DR ELIANA TRANCHITA (Orcid ID : 0000-0001-9613-8124)

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Young athletes with ventricular premature beats: Continuing or not intense training and competition?

Address for correspondence:

Eliana Tranchita: e-mail: e.tranchita@uniroma4.it

University of Rome Foro Italico
Department of Movement, Human and Health Sciences, Section of Health Sciences

P.za Lauro de Bosis 15, 00135 Rome, Italy
Phone number: +39 06 36733333; FAX +39 06 36733333

Authors

Attilio Parisi MD[1], attilio.parisi@uniroma4.it

Eliana Tranchita MD[1], e.tranchita@studenti.uniroma4.it

Carlo Minganti PhD[1], carlo.minganti@uniroma4.it

Fabio Sperandii PhD[1], fabio.sperandii@uniroma4.it

Emanuele Guerra PhD[1], emanuele.guerra@uniroma4.it

Leonardo Calò MD[2], leonardo.calo@tin.it

Paolo Borrione MD[1], paolo.borrione@uniroma4.it

Fabio Pigozzi MD[1], fabio.pigozzi@uniroma4.it

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Affiliations

[1]Department of Movement, Human and Health Sciences, Division of Health Sciences, University of Rome Foro Italico. P.za Lauro de Bosis 15, 00135 Rome, Italy

[2] Department of Cardiology - Policlinico Casilino, Via Casilina 1049, 00169 Rome, Italy

Key Words: arrhythmia, sudden death, competitive sport

Abstract

Introduction Isolated ventricular premature beats (VPBs) are commonly found during pre-participation screening in athletes. Currently the debate about the role of detraining in reducing the number of VPBs is still open.

This study evaluated the arrhythmic risk in a population of young competitive athletes who showed VPBs during eligibility evaluation and that did not undergo detraining but continued practicing competitive sports.

Methods 3746 consecutive subjects underwent pre-participation screening. Athletes who showed VPBs were selected and underwent second level evaluation (Echocardiogram, 24h Holter ECG and Exercise test). Athletes were re-evaluated after a follow-up period (6-48 months) while they continued practicing competitive sports.

Results 5,3% of the whole population showed ventricular arrhythmias. 77% of the subjects showed isolated VPBs. 88% of the subjects showed monomorphic VPBs, 22% of athletes showed polymorphic VPBs. At echocardiogram there was not any pathology which contraindicated competitive sport activity. At 24h Holter ECG recording mean number of daily VPBs was 1592 ± 3217 (range 0-16678). At Holter ECG follow-up (16±12 months) the median number of VPBs decreased from 93 (IQR 20-3065) to a new value of 72 (IQR 2-1299).

Conclusion Continuing competitive sport in subjects with ventricular arrhythmias even though frequent but with a low grade of complexity and without structural cardiomyopathy, do not increase sudden death risk.

Introduction

Isolated ventricular premature beats (VPBs) are not an unusual finding in general population and during pre-participation screening in athletes (Steriotis et al. 2013; Bisbal et al. 2012, Niwano et al. 2009). Clinical significance of VPBs was extensively debated in literature (Corrado et al. 2011; Giada et al. 2010; Maron 2006). Although several studies suggest that VPBs are a benign expression of the so called "athletes' heart" (Biffi et al. 2002), many authors advise that VPBs may represent a first sign of a concealed cardiomyopathy, which may increase the cardiovascular risk for Sudden death in athletes. Moreover, Sudden death occurs in the athletic population because of the onset of ventricular tachyarrhythmia. Longitudinal studies analyzed by McClaskey show that Sudden death risk for athletes showing VPBs is strictly linked to the presence of a structural cardiomyopathy such as Hypertrophic cardiomyopathy, Coronary arteries congenital anomalies, Myocarditis, Dilatative Cardiomyopathy and Right Ventricular Arrhythmogenic Cardiomyopathy (McClaskey et al. 2013). Several studies in the Literature analyzed the long term significance of VPBs. In the high-trained athletic population the presence of tachyarrhythmia was correlated to structural cardiomyopathy only in one third of the subjects. Even if showing complex and frequent VPBs, the other two third of the population did not show any kind of concealed cardiomyopathy. This particular condition was compatible with survival without symptoms and adverse cardiac events to the end of a long term follow-up period (Biffi et al. 2002; Verdile et al. 2015). Today the debate about the role of detraining is still open. There are authors who maintain the importance of a period of detraining in order to reduce the

number and the complexity of VPBs in athletes (Biffi et al. 2011); while recently other researchers have demonstrated that continuing sport activities in athletes without cardiomyopathy but with VPBs does not influence the number and the complexity of VPBs (Delise et al. 2011).

