

A Rare Case Report

Wolff-Parkinson-White Syndrome and Pre-excitation Induced Cardiomyopathy: Is It a Causal or Casual Relationship?

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Background

Patients with Wolff-Parkinson-White (WPW) syndrome frequently experience palpitations due to atrioventricular reciprocating tachycardia (AVRT) associated with an accessory pathway (AP). The specific mechanism underlying left ventricular (LV) dysfunction in individuals with pre-excitation syndrome is not fully understood, and its prevalence in Indonesia is also unclear. Eccentric activation of the ventricles through an AP—especially if located on the right side—can cause an asynchronous spread of ventricular depolarization. This leads to both mechanical and electrical dyssynchrony, which may worsen LV dysfunction, a condition known as Pre-excitation Induced Cardiomyopathy (PIC). This case report aims to investigate the impact of a right-sided AP in pre-excitation syndrome that results in PIC, and to discuss the outcomes of successful right AP ablation in enhancing LV systolic function in our patient at the National Cardiovascular Center Harapan Kita (NCCHK).

Case Illustration

A 12-year-old female patient with WPW syndrome and chronic heart failure symptoms presented to NCCHK. An echocardiogram indicated non-ischaemic cardiomyopathy with an initial left ventricular ejection fraction (LVEF) of 18% and global hypokinesia. A 12-lead electrocardiogram (ECG) revealed a pre-excitation pattern on the right anterior side. During radiofrequency ablation (RFA), the accessory pathway (AP) was located in the right anterior area, consistent with the ECG findings. The ablation procedure was successfully completed. One year post-ablation, the patient's LVEF gradually improved to 41%.

Summary

The patient's cardiomyopathy was likely a result of left ventricular dyssynchrony caused by significant ventricular pre-excitation. After successful ablation of the right anterior AP, the patient has not experienced any major heart failure symptoms. Continuous long-term follow-up, particularly for clinical and echocardiographic evaluations, is still needed. To date, the patient's left ventricular ejection fraction (LVEF) has improved remarkably, and the patient remains free of symptoms.

Keywords : WPW Syndrome, Cardiomyopathy, LV Dyssynchrony

INTRODUCTION

Wolf Parkinson White syndrome (WPW) is recognized as a congenital condition that involves abnormal electrical pathways between the atria and ventricles. This disorder features additional electrical circuits that bypass the AV node. Recent studies from our group, focusing on a large population of asymptomatic patients, have found that the prognosis for asymptomatic WPW, particularly in children, may not be as favorable as previously thought. WPW has been linked to issues such as syncope, sudden cardiac death, palpitations, and tachycardia-induced cardiomyopathy (TIC). There have

also been cases of non-arrhythmic dilated cardiomyopathy associated with WPW in children.^{1,2}

LV systolic dysfunction may have resulted from a mechanism similar to that noted in patients who develop cardiomyopathy from right ventricular pacing. The event of incessant tachycardia is known to be a cause of cardiomyopathy in some patients with WPW syndrome. Right-sided Accessory Pathways with prominent preexcitation may also result in a LBBB pattern, LV dyssynchrony, and Non-Ischaemic Cardiomyopathy (NICM) even in the absence of tachycardia. Most reports have shown reversal of LV dilatation and restoration of

LV function after successfully ablation of accessory pathway, it could occur for a variable period.^{3,4}

In some settings, the accessory pathway can result in the transmission of abnormal electrical impulses leading to malignant tachyarrhythmias, if untreated properly, it could lead to LV dyssynchrony and severe LV dysfunction. In this case presentation, we report an unusual case of WPW syndrome in a patient with Non-Ischaemic Cardiomyopathy (LVEF 18%) undergoing successful AP ablation in NCCHK.

CASE ILLUSTRATION

A 12-year-old female patient presented to the National Cardiovascular Center Harapan Kita (NCCHK) with a two-year history of heart rhythm disturbances. She reported experiencing symptoms such as fatigue, frequent palpitations, and exertional dyspnea, all of which improved with rest. Over the past several months, her palpitations had intensified, particularly during

physical activity. Notably, she had not experienced any episodes of syncope or chest pain. Recently, she had no specific complaints and was attending routine check-ups. Her medication regimen included Ramipril 1.25 mg once daily and Furosemide 10 mg once daily.

In her past medical history, the patient was born at term and by spontaneous delivery, with birth weight 2900 gr and birth length 48 cm. She had no family history of cardiac disease, cardiac arrhythmia or sudden death. Radiofrequency ablation (RFA) was planned for WPW syndrome she was suffering from if her body weight has reached > 30 kg. Physical examination revealed Compos Mentis, BW 37 kg, BH 155 cm, BP 117/63 mmHg, HR 71-73 bpm, RR 16 x/m, T 36,2 C, SaO₂ 98% (Room Air). Cardiac examination revealed normal heart sound, no murmur / gallop. Pulmonary examination showed normal vesicular sound, no rales / wheezing. She had no organomegaly, no sign of any oedema, CRT < 2 s.

