

ABNORMAL P WAVE MORPHOLOGY IN THE ELECTROCARDIOGRAM OF A HURDLE RUNNER WITH A HISTORY OF PRESYNCOPE – A CASE REPORT

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Abstract

Heart rhythm disturbances and their significance for health and fitness status of endurance athletes are still under investigation. In the clinical setting, it is often difficult to clearly establish the prognostic value of abnormalities found in electrocardiographic records in athletes. Hence, a case of a 29-year-old hurdle runner after an incident of pre-syncope with changes in ECG tracings typical for an athlete concomitant with non-specific P wave morphology abnormalities is presented. It is still an unresolved issue if the P morphology resembling “P mitrale” and “P pulmonale” may predict the occurrence of supraventricular arrhythmias in athletes while preliminary echocardiography and other laboratory tests results are within the normal limits. After the detailed medical investigation we concluded that the symptoms manifested by our patient could have been due to overtraining. However, the precise mechanism of the pre-syncope remains unknown and the athlete is still under our follow-up.

Key words: ECG, 24Holter monitoring, hurdle runner, athlete, P wave

Case report

We present a case of a 29 year old male athlete (professional hurdle runner) with 7 year training experience who was admitted to the hospital with a history of recent incident of pre-syncope. A pre-syncope event had occurred at home with no prodromal symptoms, was not associated with physical activity and lasted very short according to patient's report. On physical examination the patient was afebrile with heart rate of 60 beats/min, a respiratory rate of 14 breaths/min, arterial blood pressure of 120/65mmHg. No significant abnormalities including neurological dysfunctions were noted. Laboratory tests including blood levels of creatinine kinase (CK) and its isoenzyme CK MB, troponin, TSH, glucose and electrolytes were within the normal limits. The athlete was not on any permanent medication. The athlete's electrocardiogram (ECG) demonstrated: right axis deviation (+ 100 degrees, sinus rhythm with a heart rate of 76 beats per minute, a PQ interval of 160ms, a QT interval of 348 ms (corrected QT interval was 376 ms), a “narrow” QRS complex of 96ms. A notched P wave with an increase in its amplitude up to 2,5-3 mm was found in the limb leads II, III and aVF. The duration of a P wave was 120ms resembling “P mitrale” and “P pulmonale”. High amplitude R waves were present in limb lead II, III and aVF (25-30mm) with negative T waves. In

24 hour ECG Holter monitoring and in the 12-lead resting ECG record concave ST segment elevations up to 4-5 mm were found. In 24h Holter ECG monitoring minimal, maximal and mean heart rates were, respectively: 35, 114 and 74 beats/min. 24 hour Holter monitoring was performed twice within a short period of time after the syncopic incident. Nevertheless, no significant or life threatening incidents of arrhythmia were found. Echocardiogram was normal. At present, the athlete is under regular cardiological check-up. No further incidents of presyncope or even dizziness were observed in the athlete in the follow-up.

Discussion

Physiological adaptation of the heart to intense physical training may produce both electrocardiographic (ECG) and echocardiographic changes that are usually not observed in untrained persons (1). The documented incidents of sudden cardiac death among young athletes urge the precise assessment of arrhythmias in athletes with a history of syncope (2,3). Life threatening arrhythmias may produce only mild symptoms or be manifested as a syncope or even cardiac arrest (1-3). The investigated hurdle runner did not present any recurrent symptoms before admission to hospital. Arrhythmias seen in athletes include sinus bradycardia, sinus pauses and supraventricular

