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Evaluation of QRS, QTc, and JTc intervals in Congenital Heart Disease with Pulmonary Hypertension

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Abstract

Background

Pulmonary hypertension (PH) in congenital heart disease affects the patient's prognosis. Prolonged QRS and QTc intervals in ECG may intensify life-threatening dysrhythmia in patients. We aimed to investigate the correlation between QRS, QTc, and JTc intervals prolongation in ECG with PH in Congenital Heart Disease (CHD) patients.

Materials and Methods: This cross-sectional study was performed in the pediatric cardiology clinic of Be'sat Hospital in Hamadan, Iran, in 2016-2018. Patients with CHD and PH were compared with CHD patients without any evidence of PH as the control group. Afterward, QRS, QTc, and JTc intervals in ECG, RV MPI, and TAPSE echocardiography were compared between the case group (PH group) and the control group. We also compared the ECG and echocardiographic results between mild and severe PH patients in the case group.

Results: In this study, 40 patients in the case group (with CHD and PH) were compared to 40 patients in the control group (only CHD without PH). There was a significant difference in QRS (p=0.005) and QTc (p=0.036) intervals between the two groups, but no significant difference in the JTc interval was observed. Of 40 patients with PH, 19 were in the mild PH subgroup, and 21 were in severe PH subgroup, in which 9 patients had irreversible PH or Eisenmenger syndrome (ES). QTc (p<0.001) and QRS (p=0.018) intervals in the severe PH subgroup with ES were significantly different from the mild PH subgroup, but the JTc interval was not significantly different.

Conclusion

Despite longer QRS and QTc intervals in the PH group of CHD, JTc interval did not show a significant prolongation.

Key Words: Children, Congenital heart disease, Pulmonary hypertension, QTC interval, JTC interval.

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1- INTRODUCTION

One of the problems associated with congenital heart disease is an increase in pulmonary hypertension, which is commonly seen in large left-to-right shunts (1, 2). If pulmonary hypertension is left untreated in the early stages, an irreversible complication called Eisenmenger syndrome may occur (3, 4). The increase in pulmonary hypertension in the long term can lead to right ventricular dysfunction and irregular heart rhythm, which can be observed through electrocardiography (5-7).

The increased pulmonary hypertension with subsequent right ventricular dilatation and mechanical changes can lead to dangerous arrhythmias (8). A correlation between QRS interval prolongation and right ventricular dysfunction has been reported in children (10) even though the cause of some ECG interval disturbances in children is unknown (9). Studies have reported the relationship between increased pulmonary pressure and prolongation of the QT interval related to the ventricular diastole (10-12).

Considering that patients with congenital heart disease and severe pulmonary hypertension have a progressive irreversible right ventricular and pulmonary malfunction, comorbid pulmonary hypertension and diastolic phase abnormality may exacerbate the risk of life-threatening ventricular arrhythmia (11). In addition to the QTc interval, the JTc interval is considered a practical ECG marker for diastolic phase evaluation, especially in the QRS abnormality setting (13, 14). We used these ECG results to evaluate pulmonary hypertension severity.

2- MATERIALS AND METHODS

2-1. Study design and population

In this comparative cross-sectional study, patients with definite diagnoses of CHD with pulmonary hypertension (as the case group), and CHD patients without any evidence of pulmonary hypertension (PH) (as the control group) in the age range of 1 month to 15 years were compared. The study was conducted in Be'sat hospital in Hamadan, Iran, during 2016-2018. Subjects were selected based on their medical history such as echocardiography and angiography results.

2-2. Method

Patients with PH were reevaluated by physical examination, pulse oximetry, ECG, and echocardiography. If a patient had a left to right shunt or congenital great vessels anomaly, pulmonary arterial pressure (PAP) was estimated by echocardiography. At the same time, we recorded systolic, diastolic, and mean systemic blood pressure. The ratio of mean pulmonary pressure to mean systemic pressure was calculated. We measured right ventricular performance index (RVMPI) and Tricuspid annular plane systolic excoriation (TAPSE) in echocardiography, and QTc, JTc, QRS intervals in ECG. If a patient had a history of cardiac catheterization and angiography, the mean PAP was recorded and entered into a questionnaire.

