ORIGINAL ARTICLE

GMJ.2015;4(4):151-58
www.gmj.ir

Received: 2015-01-04
Revised: 2015-02-14
Accepted: 2015-06-07

Electrocardiography Findings in Iranian Premier League Football Players

Hooman Angoorani[1], Mohamadsadegh Haghi[1]✉

[1]Department of Sports and Exercise Medicine, Hazrat Rasool-e-Akram Hospital, Iran University of Medical Sciences, Tehran, Iran

Abstract

Background: Sudden cardiac death (SCD) is the leading cause of death in athletes during sport. Electrocardiography (ECG) is a useful tool to detect underlying cardiovascular conditions that may increase the risk for SCD. The aim of the present study is to evaluate common ECG changes among professional football players.**Materials and Methods:** All football players of Iranian Premier League in season 2013-2014 participated in this descriptive study (258 football players). The standard 12-lead ECGs were evaluated and ECG analysis was performed according to previously described criteria.**Results:** Electrocardiogram evaluation showed that the percent of ECG changes was as follows; Inverted T (7.7%), Depression ST (2.3%), Bradycardia (0.3%), St Elevation (2.7%), Left ventricular hypertrophy (1.5%), Left bundle branch block (0.3%), Incomplete right bundle branch (0.3%), Incomplete left anterior bundle (0.8%), branch Incomplete left posterior bundle branch (1.1%), Wolf Parkinson white (0.3%), Left axis deviation (1.5), Right axis deviation (2.3%), ECG finding in favor of HCM (3.1%) and finally ECG finding in favor of IHD(10%).**Conclusion:** Most electrocardiographic variables in Iranian professional football players were lower than the worldwide football players that may be related to the lower level of physical fitness among Iranian football players.**[GMJ.2015;4(4):151-58]**

Keywords: Professional Football Players; Echocardiography; ECG Findings; Sudden Cardiac Death

Introduction

The heart of an athlete has posed a great challenge for clinicians and scientists for more than a century. Early investigations in the late 1800s and early 1900s noted that systematic training in predominantly enduring sports and isometric sports triggers increases in cardiac mass and structural remodeling in many athletes [1].

This physiologic form of enlargement and increased volume of ventricular chambers [2], sometimes accompanied by increased thickness of the left ventricular wall 64 and an increase in the size of the left atrium, with preservation of systolic and diastolic function (athlete's heart), are regarded as a benign adaptation to systematic athletic training with no adverse cardiovascular consequences [3, 4].

On the other hand, genetic factors may also have a role in this process as the angiotensin-converting–enzyme genotype has been

GMJ

Fax: +98 731 2227091
PO Box 7461686688
Email:info@gmj.ir

✉ **Correspondence to:**
Mohamadsadegh Haghi,Department of Sports and Exercise Medicine, Hazrat Rasool-e-Akram Hospital, Iran University of Medical Sciences, Tehran, Iran
Telephone Number: +9821-88257293
Email Address: sadegh6001@yahoo.co.uk

associated with the magnitude of exercise-induced left ventricular hypertrophy in endurance athletes [5].
Other physiologic adaptations to training include a variety of abnormal patterns on 12-lead electrocardiograms in about 40 percent of athletes, some of which resemble cardiac disease (greatly increased voltages, Q waves and repolarization abnormalities) [6].
The ability to make clinical distinctions between physiologic athlete's heart and pathologic conditions has critical implications for trained athletes to reducing the risk of sudden cardiac death (SCD) or progression of cardiovascular disease [7]. The tragic nature of sudden death in young, previously asymptomatic athletes, has led to considerable efforts aimed at prevention. Causes of SCD in athletes are hypertrophic cardiomyopathy (HCM) followed by coronary artery anomalies which are more common in man (>90%) and in certain sports, such as basketball and football [3].
Electrocardiogram (ECG) is an excellent tool in the evaluation of athletes, providing important prognostic and diagnostic information on a variety of cardiovascular disorders that are associated with an increased risk of SCD during sports. Due to the ease of administration, low cost and ability to detect HCM, long QT syndrome and ischemic heart disease, ECG is proposed to be used as a routine screening tool in athletes [8].
Electrocardiogram changes in athletes are common and usually reflect structural and electrical remodeling of heart as an adaptation to regular physical training (athlete's heart) [9, 10]. However, abnormalities of athlete's ECG may be an expression of an underlying heart disease which carries a risk of SCD during sport [11]. It is important that ECG abnormalities resulting from intensive physical training and those potentially associated with an increased cardiovascular risk are correctly distinguished [12]. Studies demonstrate that without further education, the ability of many physicians to accurately interpret an athlete's ECG is relatively poor and may lead to an unacceptable rate of false-positive interpretations and unnecessary secondary evaluations [13]. However, providing physicians standardized criteria with which to evaluate an ECG considerably improves accuracy.
Because of the importance of these tests in the elimination of SCD risk in athletes and the lack of standardization in Iranian population, the aim of this study is to focus on the physiological ECG adaptations commonly found in Iranian football players in Premier League in season 2013-2014 to help physicians distinguish normal ECG changes from abnormal ECG findings related to a pathological cardiac condition associated with SCD.

