

THE EFFECT OF VENTRICULAR PRE-EXCITATION IN WPW SYNDROME PATIENTS, ON LEFT VENTRICULAR WALL MOTION AND SYSTOLIC FUNCTION, PRE AND POST RADIOFREQUENCY ABLATION OF ACCESSORY PATHWAY

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ABSTRACT

Objective: To demonstrate dys-synchrony between interventricular septal motion and left ventricular posterior wall motion in Wolff-Parkinson-White syndrome (WPW) patients resulting in left ventricular (LV) systolic dysfunction and its improvement assessed on post radiofrequency ablation.

Study design: Cross-sectional prospective study

Place and Duration of Study: Department of Cardiac Electrophysiology, Armed Forced Institute of Cardiology/National Institute of Heart Diseases, Rawalpindi Pakistan, from Oct 2020 to Mar 2021.

Methodology: Three groups of patients with different accessory pathway (AP) location using surface ECG delta wave in Wolf-Parkinson-White syndrome were made. The base line echocardiographic features of left ventricle as left ventricle ejection fraction (LVEF), regional structural changes and regional wall motion abnormalities (RWMA) were taken using 2D mode. The time from systolic posterior septal motion to early systolic posterior wall motion with reference to onset of QRS was calculated using M-Mode echocardiography in these three groups of patients with White syndrome before ablation and post radiofrequency ablation.

Results: Out of 38 patients, 17 (44.71%) patients were having right free wall (group 1), 12 (31.6%) patients were having right postero-septal (group 2) and 9 (23.7%) patients were having left free wall (group 3) accessory pathways (accessory pathways). There were significant differences in septal to posterior wall motion delay seen between the three groups ($p < 0.05$) and was very much higher in group 1 ($p < 0.05$). After ablation, left ventricle ejection fraction was raised very much in patients having right-sided free wall and post-eroseptal accessory pathway as compared to left sided free wall accessory pathway ($p < 0.05$) and LV diastolic dimension was decreased more in right sided post-eroseptal accessory pathways ($p < 0.05$).

Conclusion: Right-sided accessory pathway cause ventricular wall motion and left ventricle systolic function to be reduced, resulting in a lower left ventricle ejection fraction and a larger left ventricle end-diastolic size. These effects, including left ventricle dys-synchrony was very much improved after radiofrequency ablation

Keywords: Electrophysiological study, Echocardiographic evaluation, Radiofrequency ablation, Ventricular pre-excitation.

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INTRODUCTION

Wolff-Parkinson-White (WPW) syndrome is a ventricular pre-excitation and is a congenital condition and there is an abnormal accessory pathway (AP) between the atrium and ventricle that by passes the atrio-ventricular node present in these patients and the electrical stimulus then passes over this AP to cause ventricular depolarization^{1,2}. Thus an electrical stimulus passes directly from an atrium to a ventricle, resulting in the premature activation and later contraction of that ventricle, close to insertion of accessory pathway when in sinus rhythm. This premature contraction results further in myocardial dyskinesia. These abnormal prematurely activated segments close to insertion of AP, has been observed by echocardiography, using M-

mode and 2D mode³. As recently hypothesized, dyskinetic segments in patients with WPW syndrome, working similar to an aneurysm, might cause intraventricular hemodynamics changes, leading to progressive ventricular enlargement and cardiac dysfunction. Several studies have recently revealed a direct link between WPW syndrome and heart failure, regardless of the presence of recurrent or long-term tachyarrhythmias⁴⁻⁶. Most commonly a recurrent or sustained tachyarrhythmia, can cause the morbidity of heart failure in WPW syndrome patients⁷⁻¹⁰.

Because it is commonly linked with supraventricular tachycardia, this syndrome is also clinically significant. Further more, ventricular tachyarrhythmia caused by atrial fibrillation with rapid anterograde conduction through the AP may result in sudden cardiac death¹¹. In addition, heart failure in the WPW syndrome patients is more linked to the septal AP and has

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a reversible course having a temporal relation as seen after treatment with radiofrequency ablation (RFA) of the septal AP¹¹.