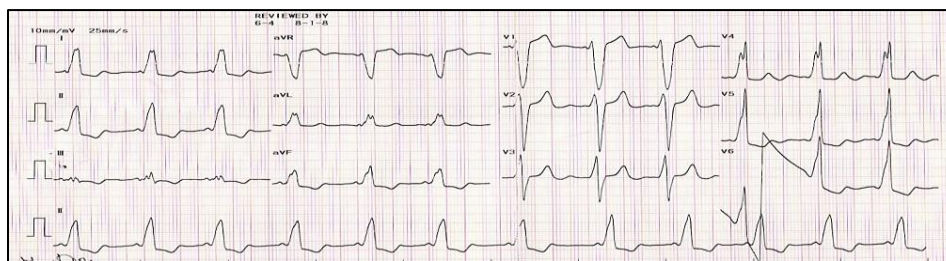


Figure 1. 12 Lead ECG showed multiple leads with shortened PR Interval and Delta Waves

Electrocardiography (ECG) showed sinus rhythm, HR 80 bpm, normal P waves, PR Interval 0,07 s, QRSd 0,14 s with Delta Waves (+), proportional ST-T segment change discordance. From the QRS deflection of ECG, delta waves in I, II, III, aVL, aVF, V4-V6. Laboratory result was within normal limit. Echocardiography before ablation procedural showed reduced LV systolic

function (LVEF 18% simpson's), Global hypokinetic, normal valves, Good RV contractility (TAPSE 24 mm), and no thrombus was seen. Chest X-Ray result showed 51% of CTR, normal aortic and pulmonic segment, loss of cardiac waist, upwarded apex, normal right heart border, no congestion / infiltrate.



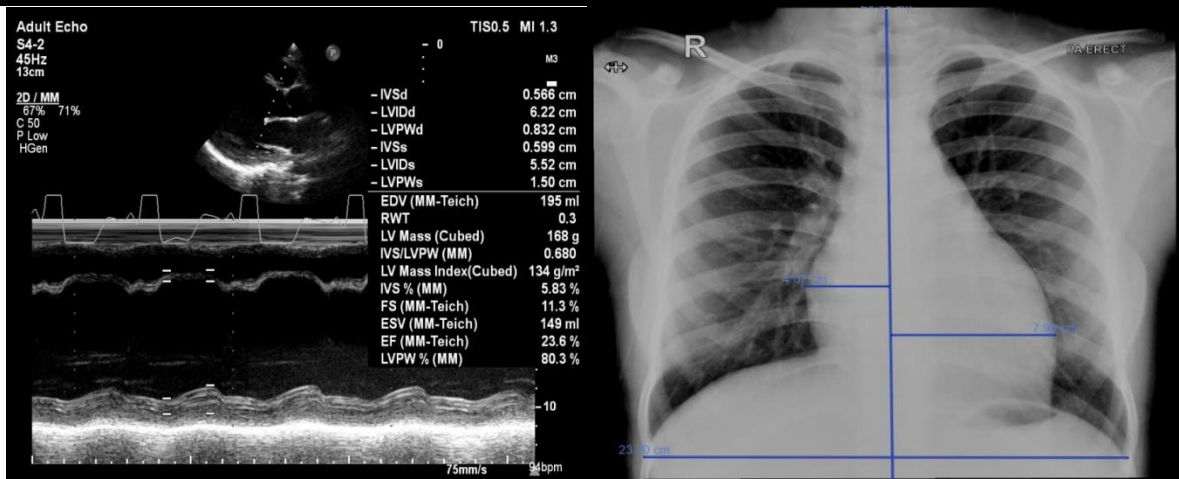


Figure 2. Echocardiography and Chest X-Ray data results before WPW AP ablation procedure

The patient was diagnosed as WPW Syndrome, Congestive Heart Failure ec Tachycardia Induced-Cardiomyopathy NYHA Fc. II. She underwent Radiofrequency Ablation of Accessory Pathway (AP) procedure in cathlab. Ablation procedure showed WP AP antegrade 310 ms, ERP AP antegrade 330 ms, WP AP retrograde 300 ms, ERP WP retrograde < 310 ms, and inducible orthodromic AVRT with the most fused VA at CS 9-10 suggesting orthodromic AVRT with right sided AP. The most fused AV signal at the right anterior area and multiple RFAs were performed. The AV

separation was noted within 3.7 second and delta waves disappeared, and Incremental ventricular pacing showed a retrograde block. Electrophysiology was conducted, basic interval measurement : PP 765 ms, AH 71 ms, HV 61 ms, PR 178 ms, QRS 119 ms, QT 440 ms, V-V: 765 RR 765 ms, QTc 511 ms. Then, Incremental atrial pacing showed antegrade WP AVN 300 ms. Conclusion of the procedure : 1) WPW Syndrome with Right Anterior Accessory Pathway; 2) Successful Ablation of Right Anterior Accessory Pathway; 3) Normal SA and AV Nodes function

