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Non-Invasive Indicators for Outcome in Children with Dilated Cardiomyopathy

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The present research aimed at defining simple prognostic indicators for children suffering DCM. A retrospective analytic study was conducted that included 284 pediatric patients diagnosed with DCM and 112 age and sex matched children as controls. All included subjects underwent; twelve lead-ECG; Holter recording and transthoracic Doppler echocardiographic examinations were carried out in all patients. Statistical Package for Social Science (SPSS) version 9.0 was used for analysis of data. Poor outcome indicators identified by logistic regression analysis included longer disease duration, higher P-wave dispersion, bigger LVEDD, lower fractional shortening and presence of mitral regurge. Risk factors identified from 24 h ECG monitoring were higher maximum and minimum were higher maximum and mean heart rates and presence of nonsustained ventricular tachycardia. Cox regression analysis, identified bigger LVEDD, lower FS% and presence of non sustained VT as predictors of bad outcome. We conclude that LVEDD, FS%, MR, non sustained ventricular tachycardia and higher maximum and average heart rates on ambulatory ECG monitoring are prognostic indicators for outcome in children with DCM. P-wave dispersion is higher among patients with poor outcome and may carry prognostic significance that will need further evaluation. QT dispersion has no prognostic significance in children with DCM.

Key words: Dilated cardiomyopathy, prognosis, outcome, pediatrics

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INTRODUCTION

Dilated cardiomyopathy (DCM) is a myocardial disease characterized by impaired systolic function and dilatation of left or both ventricles (Huh *et al*., 2004). The natural history of DCM in children is difficult to predict due to the heterogeneous nature of the disease. The outcome in infants and children is highly variable from complete recovery to death (Bostan and Cil, 2006). The identification of patients who are at increased risk of sudden death is still an unsolved issue that poses one of the greatest challenges for cardiologists (Zecchin *et al*., 2005; Leenhardt *et al*., 2001). With the exception of a few cases such as aborted sudden cardiac death, sustained ventricular tachycardia and syncope of unexplained origin, there is no consensus on the clinical findings identifying patients with idiopathic dilated cardiomyopathy with an increased risk of sudden cardiac death (Morgera *et al*., 2004).

There is growing evidence of the regional heterogeneity in repolarization in DCM patients on the basis of intracardiac monophasic action potential recordings and on measurements of the Q-T interval dispersion in the standard 12-lead surface electrocardiogram (ECG) (Berger *et al*., 1997) however the predictive value of Q-T dispersion is still under debate (Fauchier *et al*., 2005).

P-wave dispersion is a simple ECG marker that has been associated with inhomogenous and discontinuous of sinus impulses (Dilaveris *et al*., 2000). It has been reported to be significantly higher in patients with DCM (Senen *et al*., 2004).

Ambulatory electrocardiography provides useful prognostic information in patients with DCM and may identify several independent predictors of mortality including nonsustained VT and mean heart rate (Baker and Koelling, 2005). Echocardiography may be useful in predicting children with idiopathic DCM who are at increased risk of sudden cardiac death (Azevedo *et al*., 2004).

The present study aims at identifying non-invasive markers using 12 lead-surface ECG, 24 h ambulatory ECG monitoring and trans-thoracic echocardiography, that predict the outcome in children suffering of DCM.

MATERIALS AND METHODS

A retrospective analytic study was conducted that included 284 pediatric patients diagnosed with DCM, as defined by the World Health Organization and following up at the cardiomyopathy clinic, Department of Pediatrics, Cairo University in the period between January 2002 to May 2006. One hundred and twenty six age and sex matched children were included as a control group. A written consent was obtained from parents of all participating children. The study was approved by the Scientific Research Committee, Department of Pediatrics, Cairo University.

All included subjects underwent;

Twelve lead-ECG: This procedure performed during sinus rhythm. Patients on anti-arrhythmic therapy were excluded from the study and to increase accuracy all measurements were taken by both investigators who were blinded at the time as regards to the clinical status of the patients, a magnifying lens was used.

All routine measurements were taken with particular stress on P-wave, max, min and P-wave dispersion.

