□ Case Report □

Surgical removal of a left ventricular thrombus caused by acute myocarditis

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Left ventricular thrombus is mainly caused by anterior myocardial infarction or severe cardiac wall dysfunction of the apex, and is rarely caused by a complication of acute myocarditis. A 12-year-old female who developed symptoms of motor dysphasia and incomplete hemiparesis of the right side was admitted to the hospital. The brain MRI taken on the day of her admission showed acute cerebral infarction in the left basal ganglia and the frontoparietal lobe. The echocardiogram showed a movable thrombus, which was 19×28 mm sized and located in the apex of the left ventricle. So in order to prevent further thromboembolic event we performed open cardiac surgery via the atrium and removed the thrombus of the left ventricle. After the removal of the thrombus her symptoms improved and she was discharged from the hospital. Thrombus formation in acute viral myocarditis are considered to be related with endocardial injury and blood flow stasis. Treatment with anticoagulants in left ventricular thrombosis may not be effective and may even cause a major thromboembolism. When the thrombus is laminar and fixed, one should consider anticoagulant therapy. But if the thrombus is pedunculated and movable, which means that there are higher possibilities of major embolism or there may be already one, one should consider surgical removal. We report a 12-year-old girl who required surgical removal of a left ventricular thrombus caused by acute viral myocarditis. **(Korean J Pediatr 2007;50:588-591)**

Key Words : Intracardiac thrombosis, Myocarditis, Cerebral infarction, Cardiac surgery

Introduction

Left ventricular thrombus is mainly caused by anterior myocardial infarction or severe cardiac wall dysfunction of the apex[1], and is rarely caused by a complication of acute myocarditis[2, 3]. Typical left ventricular thrombi which are attached to the cardiac wall fixedly are treated with anticoagulants, but those which are at risk of thromboembolism such as pedunculated and movable thrombi may require surgical removal[4]. We report the case that surgery was required to remove the movable left ventricular thrombus caused by acute viral myocarditis in order to prevent further thromboembolic event.

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Case Report

A 12-year-old girl was admitted via the emergency room. She began to have upper respiratory infection symptoms 4 days before and began to have headaches, perspiration and dyspnea 1 day before of her admission. She was treated under the diagnosis of pneumonia at a local pediatrics. On the day of her admission she developed symptoms of motor dysphasia and right hemiparesis. She did not have any past medical history. On admission, her vital signs were; blood pressure 80/50 mmHg, pulse rate 132 beats/min, respiratory rate 36 breaths/min, body temperature 36.4℃. Her mental status was drowsy, and the pupil light reflex of her right eye was decreased. The neurologic exams revealed decreased motor and sensory function on the right side of the body.

Laboratory findings were leukocyte 6,550/mm^3 (neutrophil 50.8%), hemoglobin 13.0 g/dL, platelet 253,000/mm^3, sodium 142 mEq/L, potassium 4.2 mEq/L, urea nitrogen 10.0 mg/dL,

creatinine 0.9 mg/dL, serum total protein 5.2 g/dL, albumin 2.6 g/dL, calcium 8.2 mg/dL, phosphate 5.1 mg/dL, cholesterol 144 mg/dL, PT 14.9 C 13.3 seconds, aPTT 37 C 33 seconds. There were no abnormal findings. AST 57 U/L, ALT 74 U/L were slightly increased and LD 979 U/L, CK 564 U/L, CK-MB 13.3 ng/mL, myoglobin 663 ng/mL, Troponin 0.61 ng/mL, and NT pro-BNP 12,041 pg/mL also showed increased values. The urinary analysis showed specific gravity below 1.005, PH 7.5, and urine microscopy showed RBC 0-1/HPF, WBC 0-1/HPF.

The chest X-ray showed pulmonary edema and cardiomegaly (Fig. 1A). And the electrocardiography revealed low voltage of QRS on all leads. The brain MRI showed acute cerebral infarction in the left basal ganglia and the left frontoparietal lobe (Fig. 1B).

