Percutaneous Catheter Thrombus Aspiration of Right Renal Infarction Caused by Left Ventricular Thrombi due to Takotsubo Cardiomyopathy

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Summary

Takotsubo cardiomyopathy (TC) is a temporal dysfunction of the left ventricle (LV) due to psychological or physiological stress; however, it rarely causes LV thrombus. We report a case of a 49-year-old woman who developed LV thrombi due to TC despite anticoagulation therapy. The thrombi caused acute systemic infarction, with the most severe occlusion being in the right renal artery. The patient underwent percutaneous catheter aspiration thrombectomy of the right renal artery and her renal function recovered shortly after. The results of this case suggest that catheter aspiration thrombectomy is effective in the treatment of thromboembolism in TC.

Key words: Thromboembolic complications, Acute systemic infarction, Catheter aspiration thrombectomy

Takotsubo cardiomyopathy (TC) is a temporal dysfunction of the left ventricle (LV) due to psychological or physiological stress.1 The short-term prognosis of TC is considered as life-threatening as that of acute coronary syndrome in terms of acute heart failure, arrhythmia, or thromboembolic events.2 In the most frequent type of TC, the apical wall of the LV severely deteriorates. It may also cause thrombus formation in the LV. However, there are no guidelines for the treatment of LV thrombus in such cases. Moreover, there are only a few reports on LV thrombectomy or ventriculotomy in patients with TC.3 We aimed to introduce a treatment option for thromboembolism associated with TC through the present case. We herein report a case of TC with LV thrombi that resulted in right renal infarction despite sufficient anticoagulation therapy.

Case Report

A 49-year-old woman with a history of symptomatic seizure presented to our hospital with chest pain that had continued for more than 24 hours. Her symptom began when she was talking with her child’s schoolteacher, which was stressful for her. She had no personal or family history of cardiovascular disease, and she had never smoked. An electrocardiogram showed slight ST elevation in the II, III, and aVF leads and T-wave inversion in the II, III, aVF, and V3-6 leads (Figure 1A). Laboratory data revealed a creatinine level of 0.65 mg/dL, troponin T elevation at 0.181 ng/mL, creatine kinase of 141 U/L (within the normal range), brain natriuretic peptide elevation at 274.4 pg/mL, D-dimer elevation at 2.60 g/mL, and activated partial thromboplastin time (APTT) of 32.0 seconds. The activated protein C level was 94%, and activated protein S was 57%, both within the normal range. Coronary computed tomography (CT) showed no coronary artery stenosis (Figure 1B), and CT angiography showed 2 thrombi in the left ventricle (LV) (Figure 1C). Enhanced CT showed no evidence of pheochromocytoma. An echocardiogram revealed an akinetic area at the middle of the apex of the LV and 2 thrombi in the apex of the LV (Figure 2A-D). The LV ejection fraction was 47% with trivial mitral regurgitation. Based on electrocardiography, echocardiography, CT angiography, and enhanced CT results, we diagnosed her with TC and LV thrombi, which were presumably triggered by emotional stress. Her chest pain disappeared during admission. Heparin was initiated at 15,000 U/day (12.5 U/kg/hour), which was subsequently increased to 23,000 U/day (19 U/kg/hour) in order to maintain her APTT above 55 seconds, which was 1.5 times her initial APTT. On the sixth hospital day, the patient experienced sudden severe abdominal pain, and a CT scan revealed right renal infarction, partial left renal infarction, and partial splenic infarction (Figure 3A). The right renal artery was occluded (Figure 3B). An echocardiogram revealed no thrombus in the LV. To preserve her renal function, we performed percutaneous renal artery intervention and aspirated the thrombi (Figure 4A and B). Red blood clots were aspirated from the right renal artery (Figure 4C). On the ninth hospital day, a follow-up CT scan revealed improved contrast in her right kidney, which was better than that of the left kidney and spleen (Figure...
Figure 1. A: Electrocardiogram at admission to our hospital showing slight ST elevation in II, III, and aVF leads and T-wave depression in II, III, aVF, and V3-6 leads. B: Coronary computed tomography (CT) angiography showing no stenosis in either branch. C: CT angiography showing 2 thrombi in the left ventricle (arrowheads).

Figure 2. A: Two thrombi in the left ventricle (arrowheads). B: Left ventricle on the apical four-chamber view in diastolic phase. C: Left ventricle in systolic phase. D: Two thrombi at the apex of the left ventricle.

4D). Her serum creatinine level increased to 0.99 mg/dL. She continued receiving warfarin 1.5 mg for 3 months at an international normalized ratio of prothrombin time (PT-INR) level of 1.5-2.0 until her LV contraction fully recovered. Her D-dimer level was 9.10 μg/mL at discharge, which decreased to 1.13 μg/mL after 3 months of treatment. Her serum creatinine level recovered to 0.75 mg/dL. The activated protein S level had elevated to 76%, within the normal range, at the end of our follow-up. No recurrence of chest pain or dyspnea was noted. The patient provided informed consent for the presentation of this case.

