

Assessment of premature ventricular beats in athletes

Évaluation des extrasystoles ventriculaires chez l'athlète

Afef Ben Halima ^{1,2}, Manel Ben Halima ^{3,2}, Zied Bel Hadj ^{1,2}, Marouene Boukhris ^{1,2}, Saida Ayachi ⁴, Hayet Ben Salah ^{4,3}, Héli Addala ⁴, Anis Chaouachi ⁴, Donia Kobaa ⁴

1-Service de cardiologie, hôpital Abderrahmen Mami

2- Université Tunis El Manar, Faculté de Médecine de Tunis- Tunis

3-Service de Réanimation cardiologique, hôpital La Rabta

4- Centre Médicale de la médecine et des sciences des sports de Tunis

RÉSUMÉ

Introduction : Les extrasystoles ventriculaires (ESV) sont généralement considérées comme une anomalie électrocardiographique bénigne chez les athlètes. Cependant, elles peuvent être un indice de présence d'une cardiopathie sous-jacente qui peut augmenter le risque de survenue de mort subite. Cela implique la nécessité d'une évaluation cardiologique avant d'indiquer l'aptitude de pratiquer des sports de compétition.

Objectif: L'objectif de ce travail était d'évaluer une population d'athlètes avec des ESV et de déterminer d'éventuelles étiologies sous-jacentes afin de prendre une décision concernant la pratique du sport.

Méthodes: il s'agit d'une étude prospective qui comprenait des athlètes examinés au Centre national tunisien de médecine du sport et de sciences du sport (CNMS) de janvier 2013 à juin 2015 qui ont présenté des ESV sur un électrocardiogramme.

Résultats: 5798 athlètes ont été référés au CNMS. Nous avons identifié 42 athlètes ayant des ESV avec une prévalence de 1,8%. L'âge moyen de la population étudiée était de 21,6 ± 5,99 ans. 83% étaient des hommes. 88% étaient asymptomatiques. L'électrocardiogramme a été considéré comme normal dans 62% des athlètes selon les critères de Seattle. A l'Holter, le nombre moyen d'ESV était de 920 PVC / 24 heures. Treize athlètes avaient des doublets et 11 avaient des triplets. Un patient avait des ESV polymorphes avec phénomène R / T. L'échocardiographie transthoracique (ETT) était normale dans 71% des cas. Trois athlètes avaient une cardiomyopathie hypertrophique (CMH). Tous les patients ont eu une épreuve d'effort. Les ESV ont disparu chez 12% des athlètes. L'IRM a été réalisée chez 10 athlètes confirmant les trois cas de CMH et révélant un cas de dysplasie arythmogène du ventricule droit et un cas de compression du ventricule droit par un pectus excavatum.

Conclusion: après cette évaluation, cinq athlètes ont été classés inaptes à la pratique du sport. Cette étude montre la nécessité d'une évaluation cardiologique approfondie des athlètes avec arythmie ventriculaire afin de détecter les maladies cardiaques sous-jacentes et de prévenir la mort subite dans cette jeune population apparemment en bonne santé.

Mots-clés

Athlètes; Extrasystoles ventriculaires; Échocardiographie; Epreuve d'effort; Holter rythmique; Imagerie par résonance magnétique; Mort subite.

SUMMARY

Introduction Premature ventricular complexes (PVC) are generally considered as a benign electrocardiographic abnormality in the athletic population. However it may be indicative of underlying heart disease which may increase the risk of sudden death. This implies the need for cardiological evaluation before indicating the ability to practice competitive sports.

Aim: The aim of this study was to evaluate an athlete population with PVC and establish underlying etiologies in order to take a decision regarding practicing sports.