Since she was suspected as having acute myocarditis, an echocardiography was taken on the second day of hospitalization. It showed a movable thrombus, which was 19×28 mm sized and located in the left ventricle (Fig. 3A). The left ventricle was enlarged and the contractility was decreased to 42% of ejection fraction (Fig. 3B). Also there was a small amount of pericardial effusion and mild mitral valve regurgitation. Under a diagnosis of acute myocarditis, serologic study for coxsackievirus B, echovirus, adenovirus, CMV and HIV were done.

After mannitol was infused, her mental status became alert. She still had motor dysphasia and right hemiperesis. So in order to prevent further thromboembolic event, we performed open cardiac surgery via the atrium and removed the thrombus of the left ventricle (Fig. 2A). An endomyo-

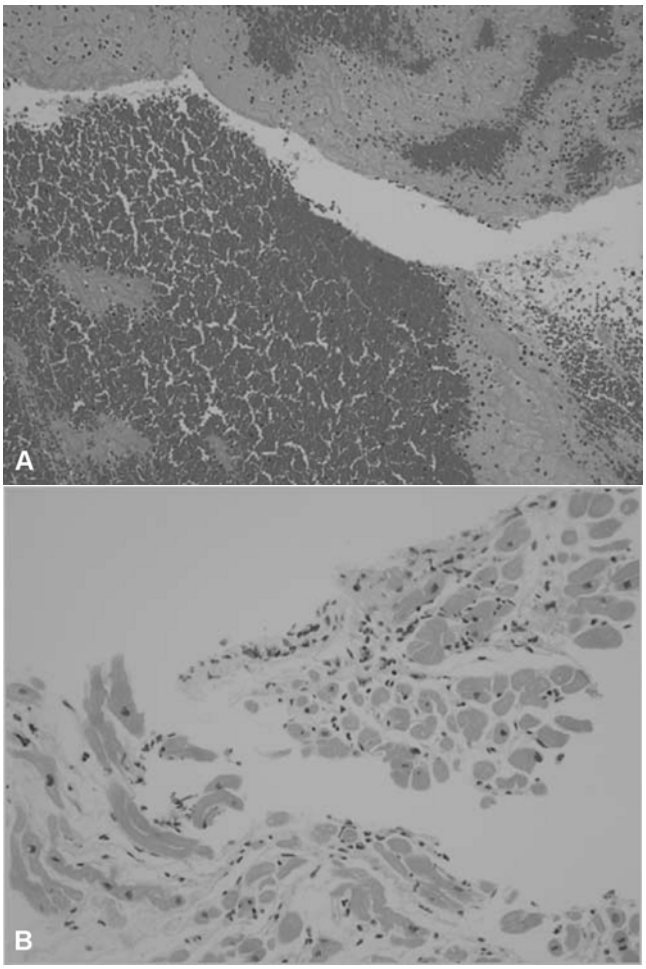

Fig. 2. (A) A microscopic finding of the thrombus in the left ventricle. H&E, ×20. (B) Microscopic finding showing inflammatory mononuclear cell infiltration in myocardium. H&E, ×40.

