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QT Interval Analysis in Individuals with Idiopathic Mitral Valve Prolapse

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Abstracts
Objectives: The goals of the study are to determine whether or not there is a correlation between the QT interval and IMVP, to provide an explanation for sudden cardiac death in some cases, and to show how IMVP symptoms are linked to certain arrhythmias.
Methods: QTC intervals were measured in 43 patients with idiopathic mitral valve prolapse (11 men and 32 women with mean age 46 ± 17 years). All patients underwent a clinical and echocardiographic examination, and ECG taking to assess the incidence of long QT in patients with IMVP.
Results: The data show that 36 of the 43 patients with IMVP have a prolonged QT interval, while only 2 of the 38 patients in the control group have. There was also an uptick in arrhythmia cases when the QT interval was prolonged.
Conclusion: Individuals with IMVP have an increased risk of arrhythmias, and this risk is even greater in patients with very long QT, which can cause life-threatening ventricular arrhythmias, as shown by this study.
Keywords: Analysis, QT interval, Idiopathic mitral valve prolapse

Introduction

MVP is a reasonably frequent, but extremely varied clinical illness caused by multiple pathogenic processes of the mitral valve apparatus. It is also known as the systolic click-murmur syndrome, Barlow's syndrome, floppy-valve syndrome, and billowing mitral leaflet syndrome. Myxomatous degeneration and substantially elevated amounts of acid mucopolysaccharide[1] are two such conditions that are linked with superfluous or excessive mitral leaflet tissue. Patients with heritable connective tissue diseases, such as the Marfan syndrome, osteogenesis imperfecta, and the Ehlers-Danlos syndrome, frequently present with MVP. When it comes to valvular heart disease, mitral valve prolapses (MVP) are the most prevalent diagnosis, and they affect 5% of the general population, notably young people.[1] Though MVP is said to have a favourable prognosis and a low frequency of complications, it is evident that even a rare problem would affect many people due to the great prevalence of MVP in the general population.[2] Most individuals with MVP, however, only experience mitral (or rarely tricuspid or aortic) myxomatous degeneration. In most cases, the posterior leaflet is more severely damaged than the anterior, and the mitral valve annulus is significantly dilated. Many patients' regurgitation is caused by or contributes to the patient's enlarged redundant chordae tendineae. Although the root aetiology of MVP remains a mystery in the vast majority of cases, it does appear to be a genetically determined collagen tissue defect in some.[3] Decreased synthesis of type III collagen has been blamed, and electron microscopy has shown that collagen fibrils have become disorganised. The papillary muscles and the myocardium just underneath them might suffer from malfunction and ischemia if MVP were to cause them undue stress. In a vicious cycle, valvular regurgitation causes more damage to the sick mitral valve's chordae tendineae and progressive annular dilatation apparatus due to the stress it takes on them. Enhanced tension on the papillary muscles appears to be the root cause of the electrocardiographic alterations and ventricular arrhythmias. A genetic arrhythmia syndrome's presentation is often the unexpected and tragic death of an otherwise healthy young individual. The long QT syndrome is the most prevalent of these conditions, and it's defined by a prolonged QT interval on a surface electrocardiogram and an elevated risk of sudden death, most often from ventricular fibrillation. Syncope or sudden death can occur in people with long-QT syndrome due to a variety of factors, including physical or mental stress, loud noises, or even while the individual is at rest. Several hundred mutations in 10 genes have been found to be associated with long-QT syndrome. Most instances are due to mutations in one of three genes that code for cardiacion channels critical for ventricular repolarization; these mutations cause distinct genetic subtypes known as long QT syndrome (LQT1, LQT2, and LQT3).[4] Disease-associated genes influence how the QT interval reacts to exercise, what causes arrhythmia, and how well treatments work. Typically, torsades de pointes, a kind of polymorphic ventricular tachycardia, is to blame for syncope in people with the long-QT syndrome. Some people with LQT3 may have syncope due to bradycardia. The most common cause of death is ventricular fibrillation.[4] Loss-of-function mutations in KCNQ1, which encodes IKs, an adrenergic-sensitive potassium channel in the heart, cause LQT1, the most prevalent type of the syndrome. Syncope or sudden death can occur in people with this condition when they experience extreme stress, such as that caused by diving or swimming.[4] Loss-of-function mutations in KCNH2 (also known as HERG), which encodes IKr, another critical potassium channel in the heart, cause LQT2. The quick onset of symptoms in response to a loud noise, such as that produced by an alarm clock, is almost diagnostic of this form 23; normal hearing is not a risk factor for this type of syncope or sudden death. Mutations in the SCN5A gene cause LQT3 by preventing the sodium channel from being quickly inactivated in the heart.[5] The aim of study is to assess the relationship between QT interval and IMVP, finding of an explanation for some cases of sudden death, presenting the association of symptoms of IMVP and some types of arrhythmias.