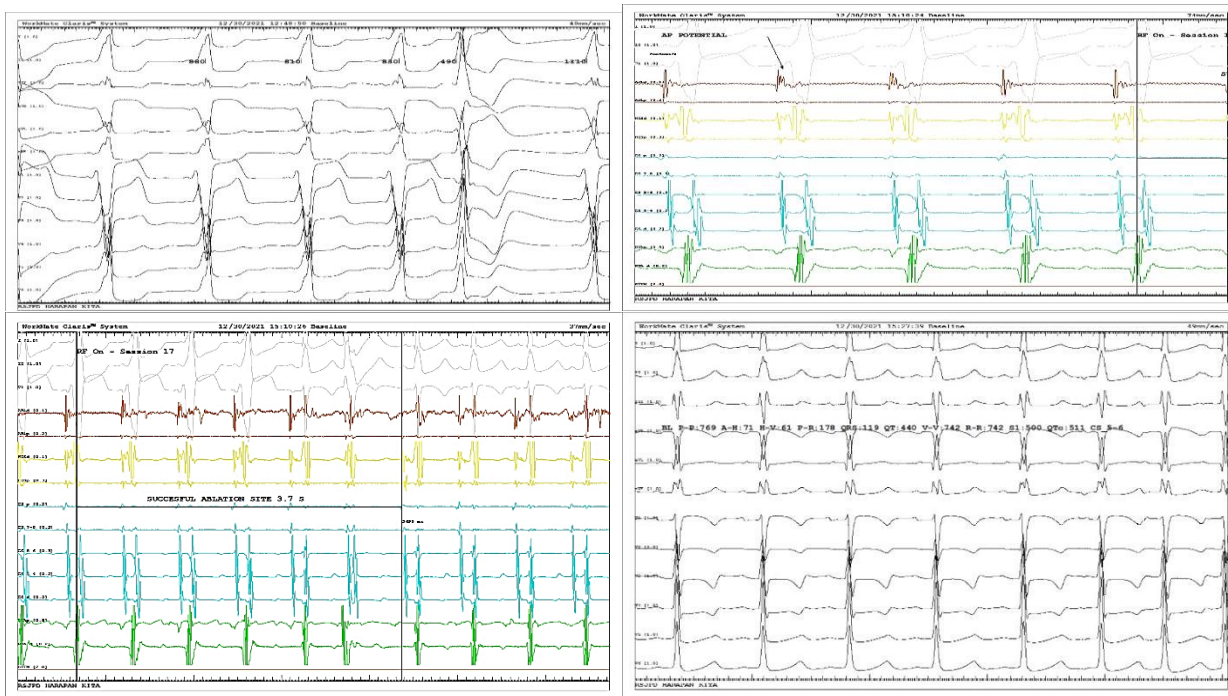


Figure 3. Electrogram (EGM) strips showed: a) Maximum Preexcitation from right anterior AP; b) Antegrade AP Potential conduction; c) Succesfull ablation episodes recorded; d) Basic Cycle-Length after ablation, 12 Lead ECG showed narrowing QRS complex and no delta waves were seen

After the RFA procedure, a bedside echocardiogram was conducted, showing no signs of pericardial effusion. The patient was then transferred to the regular pediatric ward. A 12-lead ECG indicated sinus rhythm with a heart rate of 75 bpm, normal P waves, a PR interval of 0.15 seconds, QRS duration of 0.11 seconds, IVCD in leads

II, III, aVF, and T wave inversion in leads III, aVF, and V1-V3, with no delta waves observed. The patient had a successful AP RFA and was discharged in stable condition without significant complaints. She was prescribed Ramipril 1.25 mg once daily and Furosemide 10 mg once daily.