The aim of the present study is to evaluate the arrhythmic risk in a population of young competitive athletes who showed VPBs during eligibility evaluation and that continue practicing competitive sports.

Materials and Methods

This is a retrospective longitudinal study. In a six-year period, a population of 3746 consecutive subjects underwent pre-participation screening at Centre of Sport Medicine and Physical Exercise - University of Foro Italico (Rome). The research protocol was submitted to the Department Institutional Board of our University which due to the retrospective cross-sectional nature of the protocol, verified that all the procedures were in accordance with the Helsinki declaration ethical standards. Moreover all subjects were informed verbally and in written form about the procedures of the study and gave their written informed consent.

As required by Italian law, since 1982 all the athletes who participate in competitive sports, have had to undergo a pre-participation screening for cardiovascular disease. This screening provides personal and family history, physical examination, spirometry and a 12-Lead electrocardiogram (ECG) both at rest and after a submaximal effort (step test for 3 minutes). Additional tests are required only for those athletes who have positive findings at the initial evaluation (Decree of Italian Ministry of Health, February 18, 1982). In this study, from the whole population who underwent pre-participation screening, athletes who showed more than two VPBs per 10s tracking during 12-Lead electrocardiogram (ECG) at rest or after step test, were selected (Drezner et al. 2013).

All these athletes underwent a second level evaluation through Echocardiogram, 24h Holter ECG and Maximal exercise test. Hematologic test, blood chemistry and test of thyroid function were carried out to exclude infectious diseases or thyroid diseases. All the athletes were re-evaluated after a follow-up period (6-48 months) while they continued practicing competitive sports.

Athletes who met the following inclusion criteria were selected: 1) aged 18-45, 2) both genders, 3) practising competitive sports, 4) more than 2 VPBs per 10s tracking ECG at rest or after step test; exclusion criteria were: 1) sustained ventricular tachycardia, 2) cardiomyopathy known, 3) family history positive for sudden death, 4) incomplete cardiologic second level evaluation.

All the athletes were asymptomatic and without functional limitation at the study entry. They were engaged in a variety of sport disciplines, most commonly soccer, track and field, swimming and volleyball. They practised sport at a competitive level, for almost 10 hours/week. No athletes needed anti-arrhythmic drugs.

Standard 12-lead ECG was performed with EDAN SE-12 Express® device with the subject in supine position and recorded with paper speed of 25mm/s and at a standard gain of 1mV/cm (Corrado et al. 2010).

The morphology of Ventricular premature beats was defined as "Right Ventricular Outflow Tract"(RVOT) if they showed left bundle branch block-like (LBBB) and the average directions of QRS was positive in DII, DIII and AvF ; "Left Ventricular Outflow Tract" (LVOT) if they showed right bundle branch block-like (RBBB) and the average directions of QRS was positive in DII, DIII and AvF; "Fascicular" if they showed right bundle branch block-like (RBBB) and QRS was negative in DII, DIII and AvF. Ventricular premature beats

may be isolated or organized in repetitive forms such as pairs (two beats) or runs of three or more beats (non sustained/sustained ventricular tachycardia)

Trans-thoracic Echocardiography

Trans-thoracic echocardiography was performed in left lateral decubitus by experienced cardiologists, using an AcusonX300 Siemens® ultrasound device equipped with a 3.5 MHz transducer. Images were acquired from parasternal, apical, subcostal and suprasternal windows. M-mode, bi-dimensional echocardiographic data, as well as pulsed, continuous and colour Doppler flow mapping were recorded (Calò et al. 2015).