Materials and Methods

Study Population
All football players of Iranian Premier League in season 2013-2014 with a total of 258 football players representing 18 teams from different phylum of Iran (Azarian, Kurdish, Arabian, etc.) referred to Iranian National Academy of Football for pre-competition evaluation participated in this descriptive study. All included professional athletes had been training and competing for >3 consecutive years and were between 18-35 years old (Table1).
All participants were given a brief explanation about the investigation and also were asked to sign a consent form approved by the Committee on Ethics in Research, Iran University of Medical Sciences. The competitive athletes were evaluated according to a cardiovascular program that included 12-lead ECG.

Electrocardiography
Standard 12-lead ECGs (Mac 800, GE Medical Systems: Germany) were performed with the subject in the supine position after

Table 1. Characteristics of 258 adolescent professional football players

	Min	Max	Mean	Standard deviation
Age(years)	18	35	25.55	3.90
Height(cm)	163	195	178.19	5.30
Weight(kg)	60	95	73.70	6.27
BSA (m^2)	1.6	2.8	1.82	0.15

BMA: Body Surface Area

a few minutes of rest during quiet respiration and recorded at 25mm/s. ECG analysis was performed according to previously described criteria [14]. In particular, we measured heart rate (beats/min), PR interval (ms), QRS duration (ms), QT interval corrected for the heart rate(s), presence of Q waves (>0.04 seconds in duration or 0.>25 of height of ensuring R wave or QS pattern in two or more leads) and Sokolow-Lyon criterion for LV hypertrophy, presence of left enlargement (negative potion of p wave in lead v1=>0.1 mv in depth and =>0.04 in duration) or right arterial enlargement (peaked p wave in leads 2 and 3 or v1=>0.25 mv in amplitude), presence of right or left axis deviations, presence of pathologic R wave (=>0.5 mv in amplitude + R/s ratio=>1), presence of bundle branch block (complete left, complete right, incomplete left anterior, incomplete left posterior and incomplete right) presence of ST-segment elevation (_1 mm, in _2 continuous leads), presence of J-wave (_1 mm), or ST-segment slurring [15], T-wave inversion (>1 mm in depth in two or more leads V2–V6, II and aVF, or I and aVL) and flat/biphasic T-wave pattern (in _2 continuous leads).

Results

In the present study, 258 football players of Iranian Premier League in season 2013-2014 were evaluated according to their cardiac situation. The results of their electrocardiograms are presented in Table 2. The ECG of 203 football players showed no abnormality. The rest ECG of 55 football players had some degree of abnormalities that were listed in Table 2.
ECG findings in favor of IHD were the most abnormalities among football players of Iranian Premier League (26 participants).

Discussion

Regular and long-term participation in intensive exercise(minimum of 4 h/week) is associated with unique electrical manifestations [10]. A challenge in the use of ECG for screening or diagnostic evaluations in athletes is the ability to accurately differentiate findings suggestive of a potentially lethal cardiovascular disorder from benign physiological adaptations occurring as a result of regular and sustained intensive training. In this study, clinical significance of abnormal electrocardiographic patterns in trained athletes was presented with focus on HCM as the main common cause of SCD in professional football players.