Methods: This is a prospective study which included athletes examined in the Tunisian National Centre of Sports Medicine and Sports Science (TNCSM) from January 2013 to June 2015 who presented PVC on an electrocardiogram

Results: 5798 athletes were referred to the TNCSM. We identified 42 athletes having PVC with a prevalence of 1.8%. The average age of the study population was 21.6 ± 5.99 years. 83% were men. 88% were asymptomatic. The electrocardiogram was considered normal in 62% of the athletes according to the Seattle criteria. At the Holter monitoring, the average number of PVC was 920 PVC / 24 hours. Thirteen athletes had doublets and 11 had triplets. One patient had polymorphic PVC and an R/T phenomenon. The transthoracic echocardiography (TTE) was normal in 71% of cases. Three athletes had hypertrophic cardiomyopathy (HCM). All patients underwent a stress test. The PVC disappeared in 12% of athletes. MRI was performed in 10 athletes confirming the three cases of HCM and revealing a case of arrhythmogenic right ventricular dysplasia and a case of compression of the right ventricle by pectus excavatum

Conclusion: After this assessment, five athletes were not allowed to practice sport. This study shows the necessity of a thorough cardiological assessment of athletes with ventricular arrhythmia in order to detect underlying heart disease and prevent sudden death in this young apparently healthy population

Key- words

Athletes; Premature ventricular contraction; echocardiography; stress test; Holter monitoring; Magnetic resonance imaging; Sudden death.

Premature ventricular contractions (PVC) occur in 1 % of the general population detected by standard electrocardiogram (ECG) (1) and in 40 to 75 % detected by continuous ambulatory ECG (Holter) (2-5). PVC may be asymptomatic or manifest by palpitations, chest pain, dyspnea, lipothymia or syncope. The discovery of PVC raises several controversies especially in young people engaged in strenuous activities. Their occurrence may be benign or may be a marker of a potentially life threatening cardiac disease. As ventricular arrhythmia is the most frequent cause of sudden death in athletes (6-10), eligibility or disqualification for competitive sports of athletes with PVC constitutes an important medical and legal issue.

The aim of this study was to evaluate the clinical and electrocardiographic features of an athlete population with PVC and to establish underlying etiologies in order to take a decision regarding practicing sports.

METHODS

This is a retrospective study carried out at the National Center of Sports Medicine and Science (NCSMS) over a period of 3 years (2013-2015). We included in this study all athletes who were assessed in the NCSMS in order to obtain the medical certificate of no contraindication in the sport practice and who had at least one PVC on a 12-lead ECG or at stress test.

Pre-participating screening: All competitive athletes exercising in national teams are referred to have medical certificate of no contraindication in the sport practice. They undergo a systematic medical check-up according to the consensus established by the NCSMS committee which includes medical history, physical examination, an ECG, echocardiography and stress test.

During the period of study 5798 medical examinations were carried out in order to deliver a medical certificate of no contraindication in the sport practice.

Study population : 42 athletes engaged in a variety of sports discipline having a mean age 21.6 ± 5.9 years, range 15 to 39 (34 men and 8 women) had PVC detected during the preparticipation screening. They underwent (ECG), 2-dimensional echocardiography with Doppler analysis, 24-hour Holter monitoring, exercise testing, and in selected cases contrast enhanced cardiac magnetic resonance imaging. Electrocardiograms were evaluated using digital calipers at standard paper speed (25 mm/s) using a Trismed Cardipia 800 device.

Electrocardiographic abnormalities were divided into 2 groups (common or training-related and uncommon or training-unrelated abnormalities) and interpreted according to recent recommendations (11-12).

The exercise test was performed on a bicycle (with standard 12-lead placement) up to the submaximal heart

rate. Holter monitoring was performed using 12-lead ECG (with standard lead placement). The number, morphologies, and coupling intervals of single and repetitive PVC were studied. The echocardiographic study was performed with a 2.5- to 4-MHz transducer (model Hewlett Packard Image Point HX) and included M-mode, 2-dimensional, and Doppler examinations of the traditional views. Left ventricular (LV) end-diastolic and end-systolic diameters, parietal wall thickness, and left atrial diameter were calculated in the parasternal long-axis view using M-mode imaging. LV end diastolic volume, end-systolic volume, and ejection fraction were calculated in the apical 4-chamber view (using Simpson's rule).