2-3. Laboratory measurements

Through Maylab 60 echocardiography device based on TR, PI, PDA, VSD gradient by Bernoli formula ($PG=4V^2$) pressure gradient was estimated. TR gradient was used for systolic, and PI for diastolic PA pressure assessment. If the patients had VSD or PDA, the VSD gradient was used for systolic pressure. The PDA gradient for systolic and diastolic pressures was used for mean pulmonary artery pressure estimation. Pulmonary artery pressure was estimated by reduction of these gradients from systolic and diastolic systemic pressure. Because IVC compliance in these patients was satisfactory, 5 mmHg was added to TR gradient for estimation of systolic PA

pressure. If systolic and diastolic PA pressure were simultaneously available, mean PA pressure (PAP) was estimated by two diastolic (PAP) + systolic PAP/3s. If mean pulmonary artery pressure (PAP) was calculable, mean PAP>25 mmHg was considered as pulmonary hypertension. Otherwise, a TR gradient of more than 37 mmHg was considered as systolic PH, and the end diastolic PI gradient of more than 15 mmHg was considered as diastolic PH. RVMPI in patients with a-b/b formula was calculated using spectral Doppler with continuous Doppler from tricuspid and pulmonary valves, in which (a) is the time of closing to opening the tricuspid valve, (b) is the time of blood ejection from the pulmonary valves, and TAPSE is measured by M-mode echocardiography of anterior leaflet of the tricuspid valve. QTc interval was calculated with Bazett formula QT/√R-R in lead 2 of surface ECG, and QT interval was measured from the beginning of the Q wave to the end of the T wave. QRS time was measured from the start of the Q wave to the end of the S wave. JTc interval was calculated by QTc-QRS time.

2-4. Intervention

Patients in the pulmonary hypertension group (case group) were divided into mild and severe PH groups based on mean systolic and diastolic PA pressure in comparison to systemic mean systolic and diastolic pressures. The ratio of less than 1/2 was considered as mild PH, and the ratio equal or higher than 1/2 was considered as severe PH. In the severe PH group, patients who had definite Eisenmenger diagnoses based on previous tests (echocardiography, oximetry, and catheterism) were selected as a subgroup.

2-5. Ethical considerations

This study was registered in the ethics group in Hamadan University of Medical Science with the following code: IR.UMSHA.REC.1395.331.

2-6. Inclusion and exclusion criteria

Inclusion criteria were the age range of 1 month to 15 years and a definite diagnosis of congenital heart disease with pulmonary hypertension. Exclusion criteria were congenital heart disease associated with a non-cardiac congenital anomaly, syndromic and chromosomal anomaly, and other right ventricular anomalies such as pulmonary stenosis, Ebstein's anomaly, electrolyte- and drug-associated QTc abnormality, primary long QTc syndrome, history of heart surgery, and complete atrioventricular and trifascicular block.

2-7. Data Analysis

In this study, independent T-Test was used for comparing the mean of variables in the two groups. Pearson coefficient was used to evaluate the relationship between quantitative variables. The significance level in this study was considered more than 5%. We used SPSS software version 16.0 to analyze the data.

