condition. Periprocedural stroke during LAAO is extremely unacceptable. We described the use of placement of cerebral embolic protection devices to prevent neurological events when acute thrombus formation during the implantation of LACbes (Shanghai PushMed, Shanghai, China).

Case presentation

Case 1

A 59-year-old male with hypertension, diabetes, atrial fibrillation, prior twice stroke history and prior ICD implantation due to cardiac arrest presented for selective percutaneous left atrial appendage occlusion (LAAO). The patient received catheter atrial flutter ablation therapy 6 months ago, but electrocardiogram and 24-h holter showed atrial fibrillation at present (average heart rate 86bpm). The patient's CHA2DS2-VASc score was 4 and his HASBLED score was 3. He was unwilling to take long term oral anticoagulants because of high risk fo bleeding. Before the procedure, the patient's coagulation function test was normal.

An 6F sheath was placed in the right femoral vein, intravenous heparin 3,000U was administered. After TEE-guided trans-septal puncture, heparin 4,500U was given. Then a 12F delivery sheath and pigtail catheter were positioned in the LAA. Activated clotting time (ACT) measured 254s. An angiogram and TEE was performed to assess the appendage morphology. TEE revealed a cactus shaped LAA free of thrombus. LAA emptying velocity was 40 cm/sec. Mild spontaneous echocardiographic contrast was in the LAA. The patient then underwent successfully implantation of a 22-mm LACbes device with no para-device leak. After the occlusion device release, TEE showed a 20mm length floating thrombus attached to the delivery sheath tip (Figure 1A, 1B and Video 1). ACT was measured 112s and heparin 3,000U was added immediately. We tried to suck the thrombus through long sheath but failed. ACT measured 124s after 5 min. Heparin was added in divided without any effect on the thrombus resolution. Cerebral embolic protection devices (ev3 SpiderFX) were implanted in the bilateral internal carotid arteries and urokinase 500,000U was administered

to achieve ACT >250s until TEE showed thrombus dissolved. After thrombolytic therapy, cerebral and renal artery angiogram were conducted and showed no embolism sign. Thrombus debris was detected in the filter after removal (Figure 2). Rivaroxaban and aspirin were initiated, and the patient was closely monitored post-operation. The neurological function was not impaired and cerebral CT showed old infarcts 1 day after procedure.

Follow-up

No neurological events occurred during follow up. 6 and 12 months after the procedure, TEE revealed a well-seated 22-mm LACbes device with no residual flow around the device and no device related thrombus (DRT).

Case 2

A 59-year-old male with atrial fibrillation and heart failure presented for hospital. The patient's CHA2DS2-VASc score was 2 and his HASBLED score was 1. He chose LAAO because of poor compliance with long term oral anticoagulants.

An the beginning of the procedure, TEE revealed a chicken wing shaped LAA free of thrombus. LAA emptying velocity was 28.9 cm/sec and LAA EF 34%, no spontaneous echocardiographic contrast in the LAA. Intravenous heparin 3000U was given after right femoral vein puncture. After TEE-guided trans-septal puncture, a 12F delivery sheath and pigtail catheter were delivered to the left atrial, intravenous heparin 4100U were administered. At this time, TEE suddenly showed a mobile thrombus whose proximal part was connected to the 12F delivery sheath (Figure 3A, 3B and Video 2). Immediately, the ACT measured 161s. Heparin 2000U was added and ACT measured 168s after 5 min. Heparin was added again and we tried to suck the thrombus through the long sheath, thrombus still attached to the outside of the sheath. Cerebral embolic protection devices (ev3 SpiderFX) were implanted in the bilateral internal carotid arteries, TEE showed the amount of thrombus gradually decreased until disappeared. When TEE showed thrombus dissolution, we rechecked ACT which was 260s. Then, a 24-mm LACbes device was implanted with no para-device leak and no thrombus on the device surface. The total heparin dosage used was 18000U. None was showed in the filters after withdraw cerebral protection devices. Rivaroxaban and aspirin were initiated, and the patient was closely monitored post-operation. The neurological function was not impaired after the procedure.