Abnormal septal motion has additionally been demonstrated echocardiographically in patients with other conditions such as coronary artery disease, right ventricular volume overload and conduction abnormalities such as left bundle branch block¹²⁻¹³.

According to recent literature, such left ventricular dysfunction secondary to non-physiologic activation after the implantation of pacemakers, prompting to heart failure, has also been reported. Anatomic classification of accessory pathway (fig-1).

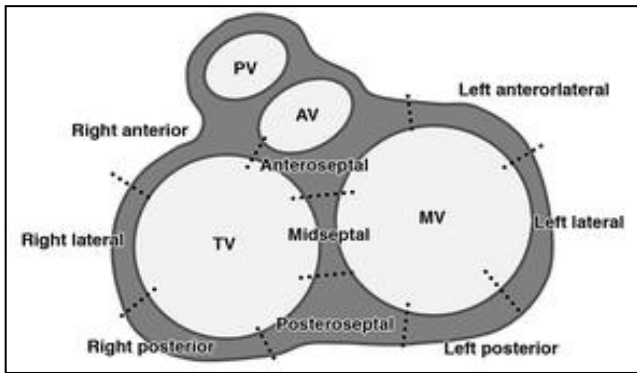


Figure-1: Classification of accessory pathway is based on anatomic position, defined with atrioventricular rings and surrounding structures in one plane.

Groups of patients with different location of accessory pathway named on basis of surface ECG, using delta wave.

Group-I: In ECG, positive delta wave in I, aVL and V₆; more positive QRS deflection in I, II, aVL and V₆; and predominant negative QRS deflection in V₁. The pre-excitation site is considered in the free wall of right ventricle (This includes no. of patients with antero-lateral and postero-lateral AP).

Group-II: In ECG, positive delta wave in I, aVL, and biphasic with predominantly negative in V₁; more negative QRS deflection in II, III, and aVF, and predominant positive QRS deflection in aVL and V₂. The accessory pathway is in the postero-septal of right ventricle.

Group-III: This group includes the patients with AP in the free wall of left ventricle (Most patients having postero-lateral and lateral wall AP and few patients having anterolateral AP).

Postero-lateral of left ventricle: Negative delta wave in II and aVF and positive in V₁; negative QRS deflection in aVL leads of ECG.

Lateral of left ventricle: Negative delta wave in I and aVL and more positive QRS deflection in V₁ and V₂ leads of ECG.

Antero-lateral of left ventricle: More positive QRS deflection in II, III, and aVF, with biphasic QRS forces in I and V₁ leads of ECG¹⁷.

METHODOLOGY

The three groups of WPW syndrome patients with different accessory pathway (AP), as mentioned above, using delta wave on surface ECG were made. The patients complaining of recurrent palpitation with shortness of breath and on echocardiography having mild LV systolic dysfunction (EF <60% and >45%), were selected. The patients having chest pain with sweating or known case of ischemic heart disease and congenital structural heart diseases were excluded. The patients having moderate to severe LV systolic dysfunction or known case of dilated cardiomyopathy were also excluded. Disappearance of AP was noted in all patients on post radiofrequency-ablation ECG with narrowing of QRS. The improvement of LV systolic function and dimension whether it was present or not present on post-ablation, were recorded.

Echocardiograms were performed with the use of a commonly available Echo ultrasound Machine. The base line echocardiographic features of LV, using M mode and 2D mode was collected by an experienced observer. The localization of AP, using Electro-physiological studies (EPS) were confirmed before radio frequency ablation (RFA). All patients were studied in sinus rhythm with controlled heart rates. The overall LV function was calculated using end-diastolic and end systolic LV dimensions in parasternal long and short axis views. The ejection fraction was measured using the biplane Simpson formula. The regional wall motion abnormality (RWMA) and regional structural changes was also evaluated from the apical four-chamber or parasternal short-axis views at end-systole and end-diastole. The time was taken from onset of QRS in ECG to peak systolic posterior septal motion on M-mode and was compared to the time taken from onset of QRS in ECG to peak systolic posterior wall motion on M-mode (fig-2). Care was taken to record septal motion below the level of the mitral valve where the inferior portion of the interventricular septum normally moves in a posterior direction during systole. The time delay from septal to posterior wall motion of >40 ms indicates interventricular dys-coordination and time delay from onset of QRS to peak posterior wall motion du-

ring systole of >150ms indicates LV dys-synchrony¹⁰ (fig-2).

