Massive Mobile Thrombus in the Left Ventricle Due to Löffler Endocarditis Complicated With Dilated Cardiomyopathy

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Figure 1. Echocardiogram showing an extremely massive mobile thrombus (25×55 mm) with several mural thrombi in the left ventricular apex. (A) Apical 4-chamber view; (B) apical 2-chamber view; (C) parasternal short-axis view at the mid-ventricular level; (D) parasternal short-axis view at the level of the apex.
26-year-old man was transferred to the emergency department of the National Cerebral Cardiovascular Center, Suita, Japan, due to acute decompensated heart failure with severely reduced left ventricular (LV) systolic function. His medical history was unremarkable. On admission, chest X-ray showed both moderate cardiomegaly and severe pulmonary congestion with bilateral moderate pleural effusion. Echocardiography indicated both LV dilatation (LV end-diastolic and end-systolic dimensions [LVDd and LVDs], 67 and 61 mm, respectively) and severely reduced systolic function (ejection fraction [EF], 11%). There was an extremely massive mobile thrombus (25×55 mm) with several mural thrombi in the LV apex (Figure 1; Movie S1). On blood test peripheral blood eosinophil count was elevated at 3,747/μl (34.7% of the total white blood cell count) and plasma brain natriuretic peptide (BNP) was 1,731 pg/ml. Cardiac catheterization showed (1) elevated pulmonary artery pressure of 43/23 mmHg (mean, 31 mmHg); (2) reduced cardiac index of 2.0 L · min⁻¹ · m⁻²; and (3) pulmonary capillary wedge pressure of 28 mmHg, but coronary angiography showed no significant stenosis. Endomyocardial biopsy of the right ventricular septum indicated eosinophilic endocarditis. Extensive eosinophilic infiltrates were seen mainly in the subendocardial interstitium and to a lesser extent in the myocardial tissue. Eosinophils and endocardium were positive for major basic protein on immunohistochemistry (Figure 2). Based on these findings, the patient was diagnosed as having Löeffler endocarditis with idiopathic dilated cardiomyopathy (DCM). He was started on 60 mg prednisolone (initial dose, 1 mg · kg⁻¹ · day⁻¹) and anticoagulation therapy with both warfarin potassium and heparin sodium, and the status of the thrombus was closely followed. Eosinophil count decreased, returning to normal on the 11th hospital day. The prednisolone dose was gradually tapered to 5 mg/week. Heart failure gradually improved after continuous drip infusion of milrinone followed by oral carvedilol and enalapril. Cardiac magnetic resonance imaging performed on the 25th hospital day showed both severely reduced LV systolic function (EF, 18%) and reduced right ventricular systolic function (EF, 23%). The thrombus in the LV apex also gradually regressed and became increasingly mobile, and disappeared on the 26th hospital day. We incidentally detected embolization in the patient’s right common femoral artery on computed tomography angiography, which fortunately regressed with continued anticoagulation therapy. Follow-up echocardiography on the 49th hospital day showed improved LV dimensions (LVDd and LVDs, 59 and 51 mm, respectively) and systolic function (EF, 22%), and mild tricuspid insufficiency with a maximum gradient of 16 mmHg. On the 57th hospital day, the plasma BNP decreased to 112 pg/ml and the patient was discharged on prednisolone 10 mg/day, carvedilol, enalapril and warfarin potassium. Follow-up echocardiography at 15 months showed further improvement, with LVDd/LVDs of 58/40 mm and LVEF of 43% with no thrombus.

In 1936, Löeffler reported 2 patients who had fibrosing endomyocarditis with marked peripheral eosinophilia, subsequently known as Löeffler endocarditis. Since then, there have been several case reports and a small series of patients with Löeffler endocarditis. This condition is typically marked by progressive subendocardial fibrosis with overlying mural thrombus formation leading to restrictive dysfunction of the LV. Thus, preserved LV systolic function and superimposed thrombus are common...
in patients with Löffler endocarditis. Indeed, in the present case we inferred that the extremely massive thrombus was due to both LV systolic dysfunction and Löffler endocarditis. To the best of our knowledge, this is the most massive mobile thrombus thus reported in the context of Löffler endocarditis.

We suggest that in patients with Löffler endocarditis, eosinophilia complicated with severely reduced LV systolic function may cause more massive thrombus formation in the LV compared with eosinophilia alone. These conditions may lead to the formation of not only superimposed mural thrombus but also mobile thrombus, especially in the LV apex. The majority of deaths in hypereosinophilic syndrome, including Löffler endocarditis, seem attributable to thromboembolic events. Therefore the combination of Löffler endocarditis and DCM is thought to confer a higher risk of thrombus formation and subsequent thromboembolism than Löffler syndrome alone, and intensive follow-up is critical.

Fortunately, in the present case the thrombus disappeared completely with aggressive anticoagulation therapy alone, and the only thromboembolic complication, peripheral thromboembolism, was not lethal. In patients with massive mobile thrombus due to Löffler endocarditis complicated with DCM, however, the indication for and efficacy of acute surgical thrombectomy in addition to aggressive anticoagulation therapy is not yet established and requires further investigation.

References

Supplementary Files
Supplementary File 1
Movie S1. Massive mobile thrombus in the left ventricle due to loffler endocarditis complicated with dilated cardiomyopathy.

Please find supplementary file(s):