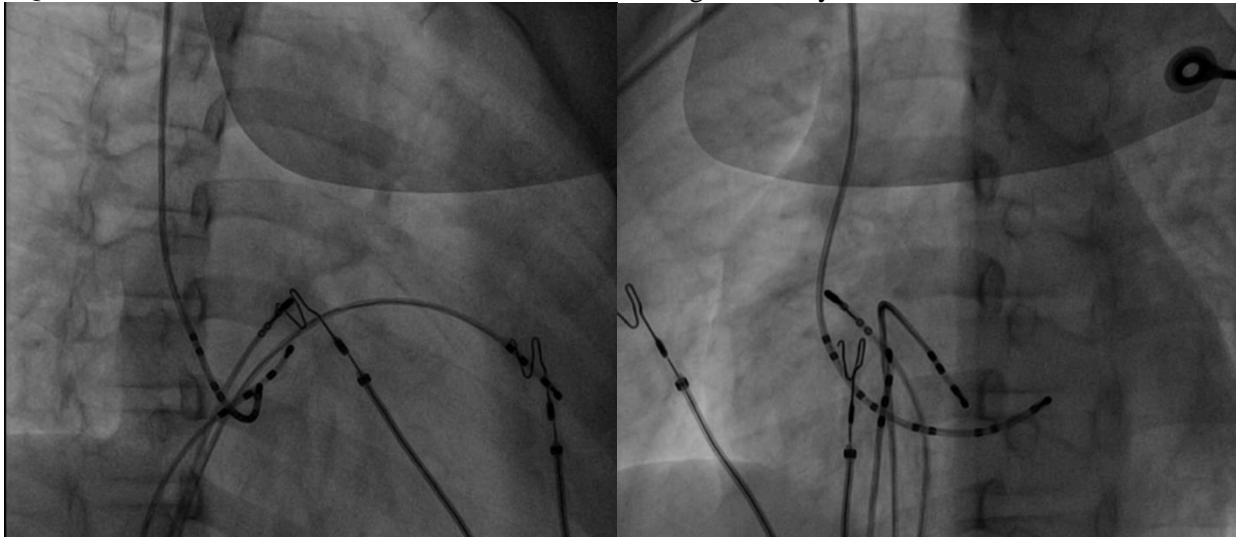
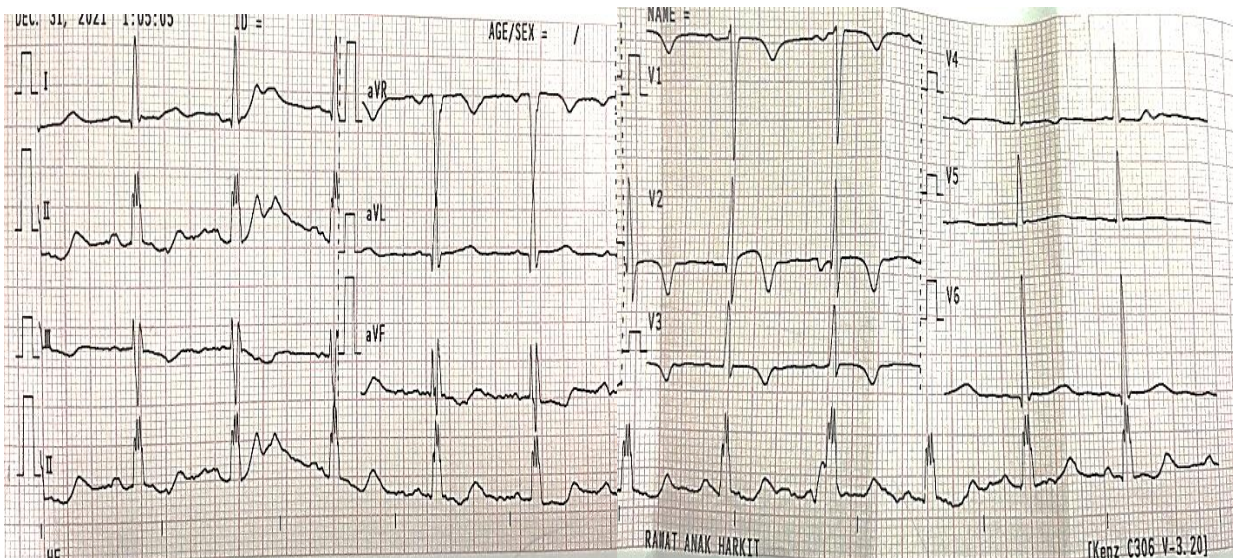


Figure 4. Fluoroscopy of Right Anterior AP RFA procedure; (left) RAO view; (right) LAO view

Ten days after the successful AP RFA ablation, the patient visited the outpatient arrhythmia clinic at NCCHK for her first follow-up evaluation. She had not experienced any episodes of palpitations, which were common before the procedure, and reported no shortness of breath or chest pain. Echocardiographic results showed a slight improvement in ejection fraction (EF) to

20% and a TAPSE of 23 mm, with normal valve function. Despite the global hypokinetic appearance of the left ventricle, predominantly in the septal area, synchronous movements were observed. The plan for the patient includes continued medication, regular follow-ups at the arrhythmia clinic, and periodic echocardiographic evaluations.