3- RESULTS

We compared 40 patients with pulmonary hypertension (case group) with 40 patients with CHD without any evidence of PH (control group). There was no significant difference in terms of age (p=0.107) and sex (p=0.182) between the two groups. In the case group, the minimum age was 45 days, and the maximum was 15 years. In the control group, the minimum age was 6 months, and the maximum was 14 years. In terms of sex, there were 21 male patients (52.5%) in the case group and 26 male patients (65%) in the control group. 19 patients (47.5%) were in the mild PH subgroup in which 11 patients were male (57.9%), and 21 patients (52.5%) were in the severe PH subgroup in which 10 patients were male (47.6%). In the severe PH subgroup (21 cases), 9 patients (42.8%) had Eisenmenger syndrome (ES), 4 (44.4%) being male. There was no

significant difference between the two groups in terms of sex (p=0.432). In the control group, the distribution of CHD was as follows: small ASD2 (n=10), moderate to large ASD2 (n=4), ASD1 (n=2), partial AVSD (n=2), small to moderate membranous VSD (n=10), muscular VSD (n=5), and PDA (n=7). The different kinds of congenital heart diseases with mild and severe PH and Eisenmenger syndrome are shown in **Table.1**. The comparative analysis between case and control groups in terms of ECG criteria and right ventricular function criteria is shown in **Table. 2**. As it has been shown in **Table.2**, there was a significant difference between case and control groups in terms of QRS time (p=0.009) and QTc interval (p=0.036), but there was no significant difference between the two groups in terms of JTc interval. RV MPI (p=0.006) and TAPSE (p<0.001) as myocardial function indices were significantly different between the two groups. There was also a significant correlation between case and control groups in terms of RVMPI (p=0.006) and TAPSE (p<0.001) (**Table.2**).

Table-1: Distribution of the CHD in the pulmonary hypertension and the Eisenmenger syndrome.

Variation of CHD in the Pulmonary Hypertension group(Case group)					
Mild PH (n=19)	Number	**Severe PH (n=21)**			
VSD	8	ES	n=9	NO-ES	**n=12**
PDA	6	VSD	4	VSD	**5**
P-AVSD	2	C- AVSD	2	PDA	**3**
ASD1	1	DTGA/VSD	1	C-AVSD	**2**
Sv ASD	1	APW	1	VSD/DORV	**1**
ASD2	1	TA	1	VSD/COA	**1**

ES: Eisenmenger syndrome, VSD: Ventricular septal defect, PDA: Patent ductus arteriosus, P-AVSD: partial AVSD, ASD: Atrial septal defect (Sv: sinus venosus, 1: primum, 2: secondum), AVSD: Atrio septal defect (C: complete, P: partial), APW: Aorta pulmonary window, TA: Truncus arterisus, ORV: Double outlet right ventricle, COA: Coarctation of Aorta.

Table-2: Comparison of ECG and Echocardiographic criteria between case and control groups.

Variables	Case (n=40) Mean ± Standard deviation	Control (n=40) Mean ± Standard deviation	P-value
Age (yr)	6 ± 4.9	4.4 ± 3.8	0.107
QRS (ms)	102 ± 20	92 ± 14	0.019
QTC (ms)	443 ± 19	434 ± 18	0.036
JTC (ms)	341 ± 23	341 ± 20	0.714
RV MPI	0.40 ± 0.16	0.32 ± 0.05	0.006
TAPSE (mm)	10.4 ± 2.2	15.4 ± 10.6	<0.001

ECG variable: QRS time-QTC: Corrected QT interval-JTC: Corrected JT interval (ms: millisecond). Echocardiography variable: RVMPI: Right ventricle myocardial performance index-TAPSE: Tricuspid annular plane systolic excoriation, Case group: Patients with definite diagnoses of CHD with pulmonary hypertension, Control group: CHD patients without any evidence of pulmonary hypertension.

