Original Article

Contractility Dispersion in Long QT Syndrome

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Background: Previous studies, using M mode echocardiography, provided unexpected evidence of a mechanical alteration in patients with long QT syndrome. The aim of this study was to evaluate entire left ventricular (LV) wall motion characteristics in patients with long QT syndrome using tissue Doppler imaging.
Methods: We enrolled 17 patients with congenital long QT syndrome [11 female and 6 male], aged 21 to 45 years. 10 subjects without cardiac disease were also selected as a control group. Two-dimensional tissue Doppler imaging (TDI) recording of the LV was obtained from the basal and mid-segments from apical four-chamber, two-chamber, and long-axis views. 'Myocardial Contraction Duration' [MCD] was defined as the time from start of R wave on ECG to end of S wave on TDI. MCD was measured in the six LV wall positions: septal, anteroseptal, lateral, inferior, posterior and anterior positions.
Results: LV contractility dispersion was significantly greater in long QT syndrome patients compared to control group [0.051 ± 0.011 vs. 0.016 ± 0.06; $P < 0.001$].
Conclusion: Our study evaluated left ventricular dispersion of contractility duration in patients with long QT syndrome. This mechanical dispersion may be a reflection of the inhomogeneity of repolarisation in the long QT syndrome.

Keywords: Tissue Doppler, Echocardiography, Long QT Syndrome, Dispersion

Introduction

Congenital long QT syndrome is characterized by abnormal ventricular repolarization and occurrence of malignant ventricular arrhythmia.[1-7] Previous studies, using M mode echocardiography, provided unexpected evidence of a mechanical alteration in patients with long QT syndrome[8] but conventional M-mode echocardiography cannot evaluate entire left ventricular (LV) wall motion.

The aim of this study was to evaluate entire left ventricular wall motion characteristics in patients with long QT syndrome using tissue Doppler imaging.

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Patients and Methods

We enrolled 17 patients with congenital long QT syndrome [11 female and 6 male], aged 21 to 45 years. Long QT syndrome was defined according to the diagnostic criteria of Schwartz, et al.[9] 10 subjects without cardiac disease were also selected as a control group for the purpose of comparison of electrocardiographic and echocardiographic parameters.

Electrocardiographic Parameters

Twelve lead standard electrocardiograms were obtained in all patients before echocardiography. The QT interval in all 12 leads was measured from the onset of the QRS complex

to the end of the T wave. When U wave was present, QT interval was measured to the nadir of the curve between the T and U wave. The QTc interval was corrected for heart rate using Bazett's formula. All long QT syndrome patients had QTc interval longer than 480 ms [range 490 to 580 msec] which was unrelated to electrolyte abnormalities or any other causes of QT prolongation.

Echocardiographic Parameters

Echocardiography was performed using a Vivid 3 [GE Healthcare] device. Standard cross-sectional, M-mode and Doppler echocardiographic studies were performed in a partial left lateral decubitus position. The echocardiography was recorded together with an electrocardiogram at lead II.

Tissue Doppler Imaging

Two-dimensional tissue Doppler imaging (TDI) recording of the LV was obtained from the basal and mid-segments from apical four-chamber, two-chamber, and long-axis views. 'Myocardial Contraction Duration' [MCD] was defined as the time from start of R wave on ECG to end of S wave on TDI. MCD was measured in the six LV wall positions: septal, anteroseptal, lateral, inferior, posterior and anterior positions. LV contractility dispersion was defined as:

LV Contractility Dispersion (msec) = Standard Deviation {MCD in six LV wall position}

Statistical Analysis

The results are presented as mean±SD. P values lass than 0.05 was considered statistically significant. Standard deviation of MCD values was defined as a parameter of mechanical dispersion of contraction.

Results

17 patients with congenital long QT syndrome [11 female and 6 male], aged 21 to 45 years were enrolled. All long QT syndrome patients had QTc interval longer than 480 ms [range 490 to 580 msec] which was unrelated to electrolyte abnormalities or any other causes of QT prolongation. Ejection fraction and systolic function were normal in all long QT syndrome patients. LV contractility dispersion was significantly greater in long QT syndrome patients compared to control group [0.051 ± 0.011 vs. 0.016 ± 0.06; $\boldsymbol{P} < 0.001$].

Discussion

Our study evaluated left ventricular dispersion of contractility duration in patients with long QT syndrome. This mechanical dispersion may be a reflection of the inhomogeneity of repolarisation in the long QT syndrome.

Long QT syndrome demonstrates inhomogeneity of regional repolarisation; this can be shown by a standard 12 lead electrocardiogram, body surface mapping, and monophasic action potentials.[10-15] The analysis of QT dispersion from the standard 12 lead electrocardiogram is a simple method for deriving regional repolarisation. Nador *et al* provided the first and unexpected evidence for mechanical alteration in long QT syndrome patients, and showed that this syndrome is not a purely electrical phenomenon.[8] They described the presence of a slow movement in the late thickening phase. This abnormality was more frequent in symptomatic patients. Nador's observation provides evidence of a structural or functional impairment of the hearts in long QT syndrome patients. In the normal heart, several mechanisms regulate myocyte repolarization. As the QT interval on the surface ECG represents the

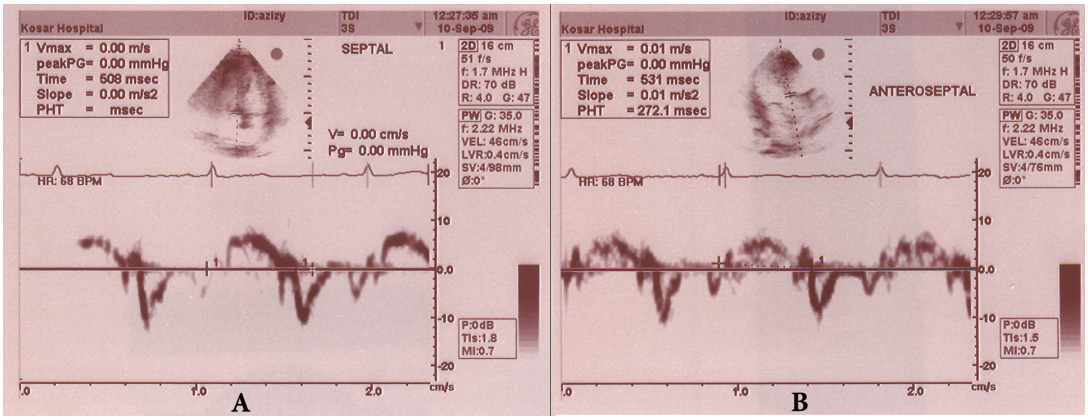

Figure1. Myocardial contraction duration was defined as the time from start of R wave on ECG to end of S wave on TDI. Myocardial contraction duration was measured in the six LV wall positions: septal, anteroseptal, lateral, inferior, posterior and anterior positions. LV contractility dispersion was defined as standard deviation of myocardial contraction duration in six LV wall position.

summation of action potentials in ventricular myocytes, QT prolongation implies action potential prolongation in at least some portions of the ventricle. A prolongation of the action potential duration is associated with an increase in the tension developed by the ventricular muscle, leading to prolonged contraction duration.

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