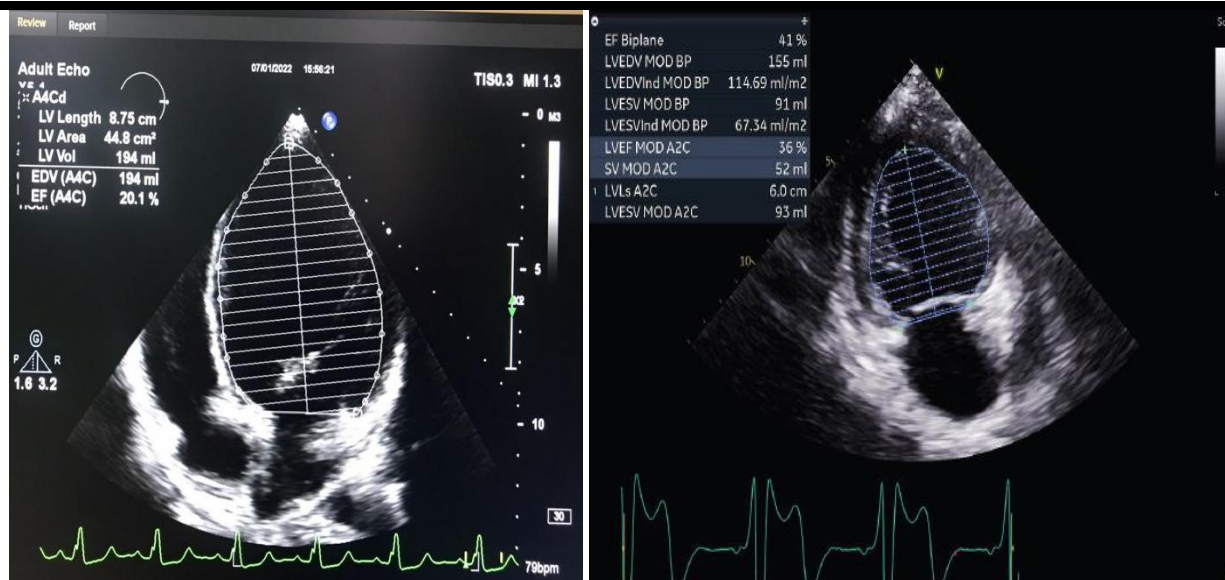


Figure 5. (Upper) Post-RFA ECG 12 lead, no delta waves and LBBB pattern; (Left Lower) Post-RFA Echocardiography evaluation test revealed slightly increased of ejection fraction; (Right Lower) The echocardiography result one year after AP ablation

DISCUSSION

Wolff-Parkinson-White (WPW) syndrome is a congenital cardiac preexcitation syndrome that arises from abnormal cardiac electrical conduction through an accessory pathway that can result in symptomatic and life-threatening arrhythmias. The electrocardiographic (ECG) finding of WPW pattern or preexcitation consists of a short PR interval and prolonged QRS with an initial slurring upstroke (“delta” wave) in the presence of sinus rhythm. The term WPW syndrome is reserved for an ECG pattern consistent with the coexistence findings of a tachyarrhythmia episodes and clinical symptoms such as palpitations, episodic lightheadedness, presyncope, syncope, or even cardiac arrest. Ventricular pre-excitation occurs when the ventricular myocardium is partially or completely depolarized through an extranodal atrioventricular connection.^{1,2}

Ventricular pre-excitation is a common condition, affecting about 0.2% of the population. Studies in both children and adults have shown a WPW (Wolff-Parkinson-White) syndrome prevalence of 1 to 4.5 per 1,000 individuals. While most people with WPW have normal cardiac anatomy, the accessory pathway associated with this syndrome is linked to syncope, sudden cardiac death (SCD), palpitations, and tachycardia-induced cardiomyopathy. Several retrospective studies indicate that the risk of life-threatening arrhythmias is higher in asymptomatic

children than in adults, with 10% to 48% of pediatric WPW cases presenting SCD as the initial event.^{2,5}

The risk of sudden cardiac death (SCD) in untreated individuals with WPW increases over time, particularly in children, where the long-term risk can significantly exceed the risks associated with the treatment procedure. Many consider transcatheter ablation to be the first-line treatment for WPW, as it has the potential to provide a definitive cure. This approach includes both radiofrequency ablation (RFA) and cryoablation techniques. RFA is regarded as the gold standard for invasive management due to its higher success rate in eliminating accessory pathways and lower recurrence rate.⁵

For many years, the initially asymptomatic WPW population has been regarded as having minimal or no risk, leading to a lack of encouragement for intensive screening programs to identify asymptomatic individuals at potential risk. Although several reports have documented severe arrhythmic events, particularly among young or asymptomatic children, the precise risk of sudden death in patients with ventricular preexcitation remains unclear. WPW patients face a low but lifetime risk of sudden death, which can be mitigated through radiofrequency catheter ablation of accessory pathways. This highlights the need for more rigorous screening and prevention programs for WPW patients globally, including in Indonesia.⁶ In our case, we performed

radiofrequency ablation (RFA) to reduce the likelihood of sudden cardiac death and eliminate tachyarrhythmia symptoms, as well as to address the dyssynchrony caused by the “LBBB” pattern resulting from right-sided WPW.

In patients with WPW syndrome, the morphology of the delta wave during sinus rhythm is dependent upon the location of the ventricular insertion of the accessory pathway, which is the site of initiation of ventricular activation. A number of investigators have correlated ECG patterns or algorithms for deciphering the location of the ventricular insertion of the AP. As the ECG algorithm accurately localizes accessory pathways prior

to ablation, it can help the physician to advise the patient regarding the likelihood of success and also complications of the procedure. According to the ECG algorithm by Arruda M. *et al*, we analysed that the Delta Wave in lead I was not *Isoelectrical* or *Negative*, then the Delta Wave in lead II was *Positive*, the morphology of the Delta Wave in V1 was not *Negative* or *isoelectrical*, its morphology was *remaining*, therefore we continued to next part of Step 4 (in Right Free Wall area). The deflection of aVF was *Positive* with *Positive* QRS Axis. Using this algorithm, we determined that the accessory pathway location of our patient could be from Right Anterior, Right Anteroseptal or Right Anterolateral.⁷