By comparing mild PH and severe PH subgroups (**Table.3**), a significant difference was found only in terms of QRS time (p=0.017) between the two groups. QTc interval (p=0.068), and JTc interval (p=0.810) were not significantly different between the two groups (**Table. 3**). Further analysis in the severe PH group (Eisenmenger and non-Eisenmenger groups) compared to mild PH group showed that QTc and QRS intervals were only different between Eisenmenger patients group with mild PH group (**Table.4**); however, this difference was not significant between the non-Eisenmenger, and mild PH groups (**Table.5**). JTc had no significant difference between mild PH group and non-Eisenmenger group. It was also not significant between mild PH and Eisenmenger groups. RVMPI was 0.50 ± 0.31 in the Eisenmenger group 0.38±0.63 in the non-Eisenmenger group. Despite this increase in the Eisenmenger group, the difference was not statistically significant (p=0.28). Spearman correlation test showed that RVMPI was not correlated to QRS time (p=0.107), QTc interval (0.304), and JTc interval (p=0.786). In the pulmonary hypertension group, there was not any correlation between RVMPI and QRS time (p=0.364), QTc interval (p=0.601), and JTc interval (p=0.816).

Table-3: Comparison of ECG criteria between Mild PH and severe PH group.

Variables	Mild PH (n=19)	Severe PH (n=21)	P- value
QRS (ms)	94.2±16	109±24	0.017
QTC ms)	437±18.2	448±19	0.068
JTC (ms)	342±20	340±26	0.810

PH: Pulmonary hypertension.

Table-4: Comparison of the ECG criteria between mild PH group and Eisenmenger group.

Variables	Mild PH group, (n=19) Mean ± SD	Severe PH ES group (n=9), Mean ± SD	P-value
QTC	437 ± 18.2	461 ± 106	<0.001
QRS	94.2 ± 16	117 ± 23	0.018
JTC	342 ± 20	348 ± 20	0.495

ES: Eisenmenger syndrome, PH: Pulmonary hypertension, SD: Standard deviation.

Table-5: Comparison of the ECG criteria between Mild PH group and non-ES group.

Variables	Mild PH group, (n=19) Mean ± SD	Severe PH No ES group, (n=12) Mean ± SD	P-value
QTC	437 ± 18.2	438 ± 17	0.873
QRS	94.2 ± 16	103 ± 20	0.208
JTC	342 ± 20	335 ± 30	0.463

ECG: Electrocardiogram, ES: Eisenmenger syndrome, PH: Pulmonary hypertension, SD: Standard deviation.

4- DISCUSSION

In this study, we evaluated the correlation of QRS, QTc, and JTc intervals in pulmonary hypertension of CHD. It was found that QRS and QTc intervals in the PH group were longer than the control group, but the JTc interval did not show a significant difference between the two groups. Pulmonary hypertension is seen in congenital heart diseases with the left-to-right shunt, and can produce pulmonary vascular disease (1) and affect long-term prognosis (3). A known complication of pulmonary hypertension in congenital heart disease is an irreversible process called pulmonary vascular obstructive disease or Eisenmenger syndrome, which converts the left-to-right shunt into a large right-to-left shunt (4).

The severe pulmonary hypertension can give rise to progressive right ventricular dilatation, symptoms of right ventricular failure, fatal arrhythmia, and sudden death in the terminal stages of the disease (5-7), which is related to the ventricular systolic stage (8). When pulmonary hypertension progresses, by increasing the systolic component, the systolic/diastolic S/D ratio increases with the worsening of right ventricular function that was observed in echocardiography and evaluation during catheterization (10).

QT abnormalities correlated with the diastolic phase are also important contributors to the development of life-threatening arrhythmia and sudden death. Since a rodent study showed that right ventricular hypertrophy is effective in increasing QTc interval over long periods (15), the importance of prolongation of QTc in human PH becomes more apparent (16, 17). Hong-Liang et al. (2009) examined the relationship between QTc and ventricular arrhythmia and sudden death in pulmonary hypertension. They concluded that the increase in pulmonary hypertension in patients with severe PH is likely associated with QTc interval prolongation, and this prolongation in QTc interval in women has a significant relationship with pulmonary hypertension (16). Rich et al. (Chicago, February 2010) examined the relationship between right ventricular dysfunction and mortality prediction in long term QTc interval prolongation and pulmonary hypertension in patients receiving PH-specific therapy (17), together with the control group.