Methods

There were two types of participants in this research. Patients in Group1 ranged in age from 18 to 73 (mean, 46 + 17 years)

and all had mitral valve prolapse. In all cases, M-mode echocardiography confirmed the presence of mitral valve prolapse (mid to late systolic type in 19 and holosystolic in 2 patients). Only when echoes from both the anterior and posterior leaflets were recorded throughout the cardiac cycle and came together at the commencement and conclusion of systole was the echocardiographic diagnosis of mitral valve prolapse accepted.

Except for digoxin (three patients) and propranolol (two patients), no other medicines known to impact the QT interval were used by the 43 patients during the research. Group 2 consisted of 38 individuals with normal results from a cardiac examination, including 21 men and 17 females aged 17 to 76 (mean, 34 & 16). Auscultatory findings, supine and upright phonocardiographic recordings, and echocardiographic findings were all within normal ranges in group 2.

Recording and Analyzing of ECCs

Following a 30-minute rest, a 12-lead ECG and 30-second rhythm strip were recorded with the patient in the supine position (speed, 25 mm/sec). Thirteen participants additionally wore 24-hour Holter monitors. The QT interval was determined by measuring the time between the first appearance of the QRS complex and the last appearance of the T wave. Before or after extrasystolic beats, QT was not measured. The QT interval and the corresponding RR interval were determined by selecting six to ten consecutive beats from lead 2 or V4-V6 (depending on which revealed the T wave's termination more clearly) for each ECG. Maximum QT interval refers to the longest QT interval among these beats, whereas mean QT interval and RR interval relate to their averages throughout all recordings. The QTC interval was calculated by dividing the QT interval by the square root of the corresponding RR interval. Maximum QTC was calculated as the longest QTC interval and mean QTC was calculated as the average QTC interval over all three measurements. Conventional electrocardiograms (ECGs) with 30-second rhythm strips were used to analyse arrhythmias and other abnormalities in all patients, whereas in 13 patients, Holter monitors were used for 24 hours or longer Table 1.

Results

In group 1, the heart rates varied between 62 and 105 beats per minute, with a mean of 83, while in group 2, the rates varied between 64 and 93 beats per minute, with a mean of 78. Patients in Group 1 had a Maximal QT Interval that was Greater Than or Equal to the 97.5 Percentile and Associated Normal Population in 36 (82%), and Greater Than or Equal to the Upper Limits of Normal Values in the Remaining 7 Patients. The maximum QT interval of the individuals in the control group (group 2) was greater than the 97.5th percentile. Maximum QT intervals for patients in groups 1 and 2 are depicted in Figure 1 and compared to normal values. QTC intervals varied between 0.37 and 0.51 seconds (mean: 0.46 0.032 seconds) in group 1, and between 0.36 and 0.47 seconds (mean: 0.39 0.025 seconds) in group 2. The statistical significance ($P < 0.003$) between the groups was found to be in the difference between the mean QTC interval. Eighteen patients in Group 1 (ranges, 0.47 to 0.51; mean, 0.48 + 0.024 Second) had a QTC interval longer than 0.46 seconds, whereas just one patient in Group 2 had a QTC interval this long.

The QTC in nine individuals with IMVP was significantly prolonged, with the maximal value ranging from 0.49 to 0.51 seconds (mean, 0.50 + 0.042 seconds). Arrhythmias affected five of these patients, or 57%, with two patients experiencing recurrent venlricular tachycardia and four patients experiencing paroxysmal atrial tachycardia with frequent premature atrial and ventricular contractions. Maximum QTC intervals in the remaining 31 IMVP patients varied from 0.42 seconds to 0.50 seconds (mean: 0.46 + 0.028 seconds). Seven patients, or 22.6%, exhibited arrhythmias such as atrial fibrillation with premature atrial or ventricular contractions, paroxysmal atrial tachycardia, or numerous premature ventricular contractions. Table 2 also displays the frequency of arrhythmias as a function of QTC duration.