24-hour Holter ECG

An ECG recorder RZ-153 Rozinn® was used to evaluate 24-hour ECG monitoring in all patients. Simultaneous three-channel 24h Holter recorded, analyzed and identified ventricular premature beats. QRS morphology was automatically analyzed and then reviewed through manual editing by an experienced Sport Physician. All arrhythmic events were analyzed to identify arrhythmias features. All the athletes were classified in four groups, on the basis of the median number of VPBs/24h: *Absent* ≤ 5 VPBs/24h, *Rare* 6 ≤ VPBs ≤ 93, *Sporadic* 94 ≤ VPBs ≤1207, *Frequent*> 1208 VPBs/24h.

Maximal exercise test

Maximal exercise test was performed with an Ergobike Medical8 Daum Electronic® linked to an ECG wireless device EDAN SE-1010®. The exercise load was increased 30-40 Watt every 2 min while keeping pedal frequency (generally 60-70 rpm) constant, until

exhaustion or when severe fatigue, weakness, dyspnea or severe ECG alterations occurred. A continuous 12-lead ECG recording was performed during the test. Arterial blood pressure and heart rate (HR) were measured before and every 2 min during the test and recovery. Arrhythmias behaviour during maximal exercise test, was classified in 5 categories: *Pattern 0*, without arrhythmias during all the test; *Pattern 1* when VPBs were present at rest and during recovery, but disappeared during the effort; *Pattern 2* when VPBs were present only during recovery; *Pattern 3* when VPBs were present throughout the test duration; *Pattern 4* when arrhythmias were stimulated by the effort.

Cardiac Magnetic Resonance

Selected on a clinical basis, some patients underwent additional testing for the purpose of detecting or defining underlying cardiovascular disease, including magnetic resonance imaging (MRI) (n 13/106). Cardiac Magnetic Resonance imaging was performed on a 1.5-T clinical scanner (Philips Intera CV, Best, the Netherlands) using electrocardiographic gating and a phased array cardiac receiver coil. Steady-state, free-precession breath-hold cines imaging was acquired in 3 long-axis planes and sequential short-axis slices from the atrioventricular ring to the apex. Delayed enhancement images (LGE) covering the whole ventricle were acquired approximately 10-15 min after intravenous administration of 0.2 mmol/kg body weight gadolinium-DTPA with breath-hold 2-dimensional segmented inversion-recovery sequence or phase-sensitive inversion-recovery sequences in identical planes as in cine images. Inversion time was optimized to null normal myocardial signal. For phase-sensitive sequences, uncorrected magnitude images were used. All measurements were performed by an experienced operator at the magnetic resonance core laboratory.

Statistical Analysis

Data analysis was performed using SPSS ver.21(IBM). Normal distribution of continuous variables was verified through Kolmogorov-Smirnov test. For continuous variables normally distributed, mean ± standard deviation was reported. For continuous variables not normally distributed, median value and interquartile range were reported; categorical variables were expressed as frequency counts, with their percentages. Statistical analysis of categorical variables was made through Pearson Chi-square test for independence, For continuous variables not normally distributed Wilcoxon test was used, while for continuous variables normally distributed ANOVA was used. Statistical significance was established as $p \leq 0,05$. Furthermore Mc Nemaar test was used to test if the proportion of subjects classified on the basis of VPBs/24h , differs at the follow-up.

Results

From the whole population who underwent pre-participation screening (3746 subjects), 199 athletes who showed VPBs were found. On the basis of inclusion and exclusion criteria, was defined a selected population of 106 competitive athletes both male (n.68) and female (n.38), mean age 22 ± 5 years.

These subjects were evaluated with second level exams that were carried out within two weeks. Athletes who underwent MRI needed four weeks and were considered temporarily not eligible until the medical report excluded cardiomyopathy.

All the athletes showed normal electrocardiographic findings based on Seattle Criteria (Drezner et al. 2013): sinus rhythm, mean Heart Rate 70±9 bpm, mean PR was 0,15±0,02 sec, mean QTc was 0,38±0,03 sec, mean QRS was 0,08. No significative anomalies of ventricular repolarization, no T wave inversion, no ST changes, no pathological Q waves were found.

