Idiopathic Fascicular Left Ventricular Tachycardia Mimicking Dilated Cardiomyopathy

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Idiopathic fascicular left ventricular tachycardia in an anatomically normal heart is a unique entity, in which management and prognosis differ from ventricular tachycardia associated with structural heart diseases. Here we report a case of a 14-year-old girl with idiopathic fascicular left ventricular tachycardia mimicking ventricular tachycardia due to dilated cardiomyopathy. **Case:** A 14-year-old girl presented to the ER with exertional dyspnea and palpitation. BP and HR were 85/65 mmHg, 160bpm. Wide QRS VT with RBBB and left superior axis deviation were observed in ECG. There was cardiomegaly in chest x-ray (Fig.1). Lab findings were as follows: CPK 88U/L (<170) CK-MB 1.8 ng/ml (<3.61) Troponin-T 0.01 ng/ml (0-0.014) NT-pro BNP 1908 pg/ml (0-125). Echocardiography revealed that global dilatation of LV and LA EF=10% (LVDD/LVSD 60/55 mm, Septum/PW 8.7/9.3 mm) (Fig.1). Cardiac arrest was occurred due to pulseless VT. It was failed to stop it. ECMO insertion was started. One week later, ECG rhythm was turned into the normal sinus rhythm, followed by verapamil infusion (Fig.2). The patient was referred to electrophysiology study right after weaning off ECMO. Intracardiac mapping showed fascicular VT originating from left ventricular posterior fascicles. For several follow-ups, function of the heart was improved with EF 41% (LVDD/LVSD 48/34mm, Septum/PW 9.5/9mm) (Fig.3).**Conclusions:** We report a unusual case of a 14-year-old girl who had idiopathic fascicular left ventricular tachycardia mimicking ventricular tachycardia due to DCMP.

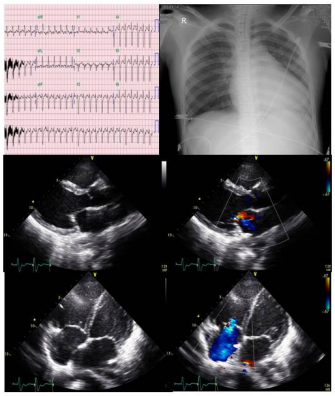

Fig.1) Initial ECG, chest xray and echocardiography
LVDD/LVSD : 60/55mm Septum/PW : 8.7/9.3mm EF =~10%

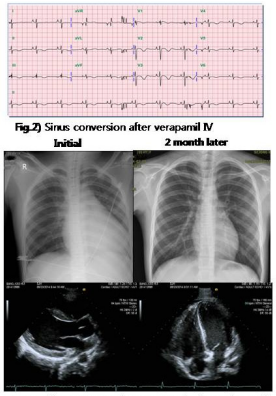

Fig.2) Sinus conversion after verapamil IV

Fig.3) Follow-up ECG, chest xray and echocardiography
LVDD/LVSD : 48/34mm Septum/PW : 9.5/9mm EF = ~41%

Idiopathic hypereosinophilic syndrome presenting as Loeffler's endocarditis

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A 61-year-old man presented with chest discomfort and dyspnea showed peripheral eosinophilia (WBC count of $13.0\times10^3/\mu l$ with 55% eosinophil). Echocardiography revealed significantly increased endocardial thickness of the left ventricle with apical cavitary obliteration (Figure.) and grade III diastolic dysfunction. Gadolinium-enhancement cardiac MR showed endocardial fibrosis, inflammation and thrombus. Chest and abdominal CT showed multiple nodular infiltrations in the lung and hypodense nodules in the liver, respectively, suggestive of eosinophilic infiltration. Bone marrow biopsy demonstrated increased number of eosinophils and its precursors. A mutation involving the FIP1L1/PDGFRA genes was not found. The patient was diagnosed with idiopathic hypereosinophilic syndrome (HES) with involvement of the heart, lung, and liver. After 1 month of treatment with corticosteriod, he was free of symptoms and peripheral eosinophilia was resolved. Follow-up echocardiography revealed improvement of endocardial thickening and chest X-ray showed no pulmonary infiltration. HES is defined by unexplained persistent eosinophilia (Absolute eosinophil count of $>1.5\times10^3/\mu l$) for at least 6 months with evidence of eosinophil-mediated organ damage. Loeffler's endocarditis is a form of cardiac involvement in HES showing apical obliteration by fibrous thickening of the endocardium, leading to restrictive cardiomyopathy and resulting in heart failure, arrhythmia or thromboembolism.

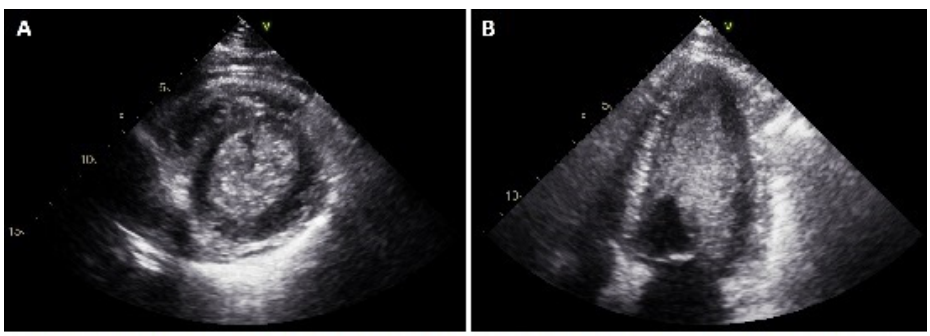

Figure. Echocardiography showed hyperechoic thickening of the endocardium and apical cavitary obliteration. (A) Mid-level short axis view at systole, (B) Apical 4 chamber view at systole