T Wave Inversion (Inverted T)
T Wave Inversion (TWI) in the lateral or inferolateral leads is seen commonly in HCM. In a series of asymptomatic patients ≤35 years old with HCM confirmed by cardiac MRI, 62% exhibited TWI [16]. Similarly, in patients with a positive HCM, genetic test and overt morphological HCM, 54% demonstrate TWI. Abnormal TWI is defined as >1mm in depth in two or more leads V2–V6, II and aVF or I and aVL (excluding leads III, aVR and V1). Deep TWI in the mid-precordial to lateral precordial leads (V4–V6) should raise the possibility of apical HCM.
In healthy athletes, TWI in the lateral or inferior leads is uncommon and the prevalence of TWI in the lateral or inferior leads is about 2% [6].
In Iranian football players in premium league, the frequency of TWI was 7.7% that is more

Table2. Electrocardiographic findings in Iranian football players in season 2013-2014

Problem in Electrocardiography	N (%)
Inverted T	20 (7.7%)
Depression ST	6 (2.3%)
Bradycardia	1(0.3%)
St Elevation	7(2.7%)
Left ventricular hypertrophy	4(1.5%)
Left bundle branch block	1(0.3%)
Incomplete right bundle branch	1(0.3%)
Incomplete left anterior bundle branch	2(0.8%)
Incomplete left posterior bundle branch	3(1.1%)
Wolf Parkinson white	1(0.3%)
Left axis deviation	4(1.5%)
Right axis deviation	6(2.3%)
ECG finding in favor of HCM	8(3.1%)
ECG finding in favor of IHD	26(10%)

than the average of this from the world wide population of elite athletes which may be due to their ethics.

ST Segment Depression

ST segment depression is a common abnormality in HCM but extremely rare in otherwise healthy athletes, making it a concerning indicator of disease if identified on an athlete's ECG. ST segment depression is reported in 46–50% of patients with HCM, but in <1% of apparently healthy athletes or adolescents undergoing ECG screening [14]. Any degree of ST depression beyond 0.5mm in two or more leads is significant and requires further investigation for cardiomyopathy. The frequency of ST segment depression in Iranian football players in premium league is about 2.3%. This statistics is close to the one usually reported in healthy athletes.

Bradycardia

In healthy adults, sinus rhythm <60 beats/min is considered as 'sinus bradycardia. In well-trained athletes, resting sinus bradycardia is a common finding due to increased vagal tone. In endurance athletes, aerobic training also may induce intrinsic adaptations in the sinus node with decreased automaticity resulting in a high prevalence of sinus bradycardia [17, 18]. In the absence of symptoms such as fatigue, dizziness or syncope, a heart rate ≥30 beats/min should be considered normal in a well-trained athlete [15]. Sinus bradycardia disappears with an increase in heart rate during physical activity. The frequency of bradycardia in Iranian football players in premium league is about 0.3%

Early Repolarization (ST Elevation)

Early repolarization is an ECG pattern consisting of ST elevation and/or a J wave (distinct notch) or slur on the downslope of the R wave. Traditional examples of early repolarization are referred to ST elevation, but newer definitions also include J waves or terminal QRS slurring. Early repolarization is a common finding in trained athletes and considered a benign ECG pattern in apparently healthy and asymptomatic individuals [15]. Depending on how it is defined, early repolarization is reported in up to 35–91% of trained athletes and is more prevalent in young males and black/Africans [19]. The early repolarization pattern in athletes typically involves a concave and ascending/upward ST segment elevation [15, 20]. Late QRS slurring or notching with horizontal ST segment elevation in the inferolateral leads has been associated with an increased risk of arrhythmic death in one study of middle-aged, nonathletic Finnish citizens [21]. However, a significant percentage of young competitive athletes (25–30%) show early repolarization with similar morphological features in either inferior or lateral leads [22, 23]. These findings are more common in athletes at times of peak fitness suggesting that early repolarization is a dynamic process and is at least in part a direct result of exercise training [22]. To date, no data support the presence of an association between early repolarization and SCD in athletes.

Although further investigation is warranted to fully characterize the prognostic implications of early repolarization in competitive athletes, all patterns of early repolarization including inferolateral subtypes should be considered normal variants in athletes [15]. In Iranian football players in premium league, the prevalence of early repolarization was 2.7% which is lower than the statistics reported in the previous surveys. This finding might be due to lower levels of physical fitness among Iranian football players.

Left Ventricular Hypertrophy

The most commonly used voltage criterion for LVH is the Sokolow-Lyon index. However, ECG QRS voltage may not be a reliable predictor of LVH. The limitation of ECG in identifying ventricular hypertrophy is due to the reliance of measuring electrical activity of the heart by electrodes on the surface of body. Consequently, anything between left ventricular myocardium and the surface electrodes will affect the voltage. ECG QRS voltage, therefore, can be influenced by a variety of factors other than LV size or mass. Males, athletes and black/African individuals have higher QRS voltage, while obesity, older age and pulmonary disease may cause lower voltage.

Correlation with echocardiography is limited and reference standards from autopsy or MRI are not available [24].