VPBs morphology

Most of the subjects showed monomorphic VPBs (88%), 13 athletes showed polymorphic VPBs (22%). The most frequent morphology was RVOT (44,3%), then LVOT (23,6) Fascicular (17%) and other morphology less frequently (15,1%) (see Fig1)

Most of ventricular premature beats were isolated (77%), only 33% of subjects showed repetitive

forms (23 athletes showed pairs, 6 athletes showed run of non sustained ventricular tachycardia, max 8 beats) There was not any relation between morphologies of VPBs and gender or the age of subjects.

Echocardiographic findings

All the athletes underwent echocardiogram in order to exclude the presence of a concealed cardiomyopathy. The mean value of Ejection fraction was 62 ± 3%, Left ventricular end diastolic diameter was 49,4 ± 4,8 mm, Left ventricular end systolic diameter was 30,6 ± 4,5 mm; posterior wall and interventricular sept thickness was 8,3 ± 1,1 mm, left atrial diameter was 33,6 ± 9,1 mm, Aortic valve diameter was 30,5 ± 3,2 mm. There was not any pathology, which contraindicated competitive sport activity; on the contrary in 58,5% of the athletes there were not any kind of echocardiographic anomalies, in 17,9% of the subjects there was arching of mitral valve and in 11,3% there was a real mitral valve prolapse. In lower percentage there were other minimal anomalies that nevertheless did not suggest any kind of cardiomyopathy (see Fig 2).

There was not any significative statistic relationship between echocardiographic findings and morphology nor the number or the complexity of arrhythmias found during 24h Holter ECG recording. (see Table 1)

Maximal exercise test findings

All the athletes underwent this test. Nobody was treated at the moment with anti-arrhythmic drugs and no one reported cardiovascular signs or symptoms. Most of the athletes did not show any kind of arrhythmias during the test (35,8%), or VPBs disappeared during the effort (34,9%). A small percentage of athletes showed VPBs throughout the test (11,3%) or only during recovery (9,4%). 9 athletes showed exercise-induced arrhythmias (8,5%). All the VPB were isolated, except for 6 athletes who showed arrhythmias rarely organized in pairs.

24-hour Holter ECG

All the athletes underwent this recording. Nobody was treated at the moment with anti-arrhythmic drugs, no one reported cardiovascular symptoms during recording. Most of the subjects (77%) showed only isolated VPBs, 23 subjects showed coupled VBPs, 6 subjects showed non sustained ventricular tachycardia (max 8 beats). Mean number of daily VPBs was 1592 ± 3217 (range 0-16678). Most of the subjects showed VBPs uniformly distributed during 24h. Some athletes (n.20) showed VPBs mainly during wakeful hours. There was not any relation between the number of BPVs/24 and the morphology nor the complexity of arrhythmias.

Cardiac Magnetic Resonance findings

13 athletes underwent magnetic resonance imaging because they showed more than 10.000 BPVs/24h e/o frequent couple, NSVT or Polymorphic VPBs. Late-enhancement was used to evaluate the presence of fibrosis, myocarditis, cardiomyopathy or myocardial scarring and no subjects presented evidence of intra-myocardial delayed enhancement nor signs of cardiomyopathy.

24-hour Holter ECG Follow-up

All the subjects were followed through a second 24h Holter ECG, after at least 6 months during which athletes continued competitive activities. Follow-up lasted on average 16±12 months (range 6-48 months). Comparing the median number of VPBs/24 between the first and the second recording, the median value changed from 93 (IQR 5-1207) to a new value of 72 (IQR 2-608). This decrease is statistically significant (p= 0,005) and suggests a tendency of VPBs to reduction during the months even though the athletes continued to practise competitive sports. Mc Nemaar test confirms this behaviour of ventricular arrhythmias after follow-up, showing a decrease in the number of subjects classified as frequent and rare VPBs and an increase in the number of subjects classified as absent and sporadic VPBs. (Tab.2)

Analysing data basing on arrhythmias morphology can be noticed that RVOT and Fascicular arrhythmias tend to reduce more than LVOT.

We noticed that 24,5% of athletes showed complex VPBs during Holter ECG recording. This finding was reduced in the second evaluation, in which 19,8% of subjects showed complex VPBs. The complexity of VPBs was reduced in subjects with normal echocardiogram and in subjects with echocardiographic anomalies, too.

