

## CAN ASYMPTOMATIC WOLFF-PARKINSON-WHITE PATTERN BE A RISK FACTOR FOR DEVELOPMENT OF DILATED CARDIOMYOPATHY?

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### Contribution

HAG conceived the idea, worked on case report, collected the pictures and did final review of the submitted case report.

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### ABSTRACT

Wolff-Parkinson-White (WPW) syndrome is a well known pre-excitation syndrome and associated with supraventricular tachycardia (SVT). Dilated cardiomyopathy (DCM) is the most common type of cardiomyopathy and it may arise in patients with symptomatic WPW. However, WPW with accessory pathway and without incessant tachyarrhythmia, may cause left ventricle septal dyskinesia and left ventricle dysfunction. Recovery of the left ventricle function after the ablation or medical treatment shows the association between DCM and ventricular pre-excitation. In this case report, we present an asymptomatic WPW patient who developed DCM and was successfully treated with ablation therapy.

**Key Words:** Accessory pathway, Asymptomatic Wolf-Parkinson-White Syndrome, Dilated cardiomyopathy

## INTRODUCTION

Dilated cardiomyopathy (DCM) is known as the most common type of cardiomyopathy, and is seen approximately 36.5 per 100000 of population. Although infectious, metabolic, ischemic, toxic and hereditary factors have been implicated in the disease pathogenesis, the primary cause of DCM is unknown in 66 % of children.<sup>1</sup> Supraventricular tachycardia (SVT) rarely causes DCM.<sup>2</sup> Wolff-Parkinson-White (WPW) syndrome is well known pre-excitation syndrome and associated with SVT. DCM may arise in patients with symptomatic WPW as a result of incessant tachyarrhythmia. Recent case reports with asymptomatic WPW patient shown that an accessory pathway is related to LV septal dyskinesia and LV dysfunction in the absence of documented SVT.<sup>3,4</sup> Here in, we present a WPW patient with DCM but did not have documented SVT in his history and was successfully treated with ablation therapy.

## CASE REPORT

Fourteen-months-old boy was admitted with complaints of respiratory distress and rapid breathing. His medical history was normal except congenital hypothyroidism. He received L-thyroxine treatment and his hypothyroidism was under good control. He had no family history of sudden cardiac death, arrhythmias or cardiomyopathy. In physical examination; body weight was 8200 gr (3-10 per), sinus tachycardia (160 beat/min), tachypnea (40 breath/min) and 2-3/6 early systolic murmur along mesocardiac area were documented. Twelve-lead electrocardiography (ECG) showed pre-excitation with delta waves and left axis deviation (Figure 1A). Transthoracic echocardiography (TTE) showed LV ejection fraction (LVEF): 44%, LV end-diastolic diameter (LVEDd) 36 mm (z score: +4.00) and moderate mitral regurgitation (Figure 1B). BNP was 105 pg/ml (0-100), Troponin-I 3,35 ng/ml (0-0,04) were found. His metabolic screening was normal and serological evidence for any bacterial, viral or fungal infection was negative. The patient was diagnosed with DCM and tachycardia accompanied with right sided accessory pathway (Figure 2). SVT was not observed at 24-hour rhythm Holter recording and there was no documented SVT attack.

Furosemide and enalapril were given for the treatment of heart failure. Amiodarone (5 mg/kg/day) treatment was started, two weeks later amiodarone dose was increased gradually to 10 mg/kg/day. The WPW pattern disappeared following amiodarone on this dosage at rhythm Holter analysis. When the dose of amiodarone was tapered to 7,5 mg/kg/day the patient showed WPW pattern at one third of the day at rhythm Holter analysis. So, the dosage of amiodarone was not increased because of ongoing hypothyroidism and side effects of drug. Therefore we decided catheter ablation of accessory pathway. The accessory pathway was seen on anteroseptal localization at electrophysiologic study by using catheters of cryoablation and coronary sinus. Cryoablation was performed at this region four times by the temperature to -80°C for 300 seconds. Any recurrence of preexcitation was not seen at basal condition and adenosine apply after the electrophysiologic study. His ECG was normal after ablation. His rhythm Holter at that time and three months after the ablation showed sinus tachycardia and LVEF 61%, LVEDd 34 mm (z score: +4.00) were measured at TTE. Anti-

congestive treatment has been continued.

## DISCUSSION

Dilated cardiomyopathy may occur in symptomatic WPW patients with sustained tachyarrhythmias. Recent reports suggested that significant left ventricular dysfunction may arise in WPW even in the absence of recurrent or incessant tachyarrhythmia.<sup>5</sup>

The pre-excitation causes septal motion abnormalities in patients who have asymptomatic WPW with right-sided septal or paraseptal accessory pathway. Premature ventricular activation over these accessory pathways induces septal wall motion abnormalities and ventricular dyssynchrony which may have detrimental effects on myocardial performance. Thus DCM may occur at asymptomatic WPW patients.

