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Evaluation of QRS, QTC, JTC Intervals in Congenital Heart Disease with Pulmonary Hypertension

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Abstract

Background

Pulmonary hypertension (PH) in congenital heart disease (CHD) affects the patient prognosis. QRS and QTC intervals prolongation in ECG may exaggerate life-threatening dysrhythmia in these patients. We aimed to investigate the correlation between QRS, QTC and JTC intervals prolongation in ECG with PH in CHD children.

Materials and Methods

In a cross-sectional study that was performed in pediatric cardiology clinic of Besat hospital (Hamadan, Iran), during 2016-2018, patients with CHD and PH as case group (n=40) were compared to simple CHD patients without any evidence of PH as control group (n=40). Based on Pulmonary Artery (PA) to systemic pressure ratio, lower than 1/2 was considered as mild PH and equal and more than 1/2 was considered severe PH; then QRS, QTC, JTC intervals in ECG and RVMPI, TAPSE in echocardiography were compared between case (PH group), and control groups. We also compared these ECG and echocardiographic findings between mild and severe PH group.

Results

There was significant difference in QRS (p=0.005), and QTC (p=0.036) intervals between two groups, but there was not any significant difference between JTC interval between two groups (p=0.714). Of 40 patients with PH, 19 subjects were in the mild PH group and 21 subjects were in severe PH group, in which nine patients had irreversible PH or Eisenmenger syndrome. QTC (p<0.001) and QRS (p=0.018) intervals in the severe PH group with Eisenmenger syndrome were significantly different from the Mild PH group.

Conclusion

Based on the results, in spite of rising QRS and QTC intervals in the PH group of CHD, JTC interval did not rise significantly.

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

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1- INTRODUCTION

One of the problems associated with congenital heart disease is the increase in pulmonary hypertension, which is commonly seen in large left-to-right shunts (1, 2). If pulmonary hypertension is left untreated in early stage, irreversible complication called Eisenmenger syndrome may occur (3, 4). The increase in pulmonary hypertension in the long term can result in right ventricular dysfunction and irregular heart rhythm, and these changes can be observed by electrocardiography (5-7). The increased pulmonary hypertension and subsequent right ventricular dilatation and mechanical changes can lead to the appearance of a dangerous arrhythmia (8). Although the cause of some ECG intervals disturbance in children is unknown (9), the correlation between QRS interval prolongation and right ventricular dysfunction was reported (10). A few studies have reported the relationship between increase in pulmonary pressure and the prolongation in 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 abnormality, coexistence of pulmonary hypertension and diastolic phase abnormality may exacerbate life threatening ventricular arrhythmia occurrence risk (11). In addition to QTC interval, JTC interval was considered a good ECG marker for diastolic phase evaluation especially in the QRS abnormality setting (13, 14). We aimed to investigate the correlation between QRS, QTC and JTC intervals prolongation in ECG with PH in CHD Children.

2- MATERIALS AND METHODS

2-1. Study design and population

In this comparative cross-sectional study, patients with definitive diagnosis of CHD with pulmonary hypertension (as case group), and CHD patients without any evidence of pulmonary hypertension (PH) were considered as control group in the age range of 1 month to 15 years were compared in Hamadan Besat hospital, Iran, during 2016-2018. We selected the subjects based on previous documents such as echocardiography and angiography.

2-2. Methods

Physical examination, pulse oximetry, ECG and echocardiography reevaluated patients with PH. If the patients 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 plan systolic excoriation (TAPSE) in echocardiography, and QTC, JTC, QRS intervals were calculated in ECG. If patients had a history of cardiac catheterization and angiography, mean PAP was recorded, too.

2-3. Measuring tools and 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 was used for diastolic PA pressure assessment. If the patients had VSD or PDA, VSD gradient was used for systolic pressure and systolic and diastolic pressure gradient of PDA was used for mean pulmonary artery pressure estimation. Pulmonary artery pressure was estimated by reducing these gradients from systolic and diastolic systemic pressure. Since IVC compliance in these patients was good, 5 mmHg was added to TR gradient for estimation of systolic PA pressure. If systolic and

diastolic PA pressure was simultaneously available, mean PA pressure (PAP) was estimated by 2 diastolic (PAP) + systolic PAP/3. If Mean Pulmonary Artery Pressure (PAP) was calculable, mean PAP>25 mmHg was considered as pulmonary hypertension. Otherwise, TR gradient more than 37 mmHg was considered as systolic PH; and if the end diastolic PI gradient was more than 15 mmHg, it was considered as diastolic PH.