The number of VPBs/24h was reduced both in subjects with normal echocardiogram and in subjects with echocardiographic anomalies; it is important to evaluate that VPBs tend to reduce more in subjects with normal echocardiogram (p <0,001) than in subjects with echocardiographic anomalies (p = 0,008) See Table 3

Clinical follow-up

We observed these athletes during follow-up period and nobody developed symptoms such as palpitations, atypical chest sensation, dyspnea or dizziness, no one experienced syncope or near-syncope. There was not any case of sudden death. No athletes during follow-up period showed clinical or instrumental changes that required additional investigations.

Discussion

In this study a population of young competitive athletes who showed ventricular arrhythmias during pre-participation screening was evaluated. A prevalence of VPBs of 5% on the whole population analysed was observed , similarly with other studies in Literature (Biffi et al. 2002; Biffi et al. 2004; D'Ascenzi et al. 2016). All the subjects underwent a second level evaluation through Echocardiogram, Maximal exercise test, 24h Holter ECG, and sometimes RMN was required. They were followed for 6-48 months while they continued practicing competitive sports since nobody showed structural cardiomyopathy. Most of the subjects showed monomorphic VPBs (88%) and prevalent morphology of BPVs was RVOT (44,3%). These findings are in agreement with those present in the Literature (Delise 2013, Verdile 2015, Steriotis 2013), reporting that in competitive athletes the most prevalent morphology of VPBs is RVOT. There was not any relation between VPBs morphology and the number of VPBs/24h nor the complexity of VPBs.

At echocardiographic evaluation nobody showed structural cardiomyopathy that contraindicated competitive sport activity. At maximal exercise test, 8,5% of athletes showed exercise-induced arrhythmias, and is known that prognosis depends more on the presence of a structured cardiomyopathy than on the presence of VPBs during exercise test (D'Ascenzi 2016). At 24h Holter ECG most of the athletes showed isolated arrhythmias, in 33% of subjects there were complex VPBs. Athletes who underwent RMN did not show structural cardiomyopathy that contraindicated competitive sport.

In none of the subjects was suspected the presence of Hypertrophic cardiomyopathy, nor infiltrative cardiomyopathy, based on family history, basal ECG, symptoms, second and third level exams.

Is known from Literature that High Sensivity Troponin T (hs-TnT) is an important marker of cardiac damage. In particular hs-TnT is a helpful diagnostic indicator for accurate differentiation between infiltrative cardiomyopathy and Hypertrophic Cardiomyopathy (Kubo T, 2015). Furthermore elevated hs-TnT is highly suggestive of acute myocarditis, if other causes of increased myocardial necrosis markers such as myocardial ischemia have been excluded (Ukena C, 2014).

Recent studies suggest the clinical utility of MRI combined with high-sensitivity troponin T assay (hs-TnT) in the diagnosis of inflammatory cardiomyopathies and for the prediction of the clinical outcome of these pathologies (Šramko M, 2013).

Considering that echocardiogram and MRI may be negative even though hs-TnT is elevated, it would be useful evaluate this marker in athletes with ventricular arrhythmias to recognize the presence of a possible concealed cardiomyopathy.

However, the study was retrospective, and this marker was not evaluated; it would be interesting in a future study to put in evidence relation between arrhythmic burden and high sensitive troponin in athletes.

All athletes were followed through 24h Holter ECG. After a median period of 16 ± 12 months, the number of VPBs/24 was significantly reduced. In particular was noticed that RVOT and Fascicular arrhythmias tend to reduce more than LVOT; these findings comply with recent studies in the Literature which define RVOT morphology as a benign pattern of VPBs.

Comparing with the studies of Biffi and colleagues (Biffi et al. 2004), who analyzed 50 competitive athletes of the same age, with healthy heart and VPBs frequent (>2000) and complex, in this study is highlighted a reduction of VPBs in a similar percentage of the population (60,3% present study vs 71% Biffi and c.) without availing of detraining. Is important to clarify that there are some differences between the population of the present

study and that analysed by Biffi and colleagues. They evaluated top level athletes while the subjects of this investigation were competitive but non elite athletes; moreover the population of athletes analysed during the present study showed a lower mean number of VPBs.