Statistical analysis: The data were entered and analyzed using SPSS software version 10.0. Data are expressed as mean SD for continuous variables and as frequencies with percentages for categorical variables. All continuous variables are expressed as mean SD. The results are presented as mean and standard deviation or number and percentage.

RESULTS

The initial detection of premature ventricular complexes (PVCs) during the preparticipation screening in the 42 athletes was at resting ECG in 26 % and on exercise tests in 74% of athletes. The athletes were engaged in 11 different sport disciplines: Handball (19.4%), Karate (19.4%), Football Boxing (5%), Basketball (5%), Gymnastics (2.7%), Golf (2.7%) and Wrestling (2.7%). The majority of athletes in our study population had practiced their sports discipline more than 5 years (74%). Eighty eight percent of the population study were asymptomatic. Five athletes reported symptoms: chest pain in a football player, palpitation in a handball player and a wrestler player and dyspnea in two handball players.

No athlete reported a family history of sudden death. Physical examination was normal in all athletes. Baseline electrocardiographic findings: All athletes were in sinus rhythm. 62% of the athletes had normal ECG. The mean heart rhythm was 69 ± 11 beats/min. The mean electrical QRS axis was 73 ± 2 °. The mean PR interval was 155 ± 29 ms. Two athletes had short PR intervals without accessory pathways at exploration. The mean QRS duration was 85 ± 21 ms. The average duration of QT was 410 ± 22 ms for girls and $385, 25 \pm 36.88$ for boys. The electrocardiographic common abnormalities were sinus bradycardia in 18 athletes (43 %) incomplete right bundle branch block (RBBB) in 5 athletes (12%), and early repolarization in 14 (33%). The electrocardiographic uncommon abnormalities were ventricular preexcitation in one athlete and complete RBBB in two athletes. Negative T waves were observed in 5 athletes (12 %) in lateral

leads in 2 athletes and inferior leads in 3 athletes and diffuse T-wave inversions in one athlete. PVC were present at ECG in 26 % of cases.

Echocardiographic findings: The mean left atrial diameter was $31 \pm 4,8$ mm ranging from 26 to 39 mm. The mean LV end-diastolic diameter was $52,1 \pm 4,63$ mm ranging from 43 mm to 61 mm. The mean LV end-systolic diameter was $32,7 \pm 1,1$ mm ranging from 26 mm to 47 mm. LV ejection fractions was $66 \pm 9\%$. The mean septal wall thickness was $8,9 \pm 1,1$ mm. The mean posterior wall thickness was $8 \pm 1,9$ mm. E/A ratios were normal in all athletes. In Three athletes the diagnoses of hypertrophic cardiomyopathy (HCM) was evoked. In one case it was predominant on the septum, the HCM was concentric in a second athlete and it was an apical hypertrophy in a third athlete. Holter monitoring showed a mean heart rate $71 \text{ bpm} \pm 2,1$. The mean number of PVC was 920 ± 1008 per day. The number of PVCs ranged from 0 to 40464. Fifty-three per cent (53%) of patients had > 700 PVC / 24 H and 47% of patients had < 700 PVC / 24 H. 31% of athletes showed ventricular couplets. The average of couplets was $375 \pm 3,13$. 28.5% showed triplets or runs. VT episodes of > 10 beats were observed in 2 athletes (4,7%).

The most frequent PVC morphologies were left bundle branch block (LBBB) with inferior axis deviation (90% of cases). In 97,6% PVC were monomorphic. One athlete had polymorphic PVC. According to the LOWN classification (13), 28.5% were in class 4 b. 10 athletes were in class 4a (18%), 16 athletes in class 2(41 %), 9 in class 1(23 %) and 1 in class 0 (2%).

Submaximal exercise tests were performed in 33 athletes with a mean theoretical maximum heart rate 87,5%. In 48% of athletes PVCs disappeared during effort and reappeared during the recovery phase. In the rest of athletes PVCs persisted during all exercise with aggravation and occurrence of couplets and one run in one athlete.