Pre-RFA, the mean value of left ventricle end diastolic dimension (LVEDD, left ventricle ejection

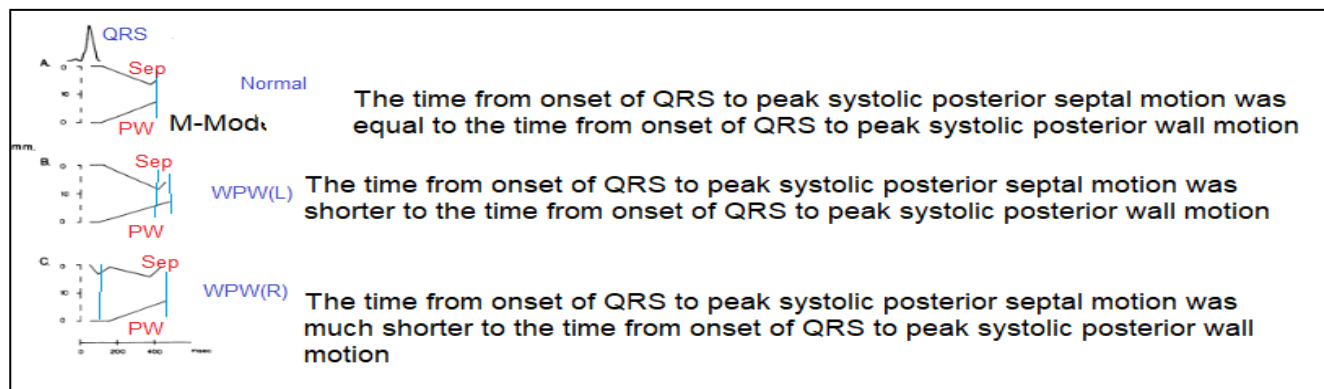


Figure-2: Schematic diagram of M-Mode synchronized with ECG showing different groups.

Standard electrophysiological study procedure was used. A 7 French, ablation catheter having distal electrode of 4mm width was used for RFA treatment with a setting of 30Watt radiofrequency current and a temperature of 60C. The endpoints of ablation was loss of the ante-grade and retrograde conduction over AP with loss of delta wave on surface ECG. The right-sided AP, ablation were performed using long sheaths in some patients to stabilize the ablation catheters. After RFA, the conducting system of heart was re-evaluated for induction of SVT or presence of residual anterograde or retrograde conduction.

All echocardiographic characteristics were recorded and compared in these three groups of WPW patients, before ablation and post radiofrequency ablation at about after one week. Mean and standard deviation was calculated for continuous data while frequency and percentage was calculated for categorical data. Inferential analysis was done by paired t-test (pre and post RFA study).

RESULTS

A total 38 patients prospectively included in the study. Out of these patient, 17 (44.7%) patients were having right free wall (group 1), 12 (31.6%) patients were having right postero-septal (group 2) and 9 (23.7%) patients were having left free wall (group 3) accessory pathways. The mean age of patients was 52.53 ± 8.43. There was 23 (60.5%) male and 15 (35.5%) female patients. All patients (n=38) were successfully ablated by RFA for accessory pathways with disappearance of delta wave and getting narrower QRS. Patients’ demographic and clinical characteristics are shown in table-I.

fraction (LVEF), septal to posterior wall motion delay (SPWMD), systolic posterior septal motion (SPSM) and systolic posterior wall motion (SPWM) was 50.16 ± 2.97 mm, 53.50 ± 3.67%, 60.76 ± 20.83ms, 70.66 ± 20.29 ms and 140.45 ± 40.88ms respectively. Post-RFA, the mean value of Left ventricle end diastolic dimension (LVEDD, left ventricle ejection fraction (LVEF), septal

Table-I: Three Groups of patients with different location of accessory pathway (AP).