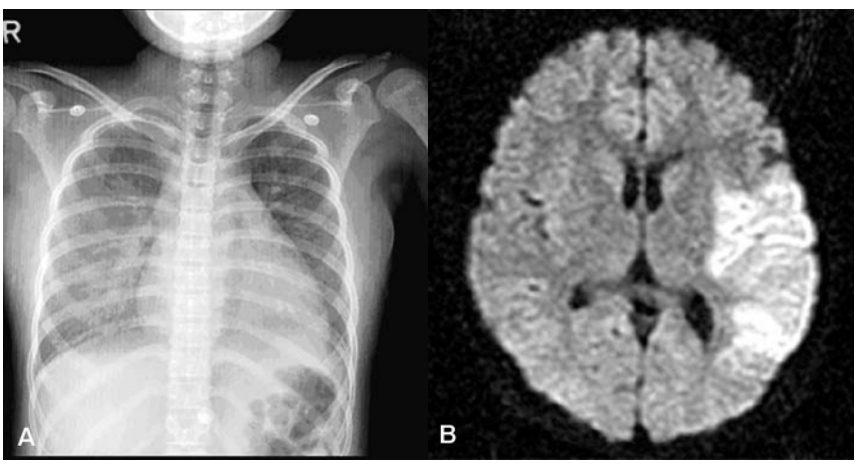

Fig. 1. (A) Chest X-ray showed pulmonary edema and cardiomegaly. Possible bilateral pleural effusion. (B) Brain MRI showed acute large infarction at the left basal ganglia and left frontoparietal lobe.

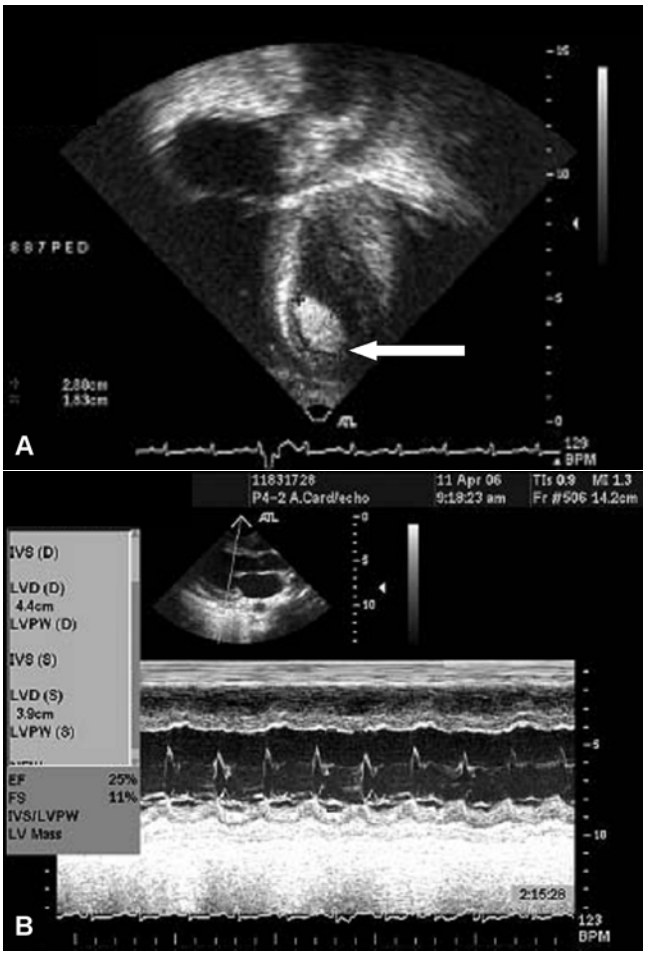

Fig. 3. (A) The posterior angulated apical 4 chamber view showed a thrombus at the left ventricular apex, which sized was measured by 2.9×1.8 cm. (B) M-mode showed decreased left ventricle ejection fraction.

cardial biopsy showed acute myocarditis (Fig. 2B).

After the surgery, dopamine was infused and oral captopril and lasix were given. Heparin was infused for 2 days after the surgery and then was changed to oral wafarin. On the third day of hospitalization, her condition was well and she was weaned from the ventilator. On the follow up echocardiogram, there was no thrombus. On the fifth day of hospitalization, her mental status was alert and the pupil light reflex was intact. And the paresis of the right lower extremities and motor dysphasia were improving. Phenobarbital was given for the small cerebral infarction in the left frontoparietal lobe shown in the brain MRI.

On the seventh day of hospitalization, all cardiotonic drugs were stopped. The ejection fraction improved from 42% to 62%. The result of the serologic study for echovirus, adenovirus, CMV and HIV were negative. The titer of neutralized coxsackievirus B type 5 antibody was 1:32.