All the athletes continued to train during the follow-up period, practicing competitive sports. Nobody manifested cardiovascular symptoms or syncope during exercise. In this kind of athletes with healthy hearts and isolated and monomorphic arrhythmias, the risk of adverse cardiac events proved to be extremely low, independently from the number of VPBs/24h. This follow-up study supports the concept that these kinds of arrhythmias, observed during evaluation of competitive athletes, have a benign behaviour and they do not necessitate stopping competitive practise. Continuing competitive training did not influence the behaviour of arrhythmias. The results of the present study comply with Delise recent studies (Delise et al. 2011).

It can be said that in absence of structural disease, a follow up with second level examination can allow continuing sport activities without an increase of cardiovascular risk for sudden death.

Perspectives

The role of VPBs in competitive athletes is an issue of concern for sport cardiology practitioners. This research evaluated a large group of competitive athletes, but they were not elite athletes like those analysed by Biffi and colleagues (Biffi et al 2002). The group of subjects analysed in this study conversely, is representative of the general population who practise sports at all level of competition which represents most people who practise sports all over the world. Moreover these athletes continued to practise competitive sports, without a period of detraining.

As known from clinical practise, the discovery of VPBs constitutes a clinical dilemma when deciding on eligibility for sport. Can be confirmed that continuing sport activities, also at a competitive level, in subjects without structural cardiomyopathy, who show ventricular arrhythmias even though frequent but with a low grade of complexity, do not increase sudden death risk. We suggest a 6-12 month re-evaluation in these athletes is important to monitor their health status.

These suggestion comply with recent European and American Guidelines which recommend an extensive cardiovascular evaluation to rule out underlying structural or electrical abnormalities in subjects who show ventricular arrhythmias (Heidbüchel H et al. 2006; Zipes DP et al 2015).

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Tab. 1: Relationship between Echocardiographic findings and VPBs features

Echocardiographic findings	Total Number	**VPBs/24h**				**Complex VPBs**		**BPVs Morphologies**			
		Absent	Rare	Sporadic	Frequent	No	Yes	RVOT	LVOT	Fascicular	Others
Normal	62	16 (26%)	12 (19%)	17 (27%)	17 (27%)	48 (77%)	14 (23%)	29 (47%)	17 (27%)	8 (13%)	8 (13%)
Mitral Arching	19	4 (21%)	3 (16%)	5 (26%)	7 (37%)	14 (74%)	5 (26%)	8 (42%)	3 (16%)	5 (26%)	3 (16%)
Mitral Valve Prolapse	12	4 (33%)	4 (33%)	3 (25%)	1 (8%)	10 (83%)	2 (17%)	4 (33%)	2 (17%)	5 (42%)	1 (8%)
Floppy IAS	4	1 (25%)	3 (75%)	0	0	3 (75%)	1 (25%)	2 (50%)	1 (25%)	0	1 (25%)
False Tendon	6	1 (17%)	3 (50%)	1 (17%)	1 (17%)	4 (67%)	2 (33%)	2 (33%)	2 (33%)	0	2 (33%)
AO Bicuspid	1	1 (100%)	0	0	0	1 (100%)	0	0	0	0	1 (100%)
IV Defect	2	0	1 (50%)	0	1 (50%)	0	2 (100%)	2 (100%)	0	0	0

IAS: Inter Atrial Septum; AO: Aortic Valve; IV: Inter Ventricular; VPBs: Ventricular premature Beats; RVOT: Right ventricular outflow tract; LVOT: Left ventricular outflow tract; Complex VPBs: pairs and non sustained ventricular tachycardia.
VPBs/24h: *Absent* ≤ 5 VPBs/24h, *Rare* 6 ≤ VPBs ≤ 93, *Sporadic* 94 ≤ VPBs ≤1207, *Frequent*> 1208 VPBs/24h.