Total (n=38)	n(%)
Group 1, Right free wall AP	17 (44.7)
Group 2, Right Postero-septal AP	12 (31.6)
Group 3, Left free wall AP	9 (23.7)
Gender	
Male	23 (60.5)
Female	15 (39.5)
Presence of LV RWMA	55.3

Table-II: Socio-demographic characteristics.

Socio-Demographic Characteristics		
Variables (n=38)	Mean ± SD	
Age (year)	52.53 ± 8.437	
Pre RFA	LVEDD Pre-RFA (mm)	50.16 ± 2.973
	LVEF Pre-RFA (%)	53.50 ± 3.674
	SPWMD Pre-RFA (mille seconds)	60.76 ± 20.833
	QRS to SPSM Duration Pre-RFA (mille seconds)	70.66 ± 20.293
	QRS to SPWM Duration PRE-RFA (mille seconds)	140.45 ± 40.881
Post RFA	LVEDD Post-RFA (mm)	46.97 ± 3.545
	LVEF Post-RFA (%)	58.185 ± 3.229
	SPWMD Post-RFA (mille seconds)	30.26 ± 10.131
	QRS to SPSM Duration Post-RFA (mille seconds)	60.76 ± 20.205
	QRS to SPWM Duration Post-RFA (mille seconds)	100.0 ± 20.61

to posterior wall motion delay (SPWMD), systolic posterior septal motion (SPSM) and systolic posterior wall motion (SPWM) was $46.97 \pm 3.54\text{mm}$, $58.185 \pm 3.22\%$, $30.26 \pm 10.13\text{ms}$, $60.76 \pm 20.20\text{ms}$ and $100.0 \pm 20.61\text{ms}$ respectively, table-II.

Table-III shows the systolic functions of LV in all the three groups before ablation. LVEF (Left ventricle Ejection Fraction) was significantly different between the three groups ($p < 0.05$). LVEF was significantly lower in patients with right-sided free wall AP (group 1) and right-sided postero-septal AP (group 2) than in patients with left-sided free wall AP (group 3), ($p < 0.05$).

Table-III: Pre-Ablation LVEF (Left ventricle Ejection Fraction) in all three groups.

Groups		n	LVEF (%)		p-value
Group 1, Right free wall AP		17	52.06 ± 2.839		0.002
Group 2, Right Postero-septal AP		12	52.83 ± 3.380		
Group 3, Left free wall AP		9	57.11 ± 2.839		
Not Improved	47.40 ± 1.94	55.40 ± 4.21	30 ± 0.7	50.8 ± 10.7	80.8 ± 20.3
p-value	0.01	0.030	1	0.055	0.07
Left free wall Accessory Pathway	Mean \pm SD				
n (%)= 9 (23.7)	LVEDD (mm)	LVEF (%)	SPWMD (mse)	QRS to SPSM (msec)	QRS to SPWM (msec)
Improved	45 ± 2.72	60 ± 1.414	20 ± 0.70	50 ± 0.88	70 ± 0.92
Not Improved	48 ± 2.72	59.50 ± 1.71	20.75 ± 0.7	40 ± 0.88	70 ± 0.92
p-value	0.25	0.749	0.351	0.798	0.626

During LV ejection the posterior motion of the septum and the anterior motion of the posterior left ventricular wall were nearly synchronous in left posterolateral accessory pathway and were not different from those found in normal subjects. There was impaired RWMA and dys-synchrony seen in right anterolateral and postero lateral APs. We had noticed 3 types of abnormal ventricular septal motion: 1) decreased interventricular septal motion. 2) the movement of the posterior wall of LV and the ventricular septum in same direction and 3) misplacement of the posterior LV wall and interventricular septum, meaning they moved in neither the same direction nor the opposite direction.

Post-RFA, overall left ventricle regional wall motion analysis had improved in 32 (84.2%) patients ($p < 0.05$) and septum to posterior wall motion delay had decreased in 26 (68.4%) patients ($p < 0.05$).