Discussion

Arrhythmias caused by IMVP have often been linked to issues with the mitral valve or the left ventricle. Normal mitral leaflet motion functioning as a mechanical stimulation for creation of ectopic beats as stated by some professionals Although catecholamines in the bloodstream might produce arrhythmias, there are numerous other possible mechanisms by which they

Table 1. **Shows the no. of patients and results**

Parameters	Patients with M.V.P	Controls
Long QT	36/43	2/38
Normal QT	7/43	36/38
Sex female/male	32 female/11 male	17 female/21 male
Mean wit mean ± S.D	0.4599 ± 0.0326 *P* = Value 0.003	0.3910 ± 0.0219 sec *P* = Value 0.0025

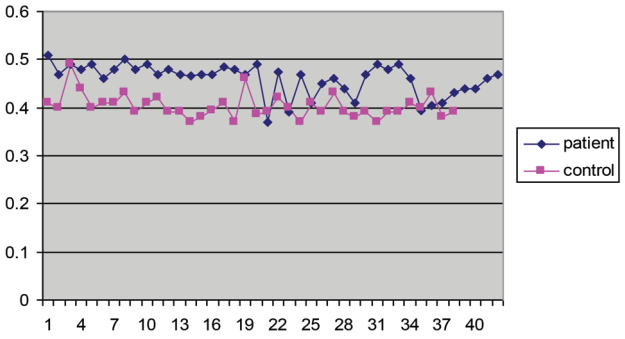

Fig. 1 **Diagram–shows the QTC in both patient with IMVP and control groups.**

Table 2. **Shows the relationship between prolongation of QTC and incidence and also the type of arrhythmias**

Maximum QTC	Total no. of patient = 43	No. of patient with arrhythmias	Type of arrhythmias
0.45 sec	14	2	PVC, PAC
0.46–0.48 sec	13	4	PVC, AF, PVC and PAC, PVC and SVT
0.49–0.51 sec	9	5	PVC, PAC and PVC, PVC and AF, VT, SVT

can do so, making explanation of the sporadic occurrence of these disorders impossible. The mechanism of rapid death in certain individuals with IMVP cannot be explained by the presence of left ventricular synergy, which is commonly shown on angiographies.[6] Preexcitation syndromes and bradycardia have also been identified as probable reasons for arrhythmias in patients with IMVP. None of our patients has demonstrated ECG indications of preexcitation or Brady arrhythmias. Previous study was made when the analysis of QT interval was made in 56 patients with idiopathic mitral valve prolapse and other control group of 62 healthy volunteers, the maximum QT interval of patients with IMVP exceeds the 97.5 percentile in 51 of 56 patients compared with only 3 of 62 subjects of control group. the difference between mean QTC in patient with IMVP and the control participants was significant ($P < 0.005$). Our investigation shows that individuals with IMVP had considerable QT prolongation on many ECG recordings. Furthermore, the high incidence of ST-T-U alterations in these individuals imply the prevalence of other repolarization disorders.[7] QT interval measurements are known to be inaccurate due to the challenge of precisely pinpointing the start of the QRS complex and the end of the T wave. In our analysis, we took special care to collect data that clearly distinguished the start of the QRS complex from the end of the T wave. Past observations of QT prolongation in people with IMVP have shown a spectrum from 0.6% to 64.0%. Our patients showed considerable variability in their QTc abnormalities. QT prolongation incidence rates may vary because people with IMVP experience natural fluctuations in their QTc interval. The rate of arrhythmias was shown to be higher in patients whose maximal QTCs were significantly longer compared to those whose QTCs were shorter. QT prolongation has been observed in other delayed repolarization syndromes,[8-10] and our finding suggests that arrhythmias in people with IMVP may share this mechanism. Variations in sympathetic discharge during the study are reflected in the wide range of ventricular refractoriness and aberrant repolarization observed here. Variations in QT intervals in our patients, as well as the appearance of many new systoles in patients with IMVP after exercise, suggest that these people may have an abnormal response to sympathetic stimuli. In people with IMVP, QT prolongation may play a substantial role in generating severe arrhythmias and sudden death, much as it does in people with inherited QT prolongation syndromes and sudden infant death syndromes.[11-15] regions where prolonged repolarization is a common cause of arrhythmias.[16-21]

Conclusion

The individuals with mitral valve prolapse may be at risk of severe arrhythmias because of the correlation with QT interval prolongation. Many of the signs and symptoms of MVP, such fainting, chest discomfort, palpitations, and dizziness, may be attributed to QT prolongation.

Conflicts of Interest

None.

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