RVMPI in patients with a-b/b formula using Spectral Doppler with continuous Doppler was calculated from tricuspid and pulmonary valves, in which (a) is from the time of closing to opening the tricuspid valve, and (b) is the time of blood ejection from the pulmonary valves, and TAPSE was measured by M-mode echocardiography of anterior leaflet of 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 Q wave to the end of T wave. QRS time was measured from beginning of Q wave to the end of S wave. JTC interval was calculated by QTC-QRS time.

2-4. Intervention

Pulmonary Hypertension group (case group) was divided into mild and severe PH group 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 group and the ratio equal or higher than 1/2 was considered as severe PH group. In the severe PH group, patients who had definite Eisemenger diagnosis based on previous studies (echocardiography, Oximetry, Catheterism) were selected as patients sub-group.

2-5. Ethical consideration

Ethic group of Hamedan University of Medical Sciences with the following code approved this study: IR.UMSHA.REC.1395.331

2-6. Inclusion and exclusion criteria

Inclusion criteria were all patients one month to 15 years-old who had definite diagnosis of congenital heart disease with pulmonary hypertension. In addition, congenital heart disease associated with non-cardiac congenital anomaly, syndromic and chromosomal anomaly, and other right ventricular anomalies for example, pulmonary stenosis and Ebstein anomaly, electrolyte and drug associated QTC abnormality, primary long QTC syndrome, history of heart surgery, Atrio ventricular and complete bundle branch block were excluded.

2-7. Data Analyses

In this study, independent t-test was used for comparing the variables' means in two groups. In order to evaluate the relationship between quantitative variables, Pearson coefficient was used. The significance level in this study was considered less than 5%. We used SPSS software version 16.0 to analyze the data.

3- RESULTS

In this study, we compared 40 patients with pulmonary hypertension (case group) with 40 patients with CHD without any evidence of PH (control group). There was not significant difference in terms of age (p=0.107), and sex (p=0.182) between two groups. In case group the minimum age was 45 days and the maximum age was 15 years, and in the control group the minimum age was 6 months and the maximum age was 14 years. In terms of sex, in the case group, there were 21 males (52.5%) and in the control group there were 26 males (65%). The mild PH group had 19 patients (47.5%), 11 were male (57.9%) and severe PH group comprised 21 patients (52.5%), 10 of whom were male (47.6%), respectively. In the severe PH group (21 cases), 9 patients (42.8%)

had Eisenmenger syndrome and in this group 44.4% were male. There was not a significant difference between two groups in terms of sex (p=0.432). In the control group, 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), PDA (n=7). The different kinds of congenital heart diseases that were accompanied by mild and severe PH group and Eisenmenger syndrome group have been shown in **Table.1**. 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 significant difference between case and control groups in terms of QRS time (p=0.009), and QTC interval (p=0.036) but there was not a 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 two groups. There was 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 PH and the ES patients.

Variation of CHD in the Pulmonary Hypertension group (Case group)					
Mild PH (n=19)		**Severe PH (n=21)**			
VSD	Number=19	ES	Number=9	NO-ES	**Number=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**

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.

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

Variables	Case (n=40) Mean ± SD	Control (n=40) Mean ± SD	P-value
Age (year)	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
Right Ventricular MPI	0.40 ± 0.16	0.32 ± 0.05	0.006
TAPSE (mm)	10.4 ± 2.2	15.4 ± 10.6	<0.001

QTC: Corrected QT interval, MPI: Myocardial performance index, JTC: Corrected JT interval, TAPSE: Tricuspid annular plane systolic excursion, SD: Standard deviation.

In comparison between mild PH and severe PH groups (**Table.3**), we found just a significant difference in terms of QRS time (p=0.017) between two groups. QTC interval (p=0.068), and JTC interval (p=0.810) were not significant between two groups (**Table.3**).

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

Variables	Mild PH	Severe PH	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

QTC: Corrected QT interval, JTC: Corrected JT interval.

Further analysis in the severe PH group (Eisenmenger and non-Eisenmenger group) in comparison to Mild PH group showed that QTC and QRS intervals were only different between Eisenmenger patients group and Mild PH group while this difference was not significant between non-Eisenmenger group and Mild PH group (**Table.4**). JTC was not significantly different between mild PH group and non-Eisenmenger. It was not also significant between mild PH group and Eisenmenger group (**Table.4**). RVMPI was 0.50 ± 0.31 in the Eisenmenger syndrome group and it was 0.38±0.63 in non-Eisenmenger group. Despite an increasing of this criteria in the Eisenmenger group, this 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-4: Comparison of the ECG criteria between mild PH group and Eisenmenger group.

Variables	Mild PH (n=19) Mean ± SD	Severe PH Eisenmenger syndrome (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

QTC: Corrected QT interval, JTC: Corrected JT interval, SD: Standard deviation.