Tab. 2 Changes of Ventricular premature Beats (VPBs) features

	1st Evaluation	**2nd Evaluation**	***P***
VPBs number at ECG	3 ± 2	2 ± 1	
Complex VPBs at ECG	0	0	
Mean VPBs/24h	1592 ± 3217	1091 ± 2958	**0.005**
Median VPBs/24h	93	72	
Athletes with pair VPBs	23	19	
Mean number of pair VPBs/24h	92 ± 340 (min 1 - max 1598)	3 ± 4 (min 1 – max 17)	0.128
Athletes with NSVT	6	3	
Mean number of NSVT/24h	1 ± 0.41	1 ± 0.0	
Number of beats per NSVT	4.50 ± 2.07	10.33 ± 8.74	
Athletes with Monomorphic VPBs	93	93	
Athletes with Polymorphic VPBs	13	13	
Athletes with Absent arrhythmias	27	41	**0.037**
Athletes with Rare arrhythmias	26	16	**0.037**
Athletes with Sporadic arrhythmias	26	32	**0.037**
Athletes with Frequent arrhythmias	27	17	**0.037**
RVOT VPBs /24h	2178.76 ± 3893.04	1505.48 ± 3873.33	**0.022**
LVOT VPBs /24h	1530.2 ± 2900.89	1586.68 ± 2755.75	0.830
Fascicular VPBs /24h	732.94 ± 1788.76	182.44 ± 303.95	**0.006**
Others VPBs /24h	931.5 ±2538.57	126.81 ± 284.3	0.778

NSVT: non sustained ventricular tachycardia; RVOT: right ventricular outflow tract; LVOT: left ventricular outflow tract
Complex VPBs: pair VPBs and non sustained ventricular tachycardia
Absent ≤ 5 VPBs/24h, Rare 6 ≤ VPBs ≤ 93, Sporadic 94 ≤ VPBs ≤1207, Frequent> 1208 VPBs/24h.

Tab 3. Relationship between Echocardiographic findings and holter ECG follow-up

Echocardiographic findings	Total Number	**Holter ECG 1**				**Complex VPBs**		**Holter ECG 2**				**Complex VPBs**	
		Absent	Rare	Sporadic	Frequent	No	Yes	Absent	Rare	Sporadic	Frequent	No	Yes
Normal	62	16 (26%)	12 (19%)	17 (27%)	17 (27%)	48 (77%)	14 (23%)	25 (40%)	11 (18%)	14 (23%)	12 (19%)	53 (85%)	9 (15%)
Mitral Arching	19	4 (21%)	3 (16%)	5 (26%)	7 (37%)	14 (74%)	5 (26%)	6 (32%)	1 (5%)	9 (47%)	3 (16%)	15 (79%)	4 (21%)
Mitral Valve Prolapse	12	4 (33%)	4 (33%)	3 (25%)	1 (8%)	10 (83%)	2 (17%)	5 (42%)	2 (17%)	4 (33%)	1 (8%)	9 (75%)	3 (25%)
Floppy IAS	4	1 (25%)	3 (75%)	0	0	3 (75%)	1 (25%)	2 (50%)	1 (25%)	0	1 (25%)	2 (50%)	2 (50%)
False Tendon	6	1 (17%)	3 (50%)	1 (17%)	1 (17%)	4 (67%)	2 (33%)	2 (33%)	1 (17%)	3 (50%)	0	5 (83%)	1 (17%)
AO Bicuspid	1	1 (100%)	0	0	0	1 (100%)	0	1 (100%)	0	0	0	0	1 (100%)
IV Defect	2	0	1 (50%)	0	1 (50%)	0	2 (100%)	0	0	2 (100%)	0	1 (50%)	1 (50%)

IAS: Inter Atrial Septum; AO: Aortic Valve; IV: Inter Ventricular; VPBs: Ventricular premature Beats; RVOT: Right ventricular outflow tract; LVOT: Left ventricular outflow tract; Complex VPBs: pairs and non sustained ventricular tachycardia.

VPBs/24h: Absent ≤ 5 VPBs/24h, Rare 6 ≤ VPBs ≤ 93, Sporadic 94 ≤ VPBs ≤1207, Frequent> 1208 VPBs/24h.