Follow-up

No neurological events occurred during follow up. TEE revealed a well-seated LACbes device with no residual flow around the device and no DRT at 1 and 3-month follow up.

Discussion

Left atrial appendage occlusion is an alternative to oral anticoagulants for prevention of stroke in patients with atrial fibrillation who are not optimal candidates for long-term anticoagulation[1]. Device related thrombus (DRT) is considered an important issue and associated with increased risk of ischemic events after LAAO[2-4]. Current published report suggests that DRT occurs about 3.7% of patients between 3 and 6 months post-procedure of LAAO[3]. The mechanism underlying DRT is incompletely understood. Some known factors such as hypercoagulability disorder, pericardial effusion, renal insufficiency, implantation depth >10 mm and non-paroxysmal atrial fibrillation are risk predictors of DRT following LAAO[5].

Intraprocedural thrombosis during LAAO has been rarely reported. A case report firstly described the acute thrombus formation on the surface of the occlusion device immediately after release[6]. The patient's recent COVID-19 infection may contribute to acute thrombus formation. Thrombus formation on the delivery sheath during LAAO has been reported[7,8]. To prevent thrombus migration, thrombus were retrieved and sucked by sheath without cerebral embolic protection device. However, suction of thrombus within the left atrial by sheath may have high risk of embolism. Prevention of periprocedural complications especially stroke is a major issue during LAAO.

In our case-series, we followed the standard LAAO procedure and heparin was used at a dosage 100 U/kg. We found that: 1) floating thrombus formed on the tip of the delivery sheath during LAAO; 2) ACT value was <200s when acute clotting formed and which only achieved acceptable low limit value after repeatedly giving heparin even thrombolytic therapy; 3) we try to suck the thrombus but failed for both cases; 4) it is feasible and safe to place cerebral embolic protection devices to prevent neurological events; 5) in the follow-up, there are no DRT and new-onset stroke.

Periprocedural stroke during LAAO which is a preventive therapy for stroke in patients with atrial fibrillation, is extremely unacceptable. Although heparin was generally administered to prevent thrombosis in the procedure, heparin-induced anticoagulation has a high interindividual variability either in terms of dosage or time duration requiring a frequent ACT monitoring. However, optimal ACT cut-off value is currently unknown during LAAO. Previous mentioned reports showed ACT >250s when thrombus formed on the deliver sheath during LAAO procedure. Preoperative coagulation tests were within normal reference values although our two patients take rivaroxaban. And persistent low ACT value in the procedure indicated that heparin resistance and stasis of left atrial dilation predispose to coagulopathy and consequently to thrombus formation. Unfortunately, antithrombin III in blood samples were not tested. A study demonstrate that lower ACT level was significantly associated with the development of procedure-related silent cerebral embolism[9]. Underlying genetic susceptibility of heparin resistance induced lower ACT level and impaired left atrial endothelial function both would be more likely to activate coagulopathy. Further studies should be conducted to determine the optimal ACT level for LAAO procedure. The use of cerebral embolic protection devices during percutaneous LAAO was a feasible and safe therapeutic option for patients with LAA thrombus[10]. It is hard to suck when acute floating thrombus formed on the sheath after occlusion device released. Placement of cerebral embolic protection devices could be a rational option for neurological protection. Indeed, our two cases showed no neurological function impairment after the procedure. In addition, continuous drip of heparin saline to the delivery and guide system may be ensure the local heparin concentration around the instruments in the left atrium.

Conclusion

Our cases showed rare event that acute thrombus formation on the delivery sheath during LAAO. The need of anticoagulation and the frequency of ACT monitoring should be highlighted. Cerebral protection device may be a feasible management for neurological function protection.

Conflict of Interests

The authors report no financial relationships or conflicts of interest

regarding the content herein.

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Figure Legends

Figure 1. (A and B) 2D and 3D TEE showed a floating thrombus attached to the delivery sheath; 3D TEE view: yellow arrow indicate sheath and red arrow indicate thrombus.

Figure 2. thrombus debris was detected in a filter (right).

Figure 3 . (A and B) 2D and 3D TEE showed a floating thrombus attached to the delivery sheath; 3D TEE view: blue arrow indicate sheath and red arrow indicate thrombus.