In athletes, intensive conditioning is also associated with morphological cardiac changes of increased cavity dimensions and wall thickness that are reflected on ECG. These changes constitute physiological LVH in trained athletes and usually manifest as an isolated increase in QRS amplitude. ECGs with increased QRS amplitudes meeting ECG voltage criteria for LVH are prevalent and present in up to 45% of athletes and 25% of sedentary young adults [25]. Therefore, the accuracy of increased QRS voltage as an indicator of pathological LVH is poor. Based on this study, only 1.5% of Iranian football players met ECG voltage criteria for LVH which is lower than expected. This finding may be due to lower levels of physical fitness among Iranian football players.

Left and Right Bundle Branch Block

Left bundle branch block (LBBB) is an abnormal finding detected in 2% of patients with HCM but not reported in screening populations of athletes or adolescents [14]. LBBB prevalence in Iranian football players was approximately 0.3%. LBBB pattern with QRS duration of 120ms or greater needs further prompt evaluation. Right bundle branch block (RBBB) is found more commonly in HCM than in athletes but the frequency of incomplete and complete RBBB in athletes is felt to limit its differentiating value [26]. The significance of anon-specific intraventricular conduction delay (IVCD) with normal QRS morphology is uncertain. However, marked nonspecific IVCD >140ms is considered abnormal and needs further prompt evaluation [27].

Incomplete RBBB (IRBBB)

IRBBB is defined as a QRS duration <120ms with an RBBB pattern: terminal R wave in lead V1 (rsR0) and wide terminal S wave in leads I and V6. IRBBB is seen in less than 10% of the general population but is observed in up to 40% of highly trained athletes, particularly those engaged in endurance training and mixed sport disciplines including both aerobic and anaerobic components. However, the prevalence of IRBBB in Iranian football players (0.3%) is completely lower than the statistics usually reported in the previous studies. This big difference might be due to several factors including ethics and training intensity [28]. It has been suggested that the mildly delayed RV conduction is caused by RV remodeling, with increased cavity size and resultant increased conduction time, rather than an intrinsic delay within the His-Purkinje system itself [29].

The occurrence of IRBBB in an asymptomatic athlete with a negative family history and physical examination does not require further evaluation. During physical examination, particular care should be devoted to the auscultation of a fixed splitting of the second heart sound because IRBBB can be an associated ECG finding in patients with an atrial septal defect [8].

Incomplete Left Bundle Branch (Anterior and Posterior)

Incomplete left anterior bundle branch is more common in men and increases in frequency with advancing age. The estimated prevalence in general population (ages less than 40 years) is 0.5–1.0% a figure similar to that reported in the athletic population in Iran. Although isolated incomplete left anterior bundle branch is usually an incidental ECG finding in subjects without evidence of structural heart disease, the association with a variety of cardiovascular disorders has been reported. Isolated incomplete left anterior bundle branch is a very rare finding, being usually associated with RBBB. Combinations of complete/incomplete bundle branch block reflect a more extensive involvement of specialized conduction system and carry an increased risk of clinically significant AV block [8].

Wolf Parkinson White (WPW)

Diagnosis of WPW is confirmed by characteristic electrocardiogram changes, which include a delta wave, short PR interval and widened QRS complex [30]. Utilization of electrocardiogram as part of a pre-participation physical evaluation may allow early identification of asymptomatic individuals with a

WPW pattern. Risk stratification techniques identify individuals at risk for malignant arrhythmias who may be candidates for curative therapy through transcatheter ablation. WPW accounts for at least 1% of sudden death in athletes and has a prevalence varying from 0.1 to 0.3%. The present study showed that the prevalence of WPW in Iranian football players was similar to previous research findings. The risk of lethal arrhythmia appears to be higher in asymptomatic children than in adults, and sudden cardiac death is often the sentinel event. Athletes with WPW should be evaluated for symptoms and the presence of intermittent or persistent pre-excitation, which dictates further consultation, treatment and monitoring strategies before returning to matches [30].

Left Axis Deviation

LAD, defined as −30° to −90°, is present in almost 12% of HCM patients but less than 1% of athletes [12]. Based on this study, LAD was seen in almost 1.5% of Iranian football players. This finding is in accordance with previous reports. LAD can be a secondary marker for pathological LV hypertrophy (LVH) and if present, warrants additional evaluation [25].

Right Axis Deviation

Right-axis deviation is defined as a frontal plane QRS axis of>120°. It is a common finding in pulmonary hypertension. Right-axis deviation was seen in about 2.5% of Iranian football players.