The onset of the P-wave was defined as the junction between the iso-electric line and the beginning of the P-wave deflection. The offset of the P-wave was defined as the junction between the iso-electric line and the end of the P-wave deflection.

Electrocardiograms with measurable P-waves in less than 9-ECG leads were repeated. P-wave dispersion was calculated as the difference between the max. and min. P-waves (Dilaveris *et al*., 1998).

QT intervals were measured in all 12 leads if possible, but in at least six leads. The average of three consecutive beats for each lead was taken wherever possible. The QT interval was measured from the beginning of the QRS complex to the end of the T-wave. The end of the T-wave was defined as the return of voltage to the iso-electric line. If a U-wave was present, measurements were taken to the nadir of the curve or notch between the T and U-waves. QT intervals were corrected for heart rate using Bazett's formula (Qtc = QT/vRR). The QT intervals reported are the maximum QT found on the 12-lead ECG (QTmax). QT dispersion (QTd) was defined as the difference between the maximum and minimum QT intervals (Day *et al*., 1990).

Twenty-four hour ambulatory ECG (Holter) recording: Digital recording with ≥20 h of artifact free data were included for all subjects. Data was analysed using the Vision ™ Holter analysis software system with manual edition and reviewing of all data. The mean, maximum and minimum heart rates, pauses (defined longer than 2 sec), supraventricular and ventricular ectopics were recorded. Ventricular tachycardia on Holter recording was defined as ≥3 consecutive premature ventricular beats.

Transthoracic Doppler echocardiographic: This examinations were carried out in all patients. The

equipment mostly used for all Doppler echocardiographic examinations was Hewelett Packard (Sonos-4500), using an 8 and 4 MHZ transducer, capable of performing 2-D studies continuous and pulsed Doppler and color flow Doppler.

The echocardiographic measurements were made in accordance with the norms suggested by the American Society of Echocardiography (Sahn *et al*., 1978). The images for 2-D studies were also obtained according to the usual standardization (Tajik *et al*., 1978). The Ejection Fraction (EF) and (FS) of the Left Ventricle (LV) were used as indicators of the magnitude of the LV systolic dysfunction. The LVEF was obtained based on the LV Systolic Diameter (LVSD) and LV Diastolic Diameter (LVDD) recordings in the longitudinal left parasternal view using the formula:

$$EF = \{[(LVDD)^3 - (LVSD)^3]/ (LVDD)^3\} \times 100$$

The FS% was calculated with the formula:

$$FS\% = [(LVDD - LVSD)/LVDD] \times 100$$

The severity of the insufficiency of the atrioventricular and pulmonary valves was detected and assessed by using Doppler echocardiography and classified as absent/mild lesion or moderate/severe lesion.

Statistical methods: Statistical Package for Social Science (SPSS) version 9.0 was used for analysis of data.

Data was summarized as mean, SD and percentage. t-test was used for analysis of quantitative data, while Chi-square test was used for analysis of qualitative data. One way ANOVA was done for analysis of more than 2 quantitative data followed by Post Hoc test for detection of significance. Logistic regression test was used for prediction of the risk factor for death. Survival follow up data were analyzed by Kaplan-Meier survival curve estimation. Significance was judged at the 2 sided 0.05 level. Cox proportional hazards regression model was used for determining the independent ECG and echocardiographic predictors of death.

RESULTS

Two hundred and eighty four children suffering of DCM were included in the present study and their data analysed. There were 148 boys and 136 girls. Their mean age at the time of the study was 49.2±40.8 months with a range of (2.4-156 months). According to the clinical status at their last follow up, they were classified into two groups; group 1 (92 boys and 80 girls) aged 48±40.8 months with good ventricular function (NYHA class I or II) and group 2 (56 boys and 56 girls) aged 50.4±42 months with poor ventricular function (NYHA class III or IV or died).

One hundred and twelve age and sex matched normal subjects (60 boys and 52 girls) with a mean age of 51.6±39 months were included as a control group. They were recruited from general pediatrics outpatient clinics referred for routine check up.