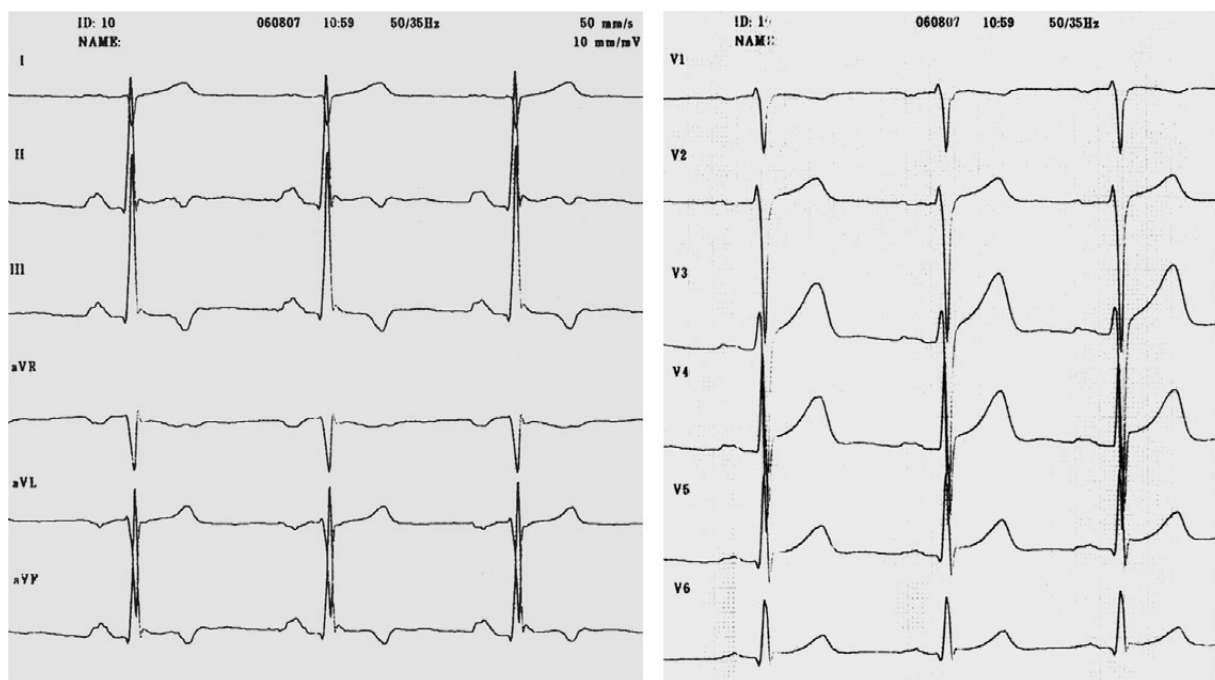


Fig. 1. 12-lead electrocardiogram of 29 year old male athlete (professional hurdle runner). The pathological P wave is observed in limb leads

