Electrocardiographic Abnormalities in Young Athletes with Mitral Valve Prolapse

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ABSTRACT

Background: Mitral valve prolapse (MVP) is the most common primary valvular abnormality in a young population. In some individuals, MVP is silent or associated with palpitations, dizziness, chest pain, and abnormal electrocardiogram (ECG) repolarization with or without ventricular arrhythmias.
Hypothesis: The aim of the present study was to assess the occurrence of the clinical and electrocardiographic abnormalities in young athletes with silent MVP.
Methods: A group of 10 children, who have been sport training intensively, with preparticipation silent MVP were examined for symptoms and/or ECG abnormalities. The diagnosis of MVP was made by echocardiography.
Results: Three athletes were asymptomatic at initial presentation. The other 7 athletes presented with symptoms. The QTc intervals >440 msec were recorded in 2 athletes (1 with syncope). Abnormal ECG repolarization was found in 7 athletes (4 athletes were symptomatic and 3 were asymptomatic). A large variety of T-waves was registered in athletes who presented with symptoms. In asymptomatic athletes, the tall and flat T-waves were recorded.
Conclusions: Young athletes with MVP are often predisposed to electrocardiographic abnormalities of ventricular repolarization, which requires annual cardiologic evaluation.

Introduction

Mitral valve prolapse (MVP) is the most common primary valvular abnormality in a young population (accounting for 5%). According to the Framingham study, MVP affects up to 2.4% of the general population, whereas the relative prevalence seems to be the same as in cases previously described in literature.[1,2] Despite its high prevalence in the population, mitral valve prolapse seems to be a rare cause of sudden cardiac death (SCD). Athletes who died suddenly rarely had myxomatous mitral valve abnormalities at necropsy.[1] An approximate 0.5% incidence of SCD during physical activity has been reported, but a higher incidence of 2.3% SCD has been reported in young competitive athletes.[3] Mitral valve prolapse is generally a benign disorder characterized by systolic protrusion of the mitral valve leaflets into the left atrium with or without mitral regurgitation. The complications like endocarditis and cerebrovascular accidents occur frequently in subjects with mitral systolic murmur, thickened mitral valve leaflets, and mitral regurgitation.[1] Electrocardiogram (ECG) is useful for diagnosis of mitral regurgitation, particularly for identifying leaflet thickening and dilation of left ventricle.[4] Although most subjects with MVP are asymptomatic, a variety of symptoms such as palpitations, dizziness, chest pain, dyspnea, presyncope, and syncope have been reported.[5,6] The MVP was suspected upon physical examination, as the characteristic auscultatory findings (mid-systolic or late systolic click and/or late systolic or holosystolic murmur) were detected.[5,6] In some individuals, MVP is associated with supraventricular or ventricular arrhythmias and repolarization abnormalities.[1,7,8] The prolonged QT intervals in MVP cases could be related to malignant ventricular arrhythmias and SCD.[9,10]

The purpose of the study was to assess the occurrence of the clinical and electrocardiographic abnormalities in young athletes with preparticipation silent MVP. It seems that symptoms and ECG patterns of abnormal repolarization play an important role in clinical diagnosis in young athletes with silent MVP.

Material and Methods

A group of 10 children, who had been sports training intensively, with preparticipation silent MVP were evaluated for symptoms and/or ECG abnormalities. The diagnosis of MVP was made by echocardiography. There were 8 boys and 2 girls. Their ages ranged from 12 to 18 years (average = 14.5 years). Athletes were engaged in 7 different sporting disciplines: football (2), basketball (2), karate (2),