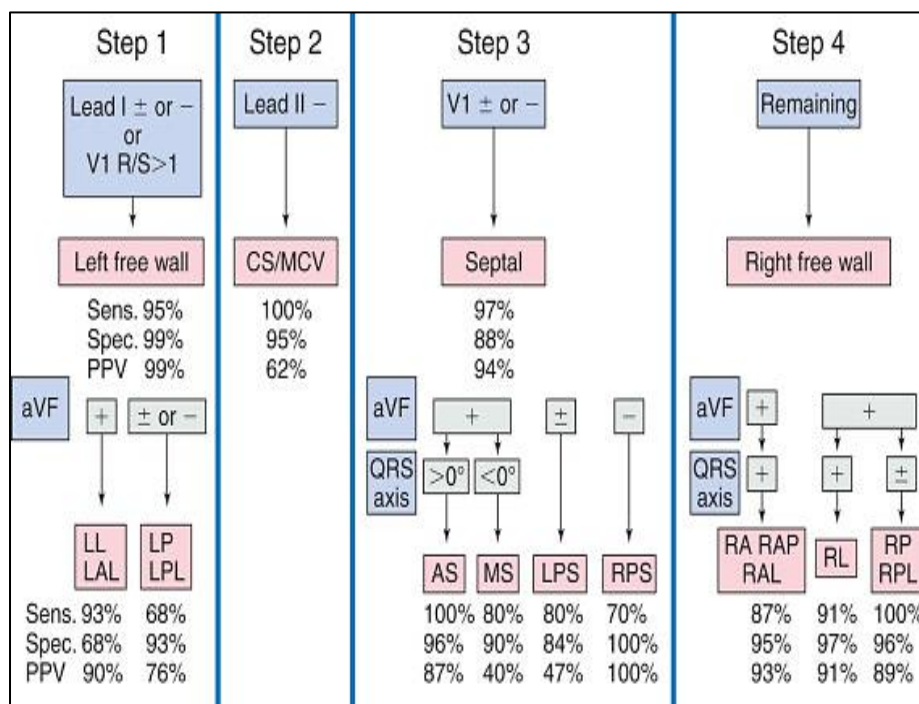


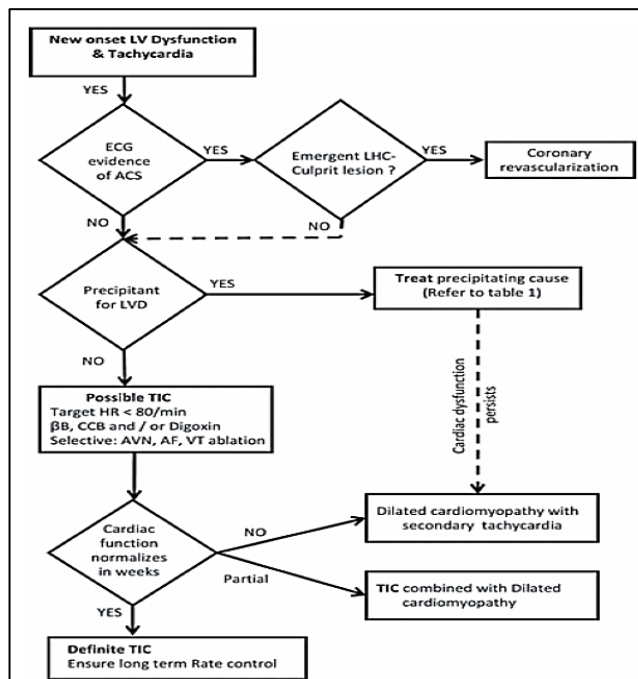
Figure 6. Stepwise ECG algorithm for determination of accessory pathway location⁷

Pambrun T. *et al*. (2018) provided an update on a new ECG algorithm for localizing manifest accessory pathways (AP) in adults, which is based on maximal pre-excitation and allows for accurate and reproducible localization.⁸ According to this updated algorithm, the accessory pathway in our patient was identified as Right Anterior (RA), which was consistent with the findings from the electrophysiological study.

Tachyarrhythmia Induced Cardiomyopathy

Various studies have employed different terms to define tachycardia-induced cardiomyopathy (TIC), including pacing-induced cardiomyopathy, tachycardiomyopathy, and arrhythmia-induced cardiomyopathy, leading to

some confusion in clinical understanding. Furthermore, significant variations in the definition of TIC in the medical literature have complicated its universal comprehension and hindered consensus on diagnostic guidelines and clinical management. A widely accepted definition describes TIC as a reversible cardiac condition marked by ventricular systolic and/or diastolic dysfunction occurring alongside chronic (incessant or persistent) atrial, supraventricular, or ventricular tachyarrhythmias.⁹ In our case presentation, there were no recorded episodes of tachycardia prior to the induced intra-ablation, aside from instances of orthodromic AVRT.