Her symptoms of motor dysphasia and right hemiparesis were nearly recovered and she was discharged from the hospital without any other special complication. During her follow up as an out patient for 12 months, she has been completely recovered of the remaining symptoms.

Discussion

Left ventricular thrombus is mainly caused by acute myocardial infarction[1] and is rarely caused by an acute myocarditis[2, 3]. Daly et al. reported that five (25%) of twenty patients with myocaridtis had evidence of pulmonary and/or systemic emboli over a 2-year period[5]. The most common causes of viral myocardidtis are enteroviruses such as coxsackievirus B and echovirus. Some other causes are adenovirus, CMV, HIV, influenza, measles, mumps, and etc[6]. Patients with myocarditis are predisposed to ventricular and atrial thrombi due to endocardial injury, procoagulant effects of cytokines and blood flow stasis[7, 8]. And congestive heart failure is partly considered to be related with the formation of thrombus[9]. The risk of systemic thromboembolism caused by left ventricular thrombus is about 5-6%[10]. But if the thrombus is movable and pedunculated, the risk of systemic thromboembolism is increased up to 60%[11]. When giving anticoagulants in acute myocarditis, the prevalence of left ventricular thrombus was not reduced[12]. And anticoagulant therapy of left ventricular thrombosis caused by acute myocarditis may not be effective[13] and may even cause a major thromboembolism[11].

Nili et al.[14] reported that early removal of the thrombus through open heart surgery (left ventriculotomy) prevented the recurrence of thromboembolism. And Lew et al.[4] asserted that early thrombectomy should be considered when a mobile and peduculated left ventricular thrombus is demonstrated by two-dimensional echocardiography.

Before deciding treatment for the left ventricular thrombus caused by acute myocarditis, one should understand the shape and location of the thrombus through the echocardiogram. When the thrombus is laminar and fixed, one should consider anticoagulant therapy. But if the thrombus is pedunculated and movable, which means that there are higher possibilities of major embolism or there may be already one, one should consider surgical removal.

In this case, on the day of her admission she developed motor dysphasia and right hemiparesis. The brain MRI

showed acute cerebral infarction in the left basal ganglia and the left frontoparietal lobe. Echocardiography showed a movable thrombus, which was 19×28 mm sized and located in the left ventricle. And the left ventricle was enlarged and the contractility was decreased to 42% of ejection fraction. LD 979 U/L, CK 564 U/L, CK-MB 13.3 ng/mL, myoglobin 663 ng/mL, Troponin 0.61 ng/mL, and NT pro-BNP 12,041 pg/mL showed increased values. And the electrocardiography revealed low voltage of QRS on all leads. Under the diagnosis of acute myocarditis she was treated with intravenous dopamine and lasix. We were able to prevent the recurrence of thromboembolic event by early removal of the left ventricular thrombus via open heart surgery.

한 글 요 약

급성 심근염에 의한 좌심실 혈전의 수술적 제거 1례

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좌심실 혈전은 전벽 심근 경색이나 심한 심첨부 심벽 운동 이상에 의해 주로 발생하며, 매우 드물게 급성 심근염에 합병되어 나타날 수 있다. 급성 심근염에 의한 혈전 형성은 심내막 손상 및 혈액의 울혈과 관계가 있는 것으로 생각되고 있다. 좌심실 혈전이 유동적이고 유경성일 경우, 전신 색전증의 위험이 증가하는 것으로 알려져 있다. 혈전이 무경성이고 유동성이 없는 경우에는 항응고 치료 요법을 고려할 수 있지만, 유경성의 과유동성 혈전이어서 전신 색전증의 가능성이 높거나 이미 전신 색전증이 발생한 경우에는 색전증의 재발을 막기 위해서 신속한 수술적 혈전 제거술이 필요하다. 저자들은 급성 심근염에 의해 생긴 좌심실 혈전을 수술적 치료로 제거하였기에 보고하고자 한다.

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