Ten athletes underwent contrast-enhanced cardiac magnetic resonance imaging. Results were normal in 5 athletes. It confirmed HCM in the three athletes. It also revealed a case of arrhythmogenic right ventricular cardiomyopathy (ARVC) and a case of compression of the right ventricle (RV) by pectus excavatum.

Results of decisions of aptitude and follow up:

After clinical evaluation, five athletes (12%) were judged to have dangerous ventricular arrhythmia on the basis of the presence of a morphologic substrate and the type of sports practiced. They were therefore disqualified from sports activity. Table 1 summarizes the main characteristics of these athletes. In 32 athletes (76%) etiological investigation did not detect a cardiovascular origin and after collegial discussion between sports physicians and cardiologists, athletes were allowed to continue their sporting activities and recommended a yearly follow up. During a mean mean follow-up time of 15 months, no athlete of this group presented with a major cardiac event.-Five athletes (12%) were lost of follow up, with no etiologic diagnosis or conclusion on their definitive skills in sport.

DISCUSSION

The prevalence of PVC in athletes in our study was 1.8 %. Cardiac abnormalities disqualifying athletes from competitive sports activity were detected in 12 % of our athletes.

Biffi et al. (14) studied 355 competitive athletes who had PVC at Holter monitoring. Cardiac abnormalities were detected in 7 % of athletes. The rate of cardiac abnormalities was significantly higher in patients having the higher number of PVC. Severe organic heart disease was detected in 12 % of the study group of Steriotis et al. which included 145 athletes with ventricular arrhythmia (15). In the study of Fuchs et al (16), the rate of organic heart disease in athletes with PVC was 7 %. Competitive athletes and women had significantly higher rates of heart disease.

In our study, the most frequent PVC morphology was left bundle branch block (LBBB) with inferior axis deviation in 90%. In 97,6% PVC were monomorphic. Steriotis et al.(15) found that the most frequent PVC morphologies were LBBB (77 %) in 145 athletes referred because of ventricular arrhythmia detected during preparticipation screening.

Table 1: Main characteristics of the disqualified athletes.

	Age	Sport	Symptoms	Number PVC Holter	Lown Classification	Echocardiography	MRI
Athlete 1	24	Karate	none	1720 PVC 52 couplets	Classe 4a	Normal	ARVC
Athlete 2	16	Gymnastics	Dizziness at exercise	6 PVC NSVT	Classe 1	Normal	Compression of the RV by pectus excavatum
Athlete 3	30	football	None	2 PVC	Class 1	Apical HCM	HCM
Athlete 4	17	Handball	none	7500 PVC polymorphic	Classe 3	HCM	HCM
Athlete 5	20	Handball	none	14 PVC	Classe 1	HCM	HCM

PVC: premature ventricular contraction- MRI : Magnetic resonance imaging-HCM : Hypertrophic cardiomyopathy- RV right ventricle. ARVC : Arrhythmogenic right ventricular cardiomyopathy

Biffi et al. (14) reported that the prevalence of LBBB morphology of PVC was similar in athletes having more than 2000 PVC/24 hour and athletes having between 100 and 2000 PVC/24 hour and athletes having less than 100 PVC /24 hour (65 % vs 78 % vs 67 % ; NS). The LBBB morphology was found more frequently in the absence than in the presence of structural cardiac disease.

Delise et al (17) also reported that the prevalent morphology of PVC was LBBB in 72.8 % of athletes.

The mechanism of ventricular arrhythmia in athletes is not well established. According to Biffi A et al. (18) frequent VPCs cannot be interpreted as a manifestation of athlete's heart (19). In their study the ventricular ectopy in elite athletes was not directly related to the magnitude of physiologic left ventricular hypertrophy. They found no statistically significant relation between left ventricular mass and the grade or frequency of ventricular tachyarrhythmias. In addition, a trend was noted in athletes with the most frequent and complex ventricular ectopy toward lower calculated LV mass.