Discussion

In this case, acute renal infarction caused by LV thrombi due to TC was treated by percutaneous catheter aspiration thrombectomy; anticoagulation therapy was not able to prevent thromboembolism. Patients with TC show transient LV dysfunction typically presenting as apical ballooning, new electrocardiogram abnormalities, and cardiac biomarker elevation. Emotional or physical stress may cause TC. LV contraction deteriorates temporarily and recovers without any medication. However, TC may be life-threatening when comorbidities exist. In an interna-
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Figure 3. A: Enhanced computed tomography (CT) scan before thrombus aspiration showing both renal infarction and splenic infarction. B: CT angiography showing occlusion of the right renal artery.

Figure 4. A: Angiography of the right renal artery before thrombus aspiration. B: Angiography of the right renal artery right after thrombus aspiration. C: Thrombi aspirated from the right renal artery. D: Enhanced computed tomography scan after thrombus aspiration. The enhancement of the right kidney had improved.

In our patient, the LV thrombi formed within 24 hours of the assumed onset of TC. In our patient, the thrombus was mobile and had a
high risk of thrombosis; moreover, we were most concerned about massive cerebral infarction. If there is any thrombus left in the LV when cerebral infarction occurs, thrombolytic therapy may release the thrombus from the LV and cause infarction in another organ; therefore, we preferred cerebral thrombectomy over thrombolytic therapy. We consulted neurosurgeons and prepared for cerebral thrombectomy. The thrombus in our patient moved to the renal artery but did not cause any lethal cerebrovascular complication. This case shows that anticoagulation therapy does not necessarily prevent thrombosis.

In a previous report, anticoagulation therapy mainly with heparin or warfarin was used for LV thrombi.8 If a thrombus appears with LV aneurysm after myocardial infarction, surgical thrombectomy with LV-plasty might be considered. There are only a few TC cases that underwent LV-plasty or thrombectomy in previous reports.13-15 Zaikokuji, et al.13 reported that surgical thrombectomy should be considered when the thrombus is mobile. If a thrombus forms due to atrial fibrillation and cannot be treated by anticoagulation, left atrial appendectomy can be considered.14 Long-term outcomes may be worse in ventricular operations than in atrial operations, but there is no known report of this. In contrast, it has been reported that heparin and warfarin failed to prevent thromboembolism.8 We considered surgical thrombectomy for preventing thrombosis, including cerebral infarction. In contrast to LV aneurysm after myocardial infarction, LV function after TC is expected to recover fully. Furthermore, the patient was young and had fewer surgical risk factors, such as coronary artery disease, symptomatic heart failure, or respiratory disease. We consulted with cardiac surgeons before making a decision. Although our patient had a relatively low risk in undergoing a heart surgery, we were concerned about the risks of surgical therapy, such as ischemic events due to the thrombi or arrhythmic events, which may affect the patient long after the surgery. Another reason we selected anticoagulation therapy was that the patient declined to undergo surgery.

Our patient experienced renal infarction during anticoagulation therapy for the LV thrombi. Percutaneous thrombus aspiration was selected for acute renal infarction in some reports.16,17 In this case, at the beginning of the treatment, since we expected thromboembolic complications, we consulted cardiologists and neurosurgeons to prepare for emergent percutaneous intervention. Thereafter, the thrombi were aspirated from the right renal artery, which was more severely occluded than the left renal artery. As a result, the right kidney achieved better reperfusion than the left kidney. This suggests that thrombus aspiration therapy is more effective in preserving renal function than conventional anticoagulation therapy alone. Thrombolytic therapy is relatively contraindicated during anticoagulation therapy with warfarin; thus, thrombus aspiration therapy is easier to initiate in such cases. Combination therapy with thrombus aspiration and anticoagulation allowed recovery of her renal function. Based on our discussion with cardiac surgeons, we chose not to perform surgical LV thrombectomy, but this might have been the only way to avoid a systemic thrombus infarction. Percutaneous catheter aspiration thrombectomy is only effective in the main vessels, which have a diameter that is large enough for the approach.

### Conclusion

We demonstrated a treatment strategy for a serious complication of TC. Anticoagulants may be insufficient for the prevention of thromboembolism, but considering the long-term risk of open-heart surgery, conventional anticoagulation therapy is currently the most appropriate treatment approach. We cannot completely prevent thromboembolic events, and prompt percutaneous catheter aspiration thrombectomy can be selected for reperfusion. However, our strategy has some limitations. We used a contrast agent, which might deteriorate renal function. Moreover, we could not prove the efficacy of this technique for other organs apart from the kidney. Considering these limitations, it is necessary to prepare for thromboembolic complications while treating patients with TC, and percutaneous catheter aspiration thrombectomy can be an effective strategy.

### Disclosure

**Conflicts of interest:** None.

### References