Cardiology in the Young

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Mid-cavitary hypertrophy after myocarditis in a patient with operated medulloblastoma

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Brief Report

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Abstract

A 16-month-old girl was referred for tachycardia and upper respiratory tract infection. Echocardiographic examination revealed pericardial effusion, mild mitral regurgitation, and left ventricle systolic dysfunction. Patient was positive for Parainfluenza type 4 virus. Her laboratory tests revealed increased troponin I level. The patient was treated with intravenous immunoglobulin considering acute viral myopericarditis. Two weeks after treatment, midventricular hypertrophy was detected.

Hypertrophic cardiomyopathy is a disease characterised by the thickening of the left ventricle wall. Although there is usually a genetic predisposition, it may develop secondary diseases such as aortic stenosis, systemic hypertension, or physiologic remodelling associated with athletic training.[1] Morphologically, there are four forms as asymmetric septal, apical, concentric, and midventricular.[2] Rarely, it can be seen as a midcavitary hypertrophy that may cause obstruction in the ventricle. In this case report, a female patient with operated medulloblastoma who was found to have midcavitary hypertrophy after myocarditis was presented. The family of the case signed an informed consent.

Case report

A 16-month-old girl operated on for medulloblastoma 2 months ago was referred to our clinic for tachycardia and symptoms of upper respiratory tract infection. Physical examination showed that blood pressure was 82/45 mmHg, and body temperature was 37.2 °C. Sinus tachycardia (heart rate: 160/m), voltage suppression on left precordial derivations, and no ST-T changes were analysed on the electrocardiogram. Echocardiographic examination revealed minimal pericardial effusion at left ventricle posterior wall, spherical and dilated left ventricle, mild mitral regurgitation, and left ventricle systolic dysfunction (ejection fraction: 54%, fractional shortening: 27%) (Fig 1). Patient was positive for Parainfluenza type 4 virus in multiplex viral PCR. Her laboratory tests revealed leukocytosis (16,300/mm^3) and increased C-reactive protein level (14.5 mg/dl, normal for laboratory: 0–0.5) and troponin I level (0.26 ng/ml, N: 0–0.04). The patient was treated with IVIG considering acute viral myopericarditis. Echocardiographic

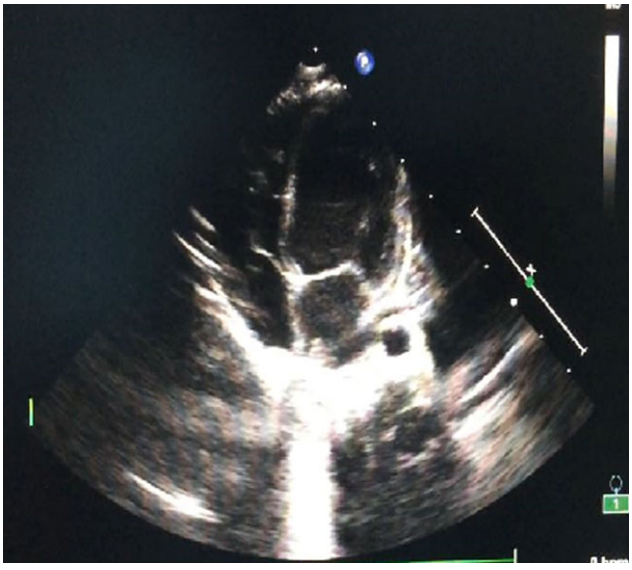

Figure 1. Echocardiographic view when the patient is diagnosed as acute myocarditis.