There was no statistically significant difference in age and gender among all groups (p = 0.1, 0.8, respectively). Patients in group 2 had slightly longer disease duration (21.6±12.4 months) than group 1 (15.6±9.2 months) that was not statistically significant p = 0.2 ECG, ECHO and ambulatory 24 h ECG characteristics were compared among the three groups (Table 1).

Controls had significantly lower heart rates than patients in both groups. Patients in group 2 had significantly higher P-wave dispersion, greater LVEDD, lower FS% and higher mean heart rates on their ambulatory 24 ECG.

QT dispersion was found to be highest in group 1 and lowest in controls, however not statistically significant.

Table 1: Comparison between good, bad cardiac function and controls in relation to ECG and ECHO findings

Variables	Good (N = 172)	Bad (N = 112)	Controls (N = 112)	p-value
ECG heart rate (beat min^{-1})	116.5±21.9a	117.6±17.6a	104.4±17.6	0.0001*
P_{Max}	85.8±20.8a	100.9±29.9b	75.0±10.2c	0.0001*
P_{Min}	53.3±13.6a	58.2±18.1b	57.5±13.0b	0.03*
$P_{Dispersion}$	32.3±18.3a	42.9±23.7b	17.9±13.5c	0.0001*
QT_{Max}	341.1±25.3a	309.5±37.7b	317.1±31.7b	0.0001*
QT_{Min}	300.2±31.4a	287.7±33.9b	300.7±32.7a	0.008*
$QT_{Dispersion}$	40.8±23.9	25.5±16.1	15.8±74.2	0.06
QTc	405.6±52.9	397.0±39.1	394.3±21.2	0.07
LVEDD (mm)	47.8±10.1a	52.6±10.5b	25.1±2.5c	0.0001*
FS (%)	20.7±7.0a	17.1±6.1b	32.7±2.1c	0.0001*
EF (%)	39.3±10.8a	34.8±6.7b	67.2±5.1c	0.0001*
$Holter_{Max\ HR}$	158.5±23.8a	171.5±28.3b	132.6±17.9c	0.0001*
$Holter_{Min\ HR}$	74.5±16.2a	76.7±24.3a	67.9±6.7b	0.0001*
$Holter_{Mean\ HR}$	110.6±19.5a	117.7±23.4b	86.4±7.9c	0.0001*

*p-value is significant if <0.05, Different symbol indicate significant HR: Heart Rate

Table 2: Logistic regression for detection of risk factor of outcome in relation to ECG and ECHO findings

Variables	B	95% C-I	Odds ratio	p-value
Duration of disease (years)	0.1414	1.0092-1.3147	1.1518	0.04*
Heart rate (beat min^{-1})	-0.0080	0.9752-1.0092	0.9921	0.40
P_{Max}	0.1592	0.8716-1.5775	1.1726	0.30
P_{Min}	-0.2799	0.4623-1.2359	0.7558	0.30
$P_{Dispersion}$	0.2163	1.0411-1.4804	1.2415	0.02*
QT_{Max}	-4.2797	0.0-1007	0.0138	0.80
QT_{Min}	3.7602	0.0-3127	42.9549	0.80
$QT_{Dispersion}$	3.5160	0.0-2453	33.6494	0.80
QTc	-0.0754	0.7854-1.0951	0.9274	0.40
LVEDD (mm)	0.0594	1.0293-1.0941	1.0612	0.0001*
FS (%)	-0.3468	0.5416-0.9228	0.7069	0.01*
EF (%)	0.0719	0.9747-1.1846	1.0746	0.10
Mitral regurge	0.8524	1.4031-3.9203	2.3453	0.001*
$Holter_{Max}$	-0.0245	0.9612-0.9906	0.9758	0.002*
$Holter_{Min}$	-0.0053	0.9772-1.0126	0.9947	0.60
$Holter_{Mean}$	-0.0274	0.9561-0.9902	0.9730	0.002*
Supraventricular tachycardia	0.6668	0.2423-1.0877	0.5133	0.08
Nonsustained VT	0.9780	0.1897-0.7456	0.3761	0.005*