In another study, Biffi et al. (20) followed 71 athletes with frequent PVC who underwent a deconditioning period of at least three consecutive months. After the deconditioning period, the overall study group showed significant reduction in PVC, couplets, and NSVT. The decrease in LV mass after detraining did not differ between athletes who experienced partial or complete reversibility of ventricular arrhythmias with deconditioning and athletes with no change in arrhythmias.

Other factors and mechanisms can potentially promote ventricular arrhythmogenicity in conditioned athletes. Autonomic nervous system adaptations induced by a varying intensity of physical training have been reported to have a role in the genesis of ventricular tachyarrhythmias (21-24). A shift in cardiovascular autonomic modulation from parasympathetic to sympathetic predominance because of an intensive training regimen might have predisposed some athletes to electrical instability, triggering ventricular tachyarrhythmias (25). The resolution of arrhythmias with deconditioning and their reappearance in the early stage of retraining could be explained by this neural hypothesis (20). Furthermore, the possible influence of the hormonal changes (in particular of the cortisol and adrenaline levels) induced by prolonged and intensive training on myocardial irritability could be considered (25).

Heavy and intensive overtraining sport endurance may overload the heart and particularly the right ventricle which may lead to some myocardial injury which could be aggravated by using doping agents. In addition the majority of arrhythmias arise from the right ventricle (26-32). But Rimensberger et al (33) studied 97 marathon runners, ultra-endurance athletes which were Compared with active controls showed no differences with regard to RV global strain and RV end-diastolic area). The number of premature ventricular contractions (PVCs) was weakly

associated with the RV size ($r=0.208$; $p=0.042$). Bohm P et al. (34) compared thirty-three healthy competitive elite male endurance athletes with a training history of 29 ± 8 years, and 33 control subjects cardiovascular magnetic right ventricular end-diastolic volume/body surface area, were significantly increased in athletes in comparison with control subjects but right ventricular ejection fraction did not differ between athletes and control. More follow-up studies are needed to clarify the nature of right ventricular abnormalities in athletes.

Other factors may be possible causative mechanisms of PVC such as old myocarditis (35,36), the presence of left ventricular false tendons (37) or adrenergic innervation abnormalities (38), alterations in blood volume and electrolytes and sinus bradycardia induced by exercise.

The prognosis of PVC in apparently normal hearts is controversial. In our study athletes whose etiological investigation did not detect a cardiovascular origin were allowed to continue their sporting activities and during a mean follow up time of 15 months, no athlete of this group presented with a major cardiac event. Biffi et al. (14) followed for 8 years 355 competitive athletes with PVC at Holter. The mortality rate was 0.3 % and the annual mortality 0.17 %.

Steriotis et al. (15) didn't report any major cardiac events in healthy athletes with ventricular arrhythmia after a follow up of 28 months. Delise et al. (17) followed for 84 months 120 healthy athletes in whom frequent PVC were discovered by chance during preparticipating screening and concluded that they had a long term benign prognosis but in 14.5% of athletes ejection fraction slightly decreased over time.

Recent guidelines recommend that the minimal level of testing in athletes with PVC to acquire prognostic information is a 12-lead ECG and exercise stress test (39-40). In most instances, an echocardiogram will also be performed to rule out a structural abnormality that cannot be identified by either the ECG or stress test. Computed tomography and magnetic resonance imaging studies can be considered, based on the circumstances of the specific arrhythmias noted. Athletes with defined structural heart disease who are considered high risk based on the specific heart disease and who have PVCs with or without treatment should be limited to low-intensity class IA competitive sports. (Class I; Level of Evidence C).

In conclusion: This study shows the necessity of a thorough cardiological assessment of athletes with ventricular arrhythmia in order to detect underlying heart disease and prevent sudden death in this young apparently healthy population. Decision for sports eligibility remain challenging and close follow up is necessary. Larger studies are needed in order to set up a Codified management approach.

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