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volleyball (1), handball (1), swimming (1), and track and field (1). They participated in vigorous training programs for periods of 1 to 5 y (median 3 y). The average intensive training program was approximately 7 h per wk. The medical examination included history collection (family history of sudden death at a young age), physical examination, ECG, Holter monitoring (Model 700 Reynolds Medical, London, England), two-dimensional (2-D) and Doppler ECG, and exercise tests. The diagnosis of MVP by echocardiography in athletes was based on either systolic billowing of 1 or both mitral leaflets across the mitral annulus in 2-D parasternal or apical long-axis views and more than 2 mm late systolic posterior displacement of mitral leaflet interfaces in the M-mode recordings.[4] Assessment of severity of mitral regurgitation was detected by 2-D and color-flow Doppler echo using the known criteria.[4] All athletes underwent an ECG 2-D, M-mode, and Doppler-ECG, which were performed using a commercially available Hewlett-Packard instrument with a 3.5 MHz transducer (Hewlett Packard Company, Palo Alto, Calif., USA). The cardiac dimensions were measured from the M-mode echocardiograms. The standard 12-leads were recorded at rest, in a lying position. Standard 12-lead ECGs were recorded with a paper speed of 50 mm/s and standardizations of 1 mV/cm. PQ, QRS, and QT intervals were measured manually. Three measurements of the QT interval were taken from the II or V_5 lead of the ECG. The QT intervals were corrected (QTc) in accordance with the heart rate using Bazett's formula.[11] On the ECG there appeared the following abnormalities: prolonged QT interval, a large variety of T-waves, flat/tall or inverted T-waves in leads I, aVL, leads V_5 and V_6, and the presentation of U-waves.[12] Twenty-four hour ambulatory electrocardiographic monitoring was carried out by a Holter recorder in all athletes. The subjects were encouraged to avoid moderate, heavy, or sustained exercise with the exception of normal daily activities during the recording. The exercise testing was a symptom-limited, graded treadmill test that used a modified Bruce protocol.[13] (Treadmill: Model ERT 100 ITAM, Zabrze, Poland; ECG: Model DEK 631 ITAM, Zabrze, Poland.) Differences between means were assessed using the paired Student *t* test. A 2-tailed p value of <0.05 was considered evidence of statistical significance.

Results

Three athletes with MVP were asymptomatic at initial presentation. The other 7 athletes presented with the following symptoms: intense chest pain (2), chest pain with excessive tiredness (1), syncope (2), presyncope during exertion (1), and excessive tiredness with headache (1). None of athletes had a family history of premature death at a young age. All the athletes with MVP showed no significant increases in cardiac dimensions. The left ventricular systolic and diastolic cavity dimensions (EDD, ESD) and wall thicknesses (IVS, PW) were normal. The MVP with first-degree mitral regurgitation was detected in 6 athletes, in which 4 presented with symptoms: 2 boys with intense chest pain and 1 girl and 1 boy with syncope. Four athletes were without mitral valve regurgitation (Table 1). The QTc intervals >440 msec were recorded in 2 children: in the girl with syncope and in the boy without symptoms. In 7 athletes with MVP, who were symptomatic, the mean QTc = 420 msec and in 3 asymptomatic athletes the mean QTc = 430 msec. Abnormal ECG repolarization, a large variety of T-wave or flat/tall T-waves in leads I, aVL, V_5 and V_6 were found in 7 athletes with MVP (in 4 symptomatic athletes and in 3 asymptomatic athletes). A large variety of T-waves without prolonged QT intervals (QTc = 380–430 msec) were often registered in athletes presented with the following symptoms: presyncope during exertion (1), headache with excessive tiredness (1), and chest pain (1). In asymptomatic athletes with MVP the flat T-waves in left leads were recorded in 2 boys (Table 1). On the Holter ECG monitoring, single monomorphic premature ventricular complexes (PVCs) <10/ h (3) and/or numerous premature supraventicular complexes (2), irregular sinus rhythm (2), and/or age-adjusted bradycardia (2) were recorded (Table 1). Two athletes presented with chest pain, and single PVCs on the Holter, without prolonged QT intervals on a standard ECG (QTc = 0.42 s–0.44 msec). But, during the treadmill test, the PVCs and prolongation QT (QTc = 460 msec) were elicited, which appear to imply risk over time. The athletes with MVP had not participated in any training programs. All the athletes remained under careful cardiologic control with recommendations regarding endocarditis prophylaxis for patients with MVP and mitral valve regurgitation. Correction of electrolyte abnormalities (magnesium and potassium) and withdrawal of any offending drugs are recommended in patients with prolonged QT.[14]

Discussion

Questions about the screening and disqualification of athletes have generated considerable debate. The controversy is magnified by the tragic, sudden, and unexpected deaths of elite young athletes. Sports, per se, are not a cause of the increased mortality. Sports activity acts as a trigger for cardiac arrest in those athletes who are affected by cardiac disease or are predisposed to life-threatening ventricular arrhythmias.[3] Mitral valve prolapse is often associated with prolongation of the QT interval and the disorder of ventricular repolarization, which predisposes them to ventricular arrhythmia and sudden death. However, the Framingham Study revealed no evidence of QT prolongation in association with mitral valve prolapse.[2] But, our study revealed that QT prolongation was a common finding in 20% of athletes with MVP. The QTc intervals >440 msec were recorded in 1 athlete with syncope and another without symptoms. We