*p-value is significant if <0.05, B: Is the estimated logit coefficient, Odds ratio = P/(1-P). P is the probability that the event y occurs, CI: Confidence Interval (it is an interval in which a true population parameters fall), Dependant variables: Outcome

Table 3: Cox regression for detection of hazard of outcome in relation to ECG, ECHO and Holter findings

Variables	B	SE	Wald	Odds ratio	p-value
Heart rate (beat min^{-1})	0.0029	0.0094	0.0962	1.0029	0.80
P_{Max}	-0.0062	0.0888	0.0049	0.9938	0.90
P_{Min}	-0.0931	0.1934	0.2317	0.9111	0.60
$P_{Dispersion}$	0.0719	0.0834	0.7417	1.0745	0.40
QT_{Max}	0.4090	0.4897	0.6976	1.5054	0.40
QT_{Min}	-0.8156	0.5007	2.6534	0.4424	0.10
$QT_{Dispersion}$	-0.8206	0.4819	2.9001	0.4402	0.09
QTc	-0.0090	0.0607	0.0219	0.9911	0.90
LVEDD (mm)	0.0304	0.0142	4.5998	1.0309	0.03*
FS (%)	-0.1478	0.0382	15.1620	0.8626	0.0001*
EF (%)	-0.0236	0.0283	0.6936	0.9767	0.40
$Holter_{Max\ HR}$	0.0054	0.0089	0.3980	1.0054	0.50
$Holter_{Min\ HR}$	0.0080	0.0075	1.1522	1.0081	0.30
$Holter_{Mean\ HR}$	-0.0136	0.0107	1.6309	0.9865	0.20

*p-value is significant if <0.05, B: Is the estimated logit coefficient, Odds ratio = P/(1-P). P is the probability that the event y occurs, CI: Confidence Interval (it is an interval in which a true population parameters fall), Dependant variables: Outcome

Table 4: Distribution of mitral regurge among cases in both groups

	Good		Bad		
Variables	No.	%	No.	%	p-value
Mitral regurge					
Class 0 and 1	60	34.9	24	21.4	0.0001*
Class 2 and 3	112	64.1	88	78.6	

*Significant p = less than 0.05

Logistic regression analysis was performed to identify risk factors of outcome (Table 2). Poor outcome in children with DCM was found to be related to longer disease duration, higher P-wave dispersion on ECG, echo measurement of bigger LVEDD, lower fractional shortening and presence of mitral regurge. Risk factors identified from 24 h ECG monitoring were higher maximum and minimum were higher maximum and mean heart rates and presence of nonsustained ventricular tachycardia.

Cox regression analysis, done for detection of hazard of outcome identified a bigger LVEDD, lower FS% and presence of non sustained VT (defined as ≥3 consecutive ventricular premature beats) on 24 h Holter recordings as predictors of worse ventricular function and poor outcome (Table 3).

Seventy nine percent of patients in group 2 as compared to 64% of patients in group I had severe mitral regurge (grade 2-3) p = 0.0001 (Table 4).

Of the 284 studied patients, 16 showed SVT on their Holter studies. P-wave dispersion however was found higher in the group that didn't develop SVT (p = 0.02). The significant discrepancy in case number may render this observation non reliable.

Thirty two patients developed nonsustained VT on their Holter monitoring. Kaplen Maier survival analysis test was performed to estimate survival in patients who developed arrhythmias. Survival was found significantly lower in patients who suffered nonsutained VT but was not related to developing SVT (Fig. 1).

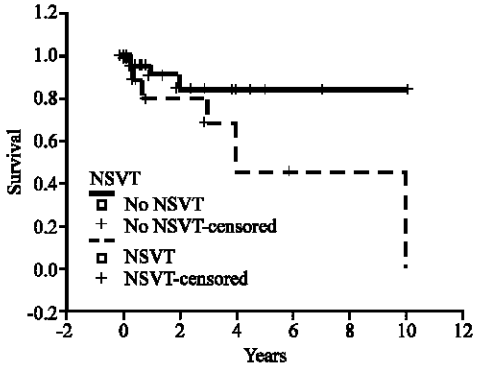

Fig. 1: Survival curve by Kaplan Meier test in relation to NSVT p = 0.0009*

DISCUSSION

Dilated cardiomyopathy is a devastating disease in childhood with grave consequences that may leave patients no hope but cardiac transplantation, a therapeutic option more often than not unavailable. Identifying patients who are more likely to deteriorate and hence receive more aggressive therapy and closer follow up is a continuous challenge for pediatric cardiologists.