Left ventricle dyssynchrony can be corrected by medical treatment such as amiodarone or particularly via radiofrequency catheter ablation (RFCA) of accessory pathway. Electromechanical resynchronization may cause recovery of LV dysfunction. RFCA may be the first therapeutic choice for accessory pathway induced DCM. On the other hand, catheter ablation is not chosen as a first step therapy in infants and small children because of its higher risk of complications and uncertain long-term effect of ablation scars on young myocardium.<sup>6</sup> Cadrin-Tourigny et al. reported two young infants with LV dysfunction caused by asymptomatic WPW syndrome who were treated with amiodarone. Their following period with amiodarone treatment resulted in recovery of LV function.<sup>6</sup> Our patient was treated with amiodarone as the first step therapy but amiodarone suppressed pre-excitation only at high doses and clinical outcome was accepted insufficient. Additionally he has congenital hypothyroidism history and have to use L-thyroxine treatment. Hypo-hyperthyroidism is known one of the most important side effects of amiodarone. Therefore we decided to apply ablation therapy of accessory pathway nevertheless patient's young age.

Kwon et al. reported 4 month of age infant diagnosed WPW who developed rapid progression of ventricular dysfunction after birth without sustained SVT. Pre-excitation of this patient could not be medically suppressed but was successfully ablated and ventricular dysfunction was completely resolved within followed 2 months.<sup>7</sup> Previous report about RFCA show that the reverse remodeling may become more considerable in follow-up investigations.<sup>3</sup> Our patient's rhythm Holter was normal after ablation, LVEF and LVEDd got better in a few months.

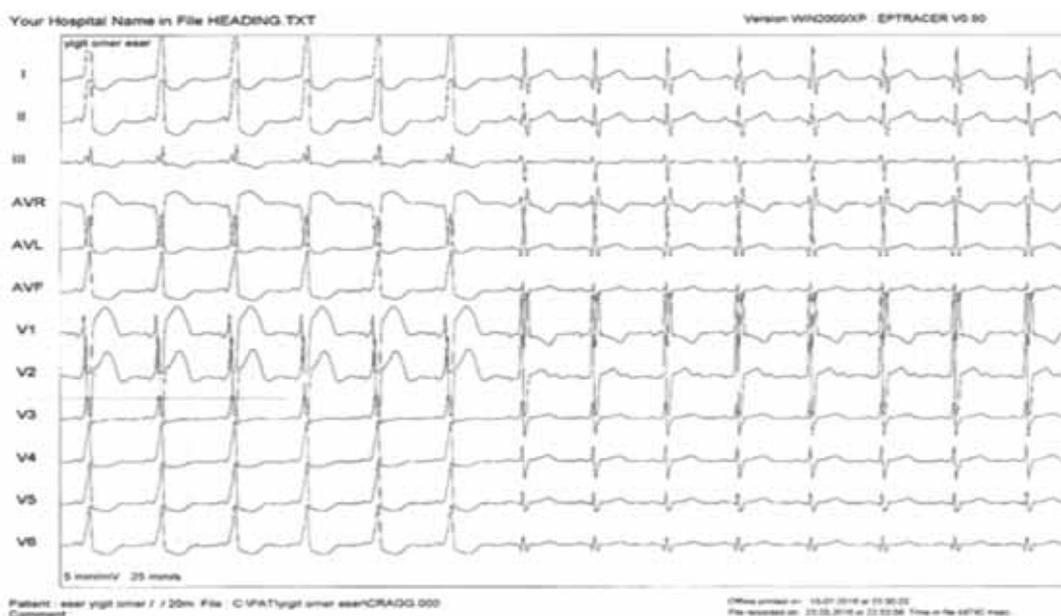
In conclusion, accessory pathway-induced DCM should be suspected in all patients presenting with heart failure and overt ventricular pre-excitation, in whom no cause for their DCM could be found. Recovery of the LV function after the ablation or medical treatment of the pre-excitation shows the association between DCM and ventricular pre-excitation. Medical treatment may be chosen as a first line therapy in infants smaller than 5 years old and RFCA may be performed in the following period.

**Figure 1A:** Electrocardiographic view of Wolff-Parkinson-White pattern before and after ablation

**Figure 1B:** Echocardiographic view of dilated cardiomyopathy and mitral regurgitation before ablation



**Figure 2: Picture Showing the Disappearance of Pre Excitation During Ablation**



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