Electrocardiogram-detectable cardiovascular diseases include cardiomyopathies such as HCM, ARVC and dilated cardiomyopathy; aortic valve stenosis; cardiac ion-channel diseases such as long-QT syndrome (LQTS), Brugada syndrome, short-QT syndrome (SQTS) and Lenègre disease and Wolff–Parkinson–White (WPW) syndrome. Based on published series from the USA and Italy, these conditions account for approximately two-thirds of SCD in young competitive athletes. Electrocardiogram abnormalities associated with these cardiovascular diseases include repolarization abnormalities such as inverted T-waves and ST-segment depression, pathological Q-waves, conduction disease including left-axis deviation, ventricular pre-excitation, long- and short-QT interval and Brugada-like repolarization changes. Based on these findings, hypertrophic cardiomyopathy and ischemic heart disease would be expected to be present in nearly 3.1% and 10% of Iranian football players, respectively.

Unlike ECG changes commonly seen in athlete's heart, such ECG abnormalities are relatively uncommon (<5%) and training-unrelated. Further diagnostic work-up is mandatory for those athletes who exhibit such ECG changes in order to confirm (or exclude) an underlying cardiovascular disease.

The future of SCD prevention in athletes by large-scale ECG screening program lies in continuing efforts to better understand the scientific basis for ECG interpretation and to define standards of ECG criteria for differentiation between athlete's heart and true heart diseases. Further studies are needed to test the accuracy, utility and cost-effectiveness of the present ECG criteria in relation to gender, age, ethnicity and different levels of training and/or type of sports.

Conclusion

Most electrocardiographic variables among Iranian professional football players were lower than worldwide football players that may be related to the lower levels of physical fitness in Iranian football players.

Acknowledgments

This research was funded by Iran University of Medical Sciences in cooperation with Medical committee of Iran Football Federation. Authors highly appreciate the cooperation of all physicians who collected the data for this project.

Conflict of Interest

The authors declare that they have no competing interests for this study.

References

1. Pelliccia A, Maron BJ, De Luca R, Di Paolo FM, Spataro A, Culasso F. Remodeling of left ventricular hypertrophy in elite athletes after long-term deconditioning. Circulation. 2002;105(8):944-9.
2. Rawlins J, Bhan A, Sharma S. Left ventricular hypertrophy in athletes. Eur J Echocardiogr: the journal of the Working Group on Echocardiography of the European Society of Cardiology. 2009;10(3):350-6.
3. Biffi A, Pelliccia A, Verdile L, Fernando F, Spataro A, Caselli S, *et al*. Long-term clinical significance of frequent and complex ventricular tachyarrhythmias in trained athletes. J Am Coll Cardiol. 2002;40(3):446-52.
4. Pelliccia A, Maron BJ, Spataro A, Proschan MA, Spirito P. The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. N Engl J Med. 1991;324(5):295-301.
5. Montgomery HE, Clarkson P, Dollery CM, Prasad K, Losi MA, Hemingway H, *et al*. Association of angiotensin-converting enzyme gene I/D polymorphism with change in left ventricular mass in response to physical training. Circulation. 1997;96(3):741-7.
6. Pelliccia A, Maron BJ, Culasso F, Di Paolo FM, Spataro A, Biffi A, *et al*. Clinical significance of abnormal electrocardiographic patterns in trained athletes. Circulation. 2000;102(3):278-84.
7. Maron BJ, Pelliccia A, Spirito P. Cardiac disease in young trained athletes. Insights into methods for distinguishing athlete's heart from structural heart disease, with particular emphasis on hypertrophic cardiomyopathy. Circulation. 1995;91(5):1596-601.
8. Corrado D, Pelliccia A, Heidbuchel H, Sharma S, Link M, Basso C, *et al*. Recommendations for interpretation of 12-lead electrocardiogram in the athlete. Eur heart j. 2010;31(2):243-59.
9. Barbier J, Ville N, Kervio G, Walther G, Carre F. Sports-specific features of athlete's heart and their relation to echocardiographic parameters. Herz. 2006;31(6):531-43.
10. Rich BS, Havens SA. The athletic heart syndrome. Current sports medicine reports. 2004;3(2):84-8.
11. Corrado D, Basso C, Thiene G. Essay: Sudden death in young athletes. Lancet (London, England). 2005;366 Suppl 1:S47-8.
12. Corrado D, McKenna WJ. Appropriate interpretation of the athlete's electrocardiogram saves lives as well as money. Eur heart j. 2007;28(16):1920-2.
13. Holly RG, Shaffrath JD, Amsterdam EA. Electrocardiographic alterations associated with the hearts of athletes. Sports medicine (Auckland, NZ). 1998;25(3):139-48.
14. Lakdawala NK, Thune JJ, Maron BJ, Cirino AL, Havndrup O, Bundgaard H, *et al*. Electrocardiographic features of sarcomere mutation carriers with and without clinically overt hypertrophic cardiomyopathy. Am J Cardiol. 2011;108(11):1606-13.
15. Tanguturi VK, Noseworthy PA, Newton-Cheh C, Baggish AL. The electrocardiographic early repolarization pattern in athletes: normal variant or sudden death risk factor? Sports medicine (Auckland, NZ). 2012;42(5):359-66.
16. Rowin EJ, Maron BJ, Appelbaum E, Link MS, Gibson CM, Lesser JR, *et al*. Significance of false negative electrocardiograms in preparticipation screening of athletes for hypertrophic cardiomyopathy. Am J Cardiol. 2012;110(7):1027-32.
17. Eckart RE, Shry EA, Burke AP, McNear JA, Appel DA, Castillo-Rojas LM, *et al*. Sudden death in young adults: an autopsy-based series of a population undergoing active surveillance. J Am Coll Cardiol. 2011;58(12):1254-61.