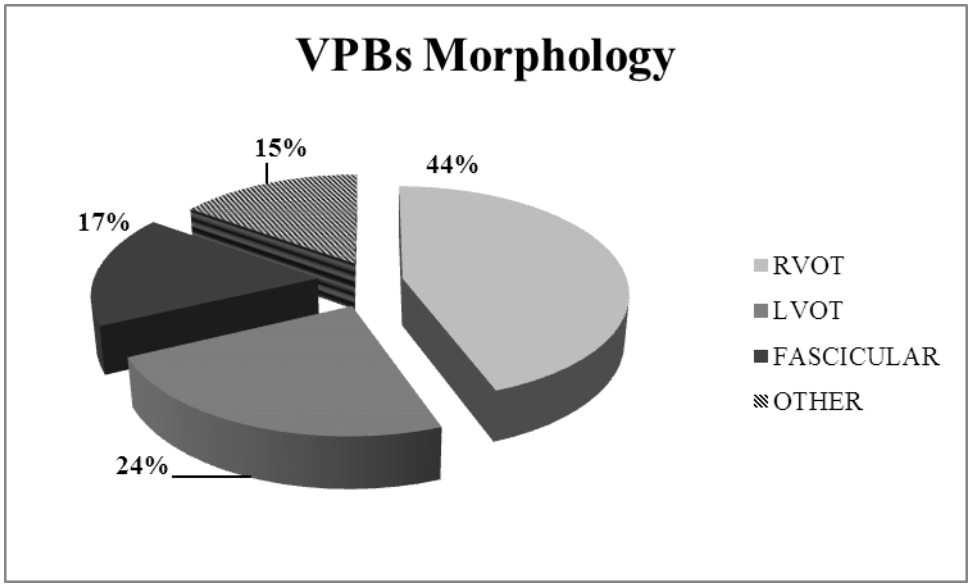

Fig 1. VPBs Morphology

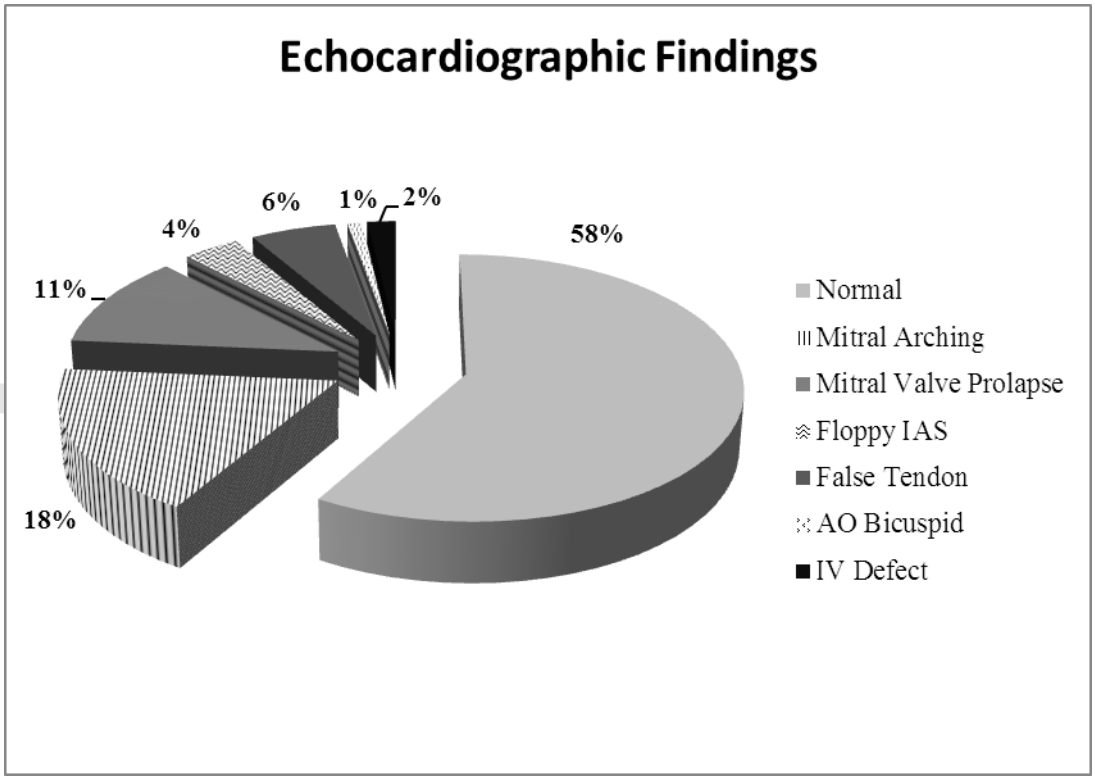

Fig 2 Echocardiographic Findings