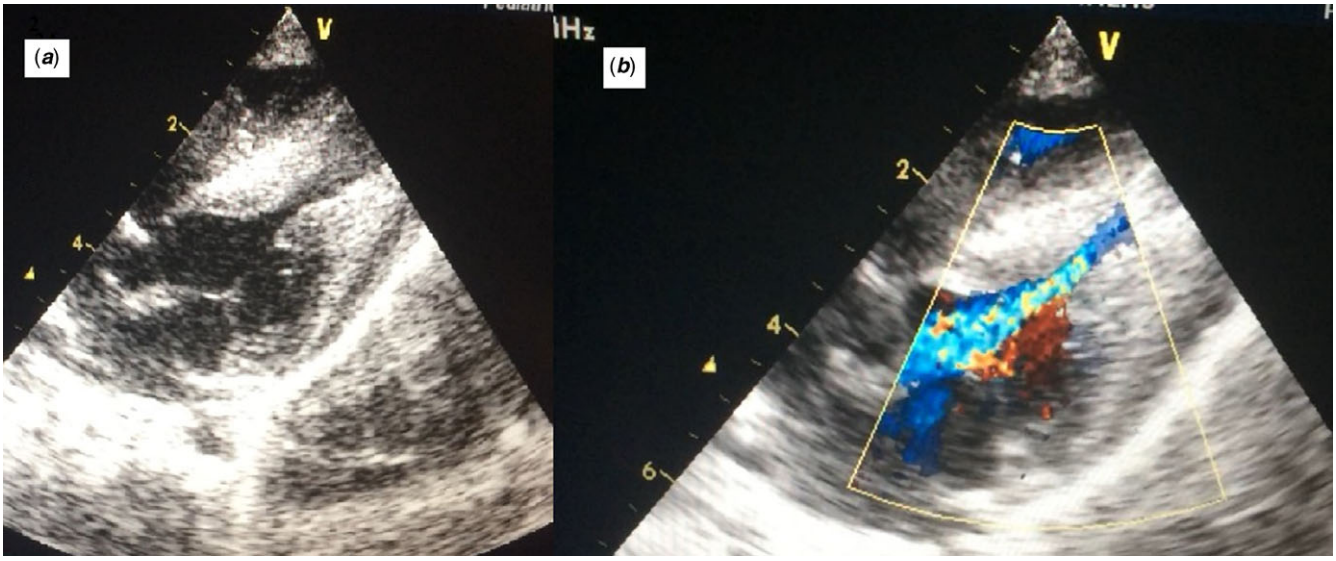

Figure 2. (*a*) 2D Echocardiographic view when the patient is diagnosed as midventricular obstruction. (*b*) Doppler echocardiographic view of midventricular obstruction.

findings and troponin I level improved after treatment. The patient's condition deteriorated 2 weeks after treatment and echocardiography revealed normal systolic function, no pericardial effusion, midventricular hypertrophy concerning hypertrophy of the midsegment of interventricular septum and LV posterior wall, with a mean 50–55 mmHg gradient (Fig 2A-2B). The patient was diagnosed with hypertrophic cardiomyopathy with midventricular obstruction and beta-blocker treatment was started. Echocardiographic findings improved on 14th day of treatment.

Discussion

Viral infection is the most common reason for myocarditis. Although myocarditis is the most frequent known cause of dilated cardiomyopathy, myocarditis does not only cause dilation of LV, but can also cause a temporary increase in wall thickness. The ventricular wall is frequently thin, although hypertrophy may be found as well. Myocardial fibrous, interstitial oedema, and oedematous tissue with infiltration of mononuclear cells were the main causes of myocardial hypertrophy.[3] However, an increment of LV wall thickness is considered as a result of the myocardial oedema.[4] Recently, studies showed that Toll-like receptors (TLR-2) are responsible for the cardiac hypertrophy which is formed by infectious or non-infectious inflammation.[5,6] When the physiological or pharmacological stress is performed on the myocardial tissue that affects TLR-2-dependent inflammation, cardiac hypertrophy occurs under the influence of heat shock protein 70 and cardiac progenitor cells.

Cardiac hypertrophy is an abnormal increment of muscle mass, which is a result of the proliferation of fibroblasts, vascular endothelium, and myocyte hypertrophy. Hypertrophic cardiomyopathy affects approximately 1:500 individuals.[7] Hypertrophic cardiomyopathy with midventricular obstruction, defined as an intracavitary gradient ≥30 mmHg, affects approximately 10% of patients with the disease, as determined in a large Japanese cohort.[8] In patients with hypertrophic cardiomyopathy, midventricular obstruction was found to be an independent risk factor in terms of sudden death and lethal arrhythmias.[7] Tezuka et al.[9] reported that beta-blocker therapy, also used in our case, may be the first choice for the initial treatment of hypertrophic cardiomyopathy with midventricular obstruction. Gradient reduction with negative inotropic agents might be expected to provide relief from dyspnoea, wall stress, and myocardial scarring in patients with midventricular obstruction.

In our case, there were no known secondary causes of left ventricular hypertrophy with normal systolic function. Our patient was presented because there was no reported case of midventricular hypertrophy following myocarditis.

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Conflict of interest. None.

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