Left Ventricular Thrombus in Hypertrophic Cardiomyopathy

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The involvement of left ventricular (LV) diastolic dysfunction and LV hypertrophy in patients with hypertrophic cardiomyopathy (HCM) seems to be closely related to the occurrence of atrial fibrillation (Af) in this disease. Left atrial thrombus associated with Af may be related to the occurrence of thromboembolism, and thromboembolic events associated with Af seem to be more common in patients with HCM than in those with other diseases. However, we seldom encounter patients with HCM who have LV thrombus, especially when the patient is in sinus rhythm.

The Table shows the backgrounds of patients with HCM and LV thrombus reported to date (1-3), including two of my own patients. Figure A shows the brain and cardiac computed tomography findings in Case 4. As the cause of multiple infarctions (a), a thrombus at the LV apex (b: yellow arrow) was confirmed. Immediately after the confirmation, anticoagulation therapy was started. Figure B shows the B-mode echocardiograms of Case 5. Figure B-b is a magnified image of the square surrounded by a dotted line (a). A thin rim at the LV apex (a: white arrows) and a circular thrombus (b: yellow arrow) were confirmed. Of the five patients presented in the Table, Af was observed in only one who had thrombi in both the left atrium and left ventricle (2). These results indicate that the existence of Af is not an absolute condition for the formation of LV thrombus, but the existence of an apical aneurysm is. In 2008, Maron et al. (4) reported that, of 1,299 patients with HCM, 22 showed an apical aneurysm, and 2 had a thrombus in the aneurysm. In 2017, the same group reported that, of 1,940 patients with HCM, 93 showed an apical aneurysm, and 18 had a thrombus in the aneurysm (5). In addition, thromboembolic events were two-fold more common in patients with apical aneurysms than in non-apical aneurysm patients, although not to a significant degree. Thromboembolic events were observed in two patients (cases 2 and 4). Thus, anticoagulant therapy is indispensable for preventing thromboembolic events. LV apical thrombus in the Table was resolved in all cases.

The patients reported by Kaku (1) and Tezuka et al. (3) had mid-ventricular obstruction. Fighali et al. (6) suggested that mid-ventricular obstruction leads to myocardial dysfunction with dilatation of the apical chamber. We have reported many HCM patients with mid-ventricular obstruction who had no apical aneurysm (7). Thus, the existence of mid-

**Table.** Backgrounds of the HCM Patients with LV Thrombus.

| Case | Ref. No. | Age (years) | LV obstruction | LV thrombus | LA thrombus | Atrial fibrillation | Thromboembolic events | Apical aneurysm | Anti-coagulant | Thrombus resolution |
|------|----------|-------------|----------------|-------------|-------------|---------------------|-----------------------|----------------|--------------|------------------|
| 1    | (1)      | 59          | Mid            | +           | -           | -                   | -                     | +              | dabigatran    | +                |
| 2    | (2)      | 60          | -              | +           | +           | +                   | +                     | +              | apixaban      | +                |
| 3    | (3)      | 80          | Mid            | +           | -           | -                   | -                     | +              | heparin & warfarin | +                |
| 4    | my case  | 55          | -              | +           | -           | -                   | +                     | +              | heparin & warfarin | +                |
| 5    | my case  | 78          | -              | +           | -           | -                   | -                     | +              | apixaban      | +                |

HCM: hypertrophic cardiomyopathy, LV: left ventricular, Ref.: reference, No.: number, LA: left atrial, Mid: mid-ventricular obstruction

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ventricular obstruction is not an indicator of an apical aneurysm. However, mid-ventricular obstruction may promote the progression of myocardial damage in the LV apex to an apical aneurysm.

The exact mechanisms underlying the formation of LV thrombus are unknown. However, LV regions affected by myocardial necrosis have been found to have decreased active blood suction from the left atrium (8). In a ballooning LV apical area, it is conceivable that blood stagnation may develop, promoting clot formation. Thus, LV thrombus in an apical aneurysm may be caused by a decrease in active blood suction.

In a clinical setting, careful observation using echocardiography, computed tomography and magnetic resonance imaging may be required for the detection of LV thrombus in patients with HCM and a deteriorated LV systolic function, even in those without Af.

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