Table-5: Comparison of the ECG criteria between mild PH group and non-Eisenmengr group.

Variables	Mild PH, (n=19) Mean ± SD	Severe PH No Eisenmenger syndrome, (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

QTC: Corrected QT interval, JTC: Corrected JT interval, PH: Pulmonary Hypertension, SD: Standard deviation.

4- DISCUSSION

In this study, we evaluated correlation of QRS, QTC and JTC intervals in pulmonary hypertension of CHD in which QRS and QTC intervals in the PH group were longer than control group but JTC interval did not have significant difference between two groups. Pulmonary hypertension is seen in different types of congenital heart disease that can produce

pulmonary vascular disease (1), and affect long-term prognosis (3). Known complication of pulmonary hypertension in congenital heart disease with large left to right shunt is an irreversible process called pulmonary vessels obstructive disease or Eisenmenger syndrome that is associated to right to left shunt (4). The severe pulmonary hypertensioncan gives rise to progressive right ventricular dilatation and symptoms of right ventricular failure and 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 increase in the systolic/diastolic S/D ratio increases with the worsening of right ventricular function that was seen in the echocardiography and evaluation during catheterization (10). Since QT– related abnormalities correlated with diastolic phase are also a major contributor to the development of life threatening arrhythmia and sudden death. As a rodent study stated that right ventricular hypertrophy is effective in increasing QTC interval over long periods (15), the importance of prolonging QTC in human PH was raised (16, 17).

Hong-liang et al. (2009) examined the relationship between QTC and ventricular arrhythmia and increased sudden death and its relationship with increased pulmonary hypertension. They concluded that the increase in pulmonary hypertension in patients with severe PH is likely associated to QTC interval prolongation, and this prolongation in QTC interval in women has a significant relationship with pulmonary hypertension (16). Rich et al. in Chicago in February 2010 examined the relationship between right ventricular dysfunction and mortality prediction in long term QTC interval prolongation and pulmonary hypertension in the patients who had PH specific therapy (17). They included the control group in their study; and measured all the relevant criteria including QTC and QRS, heart axial deviation and type of heart block in the ECG and right ventricular end diastolic volume, and the right ventricular end systolic volume and the RV ejection fraction, with cardiac resonance imaging methods and they recorded the important NT-pro BNP biomarker after the ECG of the patient. There were others studies that concluded that QTC interval prolongation and QT dispersion in the patients is indicative of RV status and an independent factor of mortality and arrhythmia (13, 18).

These studies on patients with PH (19, 20) were supported by other studies in 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 not any 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 to QRS interval abnormality (23, 24).

There have been reports of QT changes with patients with congenital heart defects and severe PH and Eisenmenger syndrome (25, 26). Regarding the fact that QTC interval is related to ventricular diastole, it can raise the risk of dangerous arrhythmia in the patients with severe ventricular systolic dysfunction. In our study, there was significant prolongation in QRS interval in the PH group in comparison to control group. Despite the significant difference between case and control groups, QTC interval was not significantly different between mild and severe PH groups (p=0.06). QTC interval prolongation in the severe PH group was

just related to Eisenmenger patients (461±106 ms) compared to mild PH group ($p<0.001$). Considering the fact that JTC interval was not significantly different between two main groups and Eisenmenger patients, QTC interval measuring might be affected by QRS interval in these patients. It seems under this situation, JTC interval maybe more reliable for assessing diastolic phase changes (27). As QRS time was significantly prolonged in the severe PH group, it can increase the risk of life threatening arrhythmia thus existence of additional risk factor such as diastolic phase abnormality can worsen this risk (28). Although in this study JTC was not significantly different between all of the pulmonary hypertension patients groups and control group, prolongation of JTC interval in the Eisenmenger patients versus normal population (29) may be important. In this study, there was not any 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 the other fields of congenital heart disease management (30), we recommend simultaneous QTC and JTC measuring in these settings together with cardiac function evaluation using right ventricular myocardial function indices (31) and sensitive cardiac biomarker (32, 33) in the future studies. It can help the evaluation of systolic and diastolic myocardial abnormalities and their effects on QRS, QTC, JTC intervals in congenital heart disease associated to severe pulmonary hypertension.

4-1. Study Limitations

To examine the myocardial function, the use of sensitive cardiac biomarkers such as NP-Pro-BNP is more appropriate.

5- CONCLUSION

In patients with congenital heart disease and severe PH, in spite of rising QRS and QTC intervals, JTC interval did not have significant increase. We suggest simultaneous measuring 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, QTC: Corrected QT interval, MPI: Myocardial performance index, JTC: corrected JT interval, TAPSE: Tricuspid annular plane systolic excursion.

7- CONFLICT OF INTEREST: None.

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