After ablation, LVEF was raised significantly more in patients with right-sided free wall APs (group 1) and postero-septal APs (group 2) as compared to left sided free wall APs (group 3) ($p < 0.05$) and LV diastolic dimension was decreased in right sided postero-septal AP (group 2) ($p < 0.05$). There were much differences in septal to posterior wall motion delay seen between the three groups ($p < 0.05$) and was significantly decreased in right free wall AP (group 1) ($p < 0.05$).

In some extreme cases, 2D echocardiography has shown that the interventricular septum's basal section moved just like an aneurysm, resulting in bulge at end of systole.

DISCUSSION

In manifest WPW syndrome, premature activation of myocardial segments were found near to the ventricular insertion of the AP. These myocardial abnormalities was seen by echocardiography, using M-mode and 2D mode. The amount of evidence that has published is limited, and previous investigations have been retrospective.

Kwon *et al*⁶, had also evaluated the echocardiographic parameters in 62 patients with WPW syndrome before and after radiofrequency ablation, to document how much the AP contributed to overall LV dysfunction. They found that the right sided septal AP subgroup had a lower LVEF compared to the right-sided and left-sided free wall APs subgroups.

In ours study, we had noticed a critical connection between AP insertion and abnormal LV RWMA prompting LV systolic dysfunction and eventually showing impact on left ventricular (LV) systolic function seen by echocardiography. But, our results showed that right-sided free wall APs had adversely negative impact on ventricular wall motion and left ventricular function more than septal APs. We hypothesized that the degree of LV dys-synchrony and abnormal ventricular septal motion were important predictors of LV dysfunction and remodeling. The pathophysiology that cause LV dysfunction and finally remodeling, in patients with right-sided AP have not yet been thoroughly understood. As timing is significant for coordinated ventricular contraction and regional cardiac function, a delay in the electrical signal activation and this

finally electromechanical dissociation of a myocardial segments may add to cardiac pump inefficiency⁹⁻²⁰.

There is arising proof that LV dys-synchrony upsets myocardial wall stress and regional workload resulting in wall motion abnormalities, alteration in the blood flow in coronaries, regional molecular abnormalities, LV cavity enlargement and dysfunctions. This asymmetrical LV wall thickness and volume changes². The pre-excited myocardial segment thins out over time, which is thought to be linked to decreased workload and hypotrophy^{2,3}.

A dys-coordinated septal segment of myocardium may work similar to an aneurysm. Patients with LV dysfunction caused by ventricular pre-excitation in WPW, may complain of easy fatigability or may have reduce exercise tolerance. A few patients were asymptomatic with fair to normal LV systolic function although LV dys-synchrony was present. In our study, all patients except only one patient, were complaining of palpitation with paroxysmal SVT. This one patient was complaining daily chest discomfort and easy fatigability during the occurrence of SVT. The echocardiography had shown dys-synchronous interventricular septal movement during even sinus rhythm. This could explain why symptoms exaggerate even when LV systolic function and size are both normal during arrhythmia episodes.

LIMITATION OF STUDY

LVEF was calculated in our study utilizing the biplane Simpson's formula which was found to be most reliably calculated by 2D echo. However, the abnormal ventricular wall motion may influence more or less the measurement of LVEF. So, Cardiac MRI could help and resolve this problem. But for financial reasons, cardiac MRI was not adopted. In our further study, we may use MRI.

CONCLUSION

Right-sided APs results in impeded ventricular segmental wall motion and LV systolic function, that results in ultimately decreased LVEF and increased LV end-diastolic measurements. These impacts, including LV dys-synchrony, had settled after RFA. A right-sided free-wall APs have more unfavorable impacts than a septal APs. The pathophysiology of LV dysfunction and ultimately remodeling seems to be caused by left ventricular dys-synchrony and abnormal interventricular septal motion. The prognosis of AP induced abnormal ventricular segmental wall motion and LV dysfunction, after ablation was superb.

CONFLICT OF INTEREST

This study has no conflict of interest to be declared by any author.

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Ventricular Pre-Excitation in WPW Syndrome Patients

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