All the relevant criteria were measured, including QTc, QRS, heart axial deviation, type of heart block in the ECG, right ventricular end diastolic volume, right ventricular end systolic volume, and the RV ejection fraction using cardiac resonance imaging methods. They recorded the important NT-proBNP biomarker after the ECG of the patients. Other studies have also concluded that QTc interval prolongation and QT dispersion in patients is indicative of RV status and an independent factor for mortality and arrhythmia (13, 18).

The mentioned studies on patients with PH (19, 20) were supported by other studies on patients with other severe causes of right ventricular hypertrophy (21, 22). In our study, although QRS and QTc intervals were prolonged in the case group, there was no correlation between these ECG findings and right ventricular myocardial functions indices (RVMPI and TAPSE). Patients with congenital defects leading to severe PH and Eisenmenger syndrome have been associated with severe systolic dysfunction of the right ventricle in the terminal stages of the disease, and are at risk of sudden death and severe arrhythmia that is associated with QRS interval abnormality (23, 24). There have been reports of QT changes in patients with congenital heart defects, severe PH, and Eisenmenger syndrome (25, 26). Since the QTc interval is related to ventricular diastole, it can raise the risk of dangerous arrhythmia in patients with severe ventricular systolic dysfunction. In

our study, there was a significant prolongation in QRS interval in the PH group compared to the control group. Despite the significant difference between case and control groups, the QTc interval was not significantly different between the mild and severe PH subgroups (p=0.06).

QTc interval prolongation in the severe PH subgroup was related to Eisenmenger patients (461±106ms) compared to mild PH subgroup (p<0.001). Considering the fact that JTc interval was not significantly different between the two main groups and Eisenmenger patients, QTc interval measuring might be affected by QRS interval in these patients. It seems that, under this situation, JTc interval is more reliable for assessing diastolic phase changes (27). As QRS time was significantly prolonged in the severe PH subgroup, it can increase the risk of life-threatening arrhythmia; therefore, the existence of an additional risk factor, such as diastolic phase abnormality, can heighten the risk (28). Although in this study, JTc was not significantly different in all of the pulmonary hypertension patients group and control group, prolongation of JTc interval in the Eisenmenger patients versus normal population (29) is important.

In this study, there were no correlation between QTc and JTc intervals and RVMPI and TAPSE. Thus, QTc and JTc prolongation in these patients may be correlated to other causes of diastolic abnormalities. As QTc prolongation was reported in other fields of congenital heart disease management (30), we recommend simultaneous measurement of QTc and JTc in these settings together with cardiac function evaluation, using right ventricular myocardial function indices (31), and sensitive cardiac biomarkers (32, 33) in the future studies. It will improve the assessment of systolic and diastolic myocardial abnormalities and their effects on QRS, QTc, JTc intervals in congenital heart disease associated with severe pulmonary hypertension.

4-1. Study Limitations

Sensitive cardiac biomarkers such as NT-Pro-BNP might be more useful in examining myocardial function.

5- CONCLUSION

In patients with congenital heart disease and severe PH, despite prolongation of QRS and QTc intervals, the JTc interval did not show a significant increase. We suggest simultaneous measurement of JTc and QTc intervals in these settings.

6- ABBREVIATIONS

CHD: Congenital heart disease, PH: Pulmonary Hypertension, ES: Eisenmenger syndrome, VSD: Ventricular septal defect, PDA: Patent ductus arteriosus, ASD: Atrial septal defect (Sv: sinus venosus, 1: primum, 2: secondum), AVSD: Atrio septal defect (C: complete, P: partial), DORV: Double outlet right ventricle, APW: Aorta pulmonary window, COA: Coarctation of Aorta, TGA: Transposition of great arteries, TA: Truncus arterisus.

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8- CONFLICT OF INTEREST: None.

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