In the present research, LVEDD and FS% measured by echocardiography were found by both logistic and cox regression analysis to be related to poor outcome. Nogueira *et al.* (2000) reported lower FS to be a poor prognostic factor in children with DCM. In a study by Azevedo *et al.* (2004), although both parameters were much worse in patients who died yet cox regression analysis didn't identify them as risk factors for death. This may be explained by the small case number in their study.

Mitral regurge in patients with DCM presents an additional volume load on an already dilated ventricle. The resulting dilation of the valve annulus worsens the DCM and pushes the patient into a vicious circle. The presence and severity of MR have been found by Azevedo *et al.* (2004), Amiya *et al.* (2006) and Barbieri *et al.* (2006) to be a predictor of poor outcome; a finding confirmed in the present study.

Present study showed that patients with DCM have longer QT intervals and higher QT dispersion than the control group which was also reported in previous studies (Nogueira *et al.*, 2000; Amiya *et al.*, 2006; Barbieri *et al.*, 2006). Other investigators have however reported that QT dispersion is not prolonged in patients with DCM (Berger *et al.*, 1997; Davey *et al.*, 1994; Fei *et al.*, 1996). Methodology to quantify QT dispersion has not been standardized and this may lead to disparate results (Alonso *et al.*, 2005; Berger, 2003; Statters *et al.*, 1994; Surawicz, 1996).

The prognostic significance of QT dispersion in patients with DCM is debatable. Although considered a reflection of heterogeneity of ventricular repolarization by Huh *et al.* (2004), Leenhardt *et al.* (2001) and Fei *et al.* (1996), others believe it not related. Present study didn't find QT dispersion of prognostic significance among children with DCM for outcome or development of ventricular arrhythmias, a finding similarly reported by Huh *et al.* (2004), Leenhardt *et al.* (2001), Martin *et al.* (1994), Berger *et al.* (1997), Galinier *et al.* (1998) and Fauchier *et al.* (2005).

P-wave dispersion and maximum P-wave are relatively new electrocardiographic markers that have been related to the prolongation of intraatrial and interatrial conduction times and inhomogenous propagation of sinus impulses (Dilaveris *et al.*, 2000; Senen *et al.*, 2004; Ozdemir *et al.*, 2004).

Present study has shown like others (Senen *et al.*, 2004) that P-wave dispersion and P-wave max are significantly increased in patients with DCM, however their prognostic significance is not clear in literature. In the present work, P-wave max and dispersion were found significantly higher among patients with poor ventricular function. Logistic regression analysis identified P-wave dispersion as a prognostic factor for poor outcome but this was not confirmed by cox regression analysis. Further studies are needed to judge the value of this marker.

Non sustained VT on Holter monitoring has been previously identified as a prognostic factor for development of major arrhythmic events and sudden cardiac death in DCM patients (Fauchier *et al.*, 2005; Baker and Koelling, 2005; Zecchin *et al.*, 2005; Grimm *et al.*, 2003). In the present research, it was related to poor outcome in patients with DCM and predicted lower survival.

Logistic regression analysis also identified higher maximum and average heart rates on ambulatory ECG as predictors of poor outcome, which is in agreement with Baker and Koeling (2005).

CONCLUSION

We conclude that LVEDD, FS%, MR, non sustained ventricular tachycardia and higher maximum and average heart rates on ambulatory ECG monitoring are prognostic indicators for outcome in children with DCM. P-wave dispersion is higher among patients with poor outcome and may carry prognostic significance that will need further evaluation. QT dispersion has no prognostic significance in children with DCM.

Study limitation: Present study is a retrospective one that was based on analyzing patient data.

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