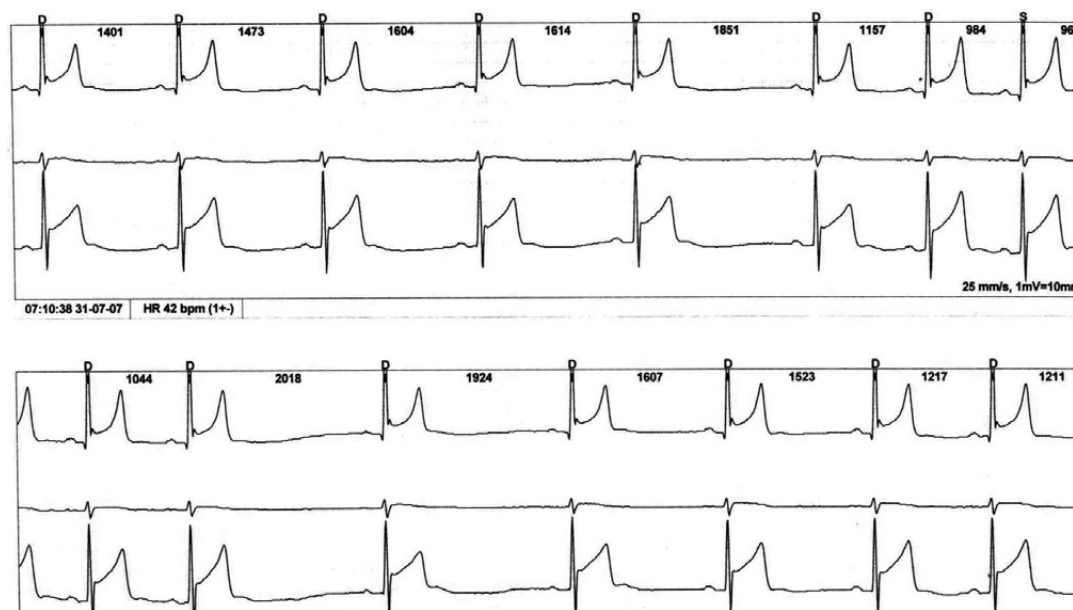


Fig. 2. A strip from 24 hour Holter monitoring of 29 year old male athlete (professional hurdle runner). Significant concave ST elevations are present. Holter leads CM5, CM1, IS.

ectopic beats (2,3). Conduction abnormalities such as prolonged P-Q interval, first degree AV heart block, Wenckebach type I AV heart block are also found (2,3). Other commonly seen ECG abnormalities include right axis deviation, increased right and left ventricular voltage, ST segment elevations, biphasic and inverted T waves (2,3). Previous investigations reported that atrial arrhythmias, particularly atrial fibrillation (AF), may be the heart's response to regular physical training in athletes, overtraining-induced inflammatory processes or even steroid abuse-induced hypertension, coronary artery disease or hypertrophic cardiomyopathy (4-6). Prolonged P wave duration and increased P wave dispersion (PWD), which is the difference between

the shortest and the longest P wave, has been reported to be a risk factor for atrial fibrillation (7). However, Karakaya et al. did not demonstrate any significant differences in P wave duration or P wave dispersion between athletes and sedentary controls (8). Although the data are equivocal, we hypothesize that abnormal P wave morphology in the hurdle runner's ECG record could potentially be a strong substrate for incidents of supraventricular arrhythmia as a cause of presyncope. Various changes found in the ECG records of the hurdle runner formerly referred to as typical for "athlete's heart" may not explain the symptoms presented by the patient. However, sometimes it is difficult to decide whether alterations found in ECG are true pathology

or typical change for athletes in case clinical symptoms are present. Regarding the presence of normal echocardiogram in the athlete, the abnormalities of the P wave could also be a phenomenon related to intensive training. However, electrocardiographic changes may forerun the ongoing structural anomaly of the athlete's heart not yet detected by echocardiography (9). For these reasons, 24h Holter monitoring was performed twice and the athlete was qualified for regular medical check-up.

Graf et al. investigated mechanisms of unexplained syncope in patients of an outpatient clinic and in majority of subjects non-invasive examinations were decisive (10). The age and number of significant prodromes were predictive factors of arrhythmic syncope (10). The authors called the diagnostic approach to patients with syncope a difficult and challenging clinical problem as syncope remains unexplained in 13-54 % of patients. Nevertheless, the most prevalent cause of syncope is vasovagal response - 23% in a group of unexplained syncope, cardio-inhibitory carotid sinus syndrome - 18 %, hypotensive (8%) and tachyarrhythmic (7%) incident. Psychogenic causes can account for less than 6 % of all syncope cases, however the authors found it in 17 % of patients with unexplained syncope (11). Moreover, Graf et al. showed that prolonged P-wave duration identifies a subgroup of patients more likely to suffer from cardiovascular disorders and arrhythmic syncope including tachy- and bradyarrhythmias (10). Haïssaguerre et al. have recently showed that in patients resuscitated after idiopathic ventricular fibrillation the prevalence of electrocardiographic early repolarization defined as an elevation of the QRS-ST junction of at least 0.1 mV from the baseline in the inferior or lateral lead manifested as QRS slurring or notching was statistically significant (12). Although the authors did not consider a group of athletes in their investigations, this suggestion prompted us to keep the patient under regular follow-up, because the similar electrocardiographic pattern as aforescribed was present in the electrocardiogram of our patient.

Conclusions

To conclude, symptoms manifested by our patient could have been due to overtraining. However, the precise pathomechanism of the pre-syncope remains unknown and it may also belong to the group of cases of unknown origin.

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