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Table 1. Clinical findings in MVP athletes with symptoms and without symptoms

No	Age	Gender	Sporting disciplines	Symptoms	Echo	Holter ECG	QTc[s]	ECG abnormalities
1.	17	M	football	–	MVP+MVR I	PSVCs Bradycardia	0.40	U-wave; tall T-wave
2.	13	M	karate	–	MVP+MVR I	PVCs<2/ h PSVCs	0.44	U-wave; flat T-wave
3.	12	F	handball	–	MVP	–	0.45	U-wave; flat T-wave
4.	17	M	track and field	intense chest pain	MVP+MVR I	PVCs<20/ h	0.42	Variety of T-waves; U-wave
5.	13	M	karate	intense chest pain	MVP+MVR I	PVCs<6/ h	0.44	U-wave; tall T-wave
6.	14	M	football	presyncope during exertion	MVP+MVR I	Irregular sinus rhythm	0.38	Variety of T-waves; U-wave
7.	16	M	basketball	syncope	MVP	PVCs<3/ h	0.37	–
8.	16	M	volleyball	chest pain with excessive tiredness	MVP	Irregular sinus rhythm, bradycardia	0.43	–
9.	14	M	basketball	excessive tiredness with headache	MVP	–	0.43	Variety of T-waves; U-wave
10.	13	F	swimming	syncope, headache	MVP+MVR I	–	0.45	–

Abbreviations: MVP, mitral valve prolapse; MVR, mitral valve regurgitation; PVCs, premature ventricular complexes; PSVCs, premature supraventicular complexes.

could not find any correlation between QT duration and the presence of cardiac arrhythmias (PVCs) in the athletes. The increased QT interval is mainly attributed to the increased cardiac vagal tone, which prolongs the repolarization phase of myocardial cells.[11] Fauchier et al., supported the theory that increased QTc in athletes is related to the increased myocardial mass.[15] Nevertheless, this finding was not verified by the present study. All the athletes with MVP showed normal left ventricle function and no significant increase in cardiac dimensions. A variety of T-waves without prolonged QT intervals were often registered in the athletes who presented with the following symptoms: chest pain, presyncope during exertion, and headache with excessive tiredness. Abnormal ECG repolarization and a variety of T-wave or flat/tall T-waves were found in 70% of athletes with MVP. These abnormal electrocardiographic patterns should also be attributed to the exercise-induced increase of the cardiac vagal tone. But in our first study (a group of 87 young athletes without MVP at the same age, with a similar training), the ECG abnormalities had only occurred in approximately 52% of young athletes.[8] Consensus-panel guidelines for the disqualification of trained athletes were proposed by the Twenty-sixth Bethesda Conference. Under the current Bethesda guidelines, young athletes with MVP are discouraged from participating in competitive sports, with the exception of low intensity sports.[16] The newest recommendations (to provide careful guidelines to physicians and consultant cardiologists regarding the evaluation of athletes with MVP and to suggest sports activities that can be safely performed) are more restrictive.[17] Every effort should be made to recognize such diseases as soon as possible, to reduce the risk for athletes, with the perspective that disqualification of affected individuals makes the prevention of an athlete's death while playing possible.[18] Young, competitive athletes who died suddenly were affected by silent cardiovascular diseases, predominantly consisting of cardiomyopathies, coronary artery disease, Marfan syndrome, and MVP.[3] Preparticipation cardiovascular screening by history, physical examination, and 12-lead ECG can easily detect the hypertrophic cardiomyopathy, coronary artery anomalies, and arrhythmogenic right ventricular dysplasia, but mitral valve prolapse is more difficult to recognize.[18] It seems that the classification of the sustained abnormal ECG patterns plays an important role in clinically recognize MVP in young athletes, but the work should be done in larger patient groups.

Conclusion

The young athletes with MVP are often predisposed to electrocardiographic abnormalities of ventricular repolarization, which requires yearly cardiologic evaluation.

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