18. Meyer L, Stubbs B, Fahrenbruch C, Maeda C, Harmon K, Eisenberg M, *et al*. Incidence, causes, and survival trends from cardiovascular-related sudden cardiac arrest in children and young adults 0 to 35 years of age: a 30-year review. Circulation. 2012;126(11):1363-72.
19. Uberoi A, Jain NA, Perez M, Weinkopff A, Ashley E, Hadley D, *et al*. Early repolarization in an ambulatory clinical population. Circulation. 2011;124(20):2208-14.
20. Tikkanen JT, Anttonen O, Junttila MJ, Aro AL, Kerola T, Rissanen HA, *et al*. Long-term outcome associated with early repolarization on electrocardiography. N Engl J Med. 2009;361(26):2529-37.
21. Tikkanen JT, Junttila MJ, Anttonen O, Aro AL, Luttinen S, Kerola T, *et al*. Early repolarization: electrocardiographic phenotypes associated with favorable long-term outcome. Circulation. 2011;123(23):2666-73.
22. Junttila MJ, Sager SJ, Freiser M, McGonagle S, Castellanos A, Myerburg RJ. Inferolateral early repolarization in athletes. J Interv Card Electrophysiol. 2011;31(1):33-8.
23. Noseworthy PA, Weiner R, Kim J, Keelara V, Wang F, Berkstresser B, *et al*. Early repolarization pattern in competitive athletes: clinical correlates and the effects of exercise training. Circulation Arrhythmia and electrophysiology. 2011;4(4):432-40.
24. Dabrowska B. [Recommendations for the standardization and interpretation of the electrocardiogram according to the American Heart Association Electrocardiography and Arrhythmias Committee and the Heart Rhythm Society 2007 and 2009 - new standards]. Kardiologia polska. 2009;67(10):1128-32.
25. Corrado D, Biffi A, Basso C, Pelliccia A, Thiene G. 12-lead ECG in the athlete: physiological versus pathological abnormalities. Br J Sports Med. 2009;43(9):669-76.
26. Le VV, Wheeler MT, Mandic S, Dewey F, Fonda H, Perez M, *et al*. Addition of the electrocardiogram to the preparticipation examination of college athletes. Clin J Sport Med. 2010;20(2):98-105.
27. Papadakis M, Carre F, Kervio G, Rawlins J, Panoulas VF, Chandra N, *et al*. The prevalence, distribution, and clinical outcomes of electrocardiographic repolarization patterns in male athletes of African/Afro-Caribbean origin. Eur heart j. 2011;32(18):2304-13.
28. Moore EN, Boineau JP, Patterson DF. Incomplete right bundle-branch block. An electrocardiographic enigma and possible misnomer. Circulation. 1971;44(4):678-87.
29. Langdeau JB, Blier L, Turcotte H, O'Hara G, Boulet LP. Electrocardiographic findings in athletes: the prevalence of left ventricular hypertrophy and conduction defects. Can J Cardiol. 2001;17(6):655-9.
30. Rao AL, Salerno JC, Asif IM, Drezner JA. Evaluation and management of wolff-Parkinson-white in athletes. Sports health. 2014;6(4):326-32.