Category	Specific Type of Tachycardia
Supraventricular	Atrial fibrillation/flutter
	Atrial tachycardia
	Permanent junctional reciprocating tachycardia (PJRT)
	Atrioventricular (AV) nodal re-entrant tachycardia (AVNRT)
	AV reentrant tachycardia (ART)
Ventricular	Inappropriate sinus tachycardia (a very rare cause)
	Right ventricular (RV) outflow tract (RVOT) ventricular tachycardia (VT)
	Fascicular tachycardia
Ectopy	Bundle branch re-entry ventricular tachycardia
	Premature ventricular complexes
Pacing	Persistent rapid ventricular pacing
	High-rate pacing
Other	Thyrotoxicosis (sinus tachycardia/atrial fibrillation)

Figure 7. (left) Diagnostic Algorithm for TIC Based on Clinical Presentation, Adapted from Lishmanov *et al* (2010); (right) Types of Tachyarrhythmias Associated with Tachycardia-Induced Cardiomyopathy, Adapted from Gupta *et al* (2014)⁹

A review article by Albakri A. (2018) summarized that from Donghua *et al* (2013) studied a cohort of 625 patients referred for radiofrequency ablation of tachyarrhythmias and found 2.7% had TIC. The incidence for specific arrhythmias has been reported between 10% for focal atrial tachycardia, 20% to 50% for permanent junctional reciprocating tachycardia and 25% for chronic atrial flutter. The incidence of AF-associated TIC ranges between 10% and 50% in HF patients. In patients with frequent ventricular tachycardia referred for electrophysiological evaluation, the incidence of TIC was 9% to 34%.⁹

The rate and duration of tachyarrhythmias impact how quickly tachycardia-induced cardiomyopathy (TIC) develops, with left ventricular (LV) dysfunction

potentially occurring as soon as 24 hours after the onset of tachyarrhythmia. The underlying mechanism may resemble that seen in premature ventricular contraction (PVC)-induced cardiomyopathy, which has gained recognition in recent years. Studies have established a link between a higher burden of PVCs and the development of cardiomyopathy. For example, Baman *et al.* found that a PVC burden of 24% in otherwise healthy patients yielded a sensitivity of 79% and specificity of 78% for diagnosing PVC cardiomyopathy.² In our case, the patient reported that her heart rhythm disturbance was identified only two years ago due to frequent episodes of palpitations and easy fatigue. She mentioned having experienced palpitations prior to that, although they were infrequent.

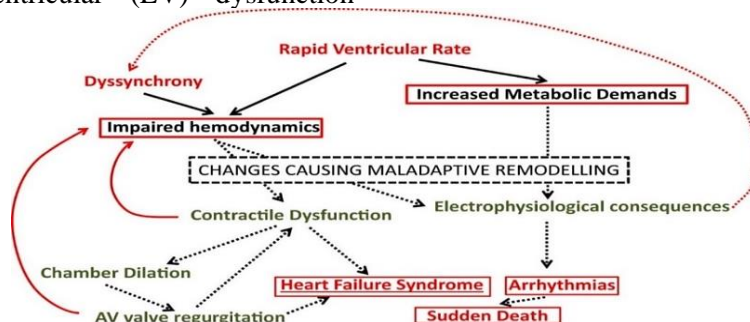


Figure 8. Mechanisms and clinical significance of Arrhythmia-Induced Cardiomyopathy¹⁰

The mechanism behind pre-excitation induced cardiomyopathy may involve ventricular dysfunction caused by non-physiological activation of the ventricular myocardium through the accessory pathway, similar to findings in patients with permanent pacemaker implantation. This type of cardiomyopathy, linked to the pacing site, is associated with structural changes in the ventricular myocardium. The eccentric activation through the accessory pathway can mimic cardiomyopathy due to the asynchronous spread of the depolarizing current. Furthermore, cardiomyopathy involving structural changes in the myocardium is unlikely to reverse quickly.¹¹

Pre-Excitation Syndrome and Cardiomyopathy

LV dysfunction has been described in patients with accessory pathways (AP). Although prospective studies are lacking, small case series show that LV dysfunction in these patients may progress over time. Recognizing pre-excitation as a cause for LV dysfunction is pertinent as it is reversible. LV dysfunction in patients with WPW may be secondary to tachyarrhythmias, either symptomatic or asymptomatic, that trigger a TIC.²

Compared to other causes of cardiomyopathy that lead to systolic dysfunction, such as ischemic heart disease, long-standing hypertension, and valvular disease, tachycardia-induced cardiomyopathy (TIC) is less recognized. Furthermore, TIC has not been systematically studied in relation to atrioventricular reentrant tachycardia (AVRT). A prospective study by Nagai T. et al. (2017) involving 30 adults (average age 40 ± 12 years) diagnosed with WPW found that these patients exhibited impaired left ventricular ejection fraction (LVEF). After undergoing radiofrequency ablation (RFA), they showed significant improvements in ejection fraction, myocardial torsion, and dyssynchrony.¹²

In a case report by Young S. *et al* (2021), they reported a case of tachycardia-mediated cardiomyopathy in a patient with WPW syndrome with low EF that was normalized with ablation. Given the location of the accessory pathway, the cardiomyopathy may have resulted from the dyssynchrony of ventricular activation similar to a left bundle branch block (LBBB) induced cardiomyopathy.¹³ As indicated by the ECG analysis, the accessory pathway in our patient was localized to the right anterior region. The pre-ablation 12-lead ECG showed a wide QRS complex with multiple delta waves, reflecting a condition of maximal preexcitation. These

findings were further confirmed by the results of the electrophysiological (EP) study.

Most studies have demonstrated a reversal of left ventricular (LV) dilation and restoration of LV function following radiofrequency ablation (RFA). Cardiac function recovery after RFA occurs over a variable time frame, with reports ranging from a few weeks to as long as 1.5 years. Notably, a septal or paraseptal accessory pathway has been identified in most patients with overt ventricular pre-excitation who developed LV dysfunction or dilated cardiomyopathy (DCM) without recurrent tachyarrhythmias. This suggests that the location of the accessory pathway plays a significant role in the development of these conditions.⁴

A study by Atta S. et al. (2013) found that among mostly asymptomatic or minimally symptomatic patients with manifest pre-excitation, 50% (24 out of 48) had left ventricular (LV) dysfunction that could not be attributed to tachyarrhythmias. Of these, two patients had severe LV dysfunction with an ejection fraction (EF) below 30%. While clinically significant arrhythmias are commonly observed in individuals with pre-excitation syndrome, LV dysfunction is a less frequent occurrence. The study also highlighted that recurrent and sustained tachyarrhythmias can lead to dilated cardiomyopathy, known as tachyarrhythmia-induced cardiomyopathy (TIC).⁴

Based on their study data, echocardiographic assessment showed that LV dysfunction (with LVEF < 55%) was present in 24/48 included patients (50%). The overall LV systolic function was impaired (EF < 55%) in 4 (17.4%) patients with left lateral APs vs 20 (80%) patients with septal accessory pathways ($p = 0.0001$). They found that over all LV systolic function was significantly less in patients with septal accessory pathways than in patients with lateral APs whether left or right. The prevalence of patients with LV dysfunction was significantly higher among patients with septal than with lateral AP. The two patients with severe LV dysfunction were found to have septal APs.⁴

An analysis by Emmel M. et al. (2004) suggested a link between the WPW pattern on ECG and cardiomyopathy occurring without arrhythmic events. For instance, among four children with left ventricular (LV) systolic dysfunction and a WPW pattern, only one patient exhibited documented tachycardia. Notably, two of the four patients saw their cardiomyopathies resolve after pre-excitation was addressed through ablation. A similar

study by Tomaske M. et al. (2008) involving 34 patients (average age 14.2 ± 2.5 years) found that WPW with a right septal or posteroseptal accessory pathway was associated with reduced left ventricular ejection fraction (LVEF), which improved following radiofrequency ablation (RFA). Importantly, none of the participants in their study had arrhythmias prior to RFA, leading the authors to conclude that tachycardia-mediated cardiomyopathy (TIC) was unlikely to be the cause of the LV dysfunction.^{11,14}

In addition, a conclusion from the study by Lee H. *et al* (2020) stated that Right-sided APs with prominent preexcitation may result in a LBBB pattern, LV dyssynchrony, and Non-Ischaemic Cardiomyopathy (NICM) even in the absence of tachycardia event. Several case reports and small case series have described non-tachycardia-mediated cardiomyopathy in pediatric patients from dyssynchrony owing to ventricular preexcitation, particularly from right-sided APs. They suggest that if there is an evidence of ventricular preexcitation, catheter ablation should be considered even in the absence of tachyarrhythmias, as successful ablation of the AP may cure not only the LBBB, but the cardiomyopathy also.³ For this case presentation, we did a similar procedure in NCCHK in the hope of restoring the LV function, not only for curing the LBBB / dyssynchrony. EGM showed a retrograde block, a sign of successful RFA ablation for AP in WPW syndrome. Accordance to 12 lead ECG result, it revealed no delta waves or LBBB pattern anymore.

Another study by Marti-Almor J. et al. (2011) demonstrated that a right-sided accessory pathway (AP) can lead to cardiomyopathy. Their findings concluded that preexcitation through a right antero-septal AP may cause heart failure due to left ventricular dysfunction and intraventricular asynchrony, resembling the effects of left bundle branch block (LBBB). Following ablation of the AP, heart failure symptoms improved, and myocardial contractility normalized. In their case report, the patient's functional class returned to normal three months after the procedure. An MRI conducted four months post-ablation showed normalization of the ejection fraction (63%) and no signs of asynchrony.¹⁵ Consistent with Marti-Almor *et al*'s study, our patient's echocardiography results significantly increased to 41% one year after successful AP ablation. We will continue to conduct periodic evaluations of the patient's clinical condition and echocardiography results.

SUMMARY

We present a case of a 12-year-old female diagnosed with pre-excitation syndrome (Right Anterior AP WPW Syndrome) and tachycardia-induced cardiomyopathy. Following the ablation of the accessory pathway, she experienced no further palpitations or shortness of breath, and there were no signs of complications. Long-term follow-up is still necessary to assess her clinical status and left ventricular function through echocardiography.

Some points to be learned from this case presentation :

1. Preexcitation from right-sided accessory pathways can lead to left ventricular dyssynchrony and contractile dysfunction.
2. Eliminating the accessory pathway may provide benefits even in the absence of atrioventricular reentrant tachycardia or atrial fibrillation.
3. Radiofrequency ablation of accessory pathways has been shown to normalize QRS duration, achieve mechanical resynchronization, and potentially enhance left ventricular function.

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